

Causes of variation in adolescent wellbeing

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ISBN: 978-94-6169-009-8

Printed by: Optima Grafische Communicatie, Rotterdam (www.ogc.nl)
Lay-out and cover design: Tiuri de Jong

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VRIJE UNIVERSITEIT

Causes of variation in adolescent wellbeing

ACADEMISCH PROEFSCHRIFT

ter verkrijging van de graad Doctor aan
de Vrije Universiteit Amsterdam,
op gezag van de rector magnificus
prof.dr. L.M. Bouter,
in het openbaar te verdedigen
ten overstaan van de promotiecommissie
van de faculteit der Psychologie en Pedagogiek
op donderdag 20 januari 2011 om 13.45 uur
in de aula van de universiteit,
De Boelelaan 1105

door

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geboren te Almelo

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1

General Introduction

Within psychology and psychiatry there is a growing interest in positive psychology. Positive psychology has its roots in humanistic psychology, and specifically in the views of William James and John Dewey (Froh, 2004). James and Dewey argued that in order to study optimal human functioning one has to consider the subjective experience of an individual, a common thread in positive psychology literature (Rathunde, 2001; Froh, 2004). Positive psychology is about valued subjective experiences, such as subjective wellbeing, contentment, satisfaction, hope, optimism, flow, and happiness (Seligman & Csikszentmihalyi, 2000). The aim of research in the field of positive psychology is to identify factors that enhance optimal human functioning. An important argument to study positive aspects of psychological functioning, such as subjective wellbeing (SWB), is that psychology and psychiatry have become increasingly focused on psychopathology, which resulted in a distorted view of what positive psychological functioning is like (Seligman & Csikszentmihalyi, 2000). The focus on psychopathology resulted in a definition of SWB as the absence of emotional and/or behavioral problems (Sin & Lyubomirsky, 2009; Ryff et al., 2006; Greenspoon & Saklofske, 2001). The increased interest in the field of positive psychology, however, has changed the conceptualization of SWB. Researchers in the field of positive psychology have developed an extensive literature on many aspects of SWB among adults (for reviews see Diener, 1984; Diener et al., 1999), whereas studies on adolescent SWB have only emerged recently (Huebner, 2004; Saha et al., 2010; Trzcinsky & Holst, 2008).

The focus of this thesis is on adolescent SWB. Studying SWB during adolescence is of particular interest because adolescence is a well-known period of important social, neurobiological, and behavioral changes (e.g. Paus et al., 2008). The main characteristic of SWB is that it represents a general positive sense of wellbeing, i.e. the majority of people feel good about themselves (Cummins, 2010). In addition, this general sense of wellbeing is highly subjective and non-specific in such a way that it is concerned with abstract feelings that individuals have about themselves in a general sense. For example, in two large scale studies on SWB among youth from a variety of different countries across Europe and North-America (Health Behavior in School aged Children project (HBSC); WHO, 2008 and UNICEF; Innocenti Research Centre, 2007), participants were asked to rate their overall quality of life, using the 10-step Cantril Ladder (Cantril, 1965). The top of the scale indicated the best possible life and the bottom the worst possible life. The range of scores on SWB clearly indicated that youth from Western countries feel good about themselves. A significant decline in SWB was observed between the ages 11 and 15 (HBSC; WHO, 2008). In other words, decreasing levels of SWB are observed during the transition from childhood into adolescence. Adolescent boys aged between 13 and 15 reported higher levels of SWB than adolescent girls in the same age range. In addition,

differences between countries were also observed: youth from The Netherlands reported the highest levels of SWB, whereas participants from the United States, Poland, and United Kingdom reported the lowest levels (HBSC; WHO, 2008).

Another important characteristic of SWB is that it shows a substantial degree of stability over time (Cummins, 2010; Wagner et al., 2007; Lucas, 2007; Huebner, 2004; Lykken & Tellegen, 1996) and situations (Diener & Larsen, 1984). The stable level of SWB over time commonly accounts for about 30-50% of the variation in adult SWB (Lucas, 2007; Lykken & Tellegen, 1996). Among adolescents in high-school, preliminary results also indicated a considerable level of stability in adolescent SWB (Huebner, 2004). Large scale information on SWB has been obtained from the German Socio-Economic Panel Survey (SOEP; Wagner et al., 2007). The SOEP data demonstrated that about 80% of this national representative adult sample reported stable levels of SWB over a twenty-year period. Using data from 21 surveys on SWB in the adult Australian population, Cummins (2010) reported that over a nine-year period during which these surveys were conducted, sample means of SWB varied over a total range of about 3 points on a scale from 0-100. These studies clearly demonstrate that self-reported SWB shows substantial levels of stability. Furthermore, results from longitudinal research on adult SWB indicated that the stable level of SWB over time is predominantly (75 – 80%) attributable to genetic factors (Nes et al., 2006; Hamer, 1996; Lykken & Tellegen, 1996). Not only has SWB been found to show substantial stability over time, it is also well established that SWB is strongly associated with stable personality traits, especially with the Big Five personality traits of neuroticism and extraversion (see DeNeve & Cooper, 1998; Diener et al., 1999 for reviews).

Causes of variation in SWB

An important question is why some individuals report high levels of SWB, whereas others report lower levels. Are these kinds of individual differences in SWB due to different circumstances and events in people's lives, are they due to genetic variation, or both? Twin family studies offer the possibility to assess the importance of genetic and environmental factors as determinants of a trait under study. With data from twins and their non-twin siblings, individual differences in a trait under study can be decomposed as due to genetic and environmental factors, by considering the different levels of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs and non-twin siblings. MZ twin pairs are genetically (nearly) identical whereas DZ twin pairs and non-twin siblings

share on average 50% of their segregating genes. Several twin studies assessed the etiology of adult SWB. Only one study to date, however, investigated the genetic architecture of SWB among adolescents.

The first study examining the genetic architecture of SWB among adults aged 20 - 40 years was conducted by Tellegen et al. (1988) using the Multidimensional Personality Questionnaire. They reported genetic (48%) and nonshared environmental effects (52%) on SWB. Part of the genetic influence on SWB reflected non-additive genetic effects (29%). Making use of the same measure, Lykken and Tellegen (1996) replicated these findings one decade later in a sample of adult twins between 20 and 30 years (Hamer, 1996). In a sample of young adults (18 - 25 years) from Norway, Røysamb et al. (2002) employed the short version of the SWB-scale (Moum et al., 1990) and reported that variation in SWB was accounted for by additive (males: 46%, females: 46%) and non-additive genetic (females: 9%) and nonshared environmental effects (males: 54%, females: 45%). In a partly overlapping sample of participants in the same age range, Nes et al. (2006) reported similar results but also found qualitative sex differences in SWB, indicating that different genetic variants affect SWB in men and women. In a large sample of adult twins and their non-twin siblings (20 - 40 years) from the Netherlands Twin Registry, Stubbe et al. (2005) reported individual differences in satisfaction with life (Satisfaction with life scale; Diener et al., 1985) to be accounted for by non-additive genetic (38%) and nonshared environmental factors (62%). No indication was found for sex differences in heritability. In the only study among adolescent twins and their siblings, Bartels and Boomsma (2009) employed four measures of SWB: satisfaction with life (Satisfaction with life scale; Diener et al., 1985), subjective happiness (Subjective happiness scale; Lyubomirsky and Lepper, 1999), and general and current quality of life (Cantril ladder; Cantril, 1965). They reported genetic and nonshared environmental effects on variation in the 4 measures of SWB. No evidence was found for quantitative and qualitative sex differences. Broad sense heritabilities ranged between 36% (quality of life at the moment) and 47% (satisfaction with life). Part of the genetic influence on these SWB measures reflected non-additive genetic effects. Finally, in a sample of young adult twins (23 - 24 years) from Italy Caprara et al., (2009) assessed satisfaction with life (Satisfaction with life scale; Diener et al., 1985) and reported individual differences in this measure to be accounted for by additive genetic (59%) and nonshared environmental factors (41%). In short, across several Western countries and making use of various measures of SWB these studies reported heritability estimates typically ranging between 40 - 55%, while the remaining variation was accounted for by nonshared environmental factors. Genetic effects on SWB appear to reflect both additive and non-additive genetic effects. The importance of shared environmental effects in the etiology of SWB has been found to be

negligible. Although most of these studies focused on adults, the only study investigating the etiology of SWB among adolescents suggested no substantial differences in the genetic architecture of SWB among adolescents as compared to adults.

The question may be raised whether the different constructs of wellbeing which have been used in these studies (personality dimension, satisfaction with life, subjective happiness, quality of life) are distinct descriptions of a single SWB concept, or whether these constructs have different etiologies. Bartels and Boomsma (2009) addressed this question using four measures of SWB (see above). They reported that the moderate to high phenotypic associations between the measures were explained by an underlying additive genetic factor and an underlying non-additive genetic factor. In addition, Caprara et al. (2009) reported that phenotypic associations between life satisfaction, optimism, and self-esteem were best explained by genetic factors common to these traits. The implication of these findings is that the phenotypic overlap between distinct constructs of SWB is determined by a single genetic factor of SWB.

Since the results from twin-family studies clearly established that SWB is a heritable trait, the logical next step would be to find the genes that are involved. One statistical method for gene-finding is linkage analysis, which localizes chromosomal regions that possibly influence the trait of interest by using pedigree information. Bartels et al. (2010) performed the first genome-wide linkage scan on subjective happiness (Subjective happiness scale; Lyubomirsky and Lepper, 1999) as a measure for SWB. They reported suggestive linkage in genomic regions on chromosome 1 and 19, harboring several protein coding genes. Which particular genetic variants in these regions affect individual differences in SWB has to be further elucidated.

Set-point theory of SWB

In summary, SWB can be characterized by a general positive sense of wellbeing which shows substantial stability over time and which is strongly associated with stable personality traits. In addition, individual differences in SWB are substantially accounted for by genetic factors. There has been a substantial body of literature in which theories have been described that aimed to integrate SWB and its characteristics. The dominant theory that has been evolved is the set-point theory of SWB. The central proposition of the theory is that individuals have differing but stable baseline levels of SWB (Headey & Wearing, 1986, 1987, 1989) which are under genetic control (Nes, 2010; Lykken, 1999; Lykken & Tellegen, 1996; Hamer, 1996). The level of SWB is assumed to fluctuate around these baseline levels as a reaction to circumstances and events in people's lives, but after a period SWB will return to its baseline level. The set-point theory of SWB has also received criticism (e.g.

Headey, 2010; Lucas, 2007; Fujita & Diener, 2005). Heady (2010) observed that 15 – 25% of the participants in the SOEP panel study (Wagner et al., 2007) reported a change in their SWB level of at least one standard deviation after controlling for major life events. He concluded that these degrees of change are not compatible with the set-point theory. Lucas (2007) reported that long-term levels of SWB change as a result of major life events and that there are individual differences in whether or not people return to their baseline levels of SWB after such life events. These individual differences are likely to result from variability in the nature of the events and from variability in people's reactions to similar events. According to Cummins (2010), however, upward or downward changes in SWB over time are plausible within the framework of the set-point theory. Based on a review of the SWB literature he proposes that individual baseline levels of SWB remain stable due to neurological systems which are thought to work in a homeostatic fashion in order to maintain the baseline levels. Specifically, when SWB is positively or negatively challenged by other factors, the homeostatic system will act to maintain the baseline level of SWB. However, once the influence of the challenging factors becomes too strong, the baseline level of SWB can no longer be maintained and the level of SWB will change according to the strength of the challenging factors. In addition, Weiss et al. (2008) reported SWB to be linked to personality traits neuroticism, extraversion, and conscientiousness by common genes, suggesting that genetic effects of personality may affect how soon SWB returns to its baseline after disturbance, and the extent to which the baseline levels change in response to challenging factors.

To increase our knowledge of the etiology of SWB insight into the factors that may cause long term stability and changes in adolescent SWB should be obtained. A variety of factors, such as physical health, relationships with parents and peers, family functioning, achievement in school, SES, life events, disabilities, and psychopathology have been found to be associated with adolescent SWB (see Proctor et al., (2009) and Huebner (2004) for extensive reviews). Positive outcomes with respect to adolescent SWB are associated with a healthy lifestyle, good physical health, exercise participation, residing in intact families, high quality relationships with parents, siblings and peers, and high levels of family functioning. In contrast, participation in risk-taking behaviors (e.g. substance abuse, violence, aggression) and suffering from some sort of psychopathology is associated with decreased levels of SWB. However, little is known about the mechanisms through which these factors affect individual differences in adolescent SWB.

One hypothesis is that increased levels of SWB in some adolescents reflect causal effects of, e.g. exercise participation and high levels of family functioning. An alternative hypothesis, however, explaining associations between SWB and related factors may be that a third underlying factor that influences SWB also influences factors which has been found

to be associated with SWB. Genetic variation among individuals is likely to represent such an underlying factor. Under this hypothesis, associations between SWB and a related trait reflect the effects of common genetic factors influencing SWB as well as the related trait, a process referred to as genetic pleiotropy. To increase our knowledge of the etiology of SWB it seems to be crucial to take such genetic effects into account, since it is well established that individual differences in SWB and factors that have been associated with SWB are substantially accounted for by genetic factors.

In addition to causal and genetic pleiotropic mechanisms, the factors mentioned above may also affect individual differences in SWB in an indirect way by complex interplays with genetic factors. One possibility is that genetic factors control the degree of sensitivity to environmental factors, a process referred to as gene-environment interaction (Eaves, 1984; Kendler & Eaves, 1986; Boomsma & Martin, 2002). Specifically, some adolescents are assumed to be genetically predisposed towards higher baseline levels of SWB, whereas others are genetically predisposed towards lower baseline levels of SWB. Those with a high baseline level of SWB may be more sensitive to positive effects from their environment, whereas those with a lower baseline level may be more sensitive to negative effects from their environment. To date, no study has focused on the possibility of gene-environment interaction with regard to SWB. This can be considered as an unfortunate state of affairs, because it is plausible that complex interactions between genetic and environmental factors play an important role in the etiology of SWB.

General aims of this thesis

The main aim of my thesis will be to integrate knowledge from behavioral genetic and social developmental research traditions by examining the co-action and interaction of genetic factors and factors from adolescents' family and individual-specific environment in the etiology of adolescent SWB. Insight into the genetic architecture of factors from adolescents' family and personal environment that have been found to be related to adolescent SWB will be obtained. Furthermore, I will focus on the question whether the associations between these factors and SWB reflect causal effects or whether genetic factors account for the associations by genetic pleiotropic effects and / or gene-environment interactions. In this way, the relationship between family environmental, individual-specific environmental, and genetic factors will be explicitly considered in explaining individual differences in adolescent SWB, and thereby advance our knowledge of the causes of individual differences in adolescents' SWB.

Family environment and SWB

In the first part of my thesis I will focus on the relation between SWB and factors of adolescents' family environment such as parental divorce and adolescents' evaluations of overall family functioning and the level of family conflict. Individual differences have been found in how adolescents react to parental divorce and how they evaluate their family functioning. The association between SWB and such family factors are presumed to be less due to true family experiences but more to the ways adolescents perceive and interpret these experiences and hence their family functioning (Millikan et al., 2002; Neiderhiser et al., 1998; Harold et al., 1997). An increasing body of evidence suggests that a substantial part of individual differences in adolescents' evaluations of family functioning is accounted for by genetic factors (e.g. Herndon et al., 2005; Jacobson & Rowe, 1999; Neiderhiser et al., 1998). Little is known, however, about the extent to which genetic pleiotropic effects account for the association between SWB and subjective evaluations of family functioning. Using general family functioning (McMaster Family Assessment Device; Epstein et al., 1983) and family conflict (Family Environment Scale; Moos, 1974) as measures of evaluations of family functioning and quality of life (Cantril Ladder; Cantril, 1965) as a measure of SWB, we investigated whether genetic factors account for individual differences in adolescents' evaluations of family functioning and, in addition, to what extent genetic factors can explain the association between SWB and these evaluations of family functioning. Information on parental divorce was used to explore whether genetic factors influencing adolescents' evaluations of SWB and family functioning interacted with parental divorce. In addition, interaction between genetic factors and unmeasured nonshared environmental factors was explored, by looking at the association between intrapair sum and difference scores on the measures of family functioning and SWB in monozygotic twin pairs (Jinks & Fulker, 1970; see chapter 2).

Exercise behavior and SWB

There is a long history of studies on the association between exercise behavior and mental health. In the adult population regular exercise has consistently been associated with higher levels of SWB and the absence of mental health problems (Stubbe et al., 2007; De Moor et al., 2006; Norris, Carrol, & Cochrane, 1992). In general, the conclusion that has been drawn from these studies is that increased levels of psychological wellbeing found in exercisers reflect causal effects of exercise. However, evidence for genetic pleiotropic effects accounting for the association between exercise behavior and the absence of mental health problems has also been found in population-based adult twin studies (De Moor et al.,

2008, 2007; Stubbe et al., 2007). It is important to further study the association between exercise behavior and SWB during adolescence because several studies reported that the prevalence of exercise participation declines during adolescence (Stubbe, Boomsma, & De Geus, 2005; Van Mechelen et al., 2000) and the genetic architecture of exercise behavior was found to be different across the life span with a peak in heritability during adolescence (Stubbe, Boomsma, & De Geus, 2005; Maia, Thomis, & Beunen, 2002; Beunen & Thomis, 1999). Exercise behavior was assessed with survey items about type and frequency of regular leisure time exercise activities. The Ainsworth's Compendium of physical activity (Ainsworth et al., 2000) was used to compute a total weekly metabolic equivalent (MET) score based on each exercise activity endorsed. Satisfaction with life (Satisfaction with Life Scale; Diener et al., 1985), subjective happiness (Subjective Happiness Scale; Lyubomirsky & Lepper, 1999), and quality of life (Cantril Ladder; Cantril, 1965) were used as measures for SWB and these were summarized into a single factor score. First, a large sample of adolescent twins made it possible to examine the genetic architecture of exercise behavior throughout adolescence by estimating genetic and environmental effects within 3 different age groups for boys and girls. Extending the procedure described by De Moor et al. (2008), we subsequently investigated whether the association between adolescent exercise behavior and SWB was best reflected by causal effects of exercise behavior or by pleiotropic genetic effects.

Sedentary behavior and SWB

Sedentary behavior has often been addressed as simply the opposite of physical activity, but research has shown this to be incorrect (e.g. Biddle, 2007; Te Velde et al., 2007). Sedentary behavior may therefore contribute to adolescents' SWB independently of their exercise participation level. Studying sedentary behavior during adolescence is of particular interest because sedentary activities such as TV viewing and engagement in personal computer and internet activities are dominant leisure time activities among youth (e.g. Swinburn & Shelly, 2008; Van den Eijnden et al., 2008). There has been some evidence that time spent on sedentary activities, such as internet use, was positively associated with low SWB (e.g. Van den Eijnden et al., 2008; Ybarra et al., 2005; Weiser, 2001; Kraut et al., 1998). However, other studies did not find any association between sedentary behavior and SWB (Gross et al., 2002; Wastlund et al., 2001; Sanders et al., 2000). To date, only one study investigated the genetic architecture of sedentary behavior and found that individual differences were substantially accounted for by genetic factors (Nelson et al., 2006). As a first step, using survey items about the weekly frequency of watching television, playing video games, and engaging in personal computer or internet activities as a measure of

sedentary behavior, the genetic architecture of sedentary behavior throughout adolescence was examined in a large sample of twins and their non-twin siblings. To increase our knowledge of the impact of sedentary activities on adolescent psychological wellbeing we examined the association between internet use and psychological wellbeing at the phenotypic level by using a diathesis-stress perspective that accounts for a progression from normal to compulsive internet use, and for individual vulnerabilities in internet use based on personality traits. Internet use was measured by using two survey items about the time spent on the internet on a daily basis and by means of the Compulsive Internet Use Scale (Meerkerk et al., 2007). Psychological wellbeing was operationalized in terms of loneliness (revised UCLA Loneliness Scale; Russell et al., 1980), self-esteem (Rosenberg Self-Esteem Scale; Rosenberg, 1989), and depressive moods (Depressive Mood List; Kandel & Davies, 1986). Personality was assessed by means of the Big Five personality traits neuroticism, extraversion, agreeableness, openness, and conscientiousness using the Quick Big Five (Vermulst and Gerris, 2005).

Psychopathology and SWB

An important question in the field of psychological research is whether SWB and psychopathology constitute the opposite ends of a mental health continuum or whether they represent distinct dimensions of mental health. Although SWB has been found to be strongly negatively associated with different types of psychopathology (e.g. Proctor et al., 2009), there is also evidence that SWB and psychopathology are distinct dimensions of mental health with separate determinants (e.g. Bergsma et al., 2010; Ryff et al., 2006; Greenspoon & Saklofske, 2001). Little is known about pleiotropic genetic effects underlying the association between psychopathology and SWB, although this could provide important information on whether SWB and psychopathology are on the same mental health continuum or whether they represent distinct dimensions of mental health. If pleiotropic genetic effects largely account for the association between psychopathology and SWB this would be in line with the hypothesis that SWB and psychopathology are on the same mental health continuum. However, if genetic factors affecting psychopathology do not or only partly overlap with genetic factors influencing SWB this would be compatible with the alternative hypothesis. Three measures of SWB (satisfaction with life, subjective happiness, and quality of life) were summarized into a single factor score. Eight syndrome scales of the Youth Self-Report (Achenbach & Rescorla, 2001) measuring eight different types of psychopathology, and the broadband scales internalizing and externalizing were

used. The association between psychopathology and SWB was examined at the phenotypic level and the genetic level in order to study the presence or absence of a mental health continuum.

Besides the association between psychopathology and SWB in general, assessing specific types of psychopathology might provide useful information with regard to SWB as well. Truancy, for example, has been found to occur in a broader context of externalizing problems and is frequent from early adolescence onwards. It has been found to be a first sign and powerful predictor of externalizing problems, with truants being more likely further penetrating the juvenile justice system (Zhang et al., 2004), and with truancy increasing the odds of initiation of drug use (Henry et al., 2007). Truancy has also been found to be associated with lower levels of psychological wellbeing (e.g. Egger et al., 2003). Therefore, we aimed to provide insight into the etiology of truancy by examining the relative importance of genetic and environmental factors using information on frequency of truancy while in high school.

Outline of this thesis

Chapter 2 addresses the association between SWB and factors from adolescents' family environment. In this chapter, the influences of genetic and environmental factors on adolescents' evaluations of SWB, family functioning, and family conflict are investigated, as well as the association between these traits. In addition, interaction between genetic factors and parental divorce as well as non-measured nonshared environmental factors will be explored for SWB and both measures of family functioning. Chapters 3 and 4 focus on exercise behavior and its relation to SWB. In chapter 3, the genetic architecture of exercise behavior throughout adolescence is examined. Chapter 4 addresses the nature of the association between exercise behavior and SWB. In chapters 5 and 6, sedentary behavior and its association with SWB is considered. Chapter 5 focuses on the genetic architecture of sedentary behavior throughout adolescence, and in chapter 6 the phenotypic relation between adolescents' psychological wellbeing and their internet use is considered. The association between psychopathology and SWB is the focus of chapters 7 and 8. The question whether SWB and psychopathology constitute the opposite ends of a mental health continuum or whether they represent distinct dimensions of a mental health continuum, is addressed in chapter 7. In chapter 8, genetic and environmental influences on frequency of truancy in high school will be disentangled. Chapter 9 describes the data collection procedures and the study sample my thesis was based on. Finally, the general discussion in chapter 10 summarizes the empirical results for the chapters 2 to 8 and the implications are discussed.

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Section I: Family environment and subjective wellbeing

2

Moderation of Genetic Factors by Parental Divorce in Adolescents' Evaluations of Family Functioning and Subjective Wellbeing

This chapter is published as:

Van der Aa, N., Boomsma, D.I., Rebollo-Mesa, I., Hudziak, J.J., & Bartels, M. (2010). Moderation of genetic factors by parental divorce in adolescents' evaluations of family functioning and subjective wellbeing. *Twin Research and Human Genetics*, 13, 143-162.

Abstract

Adolescents' evaluations of family functioning may have a significant impact on their subjective well-being and adjustment. The aim of the study was to investigate the degree to which genetic and environmental influences affect variation in evaluations of general family functioning, family conflict, and quality of life and the overlap between them. We assessed whether genetic and environmental influences are moderated by parental divorce by analyzing self-report data from 6,773 adolescent twins and their non-twin siblings. Genetic, shared, and nonshared environmental influences accounted for variation in general family functioning and family conflict, with genetic influences being relatively more important in girls than boys in general family functioning. Genetic and nonshared environmental influences accounted for variation in quality of life, with genetic influences being relatively more important in girls. Evidence was found for interaction between genetic factors and parental divorce: genetic influence on general family functioning was larger in participants from divorced families. The overlap between general family functioning and quality of life, and family conflict and quality of life was accounted for the largest part by genetic effects, with nonshared environmental effects accounting for the remaining part. By examining the data from monozygotic twins, we found evidence for interaction between genotype and nonshared, non-measured, environmental influences on evaluations of general family functioning, family conflict, and quality of life.

Introduction

The impact of family functioning, conflict, and cohesion on children's and adolescents' well-being and behavior has been a focus in research for decades. Positive family functioning characterized by supportive, close, and warm family relationships has been associated with better psychological adjustment in children and adolescents (e.g. McHale & Rasmussen, 1998; Leary & Katz, 2004; Kurdek & Fine, 1994). On the other hand, negative family functioning characterized by e.g. non-supportive family relationships and high levels of conflict has been linked to several negative outcomes such as low subjective well-being (Proctor, Linley & Maltby, 2009; McFarlane et al., 1995), internalizing problems (e.g. Hughes et al., 2008; Cui et al., 2007; Vandewater & Lansford, 2005; Formoso et al., 2000), and externalizing problems (e.g. Richmond & Stocker, 2006; Cui et al., 2007; Vandewater & Lansford, 2005; Formoso et al., 2000). However, individual differences have been found in how adolescents evaluate their family functioning and subjective well-being (SWB) and the association between family functioning and adolescent SWB is presumed to be less due to true family experiences but more to the ways adolescents perceive and interpret these experiences and hence their family functioning (Millikan et al., 2002; Neiderhiser et al., 1998; Harold, 1997). In other words, only adolescents' evaluations of how their family functions are assumed to be directly associated with their SWB and adjustment. For example, Millikan et al. (2002) found that adolescents' but not parental evaluations of family relationships are directly associated with their self-reported internalizing symptoms. Neiderhiser et al. (1998) reported that adolescent perceptions of parenting mediate the association between actual observed parental behavior and adolescent adjustment. Harold et al. (1997) found that the adverse influence of parental conflict and hostility on adolescent internalizing and externalizing behavior was completely accounted for by adolescents' evaluations of these parental behaviors.

An increasing body of behavioral genetic research suggests that a substantive part of individual differences in the way adolescents evaluate family functioning can be accounted for by genetic factors (e.g. Herndon et al., 2005; Jacobson & Rowe, 1999; Neiderhiser et al., 1998). For example, Herndon et al. (2005) investigated genetic influence on adolescents' ratings of the 10 subscales of the Family Environment Scale (FES) and reported heritability estimates ranging between 20% and 40%, with no evidence for sex-differences. Jacobson and Rowe (1999), however, reported genetic influences on adolescents' evaluations of family connectedness that were twice as large in girls as compared to boys (58% vs. 26% respectively). Shared environmental influences were higher in boys (20% vs. 0% respectively). This finding argues for potential quantitative sex differences in genetic influences on evaluations of family functioning.

Genetic factors also have been found to account for individual differences in evaluations of SWB (e.g. Bartels & Boomsma, 2009; Nes et al., 2006; Røysamb et al., 2002). For example, in a sample of Dutch adolescents Bartels and Boomsma (2009) reported that about half of the variation in SWB, including a measure of quality of life, could be accounted for by genetic influences with no evidence for sex differences. However, Røysamb et al. (2002) reported genetic influences on SWB to be larger in females than in males (55% vs. 46%), suggesting that quantitative sex differences in heritability may be present in SWB. In a recent paper, Bartels et al. (in press, this issue) found substantial differences in heritability of subjective happiness between males and females (22% vs. 41%). A variety of family factors, such as parental divorce, could contribute to individual differences in the way family functioning and SWB is evaluated. In a meta-analysis about the effect of parental divorce on well-being and adjustment, Amato and Keith (1991) reported that effect sizes are generally small. In general, literature regarding the effect of parental divorce on adolescents' evaluations of family functioning, SWB, and adjustment suggests heterogeneity in the way children and adolescents react to parental divorce. For example, some adolescents from divorced families have more conflicts with their parents (e.g. Dunn et al, 1998; O'Connor et al., 2001) and receive less emotional support, supervision, and involvement from their parents (e.g. Carlson & Corcoran, 2001) which is likely to lead to negative family functioning. Furthermore, some adolescents from divorced families show lower levels of SWB and adjustment compared to those from non-divorced families (Størksen et al., 2006; Cuffe et al., 2005). However, others have found that parental absence may only have deleterious effects on children's and adolescents' adjustment in certain circumstances. Specifically, the presence of the father in the family may only be beneficial to children's adjustment when he does not engage in antisocial behavior (Jaffee et al., 2003; Blazer et al., 2008). Others (McFarlane et al., 1995) have reported no significant differences with regard to evaluations of well-being and adjustment between adolescents from families with divorced parents and intact families. Finally, characteristics of the offspring themselves may contribute to the risk of divorce in their parents (Robbers et al. under review).

In addition to a main effect of parental divorce on evaluations of family functioning and SWB, parental divorce may affect evaluations of family functioning and SWB in an indirect way by complex interplays with genetic factors (e.g. Kendler & Baker, 2007; Price & Jaffee, 2008; Jaffee & Price, 2007). One possibility is that genetic factors control the degree of sensitivity to environmental factors such as parental divorce (i.e. gene-environment interaction; Kendler & Eaves, 1986). With respect to the current work, some adolescents may be genetically predisposed towards negative evaluations of family functioning and SWB whereas others will be genetically predisposed towards positive

evaluations. Those who are genetically predisposed towards negative evaluations will be more sensitive to negative effects of parental divorce, whereas those who are genetically predisposed towards positive evaluations will be more sensitive to positive effects of parental divorce.

Adolescents' evaluations of family functioning have been associated with their evaluations of SWB and adjustment. Since individual differences in adolescents' evaluation of family functioning and SWB are substantially accounted for by genetic influences it is plausible that these genetic influences are also responsible for at least part of the association between family functioning and SWB (Jacobson & Rowe, 1999; Neiderhiser, 1998, Pike et al., 1996). For example, Jacobson and Rowe (1999) reported high genetic overlap between adolescents' reports of family connectedness and depressed mood for girls. Pike et al., (1996) reported high genetic overlap between adolescents' evaluations of maternal negativity and adolescent depression for boys and girls. Family functioning has many characteristics (e.g. cohesion, level of conflict, warmth) and these characteristics may be perceived in various ways. For example, an adolescent might evaluate the level of family conflict as high, whereas the level of family warmth might be evaluated as high as well. Genetic influences have been reported for different characteristics of family functioning (see Kendler & Baker. 2007; Plomin & Bergeman, 1991). In the present study, we investigate whether variation in the evaluation of SWB and different aspects of family functioning share the same underlying genetic and environmental influences. Since adolescents' evaluations of family functioning and adolescent adjustment are associated, information on the underlying source of this association is crucial for prevention and intervention.

In the current study two measures of family functioning and one measure of subjective well-being (SWB) were analyzed in adolescent twins and their non-twin siblings. Family functioning measures consisted of evaluations of general family functioning (GFF; i.e. the overall health/pathology of a family) and evaluations of the level of family conflict (FC; i.e. the amount of openly expressed anger, aggression, and conflict among family members). SWB was assessed by asking adolescents to evaluate their quality of life in general (QLg). Other studies investigating the association between adolescent evaluations of family functioning and adjustment focused solely on psychopathology as an outcome measure (e.g. Millikan et al., 2002; Neiderhiser et al., 1998; Harold et al., 1997). Using QLg as an outcome measure for adolescent adjustment has the important advantage that it is sensitive to the entire spectrum of adjustment, and thus is an indicator of both well-being and psychopathology (Proctor et al., 2009). The data on GFF, FC, and QLg were collected in a large population of adolescent twins and their non-twin siblings. We tested whether genetic and environmental influences, including environmental influences

specifically shared by twins and not by their siblings, affected variation in evaluations of GFF, FC, and QLg, and the overlap between GFF and QLg, and FC and QLg. The presence of environmental influences specifically shared by twins and not by their siblings implies that twin pairs share more of their environment than non-twin siblings (Eaves et al., 1999). We investigated whether the genetic architecture of GFF, FC, and QLg was different between boys and girls, and between participants from non-divorced and divorced families.

In addition to interaction between genetic and environmental factors and measured parental divorce (i.e. by definition an environmental factor that is shared by all member of a family), interaction between genetic factors and nonshared, non-measured, environmental influences was explored. This was done by looking at the association between intrapair sum and difference scores in monozygotic (MZ) twin pairs (Jinks and Fulker, 1970). Genetic and shared environmental influences add to the similarity of MZ twin pairs, whereas nonshared environmental influences add to differences within MZ twin pairs. When individuals who evaluate GFF as negative are more similar than individuals who evaluate family functioning as positive, this indicates that the latter group is more sensitive to environmental influences that are not shared by members of the same family.

Methods

Subjects

Participants were registered with the Netherlands Twin Registry (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007; Boomsma et al., 2006). Parents of twins were first contacted to ask for consent to send their children a survey. If their parents consented, 14-, 16-, and 18-year old twins and their non-twin siblings received an online or paper & pencil self-report survey about the development of behavior, lifestyle, and well-being. When twins and siblings did not return the survey on time they were contacted by mail for a first reminder and next they were contacted by phone for a second reminder. A total of 4,912 families with twins born between 1986 and 1991 participated in this ongoing study so far. The overall family response rate was 56%.

Sibling data were included in the analyses if the siblings were older than 13 and younger than 20 years. For this paper, data from one additional sibling per family were included. From families with more than one additional participating sibling, we selected the sibling closest in age to the twin. This resulted in exclusion of 401 siblings from the

analyses because they were either too young or too old and the exclusion of another 35 siblings from families with more than one additional sibling. Data from 40 twins were excluded from the analyses due to missing data on zygosity. We excluded 261 subjects for whom it was uncertain which family they reported on, because they started a family on their own (i.e. having children themselves or living together with a partner). Another 87 individuals were excluded because one of their parents was deceased. This resulted in a total sample of 6,773 individuals of whom 5,773 were twins (44% male) and 1,000 were non-twin siblings (45% male), coming from 3,185 families. There were data from 441 (14%) incomplete and 2,666 (86%) complete twin pairs. In Table 2.1, the exact constellation of the participating families is presented. Participants came from intact families (88.5%) and families in which the biological parents were divorced (11.5%). The age of the twins ranged between 13.19 and 19.93 years, with a mean age of 16.05 years ($SD = 1.59$) and the age of the non-twin siblings ranged between 13.01 and 19.97 years with a mean age of 17.03 years ($SD = 1.77$). Participants were primarily Caucasian and they came from all regions of the Netherlands, including rural and urban areas.

Table 2.1

Sample constellation	N of individuals	N of families	1 twin	2 twins	1 twin + sib	2 twins + sib
MZM	1061	474	28	290	15	141
DZM	917	425	56	232	14	123
MZF	1540	697	54	432	11	200
DZF	1116	512	49	309	13	141
DOS	2061	999	169	566	32	232
Sibs only	78	78	--	--	--	--
Total	6773	3185	356	1829	85	837

Note. N = Number; 1 twin = families with data from 1 twin; 2 twins = families with data from both members of the twin pair; 1 twin + sib = families with data from 1 twin and 1 additional sibling; 2 twins + sib = families with data from both members of the twin pair and 1 additional sibling; MZ = Monozygotic twin pair; DZ = Dizygotic twin pair; M = male; F = female; Sibs only = families with data from non-twin siblings only

For 1,089 (36.1%) of the same-sex twin pairs zygosity was determined based on blood group or DNA typing. Zygosity for the remaining same sex twin pairs was

determined by questionnaire items about physical similarities and confusion by family members and strangers. These items allow accurate determination of zygosity in 93% of same-sex twin pairs (Rietveld et al., 2000).

Measures

General Family Functioning (GFF) refers to adolescents' evaluations of the overall health or pathology of the family with regard to problem solving, communication, roles within the household, affection, and control. GFF was assessed with the subscale General Functioning from the McMaster Family Assessment Device (FAD) (Epstein et al., 1983). We used a Dutch translation of the subscale which shows good psychometrical properties (Wenniger et al., 1993). The subscale consists of 12 items which had to be answered on a 4-point scale ranging from 1 = "strongly agree" to 4 = "strongly disagree". Six items of the scale had to be recoded, because they were asked in opposite direction. Examples of items are "planning family activities is difficult because we misunderstand each other" and "in times of crisis we can turn to each other for support". Scores on the individual items were summed to get an overall score for GFF which could range from 12 to 48 with high scores indicating high levels of GFF. A factor analysis of the items indicated one single factor which explained 40.51% of the variance. Internal consistency of the scale was good with a Chronbach's Alpha of .86.

Family Conflict (FC) refers to adolescents' evaluations of the amount of openly expressed anger, aggression, and conflict among family members. FC was assessed with the subscale Conflict from the Family Environment Scale (FES) (Moos, 1974). We used a Dutch translation of this subscale, which shows satisfactory psychometric properties (De Coole & Jansma, 1983). The subscale consists of 11 items which had to be answered on a 2-point scale ranging from 1 = "no" to 2 = "yes". Examples of items are "We argue a lot at home" and "Family members criticize each other frequently". One item in the scale ("We seldom get openly angry at each other at home") was removed from the scale because it was clearly misunderstood by the participants. Answering "yes" on this item implies a low level of family conflict, whereas answering "yes" on the other items implies a high level of family conflict. Many participants appeared not to realize this. Furthermore, the number of missing values on this item was far higher as compared to the other items (458 vs. 46-115). Scores of the remaining 10 items were summed to get an overall score for FC. These scores could range between 10 and 20 with high scores indicating low levels of FC (a positive correlation between the GFF scale and the FC scale therefore means that high

levels of GFF are associated with low levels of FC). Factor analysis of the FC scale based on 10 items revealed one single factor which explained 26.40% of the variance. Internal consistency of the scale was acceptable with a Chronbach's Alpha of .68.

Evaluations of quality of life in general (QLg) was assessed with the Cantril Ladder (Cantril, 1965). The ladder has 10 steps: the top indicated the best possible life, and the bottom the worst possible life. Participants had to indicate the step of the ladder at which they place their lives in general.

Parental divorce was assessed by asking two questions. Participants were asked whether their parents divorced: 1 = 'no', 2 = 'yes, less than 2 years ago', 3 = 'yes, more than 2 years ago'. With the second item participants were asked what living situation applied to them: 1 = living with both biological parents, 2 = living with biological mother, 3 = living with biological father, 4 = living with biological mother and her new partner, 5 = living with biological father and his new partner, 6 = other living situation. Reports on both items had to indicate that the biological parents were divorced. If the reports on both items were incongruent, parental reports on their children's behavior at age 12 were examined in order to establish whether or not the biological parents of the twins and siblings were divorced. The resulting divorce measure consisted of two categories 0 = 'intact family' and 1 = 'families in which biological parents were divorced' and it was equal for all members of the same family.

Univariate saturated and genetic analyses

The data on GFF, FC, and QLg were first analyzed with univariate genetic models. The data were structured into entire family units consisting of two or three individuals (i.e. two twins and one additional sibling) with missing data for families without siblings. In three so-called saturated models, means and variances for GFF, FC, and QLg were estimated conditional on sex and age. In addition, twin and twin-sibling correlations within traits were obtained. All parameters were allowed to differ between non-divorced and divorced families. This was done in the software package Mx (Neale et al., 2006). In the saturated models, we first tested whether sex and age influenced individual differences in mean levels of GFF, FC, and QLg. These variables were included as fixed effects (covariates) in the means model. Under this model, an individual's score (Y) on GFF, FC, and QLg for non-divorced (ND) and divorced (D) families respectively can be expressed as:

$$Y_{i,ND} = \mu_{ND} + \beta_{sex,ND} \text{sex}_i + \beta_{age,ND} \text{age}_i + \varepsilon_{i,ND} \quad (1)$$

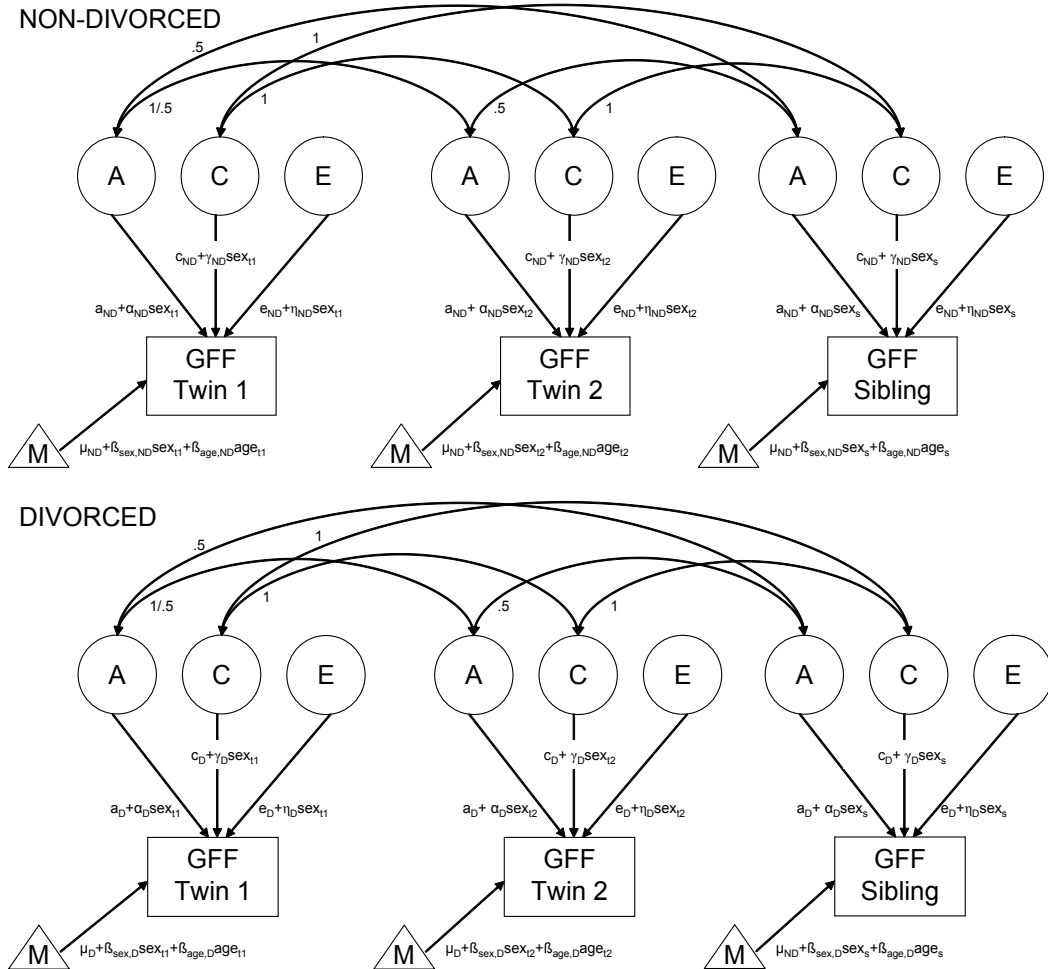
$$Y_{i,D} = \mu_D + \beta_{sex,D} \text{sex}_i + \beta_{age,D} \text{age}_i + \varepsilon_{i,D} \quad (2)$$

where subscript i indicates the individual, μ the population mean, and ε the residual. We tested whether constraining each regression weight at zero led to a significant deterioration of model fit. In addition, we assessed whether mean levels of GFF, FC, and QLg were different between non-divorced and divorced families by testing whether constraining means of both family types to be equal led to a significant deterioration of model fit.

Next, we tested whether twin and sibling correlations for GFF, FC, and QLg were equal for DZ twins and siblings and for MZ twins and DZ twins / siblings from non-divorced and divorced families. Estimating correlations for MZ twin, DZ twin, and twin-sibling pairs constitutes a first step in evaluating the relative influence of genetic and environmental factors on trait variances. More specifically, when the MZ correlation is higher for a certain trait than the DZ and the non-twin sibling correlations, it is inferred that genetic variation influences individual differences in the trait under study. DZ / sib correlations higher than half the MZ correlation implies shared environmental effects referring to environmental influences shared by all siblings reared in the same family. When the DZ correlation is higher than the twin-sibling correlation, a specific environment might exist which is shared by twins but not by non-twin siblings (Eaves et al., 1999). The remaining variation is attributed to nonshared environment which refers to environmental influences that are not shared by family members. The nonshared environmental variance component also includes measurement error variance. We estimated correlations for MZ twin, DZ, twin, and twin-sibling pairs as a function of sex and parental divorce.

Using structural equation modeling in Mx, genetic models were fitted to the data in which the genetic architecture of GFF, FC, and QLg was specified. A graphical representation of the genetic model for GFF is given in figure 1. This representation also holds for the genetic models of FC and QLg. The amount of variance in a single trait due to additive genetic effects (A), shared environmental effects (C), and nonshared environmental effects (E) can be estimated by considering the different levels of genetic relatedness between MZ and DZ twin pairs and non-twin siblings. MZ twin pairs are genetically identical, whereas DZ twin pairs and non-twin siblings share on average 50% of their genetic material. A variance component comprising twin specific environment was not modeled, because twins and siblings did not differ in correlational structure.

Figure 2.1. Univariate genetic model for general family functioning (GFF) with moderating effects of sex.



Note. GFF = general family functioning; M = moderator.

The influence of A, C and E is represented by path coefficients a, c, and e. Parameter estimates of a, c, and e were allowed to differ for individuals from non-divorced and divorced families. Significant covariates (sex and/or age) were retained in the means model.

To test for interaction between sex and genetic and environmental influences, the method as proposed by Purcell (2002) was used. The effect of sex was included as a moderator on the path coefficients a, c, and e and it was allowed to be different between non-divorced and divorced families. This can be seen in Figure 2.1, in which the path coefficients of the latent factors are expressed as linear functions of the moderator which are different between non-divorced and divorced families. Under this model, for each

individual in the family conditional on the individuals' sex, the expected trait variance (ε_i) of GFF, FC, and QLg for non-divorced and divorced families respectively can be expressed as:

$$\varepsilon_{i,ND} = (a_{ND} + \alpha_{ND} \text{sex}_i)^2 + (c_{ND} + \gamma_{ND} \text{sex}_i)^2 + (e_{ND} + \eta_{ND} \text{sex}_i)^2 \quad (3)$$

$$\varepsilon_{i,D} = (a_D + \alpha_D \text{sex}_i)^2 + (c_D + \gamma_D \text{sex}_i)^2 + (e_D + \eta_D \text{sex}_i)^2 \quad (4)$$

For pairs of relatives (i.e. MZ twins, DZ twins, and twin-sibling pairs) the within-trait covariance of GFF, FC, and QLg for non-divorced and divorced families respectively can be expressed as:

$$\text{Cov}_{\varepsilon_{1ND}, \varepsilon_{2ND}} = r_g (a_{ND} + \alpha_{ND} \text{sex}_1) (a_{ND} + \alpha_{ND} \text{sex}_2) + (c_{ND} + \gamma_{ND} \text{sex}_1) (c_{ND} + \gamma_{ND} \text{sex}_2) \quad (5)$$

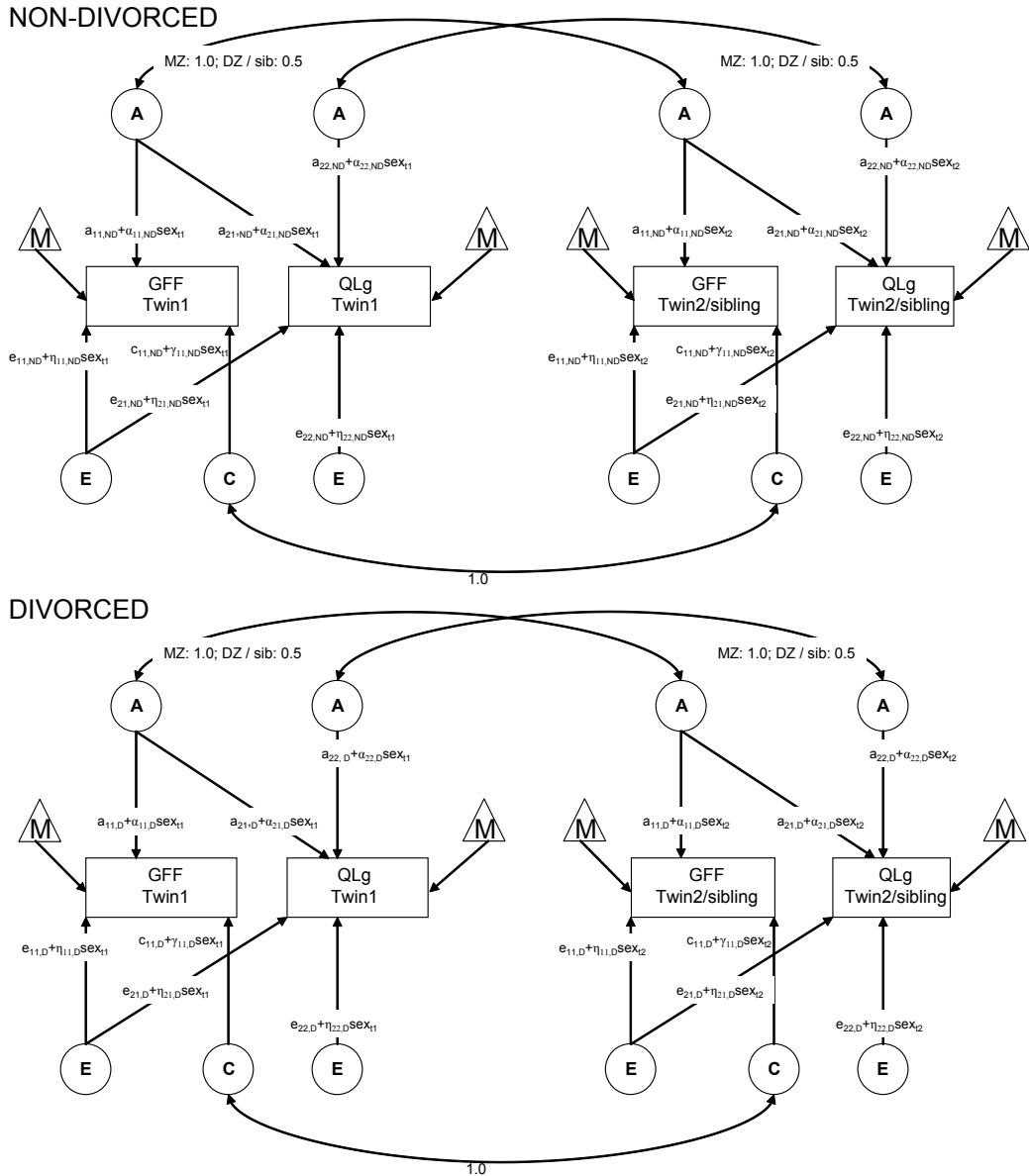
$$\text{Cov}_{\varepsilon_{1D}, \varepsilon_{2D}} = r_g (a_D + \alpha_D \text{sex}_1) (a_D + \alpha_D \text{sex}_2) + (c_D + \gamma_D \text{sex}_1) (c_D + \gamma_D \text{sex}_2) \quad (6)$$

where r_g is 1.0 for MZ twin pairs, and 0.5 for DZ twin and twin-sibling pairs.

Under this model, a , c , and e represent the unmoderated variance components, and the α -, γ - and η -coefficients represent the moderating effects of sex on the genetic, shared environmental, and nonshared environmental variance components respectively. If for example, under this model, α_{ND} is significantly different from zero, this is evidence for an interaction between the latent genetic factor of GFF, FC, or QLg and sex in non-divorced families. In the same way interactions between sex and both latent environmental factors can be detected as well. We assessed the significance of the sex effects by testing whether fixing the α -, γ - and η -coefficients to zero resulted in a significant deterioration of model fit.

To assess interaction between parental divorce and genetic and environmental influences, we tested whether constraining the genetic ($a_{ND} = a_D$), shared environmental ($c_{ND} = c_D$), and nonshared environmental ($e_{ND} = e_D$) parameter estimate of GFF, FC, and QLg to be equal between non-divorced and divorced families resulted in a significant deterioration of model fit. If for example, the genetic parameter estimate of GFF in non-divorced families (a_{ND}) is significantly different from the genetic parameter estimate in divorced families (a_D), this is evidence for an interaction between the latent genetic factor of GFF and parental divorce. In the same way interactions between parental divorce and the latent environmental factors can be detected as well.

Figure 2.2. Bivariate genetic model for general family functioning (GFF) and quality of life in general (QLg) with moderating effects of sex



Note. GFF = general family functioning; QLg = quality of life in general; M = moderator.

The presence of these interactions implies that genetic, shared environmental, and nonshared environmental influences on GFF, FC, and QLg increase or decrease as a function of parental divorce. A complicating issue is that interaction between parental divorce and genetic and environmental influences can be sex specific as well. To assess

if this is the case we tested whether constraining the α -, γ - and η -coefficients between non-divorced and divorced families to be equal ($\alpha_{ND} = \alpha_D$; $\gamma_{ND} = \gamma_D$; $\eta_{ND} = \eta_D$) led to a significant deterioration of model fit. If for example, the α -, γ - and η -coefficients of GFF in non-divorced families are significantly different from the α -, γ - and η -coefficients in divorced families, this indicates that the genetic architecture of GFF is different between non-divorced and divorced families in boys and girls.

Bivariate saturated and genetic analyses

To examine the overlap between both measures of family functioning and quality of life in general, two bivariate models were fitted to the data. First, phenotypic correlations between GFF and QLg and between FC and QLg, and cross-twin cross-trait correlations were obtained. Just as twin and twin-sibling correlations within traits provide information on the relative influence of genetic and environmental factors on trait variances, cross-twin cross-traits correlations provide information about the relative influence of these factors on the covariation between traits. Bivariate genetic models were fitted to the data in which genetic and environmental influences on the covariation between GFF and QLg, and between FC and QLg were estimated. The shared environmental covariance component was not modeled, because the contribution of shared environmental influences to variation in QLg was negligible. A graphical representation of the bivariate genetic model for the overlap between GFF and QLg is given in figure 2. This representation also holds for the genetic bivariate model for FC and QLg. Constraints on the parameter estimates of trait variances were adopted from the best fitting univariate models. Parameter estimates of the covariation between traits were estimated without constraints. The effect of sex on the covariance between traits was included as a moderator on the path coefficients and all parameter estimates were allowed to differ for individuals from non-divorced and divorced families. Under this model, for each individual (i) in the family conditional on the individual's sex, the observed (within-person) cross-trait covariance (CrossC, i.e. the covariance of the residuals (ε) for each trait) for non-divorced (ND) and divorced families (D) respectively can be expressed as:

$$\text{CrossC}_{i,ND} = (a_{11,ND} + \alpha_{11,ND} \text{sex}_i) (a_{21,ND} + \alpha_{21,ND} \text{sex}_i) + (e_{11,ND} + \eta_{11,ND} \text{sex}_i) (e_{21,ND} + \eta_{21,ND} \text{sex}_i) \quad (7)$$

$$\text{CrossC}_{i,D} = (a_{11,D} + \alpha_{11,D} \text{sex}_i) (a_{21,D} + \alpha_{21,D} \text{sex}_i) + (e_{11,D} + \eta_{11,D} \text{sex}_i) (e_{21,D} + \eta_{21,D} \text{sex}_i) \quad (8)$$

For pairs of relatives the cross-trait covariance (CrossC) for non-divorced and divorced families respectively can be expressed as:

$$\text{CrossC}_{1,2,ND} = r_g (a_{11,ND} + \alpha_{11,ND} \text{sex}_1) (a_{21,ND} + \alpha_{21,ND} \text{sex}_2) \quad (9)$$

$$\text{CrossC}_{1,2,D} = r_g (a_{11,D} + \alpha_{11,D} \text{sex}_1) (a_{21,D} + \alpha_{21,D} \text{sex}_2) \quad (10)$$

where 1 and 2 refer to different traits in twin 1 and twin 2 (see Figure 2.2) and r_g is 1.0 for MZ twin pairs, and 0.5 for DZ twin and twin-sibling pairs. We assessed the significance of the sex effects by testing whether fixing the α - and η -coefficients linked to the covariance components to zero resulted in a significant deterioration of model fit.

To assess interaction between parental divorce and genetic and environmental influences on the overlap between GFF and QLg, and FC and QLg we tested whether constraining the genetic ($a_{21,ND} = a_{21,D}$) and nonshared environmental ($e_{21,ND} = e_{21,D}$) parameter estimate to be equal between non-divorced and divorced families resulted in a significant deterioration of model fit. The presence of these interactions implies that genetic and nonshared environmental influences on the overlap between GFF and QLg, and between FC and QLg increase or decrease as a function of parental divorce. A complicating issue is that interaction between parental divorce and genetic and environmental influences on the overlap can be sex specific as well. To assess if this is the case we tested whether constraining the α - and η -coefficients between non-divorced and divorced families to be equal ($\alpha_{ND} = \alpha_D$; $\eta_{ND} = \eta_D$) led to a significant deterioration of model fit. If for example, the α - and η -coefficients of GFF in non-divorced families are significantly different from the α - and η -coefficients in divorced families, this indicates that the genetic architecture of the overlap between GFF and QLg and between FC and QLg is different between non-divorced and divorced families in boys and girls.

The fit of the different models was compared by the log-likelihood ratio test (LRT). The difference in minus two times the log-likelihood (-2LL) between two models has a χ^2 distribution with the degrees of freedom (Δdf) equaling the difference in df between the two models. The df of a model equals the difference in the number observations and the number of model parameters. If a p-value higher than 0.05 was obtained from the

χ^2 -test the fit of the constrained model was not significantly worse than the fit of the more complex model. In this case, the constrained model was kept as the most parsimonious and best fitting model.

Interaction between genetic influences and nonshared environment

To explore the interaction between genotype and nonshared, non-measured, environmental influences, correlations between the sum scores and absolute difference scores within MZ twin pairs were computed (Jinks and Fulker, 1970). By looking at the association between intrapair differences and sum scores in MZ twins the assumption of independency of genetic and nonshared environmental influences can be determined. In the present study, 1,171 families with monozygotic twins participated. In each family (n) twins have scores s_{n1} and s_{n2} on, for example, GFF. Each absolute intrapair difference score (i.e. $\text{abs}(s_{n1} - s_{n2})$) provides an estimate of the magnitude of nonshared environmental influences within families. There will be variation in these intrapair differences, because twins in some families are likely to react differently from those in other families when exposed to environmental influences, or because twins in some families are exposed to different environments than in other families. On the other hand, variation in the sum of intrapair twin scores (i.e. $s_{n1} + s_{n2}$) appears if twins belonging to different families have different genotypes and/or family environments. When there is a negative correlation between intrapair sum and absolute differences, individuals who evaluate GFF as negative are more different than those who evaluate GFF as positive, and thus individuals who evaluate GFF as positive are less susceptible to unique environmental influences (e.g. Van Leeuwen et al., 2007). Analyses were done in SPSS (version 16.0).

Results

Univariate saturated models

In Table 2.2, means and variances of GFF, FC, and QLg (uncorrected for age) are presented as a function of parental divorce and sex. Sex and age effects on means of GFF ($\chi^2(2) = .58, p = .75$) and FC ($\chi^2(2) = 1.87, p = .39$) and the age effect on the mean of QLg ($\chi^2(1) = .11, p = .74$) were not significantly different between non-divorced and divorced families. The sex effect on the mean of QLg was significantly different between non-divorced and divorced families ($\chi^2(1) = 11.54, p < .01$). The effect of sex on the means of GFF ($\chi^2(1)$

= .004, $p = .95$) and FC ($\chi^2 (1) = 3.67$, $p = .06$) were not significant, whereas the effect of sex on the mean of QLg was significant for non-divorced ($\chi^2 (1) = 12.09$, $p < .01$) and divorced families ($\chi^2 (1) = 22.09$, $p < .01$).

Table 2.2

Maximum likelihood estimates of means and variances of GFF, FC, and QLg as a function of sex and parental divorce

			M	Var
GFF	ND	Boys	38.7	24.1
		Girls	38.6	26.0
	D	Boys	37.5	23.3
		Girls	37.6	32.0
FC	ND	Boys	16.7	4.8
		Girls	16.6	4.9
	D	Boys	16.5	4.9
		Girls	16.3	5.1
QLg	ND	Boys	7.9	1.1
		Girls	7.8	1.2
	D	Boys	7.8	1.1
		Girls	7.3	1.9

Note. GFF = general family functioning; FC = family conflict; QLg = quality of life general; ND = non-divorced families; D = divorced families.

In non-divorced and divorced families boys reported higher levels of QLg than girls. There was a small but significant effect of age on the mean of GFF ($\chi^2 (1) = 25.71$, $p < .01$; standardized regression coefficient $\beta = -.07$; $r^2 = .005$) and QLg ($\chi^2 (1) = 46.50$, $p < .01$; standardized regression coefficient $\beta = -.09$; $r^2 = .008$), indicating that means were higher for younger participants. No significant age effect was found on the mean of FC. Means of GFF ($\chi^2 (1) = 18.23$, $p < .01$), FC ($\chi^2 (1) = 4.76$, $p < .05$), and QLg ($\chi^2 (1) = 5.16$, $p < .05$) were significantly lower for individuals from divorced families compared to those from non-divorced families. As can be seen in Table 2.2, variation in GFF, FC, and QLg is the same or higher for participants from divorced families compared to those from non-divorced families, indicating that participants living in divorced families form an equally or even more heterogeneous group regarding scores on GFF, FC, and QLg compared to those living in non-divorced families. This suggests that the effects of living in non-divorced or divorced families have a positive influence on reports of GFF, FC, and QLg for some participants and a negative influence for others.

Table 2.3

Familial and cross-twin cross-trait correlation for GFF, FC, and QLg

	MZM	DZM	MZF	DZF	DOS	Brother	Sister	Brother-sister
Twin correlations for GFF								
ND	.44	.37	.53	.43	.33	.34	.31	.32
D	.24	.16	.56	.29	.46	.11	.30	.20
Twin correlations for FC								
ND	.63	.58	.68	.57	.55	.38	.46	.46
D	.69	.58	.76	.56	.49	.61	.66	.61
Twin correlations for QLg								
ND	.38	.20	.46	.36	.24	.04	.18	.19
D	.23	.14	.35	.10	.11	.02	.25	.11
Cross-twin cross-trait correlations for GFF and QLg								
ND	.24	.17	.30	.24	.23	.12	.21	.18
D	.09	.16	.26	.17	.16	-.04	.14	.15
Cross-twin cross-trait correlations for FC and QLg								
ND	.29	.15	.24	.14	.17	.14	.19	.15
D	.23	.28	.20	.01	.05	.04	.13	.08

Note. ND = non-divorced families; D = divorced families; GFF = general family functioning; FC = family conflict; MZ = Monozygotic twin pair; DZ = Dizygotic twin pair; M = male; F = female; DOS = Opposite-sex twin pairs; Brother = Male twin-brother pairs; Sister = Female twin-sister pairs; Brother-sister = Male twin-sister pairs, Female twin-brother pairs

Twin and twin-sibling correlations for GFF, FC, and QLg are presented in the upper part of Table 2.3. Constraining the sex effects on the covariance structure for non-divorced and divorced families to be equal led to a significant deterioration of model fit for GFF ($\chi^2(1) = 4.78$, $p < .05$) and QLg ($\chi^2(1) = 19.21$, $p < .01$), but not for FC ($\chi^2(1) = .004$, $p = .95$). The significance of the sex effects on the covariance structure of GFF and QLg was therefore tested for non-divorced and divorced families separately. For GFF, there were significant sex differences on the covariance structure for participants from non-divorced ($\chi^2(1) = 4.61$, $p < .05$) and divorced families ($\chi^2(1) = 9.05$, $p < .01$).

Table 2.4

Univariate model fitting results for GFF, FC, and QLg

Model	vs.	-2LL	df	χ^2	df	p
GFF						
1. Full model	--	39932.350	6669	--	--	--
2. $\alpha_{ND}=\alpha_{D'}$, $\gamma_{ND}=\gamma_{D'}$, $\eta_{ND}=\eta_D$	1	39936.106	6672	3.76	3	.29
3. $\alpha_{ND}=\alpha_D=0$, $\gamma_{ND}=\gamma_D=0$, $\eta_{ND}=\eta_D=0$	2	39957.131	6675	21.03	3	< .01
4. $a_{ND} = a_D$	2	39940.401	6673	4.30	1	< .05
5. $c_{ND} = c_D$	2	39938.414	6673	2.31	1	.13
6. $e_{ND} = e_D$	2	39936.152	6673	.05	1	.83
7. $c_{ND} = c_D$, $e_{ND} = e_D$	2	39938.599	6674	2.49	2	.29
FC						
1. Full model	--	28231.223	6698	--	--	--
2. $\alpha_{ND}=\alpha_{D'}$, $\gamma_{ND}=\gamma_{D'}$, $\eta_{ND}=\eta_D$	1	28232.124	6701	.90	3	.83
3. $\alpha_{ND}=\alpha_D=0$, $\gamma_{ND}=\gamma_D=0$, $\eta_{ND}=\eta_D=0$	2	28239.099	6704	6.98	3	.07
4. $a_{ND} = a_D$	3	28239.207	6705	.11	1	.74
5. $c_{ND} = c_D$	3	28239.352	6705	.25	1	.61
6. $e_{ND} = e_D$	3	28241.622	6705	2.52	1	.11
7. $a_{ND} = a_D$, $c_{ND} = c_D$, $e_{ND} = e_D$	3	28243.079	6707	3.98	3	.26
QLg						
1. Full model	--	19662.038	6601	--	--	--
2. $\alpha_{ND}=\alpha_{D'}$, $\gamma_{ND}=\gamma_{D'}$, $\eta_{ND}=\eta_D$	1	19674.482	6604	12.44	3	< .01
3. $\alpha_{ND}=0$, $\gamma_{ND}=0$, $\eta_{ND}=0$	1	19676.436	6604	14.40	3	< .01
4. $\alpha_D=0$, $\gamma_D=0$, $\eta_D=0$	1	19688.708	6604	26.67	3	< .01
5. $a_{ND} = a_D$	1	19663.254	6602	1.22	1	.27
6. $c_{ND} = c_D$	1	19662.038	6602	.00	1	> .99
7. $e_{ND} = e_D$	1	19662.711	6602	.67	1	.41
8. $a_{ND} = a_D$, $c_{ND} = c_D$, $e_{ND} = e_D$	1	19663.294	6604	1.26	3	.74

Note. vs. = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; df = degrees of freedom of χ^2 -test; p = p-value; Full model = ACE model with differences in parameter estimates for parental divorce and sex

For QLg, there also were significant sex differences on the covariance structure for participants from non-divorced (χ^2 (1) = 8.40, p < .01) and divorced families (χ^2 (1) = 29.42, p < .01). No significant sex differences were found on the covariance structure of

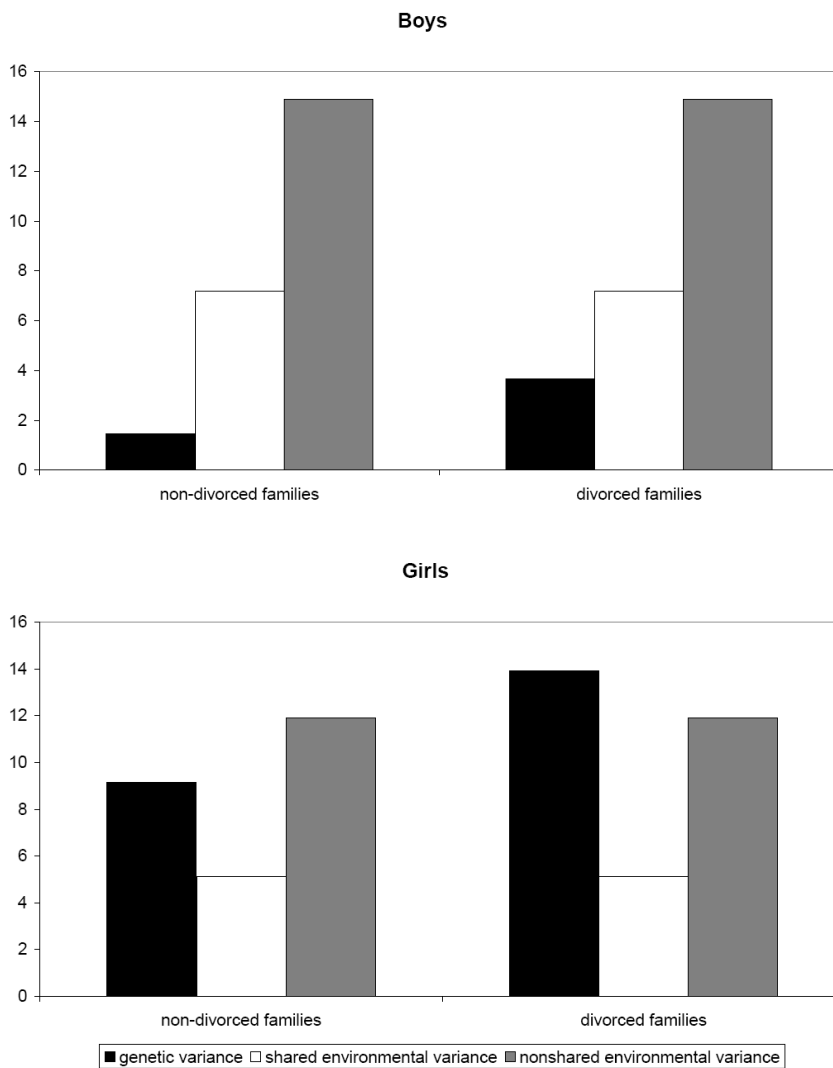
FC. No indication was found for environmental influences specifically shared by twins explaining variation in GFF, FC, and QLg for non-divorced and divorced families (p-values exceeded a 5% significance level). For GFF, FC and QLg in non-divorced families, the MZ twin correlation was larger than the DZ twin and twin – sibling correlation (all p-values < .01). For GFF and QLg in divorced families, the MZ twin correlation was equal to the DZ twin and twin – sibling correlation (both p-values > .05). For FC in divorced families, the MZ twin correlation was larger than the DZ twin and twin – sibling correlation ($\chi^2(1) = 11.71, p < .01$). Because the correlational structure of the data was not identical for participants from non-divorced and divorced families and between boys and girls, we started genetic modeling with an ACE model with different parameter estimates for participants from non-divorced and divorced families and with sex as a moderator on the genetic and environmental path coefficients.

Univariate genetic models

Table 2.4 presents the univariate model fitting results of the genetic models. In the upper part of the table the model fitting results for GFF are presented. We began with constraining the moderation effects of sex on the path coefficients of GFF to be equal between non-divorced and divorced families, which did not result in a significant deterioration of model fit (model 2). In model 3, the statistical significance of the moderation effects of sex on the path coefficients of GFF was tested, which resulted in a significant deterioration of model fit. This indicates that the magnitude of genetic, shared environmental, and nonshared environmental effects on GFF are different between boys and girls. Model 4 till 6 tested whether constraining the genetic, shared environmental, and nonshared environmental parameter estimate to be equal between non-divorced and divorced families would lead to a significant deterioration of model fit. Additive genetic effects on GFF were significantly different between participants from non-divorced and divorced families, indicating that additive genetic effects on GFF are relatively more important in divorced families compared to non-divorced families: i.e. evidence for an interaction between additive genetic effects and parental divorce. Shared and nonshared environmental effects on GFF were not significantly different between participants from non-divorced and divorced families and therefore they were constrained to be equal in model 7 simultaneously which did not result in a significant deterioration of model fit. Figure 2.3 presents the unstandardized contributions of genetic, shared environmental and nonshared environmental effects to variation in GFF as a function of sex and parental divorce.

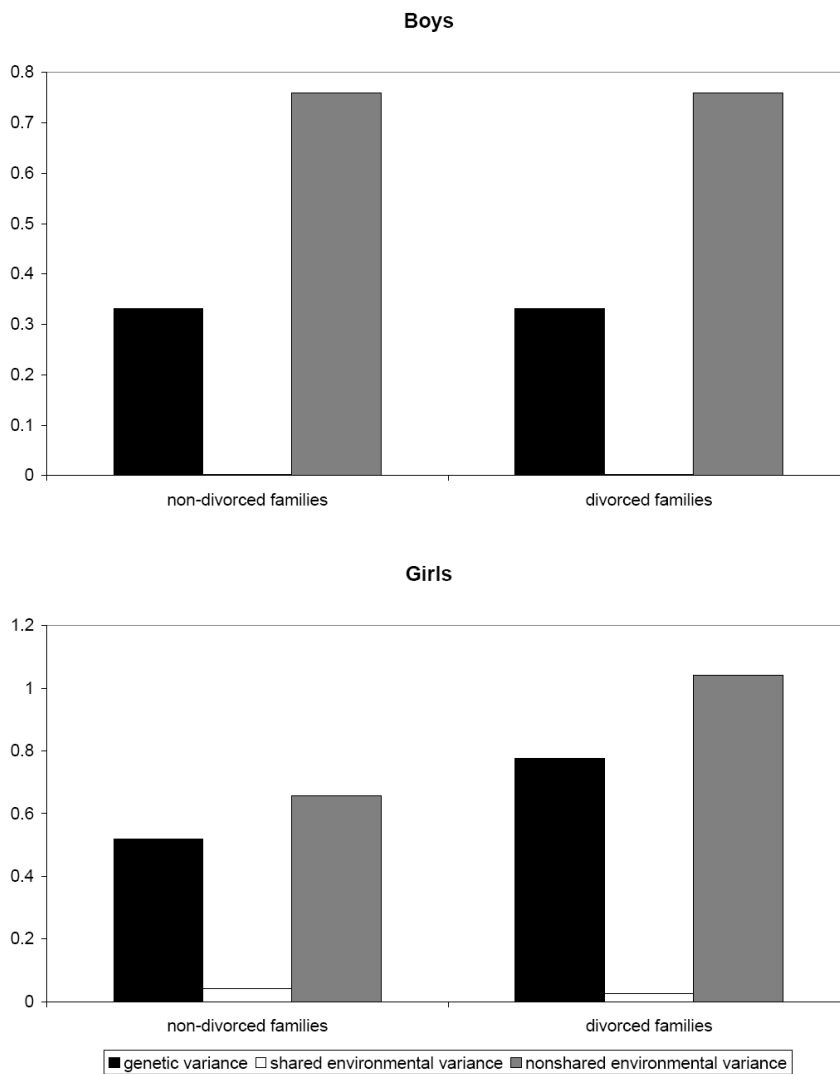
In the middle part of Table 2.4 the model fitting results for FC are presented. Constraining the moderation effects of sex on the path coefficients of FC to be equal between non-divorced and divorced families did not result in a significant deterioration of model fit (model 2). In model 3, no statistical significant moderation effects of sex on the path coefficients of FC were found, suggesting that the genetic architecture of FC is equal between boys and girls.

Figure 2.3. Changes in the absolute contribution of genetic, shared environmental, and nonshared environmental effects to variation in GFF in boys and girls.



Additive genetic (model 4), shared environmental (model 5), and nonshared environmental effects (model 6) on FC were not significantly different between participants from non-divorced and divorced families. In model 7, the genetic, shared environmental, and nonshared environmental path coefficient on FC were constrained to be equal between non-divorced and divorced families simultaneously, which did not result in a significant deterioration of model fit.

Figure 2.4. Changes in the absolute contribution of genetic, shared environmental, and nonshared environmental effects to variation in QLg in boys and girls.



In the lower part of Table 2.4 the model fitting results of QLg are presented. With regard to QLg, constraining the moderation effects of sex on the path coefficients to be equal between non-divorced and divorced families resulted in a significant deterioration of model fit (model 2). This indicates that the effects of sex on the genetic architecture of QLg are different between participants from non-divorced and divorced families. In model 3, the statistical significance of the moderation effect of sex on the genetic architecture of QLg in non-divorced families was tested, which resulted in a significant deterioration of model fit. In model 4, the statistical significance of the moderation effect of sex on the genetic architecture of QLg in divorced families was tested, which also resulted in a significant deterioration of model fit.

Table 2.5

Unstandardized and standardized parameter estimates for GFF, FC, and QLg, including 95% confidence intervals shown in parentheses

		A		C		E	
		Unstand.	Stand.	Unstand.	Stand.	Unstand.	Stand.
GFF							
Boys	ND	1.47	.06 (.00-.30)	7.20	.31 (.12-.40)	14.88	.63 (.55-.70)
	D	3.67	.14 (.00-.38)	7.20	.28 (.11-.39)	14.88	.58 (.48-.67)
Girls	ND	9.15	.35 (.20-.47)	5.10	.20 (.10-.35)	11.89	.45 (.41-.52)
	D	13.91	.45 (.30-.56)	5.10	.17 (.08-.29)	11.89	.38 (.32-.45)
FC							
Boys	ND	1.54	.31 (.24-.39)	1.75	.36 (.30-.41)	1.61	.33 (.30-.36)
	D	1.54	.31 (.24-.39)	1.75	.36 (.30-.41)	1.61	.33 (.30-.36)
Girls	ND	1.54	.31 (.24-.39)	1.75	.36 (.30-.41)	1.61	.33 (.30-.36)
	D	1.54	.31 (.24-.39)	1.75	.36 (.30-.41)	1.61	.33 (.30-.36)
QLg							
Boys	ND	.33	.30 (.18-.37)	.00	.00 (.00-.09)	.76	.70 (.63-.77)
	D	.33	.30 (.18-.37)	.00	.00 (.00-.09)	.76	.70 (.63-.77)
Girls	ND	.52	.43 (.25-.52)	.04	.03 (.00-.18)	.66	.54 (.48-.60)
	D	.78	.42 (.00-.58)	.03	.01 (.00-.38)	1.04	.56 (.42-.75)

Note. Unstand. = unstandardized variance component; Stand. = standardized variance component; GFF = general family functioning; FC = family conflict; QLg = quality of life general; ND = non-divorced families; D = divorced families

This indicates that the magnitude of genetic, shared environmental and nonshared environmental effects on QLg is different between boys and girls from non-divorced and divorced families. In model 5 till 7, the genetic, shared environmental, and nonshared environmental parameter estimates were constrained to be equal between non-divorced and divorced families, which did not result in a significant deterioration of model fit. This suggests that, independent of sex, additive genetic, shared environmental, and nonshared environmental effects on QLg were not significantly different between non-divorced and divorced families. However, the results from model 2 till 4 indicate that there is a sex specific interaction between parental divorce and the genetic architecture of QLg. In Figure 2.4 can be seen that the contribution of genetic and environmental effects to variation in QLg is equal between non-divorced and divorced families in boys, whereas for girls the contribution of genetic and nonshared environmental effects increased in divorced families.

The contributions of A, C, and E to variation in GFF, FC and QLg in non-divorced and divorced families are summarized in Table 2.5. Variation in GFF was mainly accounted for by shared and nonshared environmental influences in boys, whereas in girls additive genetic influences played a substantive role as well. Heritability of GFF increased from 6% in non-divorced families to 14% in divorced families in boys and from 35% to 45% in girls. With regard to variation in FC, additive genetic, shared environmental and nonshared environmental influences accounted equally in boys and girls from non-divorced and divorced families. Variation in QLg was accounted for by additive genetic and nonshared environmental influences. The influence of shared environmental influences was negligible. Additive genetic influences were relatively more important in girls. The absolute contribution of additive genetic and nonshared environmental influences on QLg increased for girls from divorced families compared to girls from non-divorced families, whereas the relative contribution was similar. This is due to an overall increased variation in QLg observed in girls from divorced families (see Table 2.2). In other words, this indicates that girls from divorced families are more heterogeneous with regard to their reports on QLg as compared to those from non-divorced families.

Bivariate genetic models

Phenotypic correlations between GFF and QLg were .36 for boys and .41 for girls ($\chi^2(1) = 6.14, p < .05$). Phenotypic correlations between FC and QLg were .20 for boys and .31 for girls ($\chi^2(1) = 27.59, p < .01$). Cross-twin cross-trait correlations are presented in the lower part of Table 2.3. Table 2.6 presents the bivariate model fitting results of the genetic models. Path coefficients and their corresponding beta-weights representing shared

environmental influences to covariation between GFF and QLg and FC and QLg were fixed to zero, because of the negligible contribution of shared environmental effects to variation in QLg. In the upper part of Table 2.6 the model fitting results for the overlap between GFF and QLg are presented. In model 2, constraining the moderation effects of sex on the path coefficients to be equal between non-divorced and divorced families resulted in a significant deterioration of model fit (model 2). This indicates that the effects of sex on the genetic architecture of the overlap between GFF and QLg are different in magnitude between participants from non-divorced and divorced families.

Table 2.6

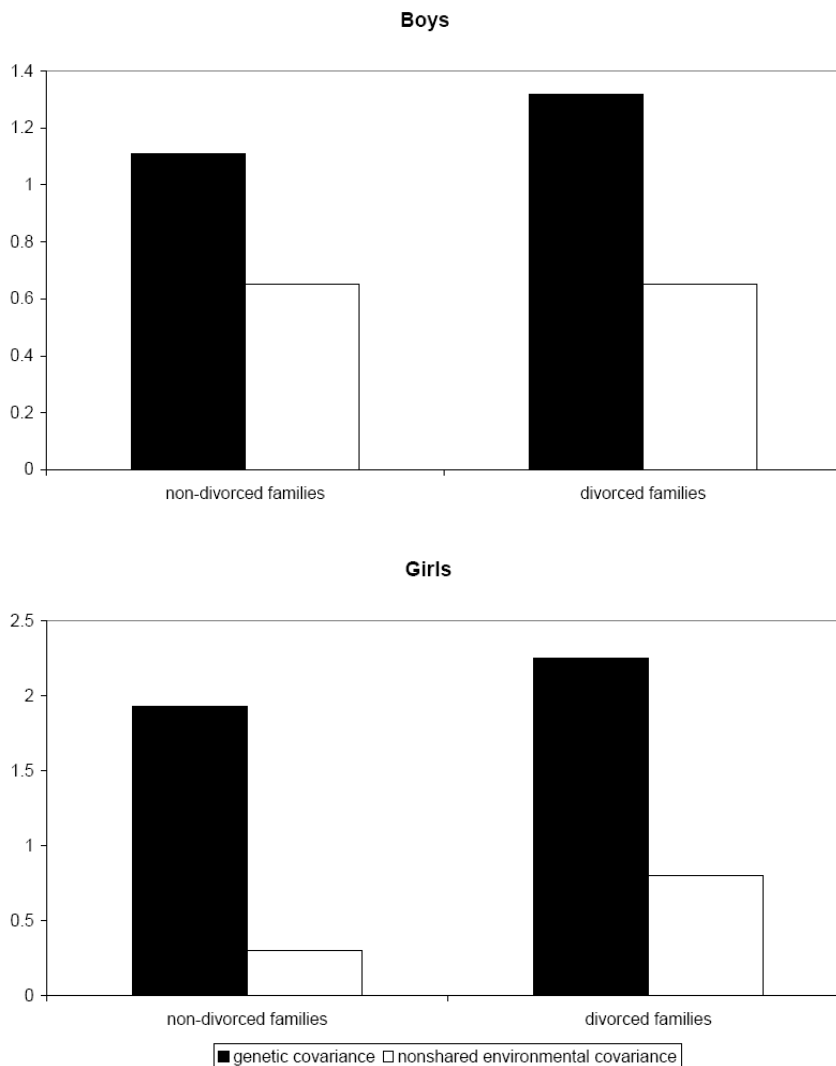
Bivariate model fitting results for the overlap between GFF and QLg, and FC and QLg						
Model	vs.	-2LL	df	χ^2	df	p
GFF - QLg						
1. Full model	--	58667.780	13270	--	--	--
2. $\alpha_{21,ND} = \alpha_{21,D}; \eta_{21,ND} = \eta_{21,D}$	1	58674.192	13272	6.41	2	< .05
3. $\alpha_{21,ND} = 0; \alpha_{21,D} = 0; \eta_{21,ND} = 0; \eta_{21,D} = 0$	1	58678.026	13274	10.25	4	< .05
4. $a_{21,ND} = a_{21,D}$	1	58668.387	13271	.61	1	.44
5. $e_{21,ND} = e_{21,D}$	1	58668.164	13271	.38	1	.54
6. $a_{21,ND} = a_{21,D}; e_{21,ND} = e_{21,D}$	1	58670.231	13272	2.43	2	.30
FC - QLg						
1. Full model	--	47475.630	13303	--	--	--
2. $\alpha_{21,ND} = \alpha_{21,D}; \eta_{21,ND} = \eta_{21,D}$	1	47482.365	13305	6.74	2	< .05
3. $\alpha_{21,ND} = 0; \alpha_{21,D} = 0; \eta_{21,ND} = 0; \eta_{21,D} = 0$	1	47510.240	13307	34.61	4	< .01
4. $a_{21,ND} = a_{21,D}$	1	47476.546	13304	.92	1	.34
5. $e_{21,ND} = e_{21,D}$	1	47476.999	13304	1.37	1	.24
6. $a_{21,ND} = a_{21,D}; e_{21,ND} = e_{21,D}$	1	47477.347	13305	1.72	2	.42

Note. vs. = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; df = degrees of freedom of χ^2 -test; p = p-value; Full model = AE model with differences in parameter estimates for parental divorce and sex

In model 3, the statistical significance of the moderation effect of sex on the genetic and nonshared environmental parameter estimate of the covariation between GFF and QLg was tested, which resulted in a significant deterioration of model fit. In model 4 and 5, the genetic and nonshared environmental parameter estimates were constrained to be equal between non-divorced and divorced families, which did not result in a significant

deterioration of model fit. This suggests that, independent of sex, the contribution of additive genetic and nonshared environmental effects to the overlap between GFF and QLg was not significantly different between non-divorced and divorced families. However, the results from model 2 and 3 indicate that there is a sex specific interaction between parental divorce and the genetic architecture of the overlap between GFF and QLg.

Figure 2.5. Changes in the absolute contribution of genetic and nonshared environmental covariation between GFF and QLg in boys and girls.

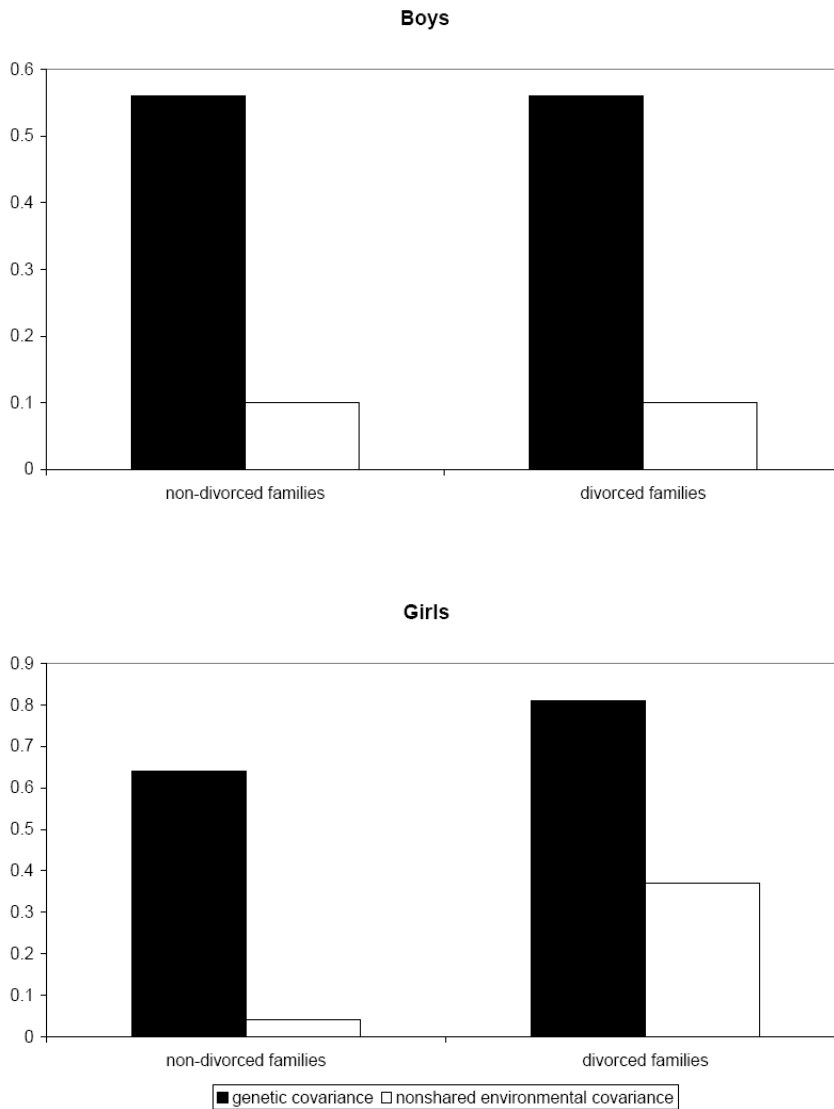


In Figure 2.5 can be seen that the contribution of genetic and nonshared environmental effects to covariation between GFF and QLg is equal for boys from non-divorced and divorced families, whereas for girls the contribution of genetic and nonshared environmental effects increased in divorced families.

In the lower part of Table 2.6 the model fitting results for the overlap between FC and QLg are presented. In model 2, constraining the moderation effects of sex on the path coefficients to be equal between non-divorced and divorced families resulted in a significant deterioration of model fit. This indicates that the effects of sex on the genetic architecture of the overlap between FC and QLg are different in magnitude between participants from non-divorced and divorced families. In model 3, the statistical significance of the moderation effect of sex on the genetic and nonshared environmental parameter estimate of the overlap between FC and QLg was tested, which resulted in a significant deterioration of model fit. In model 4 and 5, the genetic and nonshared environmental parameter estimates were constrained to be equal between non-divorced and divorced families, which did not result in a significant deterioration of model fit. This suggests that, independent of sex, the contribution of additive genetic and nonshared environmental effects to the overlap between FC and QLg was not significantly different between non-divorced and divorced families. However, the results from model 2 and 3 indicate that there is a sex specific interaction between parental divorce and the genetic architecture of the overlap between FC and QLg. In Figure 2.6 can be seen that the contribution of genetic and nonshared environmental effects to covariation between FC and QLg is equal for boys from non-divorced and divorced families, whereas for girls the contribution of genetic and nonshared environmental effects increased in divorced families.

The contributions of A and E to overlap between GFF and QLg, and FC and QLg in non-divorced and divorced families are presented in Table 2.7. Additive genetic influences accounted for the largest part of the covariation between GFF and QLg, whereas nonshared environmental effects accounted for a small part as well. Additive genetic influences were relatively more important in girls. The absolute contribution of additive genetic and nonshared environmental influences to the overlap between GFF and QLg increased for girls from divorced families compared to girls from non-divorced families. This is due to an overall increase in covariation between GFF and QLg for girls from divorced families. The relative contribution of additive genetic influences for girls from divorced families decreased in favor of nonshared environmental influences. With regard to the covariation between FC and QLg, additive genetic influences accounted (almost) exclusively for it.

Figure 2.6. Changes in the absolute contribution of genetic and nonshared environmental covariation between FC and QLg in boys and girls.



The absolute contribution of additive genetic and nonshared environmental influences to the overlap between FC and QLg increased for girls from divorced families compared to girls from non-divorced families. This is due to an overall increase in covariation between FC and QLg for girls from divorced families. The relative contribution of additive genetic influences for girls from divorced families decreased in favor of nonshared environmental influences.

With regard to overlap between GFF and QLg, the genetic and nonshared environmental correlations for boys from non-divorced and divorced families were .80 and .20 respectively. For girls from non-divorced families these correlation were .77 and .11, whereas for girls from divorced families they were .79 and .22 respectively. This suggests that adolescents' views of GFF and QLg share most of the underlying genetic influences and part of nonshared environmental influences. Regarding the overlap between FC and QLg, genetic and nonshared environmental correlations for boys from non-divorced and divorced families were .77 and .09 respectively.

Table 2.7

Unstandardized and standardized parameter estimates for covariation between GFF and QLg, and FC and QLg, including 95% confidence intervals shown in parentheses

		A		E	
		Unstand.	Stand.	Unstand.	Stand.
GFF – QLg					
Boys	ND	1.11	.63 (.50-.76)	.65	.37 (.24-.50)
	D	1.32	.67 (.67-.79)	.65	.33 (.21-.44)
Girls	ND	1.93	.86 (.86-.95)	.30	.14 (.05-.23)
	D	2.25	.74 (.63-.95)	.80	.26 (.05-.50)
FC – QLg					
Boys	ND	.56	.85 (.79-1.0)	.10	.15 (.14-.28)
	D	.56	.85 (.79-1.0)	.10	.15 (.14-.28)
Girls	ND	.64	.95 (.67-.98)	.04	.05 (.00-.17)
	D	.81	.67 (.67-.98)	.37	.31 (.09-.54)

Note. *Unstand.* = unstandardized variance component; *Stand.* = standardized variance component; *GFF - QLg* = overlap between general family functioning and quality of life general; *FC - QLg* = overlap between family conflict and quality of life general; *ND* = non-divorced families; *D* = divorced families.

For girls from non-divorced families these correlations were .68 and .04, whereas for girls from divorced families they were .79 and .29. This suggests that most genetic influences and part of nonshared environmental influences underlying adolescents' views of FC and QLg are common.

Correlation between sum and difference scores in MZ twin pairs

In Table 2.8, correlations between intrapair difference and sum scores in MZ twins are presented. Significant negative correlations were found for GFF, FC, and QLg in boys and girls, suggesting the presence of interaction between genetic and nonshared, non-measured, environmental influences. The significant correlations indicate that MZ twins reporting low levels of FC and high levels of GFF and QLg are more similar than those reporting high levels of FC and low levels of GFF and QLg. Hence, nonshared environmental influences are relatively more important in explaining variation for those who report low levels of FC, and high levels of GFF and QLg.

Table 2.8

Correlations between intrapair difference and sum scores in MZ twins

	MZM	MZF
GFF	-.11*	-.09*
FC	-.24**	-.24**
QLg	-.39**	-.41**

Note. * $p < .05$; ** $p < .001$.

Discussion

The present study investigated causes of variation in evaluations of GFF, FC, and QLg in a large sample of Dutch adolescent twins and their non-twin siblings. We tested whether the genetic architecture of GFF, FC, and QLg differed between boys and girls and between those living in non-divorced and divorced families. We found that individual differences in evaluations of GFF and FC could be accounted for by additive genetic, shared environmental and nonshared environmental influences. Heritability of evaluations of GFF was larger in girls and for participants from divorced families. Variation in QLg was accounted for by additive genetic and nonshared environmental influences, with heritability being larger in girls. Furthermore, girls from divorced families showed larger heterogeneity in their perceptions of QLg as compared to girls from non-divorced families.

We also examined the overlap between GFF and QLg and between FC and QLg by investigating to what extent genetic and environmental influences contribute to the overlap. Again, we tested whether the genetic architecture of the overlap differed between boys and girls and between those living in non-divorced and divorced families. The overlap between GFF and QLg and between FC and QLg could be accounted for by

additive genetic and nonshared environmental influences. Additive genetic influences were relatively more important than nonshared environmental influences. Girls from divorced families showed larger phenotypic overlap between GFF and QLg, and FC and QLg compared to girls from non-divorced families and boys. Finally, for evaluations of GFF, FC, and QLg we provided evidence that nonshared, non-measured, environmental influences interact with genetic factors.

With regard to adolescents' evaluations of GFF we found that genetic effects were relatively more important in girls than in boys. This finding is consistent with what was reported by Jacobson and Rowe (1999). Genetic influences on evaluations of QLg were also relatively more important in girls, which is in line with what was reported by Røysamb (2003) and Bartels et al. (in press). Genetic effects may be larger in evaluations of family functioning and subjective well-being in girls because they also have been found to be larger in traits that are likely to influence evaluations or perceptions of an individual's life. For example, sex differences have been found for depression and neuroticism with higher heritabilities for females (Boomsma et al., 2000; Kendler et al., 2001; Jardine et al., 1984). Furthermore, Jacobson and Rowe (1999) found genetic overlap between evaluations of family connectedness and depressed mood in girls. Thus, it is possible that genetic factors that influence traits such as depression and neuroticism may also influence evaluations of family functioning and SWB in girls.

Parental divorce modified the genetic architecture of evaluations of GFF. Genetic effects were larger in participants from families with divorced parents as compared to those from intact families. The increased impact of genetic factors on evaluations of GFF suggest that participants with a genetic predisposition towards negative evaluations of GFF are more sensitive to negative effects of parental divorce and those with a genetic predisposition towards positive evaluations are more sensitive to positive effects of parental divorce. For example, adolescents with a genetically predisposed tendency towards negative evaluations of family functioning may be disproportionately more likely to evaluate their family functioning as worse if one parent is absent, because they are likely to have more conflicts with their parents (e.g. Dunn et al, 1998; O'Connor et al., 2001) and receive less emotional support, supervision, and involvement from their parents (e.g. Carlson & Corcoran, 2001). On the other hand, participants with a genetically predisposed tendency towards positive evaluations of family functioning are disproportionately more likely to evaluate their family functioning as positive if the absence of one parent leads to a less stressful family situation as is for example likely to be the case when an antisocial father is absent (Jaffee et al., 2003; Blazer et al., 2008).

The genetic architecture of evaluations of the level of FC was, however, not modified by sex and parental divorce. It could be that the evaluation of the level of FC

is more dependent on true experiences of conflict and therefore is a more objective evaluation of family functioning. For example, evaluating whether family members criticize each other frequently (i.e. one example of the family conflict scale) might be more objective than the evaluation of family members get sufficient support from each other in times of crisis (i.e. one example of the general family functioning scale). Thus, the evaluation of the level of FC is assumed to be less due to the way adolescents perceive and interpret experiences in the family.

Individual differences in evaluations of QLg were larger for girls from families with divorced parents compared to those from intact families. This indicates that parental divorce has no general main effect on evaluations of subjective well-being but rather that there is large heterogeneity in the effects of parental divorce on evaluations of subjective well-being in girls. Although to a lesser extent, this also holds for boys, because boys from families with divorced parents also did not become more homogenous with regard to their evaluations of QLg which might be expected if parental divorce had a general negative main effect on the evaluation of SWB and adjustment. Heterogeneity in the effects of parental divorce on SWB is a plausible explanation of general small effect sizes found in research investigating main effects of parental divorce on well-being and adjustment (e.g. Amato & Keith, 1991; McFarlane et al., 1995). Similar results were also obtained for evaluations of GFF and FC. An important implication of these results for future research with regard to adolescents' evaluations of family functioning, subjective well-being, and adjustment is to focus on etiologies of individual differences in the effects of (negative) family events and life events in general, instead of focusing on general effects. Our results indicated that adolescents react differently to parental divorce and that heterogeneity in these reactions has different causes (e.g. genetic, factors from an adolescent's personal environment).

Moderate associations were found between GFF and QLg and between FC and QLg, indicating that higher levels of GFF and lower levels of FC are associated with higher levels of QLg, which is consistent with the results of other studies (e.g. Millikan et al., 2002; Jacobson & Rowe, 1999; Harold et al., 1997). We found that genetic factors were responsible for the largest part of the overlap between GFF and QLg and between FC and QLg. High genetic overlap between adolescents' evaluations of family functioning and depressive symptoms have previously been reported by Pike et al. (1996), Neiderhiser et al. (1998) and Jacobson and Rowe (1999), although the latter reported this high genetic overlap only for girls. A possible explanation for the high genetic overlap is that evaluations of family functioning and subjective well-being are distinct components of a more general view towards life which is influenced by genetic factors. This implicates that environmental factors, e.g. true family experiences, have a marginal role in explaining

the overlap between adolescents' evaluations between family functioning and subjective well-being. Rather, the overlap is due to a genetic predisposed way of evaluating one's life in general, which can either be positive or negative. Our results also indicated that the overlap between GFF and QLg and between FC and QLg was larger for girls from families with divorced parents.

The interaction between genetic influences and nonshared, non-measured, environmental influences for GFF, FC, and QLg reflected by the significant negative correlations between intrapair sumscores and difference scores in MZ twins, indicated that genetic influences are relatively more important in those adolescents experiencing high levels of GFF and QLg, and low levels of FC (since they were more similar) as compared to those experiencing lower levels of GFF and QLg, and higher levels of FC. One possible reason for this is that adolescents with a genetic predisposition towards positive evaluations of family functioning and subjective well-being (reflected by the experience of high levels of GFF and QLg and low levels of FC) are more sensitive to positive effects of environmental factors that are not shared by members of the same family. Thus, adolescents with a genetic tendency towards positive evaluations of family functioning and subjective well-being may be disproportionately more likely to evaluate their family functioning and subjective well-being as positive when certain nonshared environmental influences are present. This would imply that certain environmental influences unique to an adolescent play a protective role with regard to evaluations of family functioning and subjective well-being. Although these results suggest that nonshared environmental factors can play a protective role, it remains unclear which particular factors are involved because we did not measure these factors.

Research has shown that the way adolescents evaluate their family functions is positively associated with subjective well-being and psychopathology. The present study contributed to the literature in several ways. We showed that individual differences in adolescents' evaluations of family functioning and subjective well-being can be explained by the combined effect of genetic and environmental effects and that these effects can vary by sex. We found that the genetic architecture of evaluations of family functioning and subjective well-being can be modified by influences from the family environment (e.g. parental divorce as was demonstrated in this study) and the personal unique environment of an adolescent. Moreover, our results indicated that genetic factors are the main contributing factors to the association between evaluations of family functioning and subjective well-being.

Acknowledgements.

Funding was obtained from the following grants: “Spinozapremie” (NWO/SPI 56-464-14192); “Twin-family database for behavior genetics and genomics studies” (NWO 480-04-004); “Genetic and Family influences on Adolescent Psychopathology and Wellness” (NWO 463-06-001); “A twin-sib study of adolescent wellness” (NWO-VENI 451-04-034). M. Bartels is financially supported by a senior fellowship of the EMGO+ institution.

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Section II: Exercise behavior and subjective wellbeing

3

Genetic Influences on Individual Differences in Exercise Behavior during Adolescence

This chapter is published as:

Van der Aa, N., De Geus, E.J.C., Van Beijsterveldt, C.E.M., Boomsma D.I., & Bartels M. (2010). Genetic Influences on Individual Differences in Exercise Behavior during Adolescence. *International Journal of Pediatrics*, Epub, doi:10.1155/2010/138345.

Abstract

The aim of this study was to investigate the degree to which genetic and environmental influences affect variation in adolescent exercise behavior. Data on regular leisure time exercise activities were analyzed in 8,355 adolescent twins, from three age cohorts (13-14, 15-16, and 17-19 years). Exercise behavior was assessed with survey items about type of regular leisure time exercise, frequency, and duration of the activities. Participants were classified as sedentary, regular exercisers, or vigorous exercisers. The prevalence of moderate exercise behavior declined from age 13 to 19 years with a parallel increase in prevalence of sedentary behavior, whereas the prevalence of vigorous exercise behavior remained constant across age cohorts. Variation in exercise behavior was analyzed with genetic structural equation modeling employing a liability threshold model. Variation was largely accounted for by genetic factors (72% to 85% of the variance was explained by genetic factors), whereas shared environmental factors only accounted for a substantial part of the variation in girls aged 13 – 14 years (46%). We hypothesize that genetic effects on exercise ability may explain the high heritability of exercise behavior in this phase of life.

Introduction

Regular exercise has been cited to be a key contributor to health (Berlin & Colditz, 1990), whereas a sedentary lifestyle is proposed to be one of the main causes of the rise in obesity that starts at an increasingly younger age (Martinez-Gonzalez et al., 1999). Despite the well-documented benefits of exercise, many people do not exercise on a regular basis (Crespo et al., 1996). As a consequence, a sedentary lifestyle, and the accompanying risk for obesity, remains a major threat to health in today's society. Studying exercise behavior during adolescence is of particular interest because several studies reported that the prevalence of exercise participation declines with increasing age, and that this decline is most prominent during adolescence (Stubbe et al., 2005; Telema & Yang, 2000; Van Mechelen et al., 2000).

To increase the success of intervention on this important health-related behavior, much research has been devoted to the determinants of exercise behavior. The main focus of these studies has been on social, demographic, and environmental characteristics, such as low socioeconomic status and low social support by family and peers (King et al., 1992; Sallis & Hovell, 1990; Sherwood & Jeffery, 2000). None of these factors, however, have emerged as a strong causal determinant of exercise behavior, with the possible exception of gender, showing that exercise participation is higher in boys than it is in girls. Twin studies offer the possibility to assess the importance of genetic factors as determinants of exercise behavior. With data from twins, individual differences in behavior can be decomposed as due to genetic, shared environmental (environmental influences shared by members of the same family) and non-shared environmental influences (influences unique to an individual). The importance of genetic and environmental factors can be estimated by comparing the resemblance in exercise behavior between monozygotic (MZ) twins and dizygotic (DZ) twins. A greater resemblance of MZ twins, who are genetic identical, compared to DZ twins, who share on average half of their segregating genes, constitutes evidence for genetic influences on exercise behavior. If MZ twins resemble each other more than DZ twins, but not to the extent that would be expected based on their twice larger genetic resemblance, shared familial factors may also be important.

A number of twin studies have shown that genetic factors contribute to individual differences in exercise behavior and measures of exercise frequency, duration, and intensity during adolescence and adulthood (Stubbe et al., 2005; Maia et al., 2002; Beunen & Thomis, 1999; Koopmans et al., 1994; Perusse et al., 1989; Boomsma et al., 1989). The genetic architecture of exercise behavior has been found to differ across the life span with the largest differences seen during adolescence (Stubbe & De Geus, 2009). In a Dutch twin study, Stubbe, Boomsma, and De Geus (2005) found that genetic variation

was of no importance to leisure time exercise in 13- to 16-year-old adolescents. Instead, environmental influences shared by siblings from the same family accounted for the largest part of variation in exercise behavior. From age 17 the role of shared environmental influences rapidly waned and genetic influences started to dominate the individual variation in exercise behavior. A combination of genetic and shared environmental influences on exercise behavior in adolescence has also been reported by other studies (Maia et al., 2002; Beunen & Thomis, 1999). In contrast to Stubbe et al. (2005) who found no difference in the genetic architecture between boys and girls, these studies suggested clear sex differences such that the shared environment lost its importance earlier in boys than in girls. In part, the discrepancies in the sex-specific genetic architecture across these previous studies may reflect insufficient statistical power to reliably detect age by sex effects.

In the present study, we examined the relative influence of genetic and environmental factors on self-reported leisure time exercise behavior in the largest sample of adolescent twins to date. Due to the large sample size this study was able to estimate genetic and environmental influences within three different age groups (13 – 14, 15 – 16, and 17 – 19 years) and to assess quantitative sex differences (e.g. differences in heritability) as well as qualitative sex differences (are the same genes expressed in boys and girls) in the genetic architecture within these age groups.

Methods

Subjects

Participants were registered with the Netherlands Twin Registry (NTR), established by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007; Boomsma et al., 2006). The large majority of twins had been registered with the NTR as newborns. Parents of adolescent twins were asked for consent to send their children a survey. If their parents consented, twins and their non-twin siblings received an online or a paper & pencil self-report survey when they were 14, 16, and 18 years. The survey contained items about behavior, sport, lifestyle, and well-being. When twins and siblings did not return the survey on time they were contacted by mail for a first reminder and next they were contacted by phone for a second reminder. A total of 3,645 families with twins born between 1986 and 1994 participated in this ongoing study at least once so far. The overall family response rate is 56%.

Triples and non-twin siblings were not included in the present paper. Furthermore, twins with an illness or handicap interfering with their daily lives were also not included. This resulted in a total sample of 8,355 twins (42% male) from complete and incomplete pairs, coming from 3,405 families. For 1,160 twins, data were available at two time points. Participants were primarily Caucasian and they came from all regions of The Netherlands (rural and urban areas). Data were available for 754 (17%) incomplete and 3,614 (83%) complete twin pairs. In Table 3.1, zygosity of the participating twin pairs is presented. For 1,089 (36.1%) of the same-sex twin pairs zygosity was determined based on blood group or DNA typing. Zygosity for the remaining same-sex twin pairs was determined by questionnaire items about physical similarities and confusion by family members and strangers. These items allow accurate determination of zygosity in 93% of same-sex twin pairs (Rietveld et al., 2000).

Table 3.1

Zygosity of participating twin pairs for the total sample and the different age groups (complete twin pairs added in parentheses)

	Total sample	13 – 14 yr	15 – 16 yr	17 – 19 yr
MZM	662 (585)	211 (197)	282 (249)	169 (139)
DZM	567 (465)	201 (170)	210 (184)	156 (111)
MZF	1042 (918)	343 (317)	380 (333)	319 (268)
DZF	738 (621)	231 (207)	265 (225)	242 (189)
DOS	1359 (1025)	516 (428)	494 (372)	349 (225)

Note. MZM = monozygotic male twin pair; DZM = dizygotic male twin pair; MZF = monozygotic female twin pair; DZF = dizygotic female twin pair; DOS = dizygotic opposite-sex twin pair

Participants were divided into the age groups 13-14 years (33%), 15-16 years (38%), and 17-19 years (29%). Mean age in the three age groups was 14.51 years (SD = 0.31), 16.23 (SD = 0.61), and 18.06 (SD = 0.70) respectively. The age groups were not completely independent, because for a small subset of participants data were available at two time points (e.g. twins returned a survey at age 14 and 16). Furthermore, since a small subset of participants participated in a pilot and short there after in the regular survey collection data from 2 surveys were present within one age group. For this subset of participants, data from the pilot version were excluded for the analyses. As can be seen in Table 3.1, each age group had adequate numbers of monozygotic (MZ) and dizygotic (DZ) twin pairs.

Measures

Participants were asked to indicate what type(s) of regular leisure exercise they were involved in at the time of assessment. A list of 21 common individual (includes fitness centre, jogging, tennis, etc) and team-based exercise activities (soccer, field hockey) was provided plus 5 open entries for less common activities. For each exercise activity endorsed, the participants further reported how many months per year, weekly frequency and the average duration of the activity. Ainsworth's Compendium of physical activity (Ainsworth et al., 2000) was used to assign a MET score (Metabolic equivalent) to each exercise activity, reflecting its energy expenditure as a multiple of the basal energy expenditure (approximately 1 kcal/kg/hour) in an average subject engaged in that activity. When in high-school, Dutch adolescents have to attain physical education (PE) classes for 1-3 hours per week. The exact amount of MET hours weekly in these PE classes was assessed as a separate variable.

For each participant a total weekly MET score was computed across all exercise activities by summing the products of the number of hours spent weekly on each exercise activity and its MET score. Activities were only scored if participants had engaged in them for at least three months during the past year. Exercise during physical education classes at school was not included in the weekly MET score. Thus the dependent variable reflects leisure time exercise behavior only.

Participants were classified into three groups based on their total MET scores. The first category consisted of sedentary participants whose total weekly MET score was lower than 5.0. The second category of moderate exercisers consisted of participants whose total weekly MET score ranged between 5.0 and 30.0. The third category consisted of vigorous exercisers whose total weekly MET score was 30.0 or higher.

Statistical analyses

Because the data of exercise behavior were positively skewed, a liability threshold model was used to analyze individual differences in exercise behavior within each age group. The basic assumption underlying the liability threshold model, which was originally proposed by Falconer and Mackay (1996), is that a latent continuous liability underlies the skewed distribution of the observed variable (i.e., exercise behavior). The liability is assumed to be standard normal distributed (i.e., mean = 0, SD = 1). Structural equation modeling was used to estimate the relative contributions of genetic and environmental influences to individual differences in liability to exercise behavior.

Use of the liability threshold model required discretization of the observed scores on exercise behavior. Therefore, participants were classified into three groups (i.e., sedentary, moderate exercise, vigorous exercise) as described above. In this way, ordinal scores on exercise behavior were obtained that were coded 0, 1, and 2. To model the three categories of exercise behavior two thresholds were required. The thresholds, expressed in z-values, are defined by the prevalence of the three categories of exercise behavior in the sample and represent the value in the latent liability distribution above which an individual will endorse the next category.

Resemblance in the liability to exercise behavior between twins is expressed in polychoric twin correlations. Comparing MZ twin correlations with DZ twin correlations provides a first step in evaluating the relative influence of genetic and environmental factors on individual differences in liability to exercise behavior. When the MZ correlation is higher than the DZ correlation, it is inferred that genetic variation influences individual differences in liability to exercise behavior. A DZ correlation higher than half the MZ correlation implies shared environmental effects, referring to environmental factors shared by all members of the same family, on liability to exercise behavior. Variation that is not due to genetic and shared environmental effects is attributed to environmental effects which are not shared by family members. The nonshared environmental variance component also includes measurement error variance. Specific information regarding qualitative sex differences can be derived from the DZ opposite-sex (DOS) correlation. When the twin correlation in DOS twin pairs is lower than in DZ twin pairs this might be due to genetic or shared environmental effects that influence one sex but not the other (Falconer & Mackay, 1996).

As a first step, a saturated model was fitted for each age group separately, in which the thresholds and polychoric twin correlations were estimated using the software package Mx (Neale et al., 2006). Thresholds and twin correlations were estimated separately in boys and girls to take into account sex differences in the prevalence of exercise behavior and to explore sex differences in the genetic architecture of liability to exercise behavior. The saturated model simply specifies for each zygosity by sex group (i.e. MZM, DZM, MZF, DZF, and DOS) that the data from the first- and second born twin are correlated without attempting to model these correlations as a function of genes or shared environment. Within a series of nested models we tested whether constraining the thresholds to be equal between boys and girls led to a significant deterioration of model fit. In addition, we tested whether twin correlations were different for MZ and DZ twins.

Next, genetic models were fitted to the data in which the genetic architecture of liability to exercise behavior was specified for each age group. A graphical representation of the genetic model is given in Figure 3.1. The amount of variance in the underlying

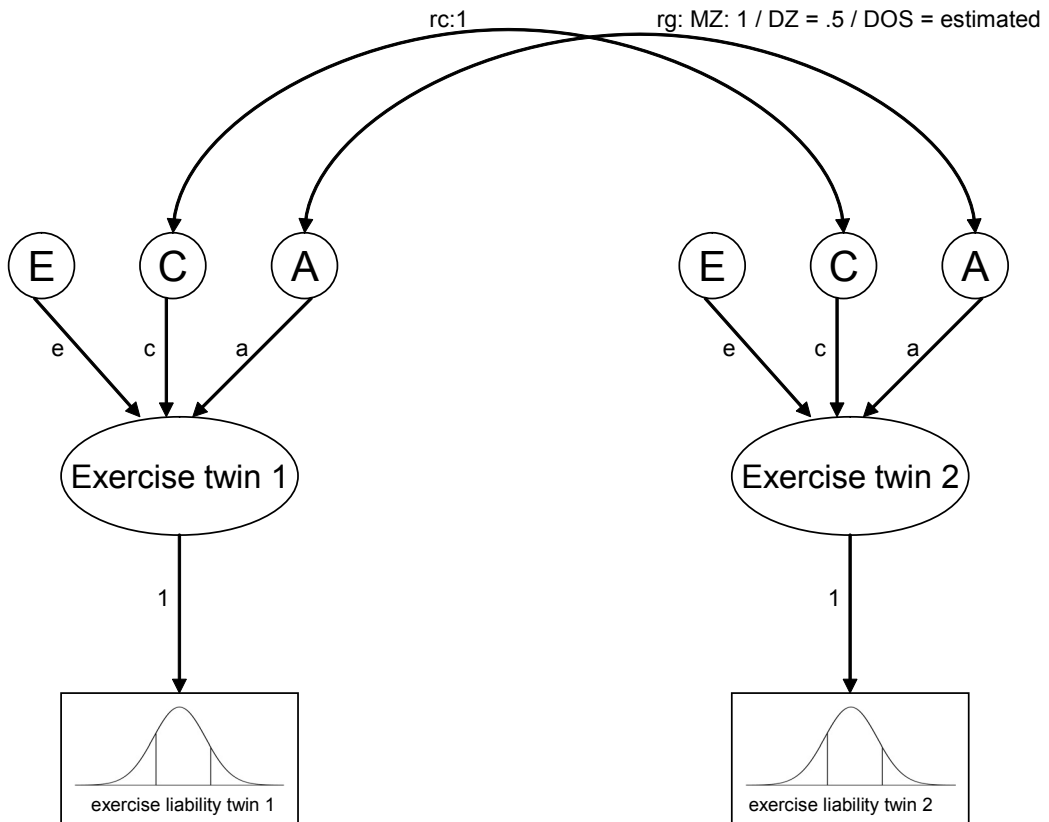
liability due to additive genetic (A), shared environmental (C), and nonshared environmental effects (E) can be estimated by considering the different level of genetic relatedness between MZ and DZ twin pairs. MZ twin pairs are genetically identical, whereas DZ twin pairs share on average 50% of their segregating genes. In the genetic models, the genetic correlation (r_g) for MZ and DZ twin pairs is therefore fixed at 1.0 and 0.5 respectively. Shared environmental effects refer to environmental factors that are shared by all siblings in the family and therefore the shared environmental correlation (r_c) is fixed at 1.0. In Figure 3.1, r_g and r_c are represented by the double headed arrows connecting the latent genetic (A) and shared environmental factors (C) of both members of a twin pair. Nonshared environmental refer to environmental factors that are unique to individuals in the family and therefore it is uncorrelated between siblings. The influence of A, C, and E is represented by path coefficients a , c and e (see Figure 3.1). To assess possible sex differences in the genetic architecture of liability to exercise behavior, parameter estimates of a , c and e were allowed to differ for boys and girls.

We fitted various models that were nested in the sense that one model could be derived from the other by the imposition of one or more constraints on the parameters. To assess the possibility that different genetic factors influence exercise behavior in boys and girls the genetic correlation (r_g) between DOS twins was estimated and we tested whether fixing r_g to 0.5 resulted in a significant deterioration of model fit.

Sex differences in the relative influence of the variance components A,C, and E, i.e. quantitative sex differences, were assessed by testing whether constraining the genetic (a), shared environmental (c), and nonshared environmental (e) parameter estimates of boys and girls to be equal resulted in a significant deterioration of model fit. Subsequently, the statistical significance of the variance components A and C was assessed by testing whether fixing the corresponding parameter estimate (i.e., a and c) to zero resulted in a significant deterioration of model fit.

The fit of the different models was compared by means of the log-likelihood ratio test (LRT). The difference in minus two times the log-likelihood ($-2LL$) between two nested models has a χ^2 distribution with the degrees of freedom (df) equaling the difference in df between the two models. If a p -value higher than 0.05 was obtained from the χ^2 -test the fit of the constrained model was not significantly worse than the fit of the more complex model. In this case, the constrained model was kept as the most parsimonious and best fitting model. The fit of the genetic models was also compared to the full ACE model by means of Akaike's Information Criterion, keeping the model with the lowest AIC as the best fitting model (Neale et al., 2006).

Figure 3.1. Univariate liability threshold model for twin data. Exercise behavior was measured with 3 categories (hence 2 thresholds are estimated). The total variance in liability is one and is modeled as caused by latent factors A (additive genetic influences), C (common or shared environment) and E (unique environment). The square of path coefficients a, c and e gives the variance due to A, C and E.



Results

Table 3.2 presents the prevalence of exercise behavior for the three age groups. The table shows that irrespective of age, sedentariness and moderate exercise behavior are more prevalent in girls, whereas vigorous exercise behavior is more prevalent in boys. Formal tests on the thresholds showed these differences to be significant. The thresholds were different between boys and girls in the 13 – 14 years ($\chi^2 (2) = 87.44, p < .01$), 15 – 16 years ($\chi^2 (2) = 84.33, p < .01$), and the 17 – 19 years ($\chi^2 (2) = 64.03, p < .01$) age groups. In boys and girls, there is an increase in the prevalence of sedentariness (i.e. decrease in

exercise behavior) in the 17 – 19 olds compared to the other age groups whereas there is a parallel decrease in the prevalence of moderate exercise behavior. The prevalence of vigorous exercise behavior remains constant throughout adolescence.

Table 3.2

Prevalence (95% confidence intervals between parentheses) of exercise participation in the different age groups as a function of sex

	13 – 14 yr		15 – 16 yr		17 – 19 yr	
	boys	girls	boys	girls	boys	girls
sedentariness	20%	31%	24%	31%	27%	38%
	(17% - 22%)	(28% - 33%)	(21% - 26%)	(29% - 34%)	(24% - 31%)	(35% - 40%)
moderate exercise	40%	45%	35%	45%	31%	38%
	(39% - 41%)	(45% - 46%)	(35% - 36%)	(44% - 45%)	(30% - 31%)	(38% - 39%)
vigorous exercise	40%	24%	41%	24%	41%	24%
	(37% - 44%)	(21% - 26%)	(38% - 44%)	(21% - 27%)	(38% - 46%)	(21% - 27%)

Table 3.3

Twin correlations for exercise participation in each age group (95% confidence intervals added in parentheses)

	13 – 14 yr	15 – 16 yr	17 – 19 yr
MZM	.85 (.79 - .90)	.76 (.67 - .82)	.73 (.60 - .82)
DZM	.23 (.01 - .42)	.48 (.32 - .62)	.48 (.27 - .65)
MZF	.83 (.78 - .88)	.83 (.77 - .87)	.71 (.63 - .78)
DZF	.67 (.56 - .75)	.52 (.39 - .63)	.34 (.15 - .50)
DOS	.32 (.21 - .42)	.36 (.25 - .47)	.29 (.12 - .44)

Twin correlations in the different age groups are presented in Table 3.3. For boys and girls, MZ twin correlations were significantly higher than DZ twin correlations in the 13 – 14 years ($\chi^2(2) = 63.20, p < .01$), 15 – 16 years ($\chi^2(2) = 43.09, p < .01$), and 17 – 19 years ($\chi^2(2) = 23.94, p < .01$) age groups, suggesting that individual differences in liability to exercise behavior are influenced by genetic factors. For girls in the youngest age group, resemblance in exercise behavior between MZ twins was similar to DZ twins, suggesting that shared environmental factors play an important role in explaining individual differences in exercise behavior. For girls in the two oldest age groups and for

boys in all age groups DZ twin correlations were about half the MZ twin correlation, suggesting that genetic factors explain the bulk of variation in exercise behavior for these age groups.

Table 3.4

Univariate model fitting results for exercise behavior in the three age groups

Model	vs	-2LL	df	χ^2	Δdf	p	AIC
13 – 14 yr							
1. ACE: sex differences (rg estimated)	--	5482.577	2812	--	--	--	--
2. ACE: sex differences (rg fixed at 0.5)	1	5482.704	2813	0.127	1	.72	-1.87
3. ACE: no sex differences (rg fixed at 0.5)	2	5502.510	2815	19.81	2	< .01	13.93
4a. CE: boys, ACE: girls (rg fixed at 0.5)	2	5536.641	2814	53.94	1	< .01	50.06
4b. ACE: boys, CE: girls (rg fixed at 0.5)	2	5497.697	2814	14.99	1	< .01	11.12
5a AE: boys, ACE: girls (rg fixed at 0.5)	2	5483.223	2814	0.52	1	.47	-3.35
5b. ACE: boys, AE: girls (rg fixed at 0.5)	2	5502.504	2814	19.80	1	< .01	15.93
15 – 16 yr							
1. ACE: sex differences (rg estimated)	--	5943.005	2986	--	--	--	--
2. ACE: sex differences (rg fixed at 0.5)	1	5944.573	2987	1.57	1	.21	-.43
3. ACE: no sex differences (rg fixed at 0.5)	2	5949.728	2989	5.16	2	.08	.72
4. CE: no sex differences (rg fixed at 0.5)	3	6024.535	2990	74.81	1	< .01	73.53
5. AE: no sex differences (rg fixed at 0.5)	3	5950.674	2990	.95	1	.33	-2.33
17 – 19 yr							
1. ACE: sex differences (rg estimated)	--	4455.979	2158	--	--	--	--
2. ACE: sex differences (rg fixed at 0.5)	1	4455.979	2159	.00	1	> .99	-2.00
3. ACE: no sex differences (rg fixed at 0.5)	2	4458.120	2161	2.14	2	.34	-3.86
4. CE: no sex differences (rg fixed at 0.5)	3	4495.737	2162	37.62	1	< .01	21.76
5. AE: no sex differences (rg fixed at 0.5)	3	4458.120	2162	.00	1	> .99	-5.86

Note. vs = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; Δdf = degrees of freedom of χ^2 test; p = p-value; AIC = Akaike's Information Criterion; rg = genetic correlation between DOS twins. Most parsimonious models are printed in boldface type.

Genetic model fitting results for all age groups are presented in Table 3.4. In model 2, rg was constrained at 0.5 which did not result in a significant deterioration of model fit in any of the three age groups, indicating that the same genetic factors act in boys and girls with regard to exercise behavior.

Model 3 tested whether constraining the parameter estimates of the full univariate ACE model to be equal for boys and girls led to a significant deterioration of model fit. In the youngest age group there appeared to be significant differences in the magnitude of the variance components explaining individual differences in liability to exercise behavior. Therefore, parameter estimates were allowed to differ between boys and girls for this age group. In the two oldest age groups constraining the parameter estimates to be equal between boys and girls did not lead to a significant deterioration of model fit.

Models 4 and 5, tested whether constraining the genetic or shared environmental parameter estimate to zero would lead to a significant deterioration of model fit. Additive genetic effects on individual differences in liability to exercise behavior were statistically significant in all age groups. Shared environmental effects were statistically significant for girls in the youngest age group. In all age groups the LRT tests and the AIC pointed to the AE model as the most parsimonious model, except for girls in the youngest age group in which the ACE model was most parsimonious.

Table 3.5

Proportions of variance explained by Additive genetic, Common environmental and unique Environmental factors from the best-fitting models for exercise participation in three age groups for boys and girls (95% confidence intervals added in parentheses)

		A	C	E
13 – 14 yr	Boys	.85 (.78 - .90)	--	.15 (.10 - .22)
	Girls	.38 (.22 - .57)	.46 (.27 - .61)	.16 (.12 - .21)
15- 16 yr	Boys	.80 (.76 - .84)	--	.20 (.16 - .24)
	Girls	.80 (.76 - .84)	--	.20 (.16 - .24)
17 – 19 yr	Boys	.72 (.65 - .77)	--	.28 (.23 - .35)
	Girls	.72 (.65 - .77)	--	.28 (.23 - .35)

The proportions of variance explained by A, C, and E in liability to exercise behavior of the three age groups are summarized in Table 3.5. For boys in all age groups, the proportion of variation in liability to exercise behavior explained by genetic factors ranged between .72 and .85. The remaining variation was accounted for by non-shared environmental factors. For girls in the youngest age group, genetic and shared environmental factors accounted for individual differences in exercise behavior, .38 and

.46 respectively. For girls in the two oldest age groups, shared environmental factors did not account for variation in liability to exercise behavior, whereas the proportions of liability explained by genetic factors were .80 and .72.

Discussion

In a large sample of Dutch adolescent twins, we found that the prevalence of sedentariness increased during late adolescence compared to early adolescence. At all ages, girls were more often sedentary than boys. When regularly engaged in exercise, girls more often exercised at a moderate rather than a vigorous level. During early adolescence, individual differences in liability to exercise behavior could be accounted for by genetic and non-shared environmental factors for boys, whereas for girls shared environmental factors accounted for a substantial part of the individual differences as well. During middle and late adolescence, genetic influences accounted for the largest part of the variation in liability to exercise behavior for boys as well as girls. No evidence was found for qualitative sex differences in the genetic factors, indicating that the same genetic variants appear to influence exercise behavior in boys and girls.

Our finding that the prevalence of moderate exercise behavior decreased during late adolescence in boys and girls in favor of the prevalence of sedentariness corresponds with the results of other studies (Stubbe et al., 2005; Telema & Yang, 2000; Van Mechelen et al., 2000). The prevalence of vigorous exercise, however, did not change across the three age groups. This finding is consistent with Van Mechelen et al. (2000) who observed a graduate decline in the prevalence of physical activities of mild intensity and non-organized sports activities, but not in the prevalence of organized sports activities. An explanation for this is that vigorous exercisers have strong intrinsic motivations to exercise leading to continuation of their exercise behavior, whereas moderate exercisers are less intrinsically motivated to exercise making them more likely to become sedentary.

The main aim of the present study was to assess to what extent genetic and environmental factors affect exercise behavior from early to late adolescence. For boys, genetic factors accounted for the major part of individual differences in exercise behavior from early to late adolescence. It has been suggested that genetic influences on exercise ability may explain part of the heritability of exercise behavior (Stubbe et al, 2006; De Geus & De Moor, 2008). The basic idea is that people will seek out the activities that they are good in. This is particularly true in male adolescents, because being good in sports is an important source of self-esteem for these adolescents and the athletic role model is

continuously reinforced by the media (Field et al., 1999; Pope et al., 2001). Therefore, genes coding for exercise ability (endurance, strength, flexibility, motor coordination) may well become genes for adolescent exercise behavior.

In contrast to boys of the same age, shared environmental factors accounted for a major part of individual differences in exercise behavior for the youngest girls, whereas from 15 years onwards the influence of these shared environmental factors had completely disappeared in favor of genetic factors. Shared environmental influences may include parents, siblings and peers who make sure the young adolescent girls regularly get to the playing field, and to provide positive feedback on their performance. The extent of positive feedback from parents, siblings and especially from peers may increasingly depend on their genotypes for exercise ability. In short, the shared environment determines exposure and encouragement in early adolescence, but, as for the boys, actual exercise ability will determine whether girls like exercising enough (by excelling in it) to maintain the behavior when the perception of peers increases in relative importance to that of parents during mid and late adolescence. The idea that a single factor like exercise ability is crucial to both boys and girls is reinforced by the fact that the same qualitative genetic variation was seen to underlie the heritability of exercise behavior in boys and girls.

The genetic architecture of exercise behavior during adolescence has been addressed in previous studies (Stubbe et al., 2005; Maia et al., 2002; Beunen & Thomis, 1999). In a sample of the Netherlands Twin Registry from an earlier birth cohort, Stubbe, Boomsma, and De Geus (2005) also found a shift from shared environmental to genetic influences during adolescence. However, they reported the shift to occur during late adolescence (i.e. around 16 years) and shared environmental effects on exercise behavior were found not only for young adolescent girls but also for the boys. The sample had very similar age groups as in the present study but the data were collected 10 to 15 years earlier, that is in a birth cohort born 10 to 15 years earlier than the current cohort. The much larger sample size of the present study and its more extensive assessment of leisure time exercise behavior may have led to increased precision of the estimated parameters.

Additional support for the pattern of sex differences in the genetic architecture of exercise behavior in adolescents found in the present study comes from other studies in different countries. In a small Flemish sample of 15 year-old twins Beunen and Thomis (1999) found that 83% and 44% of variability in exercise behavior is accounted for by genetic factors for boys and girls respectively, and 54% is accounted for by shared environmental factors only in girls. In a study based on 411 Portuguese twins aged 12 – 25 years, Maia et al. (2002) found larger heritability estimates for boys (68%) compared

to girls (40%). Unfortunately, both studies were too small to divide their samples into different age cohorts and it could not be established whether the sex differences were specific to certain age groups.

A limitation of the present study was the use of a cross-sectional twin design to examine the relative influence of genetic and environmental influences on individual differences in exercise behavior. The genetic architecture of exercise behavior during adolescence is most properly addressed in a longitudinal design. So far data at two time points are only available for a small subsample, and data throughout adolescence (13-18) are absent. Since our data collection is a continuous process at the NTR we anticipate large enough longitudinal sample size within the next 5 years. Large shifts in the genetic architecture are expected when subjects move from adolescence to adulthood. In adulthood, non-shared environmental factors become more important and heritability decrease to about 50% (Stubbe et al., 2006). Furthermore significant qualitative sex differences are found in adulthood with different genetic factors influencing male and female exercise behavior (Stubbe et al., 2006; De Moor et al., 2007).

Conclusions

The prevalence of moderate exercise behavior declined from age 13 to 19, whereas the prevalence of vigorous exercise behavior remained constant across age groups. Variation in exercise behavior could be largely accounted for by genetic factors, whereas shared environmental factors only accounted for a substantial part of the variation in girls aged 13 – 14 years. Future studies should focus on the role of exercise ability as a potential determinant of exercise behavior. If the high heritability of exercise behavior in this phase of life is indeed explained by genetic effects on exercise ability – a testable hypothesis – then the relatively high levels of sedentary adolescents may reflect an undesirable emphasis on performance rather than pleasure in current day adolescent sports culture.

Acknowledgements.

Funding was obtained from the following grants: “Spinozapremie” (NWO/SPI 56-464-14192); “Twin-family database for behavior genetics and genomics studies” (NWO 480-04-004); “Genetic and Family influences on Adolescent Psychopathology and Wellness” (NWO 463-06-001); “A twin-sib study of adolescent wellness” (NWO-VENI 451-04-034). M. Bartels is financially supported by a senior fellowship of the EMGO+ institution.

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4

Regular Exercise and Psychological Wellbeing in Adolescence: Causality or Genetic pleiotropy?

This chapter is submitted as:

Van der Aa, N., De Geus, E.J.C., De Moor, M.H.M., Boomsma, D.I., & Bartels, M.
Regular Exercise and Psychological Wellbeing in Adolescence: Causality or Genetic pleiotropy?

Abstract

Higher levels of psychological wellbeing found in exercisers are assumed to be the reflection of causal effects of exercise. However, an alternative explanation is that both exercise and wellbeing are influenced by other factors, such as genetic factors. The aim of this study was to test in a genetically informative design whether exercise behavior causally influences psychological wellbeing. Data on these traits were available in a sample of 6,317 adolescent twins and 1,180 non-twin siblings. The majority of the sample had longitudinal data with 2-year follow-up. Psychological wellbeing was defined by the absence of internalizing problems as well as by the presence of positive subjective wellbeing (SWB). Exercise behavior, internalizing problems and SWB were found to be substantially heritable. Exercise behavior was cross-sectionally and longitudinally associated with fewer internalizing problems and increased SWB. Causal effects of exercise on internalizing and SWB were tested by bivariate genetic models and by correlation of monozygotic intrapair differences in exercise behavior and wellbeing measures (cross-sectional and longitudinal). Cross-sectional and longitudinal associations were mainly accounted for by genetic factors, whereas the contribution of environmental factors was negligible. In genetically identical twin pairs, the twin who exercised more did not show higher levels of wellbeing than the co-twin who exercised less. This was found cross-sectionally and longitudinally. In conclusion, exercise behavior is associated with higher levels of wellbeing, but this cannot be explained by causal effects of exercise, but largely reflects the effects of common genetic factors on these traits.

Introduction

In the adult population regular exercise has consistently been associated with higher levels of psychological wellbeing such as fewer depressive and anxious symptoms and higher levels of subjective wellbeing (SWB; Stubbe et al., 2007; De Moor et al., 2006; Norris, Carrol, & Cochrane, 1992). Longitudinal studies showed that exercise participation at baseline predicted fewer internalizing problems at follow-up (De Moor et al., 2008; Wise et al., 2006; Camacho et al., 1991). Furthermore, there have been numerous experimental studies investigating the effect of exercise training on individuals with clinical levels of depression and anxiety (see for reviews Lawlor & Hopker, 2001; Salmon, 2000; Craft & Landers, 1998). In general, the conclusion that has been drawn from these studies is that increased levels of psychological wellbeing found in exercisers are the reflection of causal effects of exercise.

However, results on the direction of causation from experimental studies may not generalize to the population at large. It is likely that only selected groups of participants attracted to exercise may enroll and persist in these studies. Furthermore, treatment effects of exercise on clinical levels of depression and anxiety in patients may not generalize to milder forms of internalizing problems in the general population. An alternative hypothesis explaining the prospective associations between exercise behavior and psychological wellbeing found in population-based epidemiological studies may be that underlying factors influencing exercise behavior at baseline also influence psychological wellbeing at follow up. Genetic variation among individuals may represent such an underlying factor. Under this hypothesis, prospective associations between exercise behavior and psychological wellbeing reflect the effects of common genetic factors influencing a disposition for exercise participation as well as a disposition for psychological wellbeing, a process referred to as genetic pleiotropy (De Geus & De Moor, 2008). When genetic effects on exercise behavior precede effects of the same genetic factors on psychological wellbeing at a later time point, these effects can mimic the causal effect of exercise and, consequently, overestimate the beneficial effects of exercise.

Evidence for the hypothesis of genetic pleiotropy in exercise behavior and psychological wellbeing was supported in population-based adult twin studies (De Moor et al., 2008, 2007; Stubbe et al., 2007). De Moor et al. (2008) demonstrated cross-sectionally and longitudinally that genetic factors influencing a person's tendency to engage in exercise also influence lower risk for depressive and anxious symptoms, whereas environmental factors affecting exercise behavior do not affect depressive and anxious symptoms. Strong support for a non-causal explanation came from the finding that in genetically identical twin pairs, the twin who exercised more did not have fewer

depressive and anxious symptoms than his or her co-twin who exercised less. Moreover, individuals who increased their exercise participation over time did not show a parallel decrease in depressive and anxious symptoms.

Many studies, like the above, have defined wellbeing as the absence of depressive and anxious symptoms. Stubbe et al. (2007) added to this by using happiness and satisfaction with life as an indicator of positive wellbeing. Using both internalizing and subjective wellbeing (SWB) as outcome measures for adolescent psychological wellbeing has the important advantage that it is sensitive to the entire spectrum of psychological wellbeing (Proctor et al., 2009). In a co-twin control study, Stubbe et al. (2006) reported that although exercisers were more satisfied with their lives and happier than non-exercisers, the odds ratios decreased and were no longer significant in MZ and DZ twin pairs discordant for exercise participation. Taken together these results indicate that the association between exercise and SWB was most likely explained by genetic factors influencing both traits.

Most research up to date has focused on adults. It is important to further study the association between exercise behavior and psychological wellbeing during adolescence because several studies reported that the prevalence of exercise participation declines during adolescence (Van der Aa et al., 2010a; Stubbe, Boomsma, & De Geus, 2005; Van Mechelen et al., 2000) while the prevalence of internalizing problems shows its most prominent increase (Costello, Egger, & Angold, 2005). Furthermore, the genetic architecture of exercise behavior was found to be vastly different across the life span with a peak in heritability during adolescence (Van der Aa et al., 2010a; Stubbe, Boomsma, & De Geus, 2005; Maia, Thomis, & Beunen, 2002; Beunen & Thomis, 1999). Van der Aa et al. (2010a), for instance, showed that variation in exercise behavior during adolescence was largely accounted for by genetic factors (70% to 85%), whereas shared environmental factors only accounted for part of the variation in young adolescent girls (46%). Among adolescents, individual differences in internalizing (Lamb et al., 2010; Rice et al., 2002; Bergen et al., 2007) and SWB (Van der Aa et al., 2010b; Bartels & Boomsma, 2009; Røysamb et al., 2002) have also been found to be substantially influenced by genetic factors, whereas shared environmental factors accounted for variation in internalizing only during young adolescence (Lamb et al., 2010; Rice et al., 2002) and were absent with regard to SWB (Van der Aa et al., 2010b; Bartels & Boomsma, 2009; Røysamb et al., 2002).

In the present study we tested whether exercise behavior causally influences internalizing problems and SWB in a large population-based sample of adolescent twins and their non-twin siblings. Cross-sectional and longitudinal data were used, providing a strong non-experimental design to test the causal hypothesis. Extending the procedure as

described by De Moor et al. (2008), four predictions generated by the causal hypothesis were tested. A graphical representation of the 4 models used to test causal effects of exercise behavior on internalizing problems and SWB is presented in Figure 4.1.

A first prediction is that, if exercise causally influences internalizing problems and SWB, all genetic and environmental factors influencing individual differences in exercise behavior will also, through the causal chain, influence individual differences in internalizing problems and SWB. This can be tested in a bivariate genetic model by computing genetic and environmental correlations between 2 traits (Neale & Cardon, 1992). If genetic and environmental correlations are both significant this would be in line with the causal hypothesis (see Figure 4.1A). If only the genetic correlation is significant this is compatible with the hypothesis of common genetic factors underlying exercise behavior, internalizing problems, and SWB.

A second prediction is that under the causal hypothesis, monozygotic within-twin pair differences in exercise behavior should be associated with within-twin pair differences in internalizing problems and SWB. The twin who exercises more should display fewer internalizing problems and higher levels of SWB than the co-twin who exercises less (see Figure 4.1B). This would be in line with the causal hypothesis because it excludes confounding by genetic factors (the twins are genetically identical), whereas if the association is not significant this would falsify the causal effect of exercise.

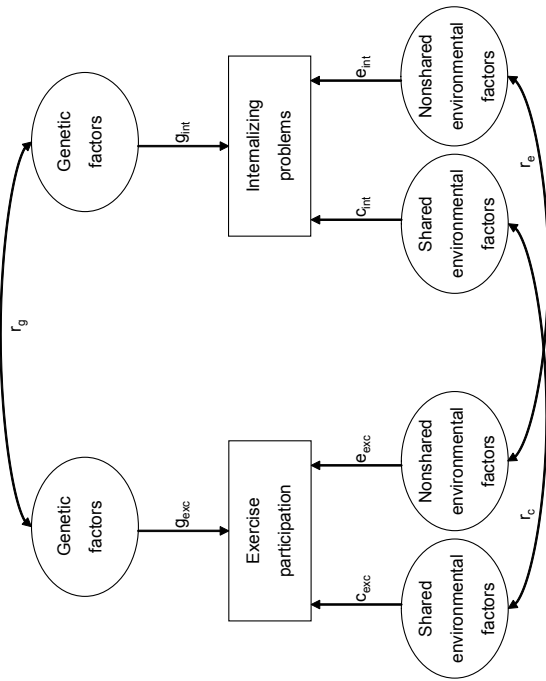
A third prediction made by the causal hypothesis is that, if exercise causes fewer internalizing problems and higher levels of SWB, there should be a significant longitudinal association between exercise at baseline and internalizing problems and SWB at follow-up. This should be independent of common genetic factors influencing exercise behavior, internalizing problems, and SWB. This can be tested in a bivariate genetic model by computing genetic and environmental correlations between exercise behavior at baseline and internalizing problems and SWB at a successive time point. If genetic and environmental correlations between exercise behavior at baseline and internalizing problems or SWB at follow up are both significant this would be in line with the causal hypothesis (see Figure 4.1C). If only the genetic correlation is significant this is compatible with the hypothesis of common genetic factors underlying exercise behavior, internalizing problems, and SWB.

A fourth prediction is that, under the causal hypothesis, monozygotic intrapair differences in within-individual changes in exercise behavior over time should be associated with parallel intrapair differences in within individual changes in internalizing problems and SWB over time.

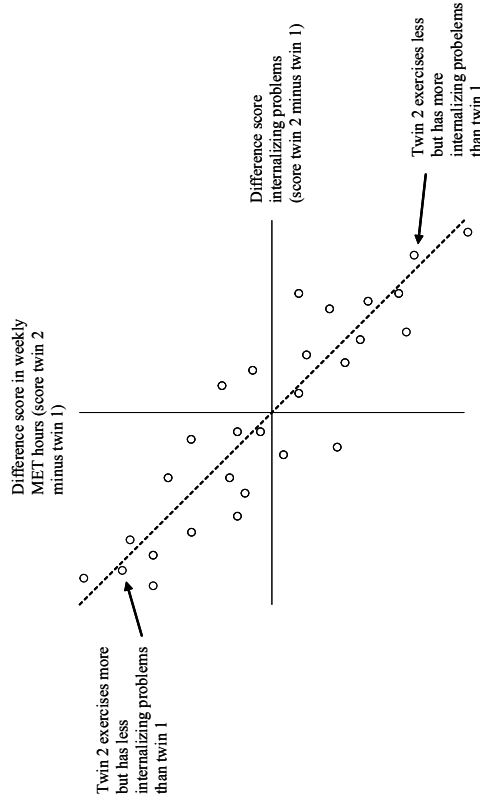
Figure 4.1. Graphic representation of 4 models used to test the hypothesis that exercise causes lower levels of internalizing problems.

A: a causal effect of exercise predicts a correlation between genetic, shared environmental, and nonshared environmental factors influencing exercise behavior and internalizing problems. Cross-sectional correlation between exercise and internalizing problems = $(g_{exc} * r_g * g_{int}) + (c_{exc} * r_c * c_{int}) + (e_{exc} * r_e * e_{int})$. B: A causal effect of exercise predicts that in genetically identical twins, the twin who exercises more has lower levels of internalizing problems. C: a causal effect of exercise predicts a correlation between genetic, shared environmental, and nonshared environmental factors influencing exercise behavior at time 1 and internalizing problems at time 2 = $(g_{exc} * r_{g,1,2} * g_{int}) + (c_{exc} * r_{c,1,2} * c_{int}) + (e_{exc} * r_{e,1,2} * e_{int})$. D: A causal effect of exercise predicts that in genetically identical twins, the twin who increases exercise intensity over time also shows decreased levels of internalizing problems over time.

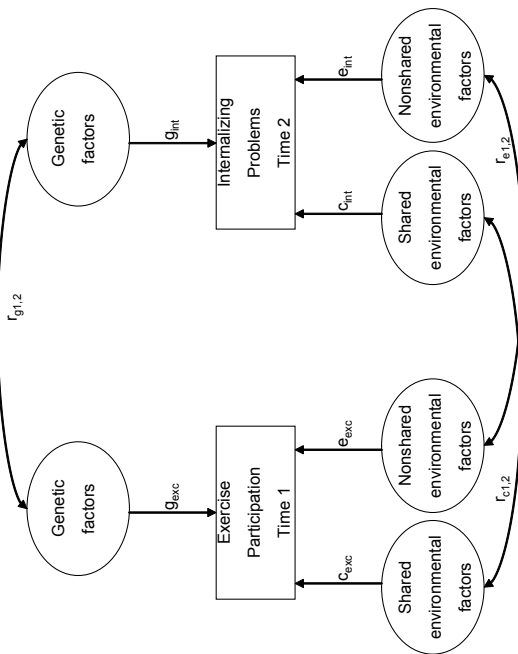
A. Cross-sectional bivariate genetic model



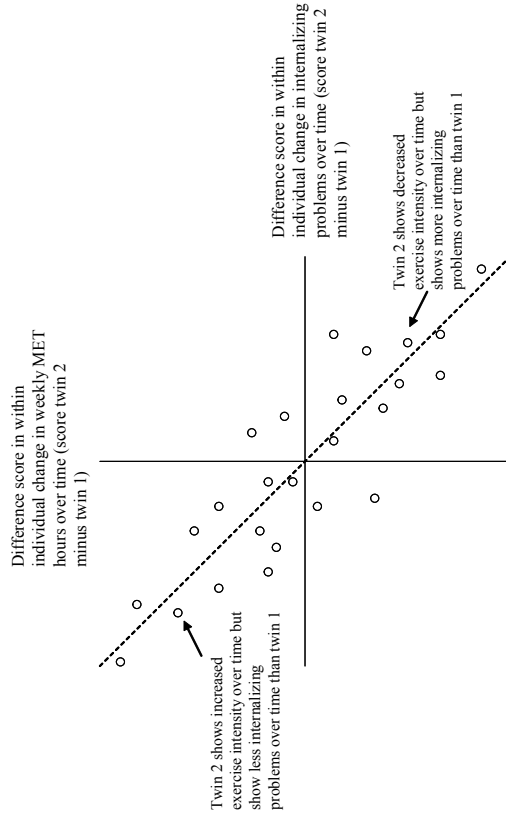
B. Cross-sectional MZ twin intrapair differences model



C. Longitudinal bivariate genetic model



D. Longitudinal MZ twin intrapair differences model



In other words, the twin showing the largest increase in frequency and intensity of exercise behavior over time should display larger decreases in internalizing problems and increases in SWB over time than the co-twin showing a lower increase or even a decrease in frequency and intensity of exercise behavior over time (see Figure 4.1D). This would be compatible with the causal hypothesis because it excludes confounding by genetic factors that would independently cause increases in exercise behavior as well as wellbeing over time.

Methods

Subjects

Participants were registered with the Netherlands Twin Registry (NTR), established by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007, Boomsma et al., 2006). The large majority of twins had been registered with the NTR as newborns and they were followed throughout childhood. At the time twins were 13 years old, their parents were asked for consent to send their children a survey. If their parents consented, twins and their non-twin siblings received an online or a paper & pencil self-report survey when they were 14, 16, and 18 years. The survey contained items about behavior, exercise, lifestyle, and wellbeing. In this way, longitudinal data on exercise behavior, internalizing problems, and SWB are collected on 3 different occasions during adolescence. Data collection started in 2005 and currently data have been collected on 2 occasions (14 and 16 or 16 and 18). Since our data collection is an ongoing process, data on 3 different occasions during adolescence will become available in the coming years. When twins and siblings did not return the survey on time they were contacted by mail for a first reminder and next they were contacted by phone for a second reminder. A total of 3,645 families with twins born between 1986 and 1994 participated in this ongoing study at least once so far. The overall family response rate is 56%.

Twins with an illness or a physical handicap interfering with their daily lives were not included in the present paper ($N = 56$ twins). Data from one additional sibling per family were included. From families with more than one additional participating sibling, we selected the sibling closest in age to the twin. Maximum age difference between twins and siblings was 5.7 years (Mean age difference = 2.4 years, $SD = 1.0$). This resulted in exclusion of 157 siblings from the analyses. The total sample consisted of 6,317 twins (44% male) and 1,180 non-twin siblings (44% male), from 3,486 families, for whom data were available on at least 1 time point. For the cross-sectional analyses, data from the measurement occasion at the youngest age were selected. The age of the twins in the

cross-sectional dataset ranged between 13.2 and 19.9 years, with a mean age of 15.7 years ($SD = 1.4$) and the age of the non-twin siblings ranged between 13.0 and 20.0 with a mean age of 16.9 years ($SD = 1.9$). The cross-sectional dataset consisted of 528 MZ male, 455 DZ male, 778 MZ female, 559 DZ female, 1,085 DZ opposite-sex twin pairs, 519 brother, and 661 sisters. Of these, there were 2,912 (86%) complete and 493 (14%) incomplete twin pairs. There were 81 families for which only non-twin sibling data were available. For 802 (34.6%) of the same-sex twin pairs zygosity was determined based on blood group or DNA typing. Zygosity for the remaining same-sex twin pairs was determined by longitudinally collected questionnaire items about physical similarities and confusion by family members and strangers. These items allow accurate determination of zygosity in 93% of same-sex twin pairs (Rietveld et al., 2000).

Longitudinal analyses were based on a subsample of 1,658 twins (38% male) and 150 non-twin siblings (39% male), from 979 families for whom data on exercise behavior, internalizing problems, and SWB were available at 2 time points (at age 14 and 16 or 16 and 18). The longitudinal dataset consisted of 135 MZ male, 91 DZ male, 259 MZ female, 179 DZ female, and 289 DZ opposite-sex twin pairs, 58 brothers, and 92 sisters. Of these, there were 705 (74%) complete and 248 (26%) incomplete twin pairs. There were 26 families for which longitudinal data were available for non-twin siblings only.

Measures

Participants were asked to indicate what type(s) of regular leisure exercise they were involved in at the time of assessment. A list of 21 common individual (includes fitness centre, jogging, tennis, etc) and team-based exercise activities (soccer, field hockey) was provided plus 5 open entries for less common activities. For each exercise activity endorsed, the participants further reported on the number of months per year, weekly frequency and average duration of the activity. Ainsworth's Compendium of physical activity (Ainsworth et al., 2000) was used to assign a MET score (Metabolic equivalent) to each exercise activity, reflecting its energy expenditure as a multiple of the basal energy expenditure (approximately 1 kcal/kg/hour) in an average subject engaged in that activity.

For each participant a total weekly MET score was computed across all exercise activities by summing the products of the number of hours spent weekly on each exercise activity and its MET score. Activities were only scored if that participant had engaged in them for at least three months during the past year. Exercise during physical education classes at school was not included in the weekly MET score. Thus the dependent variable reflects leisure time exercise behavior only. The distribution of the total weekly MET score was skewed (distribution, 2.58) and demonstrated kurtosis (distribution, 16.06).

Therefore, for the bivariate genetic models, log-transformation was applied to the weekly MET score which reduced the skewness and kurtosis significantly to $-.51$ and -1.12 , respectively. For computation of the difference scores, we used untransformed weekly MET scores, because the difference scores were normally distributed.

Self-reported internalizing problems were collected with the Youth Self Report (YSR; Achenbach & Rescorla, 2001). The YSR was developed for adolescents to rate behavioral and emotional problems. The questionnaire consisted of 112 items from the 2001 version supplemented with 6 items from older versions of the YSR. The items had to be answered on a 3-point scale based on the occurrence of the behavior during the past 6 months: 0 if the item was not true, 1 if the item was somewhat or sometimes true, and 2 if the item was often or very true. We used a Dutch translation of the YSR which shows good psychometrical properties (Verhulst, Van der Ende, & Koot, 1997). The internalizing scale consists of the subscales Anxious/Depressed, Somatic Complaints, and Withdrawn and it consists of 31 items. Example items are “I’m too shy” and “I feel lonely”. Scores on the individual items were summed to get an overall score of internalizing problems which could range from 0 to 62 with high scores indicating high levels of internalizing problems. Internal consistency of the scale was good with a Chronbach’s Alpha of $.88$.

Three measures of self-reported evaluations of subjective wellbeing (SWB) were used in the present study: (1) satisfaction with life (SAT) was assessed with the Satisfaction with Life Scale (Diener et al., 1985). The scale consists of 5 items which had to be answered on a 7-point scale ranging from 1 = ‘strongly disagree’ to 7 = ‘strongly agree’. Example items are “My life is going more or less as I wished” and “I’m satisfied with my life”. Internal consistency of the scale was good with a Chronbach’s Alpha of $.86$. (2) Subjective happiness (HAP) was assessed with the Subjective Happiness Scale (Lyubomirsky & Lepper, 1999). The scale consists of 4 items which had to be answered on 7-point scale ranging from 1 = ‘strongly disagree’ to 7 = ‘strongly agree’. Example items are “On the whole I’m a happy person” and “On the whole, I’m not very happy”. Internal consistency of the scale was good with a Chronbach’s Alpha of $.84$. (3) Quality of life in general (QLg) as assessed with the Cantril Ladder (Cantril, 1965). The ladder has 10 steps: the top indicated the best possible life, and the bottom the worst possible life. Participants had to indicate the step of the ladder at which they place their lives in general.

A genetic factor analysis of SAT, HAP, and QLg demonstrated that these measures assess the same genetic construct (Bartels and Boomsma, 2009). Therefore measures of SAT, HAP, and QLg were summarized into a single score for SWB. For all participants, a latent factor score for SWB was estimated in Mplus (Muthén & Muthén, 2007). Phenotypic correlations between SAT, HAP, and QLg ranged between $.54$ and $.74$.

Correlations between the resulting latent factor scores and SAT, HAP, and QLg ranged between .70 and .95, indicating that the latent factor score is a good representation of the three observed measures of SWB.

Statistical analyses

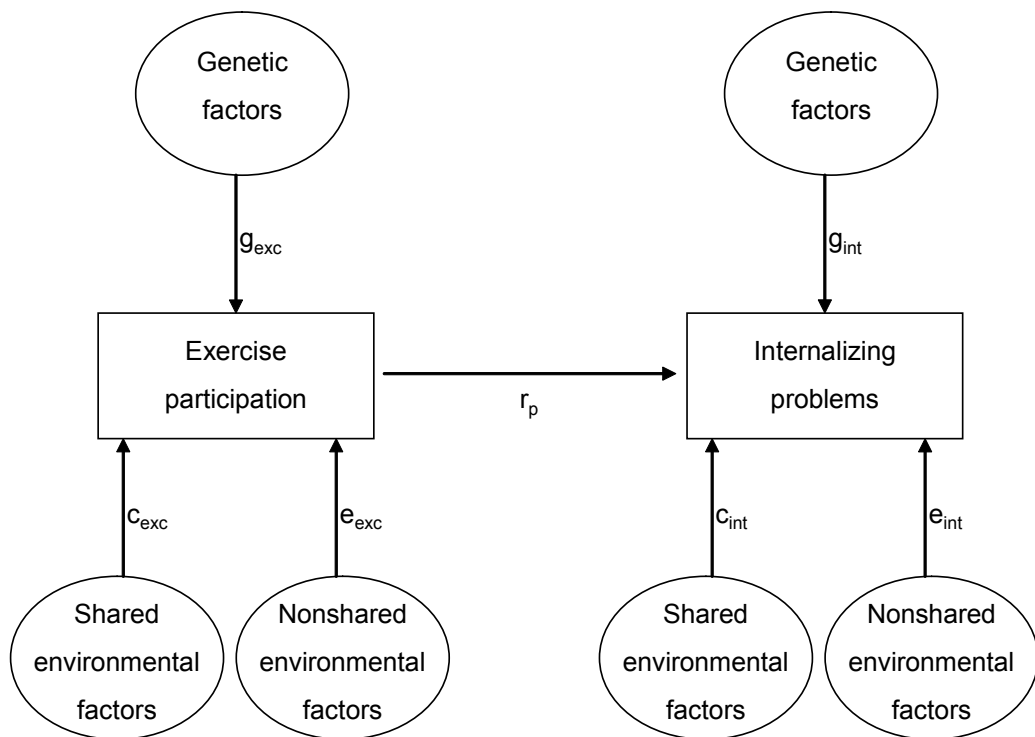
As a first step, using structural equation modeling in Mx (Neale et al, 2006), univariate genetic models were fitted to the data to examine the relative influence of genetic and environmental factors on exercise behavior, internalizing problems, and SWB. The amount of variation in a single trait due to additive genetic (A), shared environmental (C) and nonshared environmental factors (E) can be estimated by considering the different levels of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs and non-twin siblings. MZ twin pairs are genetically identical, whereas DZ twin pairs and non-twin siblings share on average 50% of their genetic material. Shared environmental effects refer to environmental factors shared by all members of the same family. Variation that is not due to genetic and shared environmental effects is attributed to environmental effects which are not shared by family members. The nonshared environmental variance component also includes measurement error variance. Parameter estimates representing the influence of A, C and E were allowed to differ between boys and girls to assess sex differences in the genetic architecture of the traits.

Causal effects of exercise on internalizing problems and SWB were tested in 4 different models. The first two tests were based on cross-sectional data. In the first test it was assessed whether the genetic (r_g), shared environmental (r_c) or nonshared environmental (r_e) correlation between exercise behavior and internalizing problems and SWB were significantly different from zero. If the genetic (r_g), shared environmental (r_c) or nonshared environmental (r_e) correlation between exercise behavior and internalizing problems, and between exercise behavior and SWB is not significantly different from zero, a causal effect cannot be the source of the association (De Moor et al., 2008).

A bivariate genetic model was fitted to the data in which the genetic architecture of the covariation between exercise behavior and internalizing problems and between exercise behavior and SWB was specified and r_g , r_c and r_e were computed (see Figure 4.1A). Based on the amount of variance in and covariance between traits due to A, C and E, r_g , r_c and r_e can be computed. r_g , r_c , and r_e were allowed to differ between boys and girls. Sex differences in the magnitude of r_g , r_c , and r_e were assessed by testing whether constraining r_g , r_c , and r_e for boys and girls to be equal resulted in a significant deterioration of model fit. The hypothesis of a causal effect was assessed by testing whether constraining r_g , r_c , and r_e to zero one by one would lead to a significant deterioration

of model fit. The bivariate genetic model also allowed us to test whether only r_g was significant. This would be compatible with the hypothesis of a common genetic factor underlying the cross-sectional association.

Figure 4.2. Graphical representation of a model in which exercise behavior causally influences internalizing problems. If this model is completely standardized, r_p represents the phenotypic correlation between exercise behavior and internalizing problems, and the g -, c - and e - coefficients represent the standardized genetic, shared environmental and nonshared environmental path coefficients for exercise behavior and internalizing problems.



Based on the magnitude of genetic, shared environmental and nonshared environmental effects on variation in exercise behavior, internalizing problems and SWB, and phenotypic correlations between the traits, the expected r_g , r_c , and r_e under a model in which exercise behavior causally influences internalizing problems and SWB can be computed. In Figure 4.2, a graphical representation of a model in which exercise behavior causally influences internalizing problems is presented. If the causal model as depicted in

Figure 4.2 is the true model that generated the data, the expected r_g ($r_{g,exp}$), r_c ($r_{c,exp}$) and r_e ($r_{e,exp}$) under a causal model can be derived. We assume in this derivation that the solution is standardized so that r_p in Figure 4.2 represents a phenotypic correlation.

$$r_{g,exp} = r_p \frac{g_{exc}}{g_{int}} \quad (1)$$

$$r_{c,exp} = r_p \frac{c_{exc}}{c_{int}} \quad (2)$$

$$r_{e,exp} = r_p \frac{e_{exc}}{e_{int}} \quad (3)$$

Here, r_p represents the phenotypic correlation between exercise behavior and internalizing problems, and the g -, c - and e - coefficients represent the standardized genetic, shared environmental and nonshared environmental path coefficients for exercise behavior and internalizing problems. In the same way $r_{g,exp}$, $r_{c,exp}$ and $r_{e,exp}$ between exercise behavior and SWB can be derived. If the estimates of r_g , r_c and r_e are significantly different from $r_{g,exp}$, $r_{c,exp}$ and $r_{e,exp}$, this would not be compatible with the hypothesis that the association between exercise behavior, internalizing problems and SWB is fully explained by causal effects of exercise behavior. We assessed whether the estimates of r_g , r_c and r_e from the bivariate genetic models were significantly different from $r_{g,exp}$, $r_{c,exp}$ and $r_{e,exp}$ by testing whether fixing r_g , r_c and r_e at the expected values would lead to a significant deterioration of model fit.

The fit of the different models was compared by means of the log-likelihood ratio test (LRT). The difference in minus two times the log-likelihood (-2LL) between two nested models has a χ^2 distribution with the degrees of freedom (df) equaling the difference in df between the two models. If a p -value higher than 0.05 was obtained from the χ^2 -test the fit of the constrained model was not significantly worse than the fit of the more complex model. In this case, the constrained model was kept as the most parsimonious and best fitting model.

In the second test of the causal hypothesis we assessed whether MZ twin intrapair differences in levels of exercise participation are associated with intrapair differences in internalizing problems and SWB. Significant associations between intrapair differences in

exercise behavior and internalizing problems, and exercise behavior and SWB would be compatible with the causal hypothesis, whereas non-significant associations would falsify this hypothesis (De Moor et al., 2008). Differences of the measures of exercise behavior, internalizing problems, and SWB of an MZ twin and his or her co-twin were computed. The resulting intrapair difference scores of exercise behavior, internalizing problems, and SWB were normally distributed. Using maximum likelihood estimation in Mplus, the MZ intrapair differences in internalizing problems and SWB were regressed on the difference in weekly MET hours.

The third and fourth tests of the causal hypothesis were based on the longitudinal dataset. Longitudinal correlations between exercise behavior and internalizing problems and SWB for a 2-year time interval were computed in Mx. If exercise participation causes fewer internalizing problems and increased levels of SWB, there should be a significant longitudinal correlation between exercise behavior at baseline and subsequent internalizing problems and SWB. The hypothesis of a causal effect is falsified if these longitudinal correlations are non-significant. However, the reverse is not true. A significant longitudinal correlation does not necessarily provide evidence for causality if common genetic factors drive the association between exercise behavior at baseline and subsequent internalizing problems and SWB (De Moor et al., 2008). The genetic information in our sample was again used to more robustly test causality by fitting a longitudinal bivariate genetic model in Mx (see Figure 4.1C). If the effect of exercise behavior on internalizing problems and SWB is causal, $r_{g1,2}$, $r_{c1,2}$ and $r_{e1,2}$ should be significant (De Moor et al., 2008). $r_{g1,2}$, $r_{c1,2}$ and $r_{e1,2}$ represent the genetic, shared environmental, and nonshared environmental correlation between the measurement of exercise behavior at baseline and successive measurement of internalizing problems and SWB with a 2-year interval.

As with the cross-sectional bivariate genetic model, the genetic architecture of the covariation between exercise behavior at baseline and internalizing problems and SWB at follow-up was specified, and $r_{g1,2}$, $r_{c1,2}$, and $r_{e1,2}$ were estimated. Sex differences in the magnitude of $r_{g1,2}$, $r_{c1,2}$, and $r_{e1,2}$ were assessed by testing whether constraining $r_{g1,2}$, $r_{c1,2}$, and $r_{e1,2}$ for boys and girls to be equal resulted in a significant deterioration of model fit. The hypothesis of a causal effect was assessed by testing whether constraining $r_{g1,2}$, $r_{c1,2}$, and $r_{e1,2}$ to zero would lead to a significant deterioration of model fit. As in the cross-sectional genetic analysis, the bivariate genetic model also allowed us to test whether only $r_{g1,2}$ was significant. This would be compatible with the hypothesis of a common genetic factor underlying the longitudinal association (De Moor et al., 2008). In the longitudinal bivariate genetic model, we also assessed whether the estimates of $r_{g1,2}$, $r_{c1,2}$ and $r_{e1,2}$ from

the longitudinal bivariate genetic models were significantly different from $r_{g1,2,exp}$, $r_{c1,2,exp}$ and $r_{e1,2,exp}$ under a causal model, by testing whether fixing r_g , r_c and r_e at the expected values would lead to a significant deterioration of model fit.

In the fourth test of the causal hypothesis we assessed whether MZ twin intrapair differences in individuals' changes in exercise behavior over time are associated with intrapair differences in individuals' changes in internalizing problems and SWB over time (see Figure 4.1D). Significant associations between these intrapair differences would be compatible with the causal hypothesis, whereas non-significant associations would falsify this hypothesis. Within-individual differences between baseline and follow-up scores were computed for the measures of exercise behavior, internalizing problems, and SWB. Subsequently, differences between an MZ twin and his or her co-twin were computed for the within-individual changes in exercise behavior, internalizing problems, and SWB over time. The resulting MZ intrapair difference scores were normally distributed. Using maximum likelihood estimation in MPlus, the MZ intrapair differences in within-individual changes in internalizing problems and SWB over time were regressed on the MZ intrapair differences of within-individual changes in exercise behavior over time.

Results

In Table 4.1, means and variances of exercise behavior, internalizing problems and SWB (uncorrected for age) are presented as a function of sex. Significant effects of age were found on the means of exercise behavior ($\chi^2(1) = 43.28$, $p < .01$; standardized regression coefficient $\beta = -.08$), internalizing problems ($\chi^2(1) = 7.83$, $p < .01$; standardized regression coefficient $\beta = .03$) and SWB ($\chi^2(1) = 26.74$, $p < .01$; standardized regression coefficient $\beta = -.06$), indicating that mean scores for exercise behavior and SWB were higher for younger participants, whereas the mean score for internalizing problems was higher for older participants. Means of exercise behavior ($\chi^2(1) = 149.75$, $p < .01$) and SWB ($\chi^2(1) = 355.84$, $p < .01$) were significantly higher for boys, whereas the mean of internalizing problems ($\chi^2(1) = 18.69$, $p < .01$) was significantly higher for girls. Variance in exercise behavior ($\chi^2(1) = 4.13$, $p < .05$) was significantly larger for boys, whereas variance in internalizing problems ($\chi^2(1) = 251.31$, $p < .01$) and SWB ($\chi^2(1) = 23.78$, $p < .01$) were significantly higher for girls.

Twin and twin-sibling correlations for exercise behavior, internalizing problems and SWB are presented in the left part of Table 4.2. The DZ twin correlations for the traits were equal to twin-sibling correlations (p -values $> .05$).

Table 4.1

Maximum likelihood estimates of means and variances of exercise behavior, internalizing problems, and SWB as a function of sex

		M	Var
Exercise behavior (weekly MET score)	Boys	26.8	664.8
	Girls	18.4	521.9
Internalizing problems (scale score)	Boys	6.8	31.1
	Girls	9.6	55.3
SWB (factor score)	Boys	.06	.79
	Girls	-.04	.93

Table 4.2

Twin and twin-sibling correlations for exercise behavior, internalizing problems and SWB, and the cross-twin cross-trait and cross-sibling cross-trait correlations of exercise behavior, internalizing problems and subjective wellbeing

	Exercise	Internalizing	SWB	Exercise - internalizing	Exercise - SWB
MZM	.69 (.65 to .73)	.63 (.56 to .68)	.41 (.33 to .48)	-.11 (-.16 to -.06)	.10 (.06 to .15)
DZM / sibling	.32 (.26 to .38)	.34 (.25 to .43)	.11 (.03 to .19)	-.07 (-.13 to -.01)	.07 (.02 to .12)
MZF	.72 (.69 to .75)	.50 (.45 to .55)	.47 (.41 to .52)	-.12 (-.16 to -.09)	.14 (.11 to .18)
DZF / sibling	.42 (.36 to .47)	.30 (.25 to .35)	.24 (.18 to .30)	-.08 (-.12 to -.04)	.11 (.06 to .15)
DOS / sibling	.28 (.24 to .33)	.24 (.19 to .28)	.20 (.15 to .24)	-.05 (-.09 to -.02)	.08 (.05 to .12)

Note. MZ = Monozygotic twin pair; DZ = Dizygotic twin pair; M = male; F = female; DOS = Opposite-sex twin pair.

The MZ twin correlations were significantly larger than the DZ twin and twin-sibling correlations (p -values $< .01$), indicating that genetic influences accounted for individual differences in exercise behavior, internalizing problems and SWB. For girls, DZ twin and twin-sibling correlations for exercise behavior and internalizing problems were higher than half the MZ twin correlations, suggesting that shared environmental factors play a role in explaining variation in exercise behavior and internalizing problems. For exercise

behavior and internalizing problems in boys and SWB in boys and girls, DZ twin and twin-sibling correlations were about half the MZ twin correlations, suggesting no shared environmental effects.

The proportions of variance explained by additive genetic, shared environmental and nonshared environmental derived from univariate genetic models for exercise behavior, internalizing problems, and SWB are presented in Table 4.3. There appeared to be significant sex differences in the magnitude of additive genetic, shared environmental, and nonshared environmental effects on exercise behavior ($\chi^2(3) = 23.36, p < .01$), internalizing problems ($\chi^2(3) = 263.70, p < .01$), and SWB ($\chi^2(3) = 29.56, p < .01$). Variation in exercise behavior and internalizing problems was accounted for by genetic and nonshared environmental factors in boys, whereas in girls shared environmental factors played a significant role as well. Additive genetic influences were relatively more important in boys. Variation in SWB was accounted for by genetic and nonshared environmental factors in boys and girls, with additive genetic influences being relatively more important in girls. Shared environmental played no significant role in explaining individual differences in SWB.

Table 4.3

Proportions of variance explained by additive genetic, shared environmental and nonshared environmental factors derived from univariate genetic models for exercise participation, internalizing problems, and SWB (95% confidence intervals added in parentheses)

		A	C	E
Exercise behavior	Boys	.70 (.63 - .76)	--	.30 (.27 - .34)
	Girls	.56 (.47 - .66)	.15 (.07 - .23)	.29 (.26 - .32)
Internalizing problems	Boys	.50 (.43 - .57)	--	.50 (.45 - .56)
	Girls	.45 (.34 - .57)	.12 (.02 - .21)	.43 (.39 - .48)
SWB	Boys	.34 (.27 - .41)	--	.66 (.60 - .73)
	Girls	.50 (.43 - .56)	--	.50 (.46 - .56)

CROSS-SECTIONAL ASSOCIATIONS

The phenotypic correlation between exercise behavior and internalizing problems was $-.13$ (95% CI: $-.14$ to $-.10$; $\chi^2(1) = 93.81, p < .01$) and between exercise behavior and SWB it was $.12$ (95% CI: $.09$ - $.15$; $\chi^2(1) = 91.90, p < .01$). The phenotypic correlations were equal between boys and girls (p -values $> .05$). This indicates that increased levels

of exercise behavior are cross-sectionally associated with decreased levels of internalizing problems and increased levels of SWB. The phenotypic correlation between internalizing problems and SWB was $-.51$ (95% CI: $-.53$ to $-.49$; $\chi^2(1) = 1766.78$, $p < .01$). Cross-twin cross-trait correlations are presented in the right part of Table 4.2. The cross-twin cross-trait correlations in MZ and DZ twin pairs, and twin-sibling pairs were significant (p -values $> .05$). This indicates that exercise behavior in one twin is predictive of internalizing problems and SWB in his or her co-twin or non-twin sibling. The DZ cross-twin cross-trait correlations were equal to twin-sibling cross-trait correlations (p -values exceeded a 5% significance level). The MZ cross-twin cross-trait correlations were significant larger than the DZ cross-twin cross-trait and the twin-sibling cross-trait correlations (p -values $< .05$), indicating that genetic influences account for the covariation between exercise behavior and internalizing problems, and between exercise behavior and SWB. MZ and DZ cross-twin cross-trait correlations were equal between boys and girls (p -values $> .05$).

Bivariate genetic model

Bivariate genetic model fitting results are presented in Table 4.4. In the upper part of the table the results of the bivariate genetic model for exercise behavior and internalizing problems are presented. r_c was not modeled for boys, because shared environmental influences did not contribute to variation in exercise behavior and internalizing problems in boys. In model 2 the genetic (r_g) and nonshared environmental correlation (r_c) were constrained to be equal for boys and girls which did not result in a significant deterioration of model fit. Subsequent models were therefore compared to this model. Models 3 to 5 tested whether constraining r_g , r_c for girls, and r_c to zero would lead to a significant deterioration of model fit. r_g was statistically significant for boys and girls, and r_c was statistically significant for girls, whereas r_c was not statistically significant. r_g was $-.19$ (95% CI: $-.26$ to $-.15$) and r_c for girls was $-.45$ (95% CI: -1.0 to $-.19$). Model 6 to 8 assessed whether the estimates of r_g , r_c for girls and r_c were significantly different from $r_{g,exp}$, $r_{c,exp}$ for girls and $r_{c,exp}$ under a model in which exercise behavior causally influences internalizing problems, by testing whether fixing r_g , r_c for girls and r_c at the expected values resulted in a significant deterioration of model fit. The estimates of r_g and r_c were significantly larger than $r_{g,exp}$ ($-.19$ versus $-.14$) and $r_{c,exp}$ ($-.45$ versus $-.14$), whereas the estimate of r_c was significantly smaller compared to $r_{c,exp}$ ($-.02$ versus $-.10$). These results suggest that the association between exercise behavior and internalizing problems in boys seems to be mainly due to genetic factors which are common (i.e. overlapping) for exercise

behavior and internalizing problems. For girls, genetic and shared environmental factors both account for the association. Because r_c was not different from zero and significantly smaller compared to $r_{c,exp}$ the causal hypothesis was not supported in either boys or girls.

Table 4.4

Bivariate model fitting results for exercise behavior and internalizing problems and SWB

Model	vs.	-2LL	df	χ^2	df	p
Exercise behavior – internalizing problems						
1. r_g, r_c boys; r_g, r_c, r_e girls: sex differences	--	58490.629	14427	--	--	--
2. r_g, r_c boys; r_g, r_c, r_e girls: no sex differences	1	58492.491	14429	1.86	2	.39
3. $r_g = 0$	2	58514.756	14430	22.26	1	< .01
4. r_c girls = 0	2	58503.387	14430	10.90	1	< .01
5. $r_e = 0$	2	58493.414	14430	.92	1	.34
6. $r_g = r_g$ expected	2	58496.720	14430	4.23	1	< .05
7. r_c girls = r_c girls expected	2	58497.718	14430	5.33	1	< .05
8. $r_e = r_e$ expected	2	58527.715	14430	35.22	1	< .01
Exercise behavior – SWB						
1. r_g, r_e : sex differences	--	32001.879	14602	--	--	--
2. r_g, r_e : no sex differences	1	32009.817	14604	7.94	2	< .05
3a. r_g boys = 0	1	32017.844	14603	15.97	1	< .01
3b. r_g girls = 0	1	32064.555	14603	62.68	1	< .01
4a. r_e boys = 0	1	32002.042	14603	.16	1	.69
4b. r_e girls = 0	1	32006.982	14603	5.10	1	< .05
5a. r_g boys = r_g boys expected	1	32002.217	14603	.34	1	.56
5b. r_g girls = r_g girls expected	1	32022.662	14603	20.78	1	< .01
6a. r_e boys = r_e boys expected	1	32004.310	14603	2.43	1	.12
6b. r_e girls = r_e girls expected	1	32025.859	14603	23.98	1	< .01

Note. r_g = genetic correlation; r_c = shared environmental correlation; r_e = nonshared environmental correlation; vs. = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; df = degrees of freedom of χ^2 -test; p = p-value

In the lower part of Table 4.4 the results of the bivariate genetic model for exercise behavior and SWB are presented. For SWB, r_c was not modeled, because shared environmental influences did not contribute to variation in SWB in boys and girls. In model 2, r_g and r_e were constrained to be equal for boys and girls which resulted in a significant deterioration of model fit. Therefore, r_g and r_e were allowed to differ between boys and girls. Model 3 and 4 tested whether constraining r_g and r_e to zero would lead to a significant deterioration of model fit. r_g was statistically significant in boys and girls and estimated at .22 (95% CI: .14 - .29) and .31 (95% CI: .23 - .39) for boys and girls respectively. r_e was close to zero in both sexes, and estimated at .02 (95% CI: -.06 to .10) for boys and .04 (95% CI: .09 - .01) for girls. Model 5 and 6 tested whether the estimates of r_g and r_e were significantly different from $r_{g,exp}$ and $r_{e,exp}$ under a model in which exercise behavior causally influences SWB, by testing whether fixing r_g and r_e at the expected values resulted in a significant deterioration of model fit. For boys, the estimates of r_g and r_e were not statistically different from $r_{g,exp}$ (.22 versus .17) and $r_{e,exp}$ (.02 versus .08). For girls, the estimate of r_g was significantly larger (.31 versus .13), whereas the estimate of r_e was significantly smaller (.04 versus .09) compared to $r_{g,exp}$ and $r_{e,exp}$. These results suggested that the association between exercise behavior and SWB in girls and boys is mainly due to genetic pleiotropy. Because r_e was not different from zero (boys) or significantly smaller compared to $r_{e,exp}$ (girls) the causal hypothesis was not supported.

MZ twin intrapair differences model

Regression of the intrapair difference scores in internalizing problems on the intrapair difference scores in exercise behavior was non-significant (standardized regression coefficient $\beta = -.03$; $\chi^2(1) = 1.21$, $p = .27$). Regression of the intrapair difference scores in SWB on the intrapair difference scores in exercise behavior was non-significant (standardized regression coefficient $\beta = -.01$; $\chi^2(1) = .03$, $p = .85$). This indicates that in genetically identical twin pairs, a twin who exercises more does not have fewer internalizing problems or higher levels of SWB than his or her co-twin who exercises less. This is not in line with the causal hypothesis that increased levels of exercise cause decreased levels of internalizing problems and increased levels of SWB.

LONGITUDINAL ASSOCIATIONS

Based on the longitudinal subsample, twin and twin-sibling correlations for exercise behavior, internalizing problems and SWB at the first (T1) and second (T2) measurement occasion are presented in the left part of Table 4.5.

Table 4.5

Twin and twin-sibling correlations for exercise behavior, internalizing problems and SWB at the first (T1) and second (T2) measurement occasion, and the longitudinal cross-twin cross-trait and cross-sibling cross-trait correlations for exercise behavior, internalizing problems and subjective wellbeing

	Exercise		Internalizing		SWB		Exercise T1 – internalizing T2	Exercise T1 – SWBT2
	T1	T2	T1	T2	T1	T2		
MZM	.74 (.66 to .81)	.66 (.56 to .74)	.71 (.60 to .78)	.40 (.09 to .59)	.28 (.09 to .44)	.43 (.25 to .57)	-.13 (-.23 to -.02)	.13 (.04 to .22)
DZM / sibling	.27 (.12 to .40)	.21 (.02 to .38)	.27 (.08 to .43)	.00 (-.22 to .25)	.03 (-.15 to .22)	-.01 (-.18 to .17)	.07 (-.10 to .23)	-.05 (-.19 to .09)
MZF	.81 (.76 to 84)	.75 (.69 to .79)	.57 (.48 to .65)	.44 (.32 to .55)	.52 (.41 to .60)	.36 (.24 to .46)	-.14 (-.22 to -.07)	.16 (.10 to .22)
DZF / sibling	.42 (.33 to .50)	.45 (.33 to .55)	.32 (.23 to .40)	.39 (.24 to .51)	.22 (.12 to .30)	.12 (-.06 to .28)	-.07 (-.17 to .03)	.19 (.09 to .28)
DOS / sibling	.36 (.28 to .43)	.24 (.14 to .34)	.24 (.14 to .33)	.20 (.00 to .36)	.15 (.06 to .23)	.15 (.02 to .27)	-.08 (-.18 to .02)	.09 (.01 to .18)

Note. T1 = first measurement occasion; T2 = second measurement occasion; MZ = Monozygotic twin pair; DZ = Dizygotic twin pair; M = male; F = female; DOS = Opposite-sex twin pair.

The DZ twin correlations for the traits at both measurement occasions were equal to twin-sibling correlations (p-values > .05). Twin and twin-sibling correlations on T1 and T2 were largely similar to those presented in Table 4.2 which were based on the cross-sectional sample.

The longitudinal phenotypic correlation between exercise behavior and internalizing problems two years later was -.15 (95% CI: -.21 to -.08; $\chi^2(1) = 19.70$, $p < .01$) and between exercise behavior and SWB it was .16 (95% CI: .11 - .21; $\chi^2(1) = 37.66$, $p < .01$) which is comparable with the magnitude of the cross-sectional phenotypic correlations. This means that exercise behavior at baseline is associated with internalizing problems and SWB at follow-up. Longitudinal cross-twin cross-trait correlations between exercise behavior at T1 and internalizing problems and SWB at T2 are presented in the right part of Table 4.5. The DZ cross-twin cross-trait correlations were equal to twin-sibling cross-trait correlations (p-values exceeded a 5% significance level). For boys, the MZ cross-twin cross-trait correlations were significant larger than the DZ cross-twin cross-trait and the twin-sibling cross-trait correlations (p-values < .05), indicating that genetic influences account for the covariation between exercise behavior and internalizing problems, and between exercise behavior and SWB. For girls, the MZ cross-twin cross-trait correlations were not significantly different from the DZ cross-twin cross-trait and the twin-sibling cross-trait correlations (p-values > .05), indicating that shared environmental influences account for the covariation between exercise behavior and internalizing problems, and between exercise behavior and SWB.

Longitudinal bivariate genetic model

Longitudinal bivariate model fitting results are presented in Table 4.6. In the upper part of the table results of the longitudinal genetic model for exercise behavior and internalizing problems are presented. Model 2 tested whether constraining the genetic ($r_{g1,2}$) and nonshared environmental correlation ($r_{e1,2}$) to be equal for boys and girls led to a significant deterioration of model fit. $r_{g1,2}$ and $r_{e1,2}$ were not significantly different for boys and girls and were therefore constrained to be equal. Models 3 to 5 tested whether constraining $r_{g1,2}$ and $r_{e1,2}$ for boys and girls, and $r_{e1,2}$ for girls, to zero would lead to a significant deterioration of model fit. $r_{g1,2}$ was -.27 (95% CI: -.56 to -.10) and significantly larger than zero, whereas $r_{e1,2}$ for girls and $r_{e1,2}$ were not different from zero. Model 6 to 8 assessed whether the estimates of $r_{g1,2}$, $r_{e1,2}$ for girls and $r_{e1,2}$ were significantly different from $r_{g1,2,exp}$, $r_{e1,2,exp}$ for girls and $r_{e1,2,exp}$ under a model in which exercise behavior causally influences internalizing problems. The estimates of $r_{g1,2}$ and $r_{e1,2}$ for girls were not significantly different from $r_{g1,2,exp}$ and $r_{e1,2,exp}$ for girls, whereas the estimate of $r_{e1,2}$ was significantly

smaller compared to $r_{e,1,2,exp}$ (-.02 versus -.12) under a causal model. This suggests that the longitudinal association between exercise behavior and internalizing problems in both sexes is mainly accounted for by genetic factors which are common for exercise behavior and internalizing problems. Because r_e and r_c for girls were not different from zero and r_c was significantly smaller compared to $r_{e,exp}$ the causal hypothesis was not supported.

Table 4.6

Longitudinal bivariate model fitting results for exercise behavior and internalizing problems and SWB

Model	vs.	-2LL	df	χ^2	df	p
Exercise behavior – internalizing problems						
1. $r_{g1,2}, r_{e1,2}$ boys; $r_{g1,2}, r_{c1,2}, r_{e1,2}$ girls: sex differences	--	9583.709	2838	--	--	--
2. $r_{g1,2}, r_{e1,2}$ boys; $r_{g1,2}, r_{c1,2}, r_{e1,2}$ girls: no sex differences	1	9584.841	2840	1.13	2	.57
3. $r_{g1,2} = 0$	2	9588.744	2841	3.90	1	< .05
4. $r_{c1,2}$ girls = 0	2	9586.936	2841	2.10	1	.15
5. $r_{e1,2} = 0$	2	9585.028	2841	.19	1	.67
6. $r_{g1,2} = r_{g1,2,exp}$	2	9585.203	2841	.36	1	.55
7. $r_{c1,2}$ girls = $r_{c1,2,exp}$	2	9585.559	2841	.72	1	.40
8. $r_{e1,2} = r_{e1,2,exp}$	2	9589.375	2841	4.53	1	< .05
Exercise behavior – SWB						
1. $r_{g1,2}, r_{e1,2}$: sex differences	--	7237.608	3455	--	--	--
2. $r_{g1,2}, r_{e1,2}$: no sex differences	1	7239.709	3457	2.10	2	.35
3. $r_{g1,2} = 0$	2	7265.019	3458	25.31	1	< .01
4. $r_{e1,2} = 0$	2	7239.716	3458	.01	1	.93
5. $r_{g1,2} = r_{g1,2,exp}$	2	7245.787	3458	6.08	1	< .05
6. $r_{e1,2} = r_{e1,2,exp}$	2	7248.324	3458	8.62	1	< .01

Note. $r_{g1,2}$ = genetic correlation; $r_{c1,2}$ = shared environmental correlation; $r_{e1,2}$ = nonshared environmental correlation; vs. = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; df = degrees of freedom of χ^2 -test; p = p-value.

In the lower part of Table 4.6 results of the longitudinal genetic model for exercise behavior and SWB are presented. Model 2 tests whether constraining $r_{g1,2}$ and $r_{e1,2}$ to be equal for boys and girls resulted in a significant deterioration of model fit. $r_{g1,2}$ and $r_{e1,2}$ were not significantly different for boys and girls. Models 3 and 4 tested whether

constraining $r_{g1,2}$ and $r_{e1,2}$ to zero would lead to a significant deterioration of model fit. $r_{g1,2}$ was statistically significant, whereas $r_{e1,2}$ was not significantly larger than zero. $r_{g1,2}$ was .31 (95% CI: .19 - .43). Model 5 and 6 assessed whether the estimates of $r_{g1,2}$ and $r_{e1,2}$ were significantly different from $r_{g1,2,exp}$ and $r_{e1,2,exp}$ under a model in which exercise behavior causally influences SWB. $r_{g1,2}$ was significantly larger (.31 versus .20), whereas $r_{e1,2}$ was significantly smaller (.00 versus .11) than $r_{g1,2,exp}$ and $r_{e1,2,exp}$ respectively. This suggests that the longitudinal association between exercise behavior and SWB is also best explained by genetic factors which are common (i.e. overlapping) for exercise behavior and SWB. Because r_e was not different from zero and r_e was significantly smaller compared to $r_{e,exp}$ the causal hypothesis was not supported.

Longitudinal MZ twin intrapair differences model

Regression of the intrapair difference scores in individuals' changes in internalizing problems over time on intrapair difference scores in individuals' changes in exercise behavior over time was non-significant (standardized regression coefficient $\beta = .07$; $\chi^2 (1) = .915$, $p = .34$). Regression of the intrapair difference scores in individuals' changes in SWB over time on intrapair difference scores in individuals' changes in exercise behavior over time was also non-significant (standardized regression coefficient $\beta = .12$; $\chi^2 (1) = 3.70$, $p = .06$). This indicates that in genetically identical twin pairs, a twin showing increased frequency and intensity of exercise behavior over time does not show lower levels of internalizing problems or higher levels of SWB over time than his or her co-twin with decreasing levels of exercise behavior over time. This is again not in line with the causal hypothesis that increased levels of exercise cause decreased levels of internalizing problems and increased levels of SWB.

Discussion

The present study investigated whether exercise behavior causally influences internalizing problems and SWB or whether the association between these traits reflects the effects of underlying genetic factors, in a large sample of Dutch adolescent twins and their non-twin siblings. In keeping with population based studies investigating adult samples (Stubbe et al., 2007; De Moor et al., 2006; Norris, Carroll, & Cochrane, 1992; De Moor et al., 2008; Wise et al., 2006; Camacho et al., 1991), we found that exercise behavior was cross-sectionally and longitudinally associated with fewer internalizing problems and increased levels of SWB across the total sample of adolescents. However, within genetically identical twins, a twin who exercised more did not have fewer internalizing problems or higher

levels of SWB than his or her less exercising co-twin and, in addition, a twin showing increased frequency and intensity of exercise behavior over time did not show lower levels of internalizing problems or higher levels of SWB over time than his or her co-twin with unchanged or decreased levels of exercise behavior. The cross-sectional and longitudinal associations between exercise behavior and internalizing problems and SWB were entirely accounted for by genetic factors in boys and genetic and shared environmental factors in girls, whereas the unique environmental factors causing them to participate in exercise did not cause lower levels of internalizing problems and increased levels of SWB, currently or 2 years later.

Taken together, these results do not support the hypothesis that increased levels of psychological wellbeing found in adolescent exercisers reflect a causal effect of exercise. Instead, the results are compatible with the alternative hypothesis that the association between exercise behavior and psychological wellbeing in adolescence is mainly due to an underlying factor. Pleiotropic genes seem to be the major component of this underlying factor although shared environmental influences also contribute to both exercise behavior and internalizing behavior in girls. In adults, pleiotropic genetic factors have also been found to explain the association between exercise and depressive and anxious symptoms (De Moor et al., 2008) as well as between exercise and SWB (Stubbe et al., 2007). As in the adolescents, the causal hypothesis had to be rejected as the source of the association between exercise behavior and wellbeing in adults.

The lack of support for the hypothesis that increased levels of psychological wellbeing found in exercisers reflect a causal effect of exercise needs to be reconciled with findings from training studies that reported improved mood or reductions in coping behavior, depression, and anxiety after a program of aerobic exercise training in comparison to control manipulations (Barbour & Blumenthal, 2002; Brosse et al., 2002; Steptoe et al., 1989). Although others have failed to replicate these training effects (de Geus et al., 1993; King et al., 1989) and critical reviews on this topic express only cautious optimism about the causal role of exercise (Lawlor & Hopker, 2001; Dunn et al., 2005) recent well-designed studies in depression patients (Blumenthal et al., 2007) report beneficial psychological effects of exercise that match or even exceed those of pharmacological treatment. It should be recognized, however, that these studies examined the effects of prescribed and externally monitored exercise treatments in select subgroups and that methodological problems cling to even the most well designed randomized controlled trial (Ekkekakis, 2008) due to the strong societal beliefs about the efficacy of this intervention. Taken our evidence for genetic pleiotropy and the pitfalls in existing

randomized controlled trials (i.e. assuming that regular exercise may prevent depression in a non-clinical adolescent sample, because it has been successfully used as a therapy in subsets of clinically depressed adult patients) is still a big leap of faith.

In spite of the above, we do not rule out the possibility that exercise might have a causal effect on psychological well-being in specific subgroups of adolescents. In fact, a model has been proposed that is compatible with genetic pleiotropy and still allows exercise to causally increase well-being in specific subgroups (de Geus & de Moor, 2008). To maintain regular exercise participation, appetitive effects of exercise need to outweigh the aversive effects. Individuals for who the aversive effects of exercise are stronger than the rewarding effects will eventually cease their exercise participation. In contrast, individuals for who the appetitive effects are stronger than the aversive effects will maintain exercise participation and become regular exercisers. It has indeed been shown that regular exercisers report greater acute exercise-induced mood enhancement than non-exercisers (Hoffman & Hoffman, 2008; Gauvin, 1990). These positive mood experiences after exercise activities may be a positive reward and therefore an important component of the higher wellbeing reported by regular exercisers. Genetic variants tipping the balance between appetitive and aversive effects may be a major source of individual differences in the effects of exercise on these acute mood responses. These genetic factors may not only keep people maintaining regular exercise participation. The repeated exercise-induced increases in mood may influence their overall psychological wellbeing. It is important to note that this causal effect of exercise is limited to a genetically sensitive subgroup and does not apply to the total adolescent population.

In addition, there is also a social-psychological mechanism through which the association between exercise behavior and psychological wellbeing may be explained by genetic pleiotropy (Stubbe et al., 2007; De Geus & De Moor, 2008). The basic idea is that people will seek out the activities that they are good in. This is particularly true during adolescence, because being good in sports is an important source of self-esteem for adolescents and the athletic role model is continuously reinforced by the media (Field et al., 1999; Pope et al., 2001). Therefore, genes coding for exercise ability (endurance, strength, flexibility, motor coordination) may well become genes for adolescent exercise behavior. These genetic factors may further influence psychological wellbeing because high exercise ability will coincide with the experience of strong feelings of competence and mastery as well as positive feedback from peers, leading to an increased sense of self-esteem and psychological wellbeing. In short, genetic variants influencing exercise ability will act to maintain exercise behavior and to increase psychological wellbeing, but again are limited to a selected group of adolescents, i.e. those with high exercise ability.

Apart from the gene-by-exercise interactive effects sketched above, we cannot rule out the presence of more complex mechanisms, like reverse or reciprocal causality, or combinations of causal, reverse causal and pleiotropic mechanisms. These complex mechanisms should be assessed in future studies making use of more complex causation models and larger sample sizes. Nonetheless, our results already have implications for the prevention of internalizing problems and decreasing levels of SWB during adolescence and the maintenance of exercise behavior at a sufficient level throughout adolescence and during the transition to adulthood. Current intervention strategies are mainly based on the idea that the associations between exercise and psychological wellbeing reflect causal effects of exercise and that exercise interventions exert beneficial effects on all participants. While it remains possible that exercise behavior can prevent some adolescents from developing internalizing problems, our results show that those with a disposition towards internalizing problems also have a higher chance to be a non-exerciser because of their genetic make-up. Approaching these adolescents at risk for internalizing problems with demanding exercise programs may be doomed to fail and may even amplify feelings of low self-esteem and consequently internalizing problems. Instead, behavioral therapy aimed to improve psychological wellbeing may need to coincide or even precede intervention on exercise behavior.

Conclusion

Among Dutch adolescents, regular exercise was found to be associated with fewer internalizing problems and higher levels of subjective wellbeing. The association could not be explained by causal effects of exercise behavior. Instead, the association was largely accounted for by genetic factors influencing a tendency towards exercise as well as a disposition towards low levels of internalizing problems and high levels of subjective wellbeing.

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Section III: Sedentary behavior and subjective wellbeing

5

Genetic and Environmental Influences on Individual Differences in Sedentary Behavior during Adolescence

This chapter is submitted as:

Van der Aa, N., Bartels, M., Te Velde, S.J., Boomsma, D.I., De Geus, E.J.C., & Brug, J. Genetic and environmental influences on individual differences in sedentary behaviour during adolescence – a twin-family study. *British Medical Journal*, under review.

Abstract

Sedentary behavior, and screen-viewing behaviors like watching TV and computer use more particularly, has been identified as a risk factor for obesity. Little is known, though, about the causes of individual differences in adolescents' sedentary behavior. The main aim of this study was to investigate the degree to which genetic and environmental influences affect variation in sedentary behavior throughout adolescence. Data on screen-viewing sedentary activities during leisure time were analyzed in a sample of 5,074 adolescent twins (age 12-20 years) and 937 siblings from 2,777 families. Screen-viewing sedentary behavior was assessed with survey items about weekly frequency of TV viewing, playing electronic games and computer/internet use. Results indicated that the genetic architecture of sedentary behavior changed during adolescence. Variation in sedentary behavior at age 12 was accounted for by genetic (boys: 35%, girls: 19%), shared environmental (boys: 29%, girls: 48%), and nonshared environmental factors (boys: 36%, girls: 34%). Variation in sedentary behavior at age 20 was accounted for by genetic (boys: 48%, girls: 34%) and nonshared environmental factors (boys: 34%, girls: 66%). The shift from shared environmental factors in the etiology of sedentary behavior in early adolescence to genetic and nonshared environmental factors in late adolescence requires tailoring and targeting of interventions to age groups.

Introduction

Overweight, obesity, and associated metabolic disorders are among the most important determinants of avoidable burden of disease (WHO, 2007). Engaging in sedentary behaviors, and screen viewing behaviors such as TV watching and computer use in particular, has been identified as an important risk factor for weight gain and metabolic disorders (Martínez-Gómez et al., 2010; Mark & Janssen, 2008; Wong et al., 1992). Such sedentary behaviors have been found to be obesity-related behaviors independent of physical activity, with separate determinants and independent contributions to obesity and metabolic disease (e.g. Patel et al., 2010; Biddle, 2007; Te Velde et al., 2007). Studying sedentary behavior during adolescence is of particular interest because overweight and obesity is likely to track into adulthood (see Serdula et al. (1993) for a review) and sedentary activities such as TV viewing and engagement in personal computer and internet activities are dominant leisure time activities during childhood and adolescence (e.g. Swinburn & Shelly, 2008; Van den Eijnden et al., 2008).

To inform prevention and intervention strategies aiming to curb the obesity and metabolic disorders epidemics, insight into the etiology of important and modifiable risk factors is needed. Although much research has focused on physical activity, research into the determinants of sedentary behavior is scarce. The few available studies indicate that family environmental factors such as parental modeling (e.g. rules and restrictions, parental sedentary behavior) and availability of screen-viewing opportunities in the home are important correlates of engaging in sedentary behaviors in youth (e.g. Van Zutphen et al., 2007; Salmon et al., 2005). Such etiological studies were, however, unable to focus on the possible genetic influence on likelihood of engaging in sedentary activities, while recent research indicates that engagement in physical activity, sport participation in particular, is importantly influenced by genetic factors (e.g. Van der Aa et al., 2010; Stubbe et al., 2005; Maia et al., 2002; Beunen & Thomis, 1999).

Twin-family studies offer the possibility to disentangle genetic and environmental factors as determinants of sedentary behavior. With data from twins and their non-twin siblings individual differences in behavior can be decomposed as due to genetic, shared environmental (environmental influences shared by members of the same family) and nonshared environmental influences (environmental influences unique to an individual). If a twin study is enriched with data from their non-twin siblings, comparison between twin and non-twin siblings also enables distinguishing between environmental factors specifically shared by twins and those shared by all siblings in the family. To our knowledge, only one study examined the relative influence of genetic factors to individual differences in sedentary behavior (Nelson et al., 2006). In a large sample of US adolescent

siblings (including twins) with a mean age of 16.5 years, variation in sedentary behavior was accounted for by additive genetic (34%), shared environmental (10%), and nonshared environmental factors (56%). When the same sample was assessed during early adulthood (mean age: 22.4 years), individual differences in sedentary behavior were accounted for by genetic (32%) and nonshared environmental factors (68%). These results indicated that the genetic architecture changed from adolescence to early adulthood. This repeats the pattern found for other behavioral traits, e.g. exercise behavior (Van der Aa et al., 2010; Stubbe et al., 2005) and internalizing psychopathology (Lamb et al., 2010; Bergen et al., 2007), where the genetic architecture has been found to change throughout adolescence, particularly during early adolescence.

In the present study we assessed sedentary behaviors in a large sample of Dutch adolescent twins and their non-twin siblings in the age range 12 to 20. Sedentary behavior was defined as the frequency of TV viewing, playing electronic games and engagement in personal computer / internet activities. The main objective was to estimate the genetic and environmental contribution to individual differences in self-reported sedentary behavior in adolescence as a function of age. In addition, we assessed whether there were sex differences in the heritability (i.e. quantitative sex differences) of sedentary behavior and whether different genetic variants play a role in sedentary behavior for boys and girls (i.e. qualitative sex differences).

Methods

procedures

Participants were registered with the Netherlands Twin Registry (NTR), established by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007; Boomsma et al., 2006). The large majority of twins were registered with the NTR as newborns. Parents of adolescent twins were asked for consent to send their children a survey. If their parents consented, twins and their non-twin siblings received an online or a paper & pencil self-report survey when they were 14, 16, and 18 years. The survey contained items about behavior, exercise, sedentariness, lifestyle, and well-being. When twins and siblings did not return the survey on time they were contacted by mail for a first reminder and next they were contacted by phone for a second reminder. For the present study, twins born between 1986 and 1992 and their non-twin siblings were selected. Twins and siblings with an illness or handicap interfering with their daily lives were not selected for the present study. Information on this came from parental reports

from questionnaires in previous years, and from the parental consent form on which parents could indicate if a twin or sibling was not able to participate in questionnaire research.

Subjects

Data from one additional non-twin sibling per family were included for the present study. From families with more than one additional participating sibling, we selected the sibling closest in age to the twin. This resulted in exclusion of 158 siblings from the analyses. The total sample consisted of 5,101 twins (44% male) and 980 non-twin siblings (45% male) from 2,777 families. Age of the twins and siblings ranged between 12 and 20 years with a mean age of 15.93 years ($SD = 1.60$). Participants were primarily Caucasian and they came from all regions of The Netherlands (rural and urban areas). Data were available for 440 monozygotic male (MZM), 384 dizygotic male (DZM), 627 monozygotic female (MZF), 461 dizygotic female (DZF), and 822 dizygotic opposite-sex (DOS) twin pairs. Of these, 367 (13%) were incomplete and 2,367 (87%) were complete twin pairs. For 743 (39.0%) of the same-sex twin pairs zygosity was determined based on blood group or DNA typing. Zygosity for the remaining same-sex twin pairs was determined by questionnaire items about physical similarities and confusion by family members and strangers, which were provided by parents on multiple time points in previous questionnaires. These items allow accurate determination of zygosity in 93% of same-sex twin pairs (Rietveld et al., 2000).

Measures

Participants were asked to report their weekly frequency of (1) watching TV, (2) gaming, and (3) engaging in personal computer / Internet activities during leisure time on 7-point scales (1 = 'never', 2 = 'once until now', 3 = 'less than 1 time per week', 4 = 'once a week', 5 = 'a couple of days per week', 6 = 'almost everyday', 7 = 'every day'). Scores on these 3 items were summed to get an overall score for the frequency of sedentary behavior per week, ranging from 3 to 21. This variable was normally distributed (skewness distribution: $-.03$; kurtosis distribution: $.30$). A factor analysis of the items indicated one single factor which explained 43.1% of the variance.

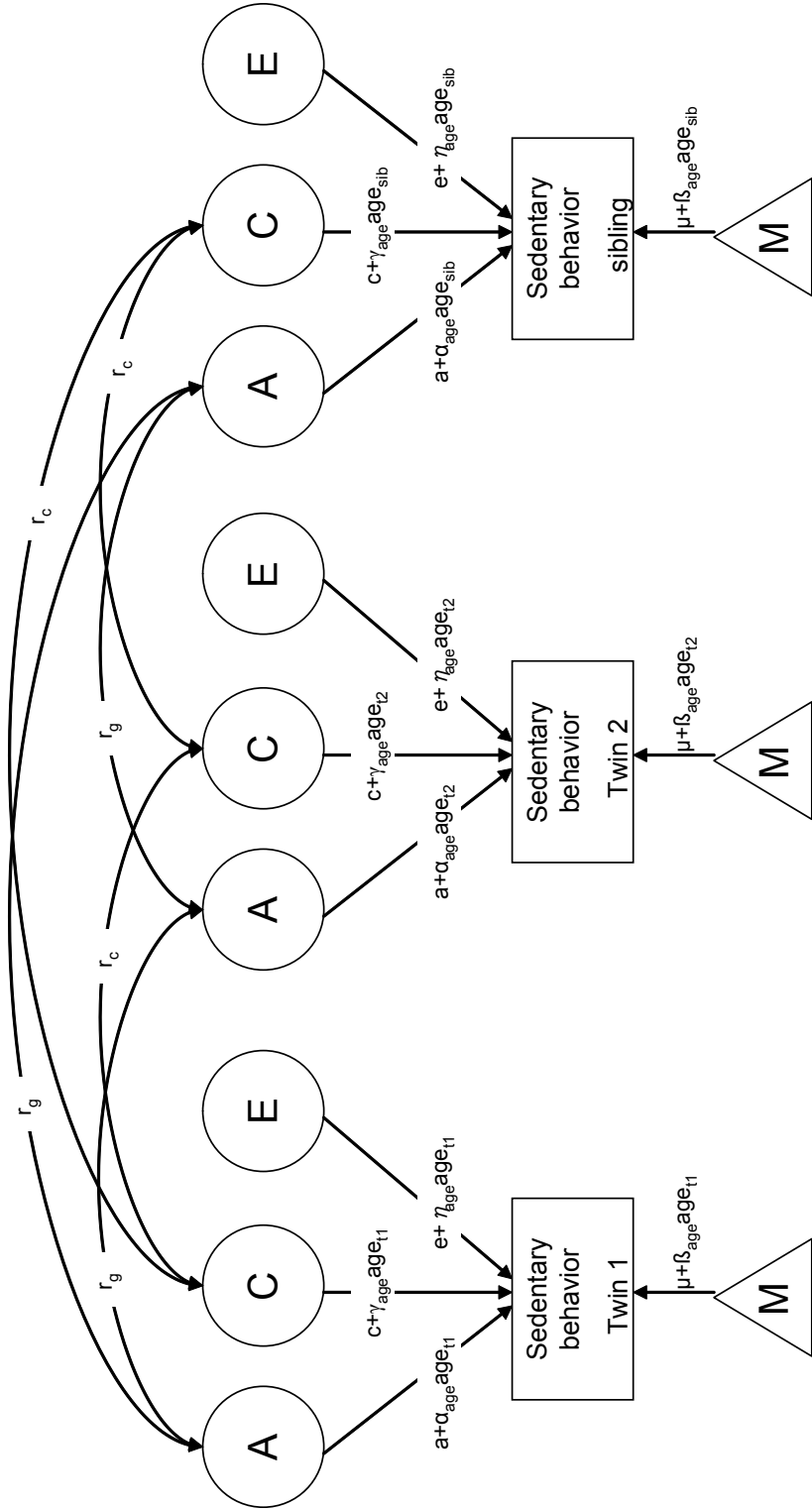
Statistical analyses

After obtaining correlations between twin and twin-sibling pairs for sedentary behavior, we employed genetic structural equation modeling to estimate the relative influence of genetic and environmental influences to individual differences in sedentary behavior.

Resemblance in sedentary behavior for twin and twin-sibling pairs is expressed in twin and twin-sibling correlations. Comparing correlations for MZ twin, DZ twin, and twin-sibling pairs constitutes a first step in evaluating the relative influence of genetic and environmental factors on variation in sedentary behavior. When the MZ correlation is higher than the DZ correlation and the twin-sibling correlation, it is inferred that genetic variation influences individual differences in sedentary behavior. A DZ or twin-sibling correlation higher than half the MZ correlation implies shared environmental effects. When the DZ twin correlation is higher than twin-sibling correlation, a specific environment might exist which is shared by twins but not by non-twin siblings (Eaves et al., 1999). Variation that is not due to genetic and shared environmental effects is attributed to nonshared environmental effects. The nonshared environmental variance component also includes measurement error variance. Comparing MZ, DZ, and twin-sibling correlations in boys and girls provides specific information regarding quantitative sex differences. For example, when the difference between MZ and DZ / sibling twin correlations is larger in boys than in girls, it can be concluded that genetic influences are stronger in boys compared to girls. Specific information regarding qualitative sex differences can be derived from the DZ opposite-sex (DOS) correlation. When the twin correlation in DOS twin pairs is lower than predicted from the correlation in male DZ / twin-sibling pairs and female DZ / twin-sibling pairs this might be due to genetic or shared environmental effects that influence one sex but not the other (Falconer & Mackay, 1996).

Means, variances, twin and twin-sibling correlations were estimated for each of the 5 sex by zygosity groups (i.e. MZM, DZM, MZF, DZF, and DOS) using the software package Mx (Neale et al., 2006). This was done in a so called saturated model which simply specifies for each sex by zygosity group that the data from the first- and second-born twin and the non-twin sibling are correlated without attempting to model these correlations as a function of genes or shared environment. Means and the covariance structure were estimated conditional on sex and age to take these variables into account. Age, which was transformed into a z-score, was included as a fixed effect (covariate) in the means model and on the covariance structure. Within a series of nested models we tested whether constraining the regression weight of age at zero led to a significant deterioration of model fit.

Figure 5.1. Univariate genetic model for sedentary behavior. The total variance in liability is modeled as caused by latent factors A (additive genetic influences), C (common or shared environment), and E (nonshared environment). r_g : MZ twin pairs = 1, DZ twin pairs and twin-sibling pairs = 0.5, DOS = estimated; r_c = 1.



Sex differences in means and variances for sedentary behavior were tested by constraining means and variances to be equal between boys and girls. In addition, we tested whether twin and twin-sibling correlations were significantly different for MZ and DZ / twin-sibling pairs.

Next, genetic models were fitted to the data in which the genetic architecture of sedentary behavior was specified. A graphical representation of the genetic model is given in Figure 5.1. The amount of variance in sedentary behavior due to additive genetic (A), shared environmental (C), and nonshared environmental effects (E) can be estimated by considering the different level of genetic relatedness between MZ, DZ, and twin-sibling pairs. MZ twin pairs are genetically identical, whereas DZ twin pairs and twin-sibling pairs share on average 50% of their segregating genes. In the genetic models, the genetic correlation (r_g) for MZ twin pairs is therefore fixed at 1, whereas for DZ and twin-sibling pairs it is fixed at 0.5. Shared environmental effects refer to environmental effects that are shared by all siblings in the family and therefore the shared environmental correlation (r_c) is fixed at 1. In Figure 5.1, r_g and r_c are represented by the double-headed arrows connecting the latent genetic (A) and shared environmental factors (C) for twin and twin-sibling pairs. Nonshared environmental effects refer to environmental factors that are unique to individuals in the family and therefore these are uncorrelated between siblings. A variance component comprising twin specific environment was not modeled, because twins and siblings did not differ in correlation. The influence of A, C, and E is represented by path coefficients a, c, and e (see Figure 5.1).

To assess whether there was evidence for changes in the genetic architecture of sedentary behavior throughout adolescence, the method as proposed by Purcell (2002) was used. The effect of age, which was transformed into a z-score, was included as a moderator on the path coefficients a, c, and e. This can be seen in Figure 5.1, in which the path coefficients of the latent factors are expressed as linear functions of the moderator. Under this model, a, c, and e represent the unmoderated variance components, and the α -, γ -, and η -coefficients represent the moderating effects of age on the genetic, shared environmental, and nonshared environmental variance components respectively. If for example, under this model, α is significantly different from zero, this indicates that the magnitude of the genetic variance component changes as a linear function of age. We assessed the significance of the age effects by testing whether fixing the α -, γ -, and η -coefficients to zero resulted in a significant deterioration of model fit. A complicating issue is that the effects of age can be sex specific as well. To assess if this is the case, the α -, γ -, and η -coefficients were allowed to be different between boys and girls and we tested whether constraining the α -, γ -, and η -coefficients between boys and girls to be equal

resulted in a significant deterioration of model fit. If these coefficients are significantly different between boys and girls, this indicates that the effect of age on the genetic architecture of sedentary behavior is different for boys and girls.

If qualitative sex differences in variation in sedentary behavior are present, the genetic correlation for DOS twins should be lower than the genetic correlation for DZ twins. To assess qualitative sex differences in the analyses for the total sample the genetic correlation (r_g) between DOS twins was estimated and we tested whether fixing r_g to 0.5 resulted in a significant deterioration of model fit. Quantitative sex differences in the genetic architecture of sedentary behavior were assessed by allowing the genetic (a), shared environmental (c), and nonshared environmental (e) parameter estimates to differ for boys and girls and we tested whether constraining these parameter estimates to be equal for boys and girls resulted in a significant deterioration of model fit. The statistical significance of the variance components A and C was assessed by testing whether fixing the corresponding parameter estimate (i.e. a and c) to zero resulted in a significant deterioration of model fit. The unmoderated estimates of a, c, and e represent the variance components A, C, and E at the mean age in the sample, since age was transformed into a z-score (i.e. z-distribution: mean = 0 and SD = 1).

We fitted various models that were nested in the sense that one model could be derived from the other by the imposition of one or more constraints on the parameters. The fit of the different models was compared by means of the log-likelihood ratio test (LRT). The difference in minus two times the log-likelihood (-2LL) between two nested models has a χ^2 distribution with the degrees of freedom (df) equaling the difference in df between the two models. If a p-value higher than 0.05 was obtained from the χ^2 -test the fit of the constrained model was not significantly worse than the fit of the more complex model. In this case, the constrained model was kept as the most parsimonious and best fitting model. The fit of the genetic models was also compared to the full ACTE model by means of Akaike's Information Criterion (AIC), keeping the model with the lowest AIC as the best fitting model (Neale et al., 2006).

Results

In the upper part of Table 5.1 means and variances (uncorrected for age) for sedentary behavior are presented as a function of sex. Mean level of sedentary behavior was significantly higher for boys compared to girls ($\chi^2(1) = 755.56, p < .05$). Figure 5.2 presents mean levels of sedentary behavior as a function of sex and age. A significant

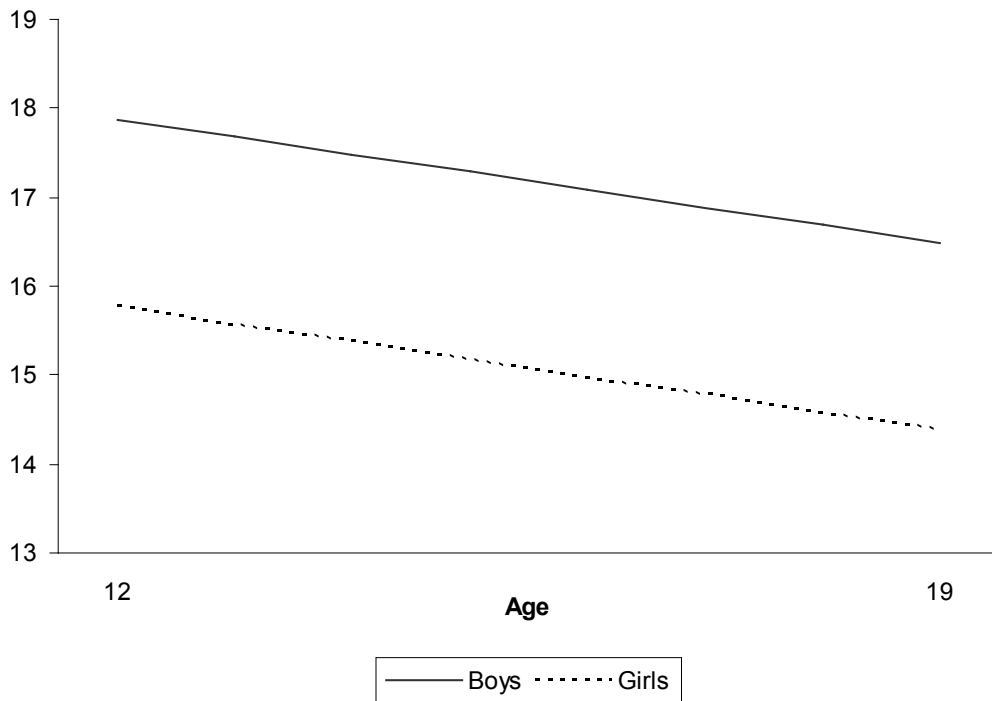
negative effect of age was found on the variance ($\chi^2 (1) = 19.66, p < .05$), indicating that variance in sedentary behavior declines with increasing age. Variation in sedentary behavior was also larger for boys compared to girls ($\chi^2 (1) = 104.58, p < .05$).

Table 5.1

Maximum likelihood estimates of means and variances of sedentary behavior as a function of sex

		M	Var
Total sample	boys	17.1	8.4
	girls	15.0	5.8

Figure 5.2. Changes in mean levels of sedentary behavior as a function of sex and age.



Twin and twin-sibling correlations for sedentary behavior are presented in Table 5.2. DZ twin correlations were not significantly different from twin-sibling correlations ($\chi^2 (3) = 5.41, p = .14$). MZ twin correlations were significantly higher than the DZ twin / twin-sibling correlations for boys ($\chi^2 (1) = 21.22, p < .05$) and girls ($\chi^2 (1) = 25.45, p < .05$). This suggests that individual differences in sedentary behavior are influenced by

genetic factors. For boys, DZ twin / twin-sibling correlations were about half the MZ twin correlation, suggesting that shared environmental factors play no role in explaining variation in sedentary behavior. For girls, DZ twin / twin-sibling correlation was higher than half the MZ correlation, suggesting shared environmental influence. Because there were sex differences in the correlational structure of the data, genetic modeling was started with an ACE model with different parameter estimates for boys and girls.

Table 5.2

Age corrected twin and twin-sibling correlations and their 95% confidence intervals for sedentary behavior

	r	95% CI
MZM	.47	.40 - .53
DZM	.25	.14 - .34
MZF	.58	.52 - .63
DZF	.44	.34 - .51
DOS	.25	.18 - .31
Brother	.25	.16 - .33
Sister	.28	.22 - .34
Brother – sister	.29	.19 - .38

Note. 95% CI = 95% confidence interval; Brother = male twin-brother pairs; Sister = female twin-sister pairs; Brother-sister = male twin-sister pairs, female twin-brother pairs

Table 5.3 presents the univariate model fitting results of the genetic models. In model 2, r_g was constrained at 0.5 which did not result in a significant deterioration of model fit, indicating that the same genetic factors act in boys and girls with regard to sedentary behavior.

In model 3, the moderation effects of age on the path coefficients of sedentary behavior were constrained to be equal between boys and girls, which did not result in a significant deterioration of model fit. Model 4 tested the statistical significance of the moderation effects of age on the path coefficients of sedentary behavior which resulted in a significant deterioration of model fit, indicating that the magnitude of genetic, shared environmental, and nonshared environmental effects on variation in sedentary behavior changes as a function of age. Figure 5.3 presents the unstandardized (presented at the left side) and standardized (presented at the right side) contributions of genetic, shared environmental, and nonshared environmental effects to variation in sedentary behavior as a function of age and sex. It can be seen that the contribution of shared environmental effects to variation in sedentary behavior diminished with increasing age.

Table 5.3
Genetic model fitting results for sedentary behavior

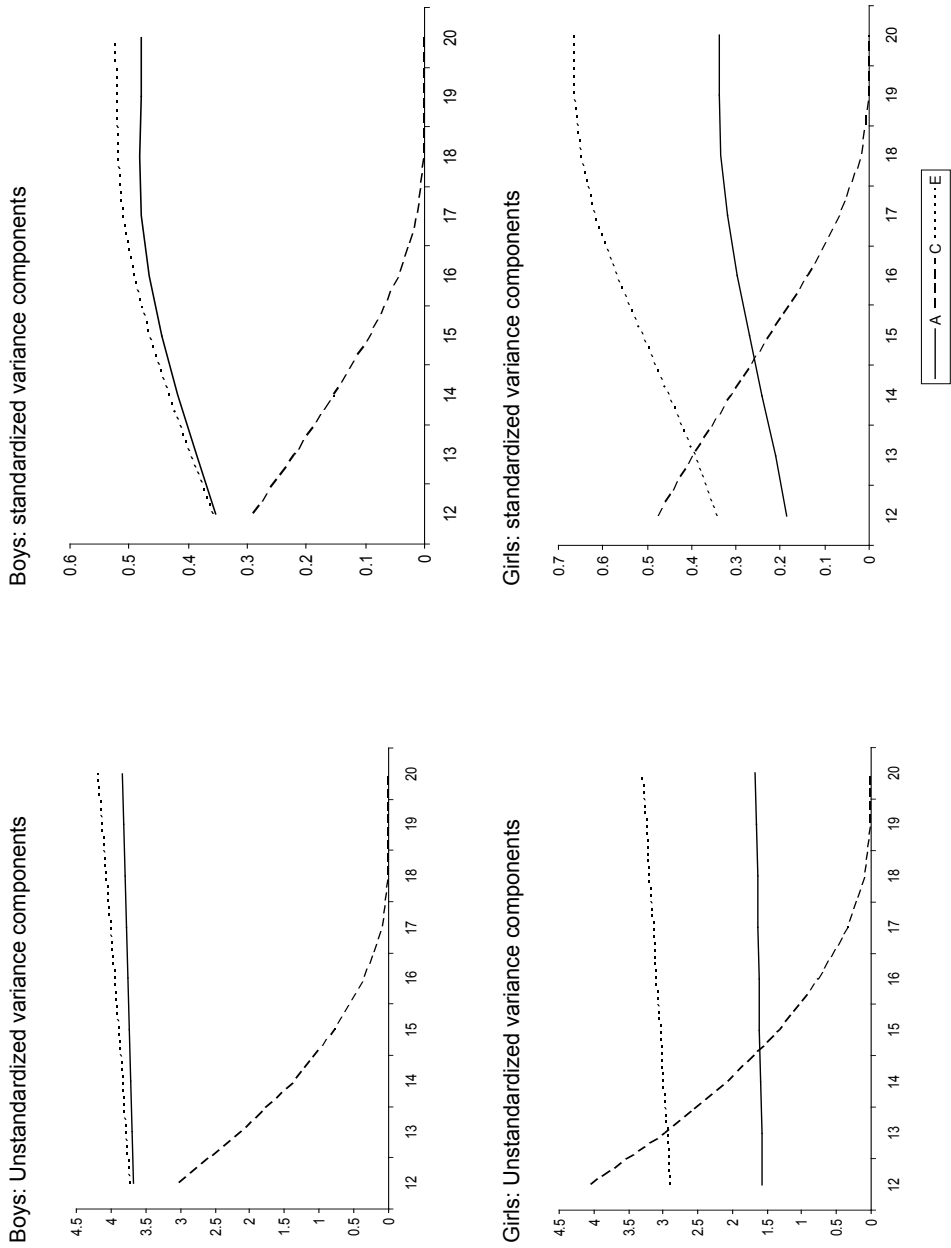
Model	vs	-2LL	df	χ^2	Δdf	p	AIC
1. ACE model	--	27977.339	6007	--	--	--	--
2. r_g DOS = 0.5	1	27977.981	6008	.64	1	.42	-1.36
3. $\alpha_{\text{boys}} = \alpha_{\text{girls}}, \beta_{\text{boys}} = \beta_{\text{girls}},$ $\eta_{\text{boys}} = \eta_{\text{girls}}$	2	27982.151	6011	4.17	3	.24	-3.19
4. $\alpha_{\text{boys}} = \alpha_{\text{girls}} = 0,$ $\beta_{\text{boys}} = \beta_{\text{girls}} = 0,$ $\eta_{\text{boys}} = \eta_{\text{girls}} = 0$	3	28019.761	6014	37.61	3	< .05	28.42
5. $a_{\text{boys}} = a_{\text{girls}}, c_{\text{boys}} = c_{\text{girls}},$ $e_{\text{boys}} = e_{\text{girls}}$	3	28091.621	6014	109.47	3	< .05	100.28
6. $a_{\text{boys}} = 0$	3	28006.402	6012	24.25	1	< .05	19.06
7. $a_{\text{girls}} = 0$	3	28004.646	6012	22.50	1	< .05	17.31
8. $c_{\text{boys}} = 0$	3	27987.018	6012	4.87	1	< .05	-0.32
9. $c_{\text{girls}} = 0$	3	27990.879	6012	8.73	1	< .05	3.54

Note. Most parsimonious model is printed in boldface type; ACE model = ACE model with moderating effects of age, different parameter estimates for boys and girls and r_g for DOS twins estimated; vs = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; Δdf = degrees of freedom of χ^2 test; p = p-value; AIC = Akaike's Information Criterion; r_g DOS = genetic correlation between DOS twins.

The absolute contribution of genetic and nonshared environmental effects to variation in sedentary behavior was similar with increasing age, whereas the relative contribution of genetic and nonshared environmental effects showed substantial increase. This is due to the diminishing part of variation accounted for by shared environmental factors with increasing age, leading to an overall decreased variation in sedentary behavior.

Model 5 tested whether constraining the genetic, shared environmental, and nonshared environmental parameter estimate to be equal for boys and girls led to a significant deterioration of model fit. There appeared to be significant differences in the magnitude of the variance components explaining individual differences in sedentary behavior. Therefore, parameter estimates were allowed to differ between boys and girls.

Figure 5.3. Changes in the absolute and relative contribution of genetic, shared environmental, and nonshared environmental effects to variation in sedentary behavior as a function of age for boys and girls.



Model 6 through 9, tested whether constraining the genetic or shared environmental parameter estimate to zero would lead to a significant deterioration of model fit. Additive genetic and shared environmental effects on individual differences in sedentary behavior were statistically significant for boys and girls. The LRT tests and the AIC pointed to the ACE model with significant age effects on the path coefficients as the most parsimonious models for boys and girls.

Table 5.4

Proportions of variance explained by additive genetic, shared environmental, and nonshared environmental factors for sedentary behavior at mean age (15.93 years) in the sample as a function of sex (95% confidence intervals added in parentheses)

	A	C	E
Boys	.47 (.35 - .57)	.05 (.001 - .12)	.48 (.43 - .55)
Girls	.30 (.16 - .42)	.14 (.04 - .26)	.56 (.52 - .61)

The proportions of variance in sedentary behavior explained by A, C, and E are summarized in Table 5.4. These are based on the mean age in the sample (i.e. z-score of age = 0). The best fitting models for boys and girls indicated that the proportions of variation in sedentary behavior explained by genetic factors were .47 and .30 respectively. Shared environmental effects also accounted for individual differences in sedentary behavior, .05 and .14 for boys and girls respectively. The remaining variation was accounted for by nonshared environmental factors.

Discussion

In a large sample of Dutch adolescent twins and their non-twin siblings, we found that adolescents become less frequently engaged in sedentary behaviors (TV viewing, electronic games, internet activities) throughout adolescence and that boys were more often sedentary than girls. Variation in sedentary behavior was accounted for by genetic, shared environmental, and nonshared environmental factors. Heritability of sedentary behavior was larger in boys compared to girls. No evidence was found for qualitative sex differences in the genetic factors, indicating that the same genetic variants appear to influence sedentary behavior in boys and girls. In addition, we found that the genetic architecture changed throughout adolescence. For boys and girls, shared environmental effects on sedentary behavior were important during early adolescence but diminished during middle and late adolescence in favor of genetic and nonshared environmental effects.

Our finding that the frequency of sedentary activities decreased during adolescence corresponds with the results of other studies (Barr-Anderson et al., 2009; Van den Bulck & Van Mierlo, 2004). For example, Barr-Anderson et al. (2009) reported that the prevalence of limited television use (less than 2 hours a day) increased while there was a parallel decrease in the prevalence of heavy television use (more than 5 hours a day) from early to late adolescence. We also observed (data not presented) that the overall decline in frequency of sedentary behavior during adolescence was due to a lower frequency of TV viewing. In addition, frequency of gaming also decreased, while the frequency of engagement in personal computer and internet activities did not change during adolescence. An explanation for the overall decreasing levels of sedentary behavior may be that other activities, such as study, jobs and going out increasingly compete with these sedentary activities. Sex differences in frequency of sedentary behavior were mainly due to a higher frequency of playing electronic games for boys (data not presented). Frequency of watching TV and engaging in personal computer and internet activities were similar for boys and girls.

The main aim of the present study was to assess to what extent genetic and environmental factors affect sedentary behavior during adolescence. Age significantly modified the genetic architecture of sedentary behavior. Shared environmental factors accounted for a major part of individual differences in sedentary behavior during young adolescence, whereas the influence of shared environmental factors decreased rapidly throughout adolescence to be completely disappeared during late adolescence. It is important to note that the relative increased contribution of genetic and nonshared environmental effects to individual differences in sedentary behavior during adolescence is not due to an increase in genetic and nonshared environmental variation, since the absolute contribution of genetic and nonshared environmental effects was similar during adolescence. The relative increased importance of genetic and nonshared environmental effects on sedentary behavior was due to the diminishing contribution of shared environmental effects to variation in sedentary behavior, leading to an overall decrease in the variation in sedentary behavior with increasing age.

The substantial influence of genetic factors on sedentary behavior in adolescence might have important implications for intervention strategies aiming at reduction of screen-time activities. For adolescents, sedentary activities such as TV viewing and engagement in personal computer and internet activities are frequent leisure time activities (e.g. Swinburn & Shelly, 2008; Van den Eijnden et al., 2008). The substantial genetic influence on these sedentary behaviors suggests that there is a genetic liability towards such sedentary activities, which might complicate prevention and intervention strategies aiming to reduce sedentariness. It may be that interventions aiming to reduce

the availability and accessibility of screen time opportunities, i.e. interventions that may restrict opportunities to act according to a genetically defined ‘preference’, may have better perspectives than health education-like interventions trying to educate and convince youngsters to change their behavior. Earlier studies indicated that adolescents with easier access to screen time opportunities, e.g. having a TV in the bedroom, are more likely to engage in screen time activities. However, restricting certain sedentary activities, particularly in late adolescence may not be feasible. In addition, it may not make adolescents give up sedentary activities but rather may result in compensatory forms of sedentary activities. Van den Bulck and Van den Bergh (2000) for example, showed that parental attempts to restrict media use in their children often led to an increase in the use of other media. Interventions focusing on offering alternative activities and promoting regular interruptions of sedentary behavior may have more potential. Preliminary research indicates that prolonged duration, but not interrupted duration of sedentary behavior is most unfavorable for metabolic disorders (Healy et al., 2008).

Our results indicate that as adolescents grow older and become more independent, adolescents’ sedentary behavior increasingly depends on their genetic make-up and factors from their personal environment. Factors from an adolescents’ personal environment may include activities, such as study, jobs, and going out. In other words, changes in the social and economical environments of adolescents may cause some adolescents to spend less time on sedentary activities, whereas for other adolescents sedentary activities remain dominant leisure time activities.

Shared environmental factors accounted for a substantial part of individual differences in the sedentary behaviors of the younger adolescents. Shared environmental factors may include the influence of parents’ sedentary behavior and parental monitoring of their children’s sedentary behavior by setting rules and restrictions regarding sedentary activities. Such factors have already been shown to be associated with sedentary behavior in youth (Van Zutphen et al., 2007; Salmon et al., 2005). It is important to note, however, that genetic factors may affect these influences from adolescents’ family environment as well (i.e. gene-environment correlation; Kendler & Baker, 2007; Plomin & Bergeman, 1991). Parents with a genetic liability towards sedentariness may also be more likely to create a family environment in which sedentary behavior is promoted rather than restricted, by e.g. allowing a TV in their children’s bedrooms and having multiple personal computers in the household. In addition, these parents may also set few rules and restrictions regarding sedentary activities resulting in undisturbed engagement in such activities by their children. Given the importance of shared environmental effects on sedentary behavior, especially during young adolescence, supports prevention and intervention strategies to target on families rather than on the individual adolescent.

The genetic architecture of adolescent sedentary behavior has been addressed in one previous study. Our findings are consistent with those reported by Nelson et al. (2006). They reported genetic influence on variation in sedentary behavior and reported a heritability estimate of 34%. They also reported shared environmental factors (10%) to account for a small part of the variation in sedentary behavior. In the present study, however, sex differences were examined extensively. Nelson et al. (2006) also assessed changes in the genetic architecture of sedentary behavior during the transition from adolescence to young adulthood. They reported shared environmental effects on sedentary behavior to disappear with increasing age, which is in line with findings from the present study. They also reported nonshared environmental effects to be relatively more important during young adulthood compared to adolescence, whereas genetic effects were relatively less important. This may suggest nonshared environmental factors to become increasingly important at the expense of genetic factors during the transition from adolescence to adulthood.

A limitation of the present study was the use of a cross-sectional twin – sibling design to examine the relative influence of genetic and environmental influences on individual differences in sedentary behavior. The genetic architecture of sedentary behavior during adolescence is most properly addressed in a longitudinal design. So far data at two time point are only available for a small subsample, and data throughout adolescence (12 – 19) are absent. Since our data collection is a continuous process at the NTR we anticipate large enough longitudinal sample size within the next 5 years.

Conclusion

Our data showed that variation in sedentary behavior was largely accounted for by genetic and nonshared environmental factors during adolescence, whereas shared environmental factors account for a substantial part of the variation during young adolescence. The shift from shared environmental factors in the etiology of sedentary behavior in early adolescence to genetic and nonshared environmental factors in late adolescence requires tailoring and targeting of interventions to age groups.

Acknowledgements.

Funding was obtained from the following grant: “Genes and environment in obesogenic behaviour: Exploring the relative importance of environmental and genetic factors on sedentary behaviour and sleep duration during adolescence – a study in adolescent twins”

(WCRF); “Twin-family database for behavior genetics and genomics studies” (NWO 480-04-004); M. Bartels is financially supported by a senior fellowship of the EMGO+ institute for health and care.

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6

Daily and Compulsive Internet Use and Wellbeing in Adolescence: A Diathesis-Stress Model Based on Big Five Personality Traits

This chapter is published as:

Van der Aa, N., Overbeek, G., Engels, R.C.M.E., Scholte, R.H.J., Meerkerk, G.J., & Van den Eijnden, R.J.J.M. (2009). Daily and compulsive internet use and well-being in adolescence: a diathesis-stress model based on big five personality traits. *Journal of Youth and Adolescence*, 38, 765-776.

Abstract

This study examined the associations between adolescents' daily Internet use and low well-being (i.e., loneliness, low self-esteem, and depressive moods). We hypothesized that (a) linkages between high levels of daily Internet use and low well-being would be mediated by compulsive Internet use (CIU), and (b) that adolescents with low levels of agreeableness and emotional stability, and high levels of introversion would be more likely to develop CIU and lower well-being. Data were used from a sample of 7,888 Dutch adolescents (11-21 years). Results from structural equation modeling analyses showed that daily Internet use was indirectly related to low well-being through CIU. In addition, daily Internet use was found to be more strongly related to CIU in introverted, low-agreeable, and emotionally less-stable adolescents. In turn, again, CIU was more strongly linked to loneliness in introverted, emotionally less-stable, and less agreeable adolescents.

Introduction

During the past few decades, the Internet has become increasingly important in adolescents' lives. According to recent Dutch research, 99% of Dutch adolescents are actively using the Internet and 97% has Internet access at home (Van den Eijnden et al., 2008). The most popular Internet function among Dutch and American adolescents is online communication (Kraut et al., 2002; Valkenburg & Peter, 2007; Subrahmanyam et al., 2001). Recently, among scholars and in the popular media a heated debate has unfolded about the impact of daily Internet use - especially online communication - on adolescents' well-being. In accordance, previous research on the linkages between adolescents' daily Internet use and well-being has provided contrasting findings (Caplan, 2003). While some studies found that daily Internet use was associated with lower well-being (e.g., Kraut et al., 1998; Ybarra et al., 2005) other studies found no evidence for this contradicting evidence (e.g., Gross et al., 2002; Kraut et al., 2002). In this paper, we aim to overcome this state of affairs by using a diathesis-stress perspective on the relationship between daily Internet use and low well-being, that (a) accounts for a progression from 'normal' to compulsive Internet use, and (b) accounts for individual vulnerabilities in Internet use based on Big Five personality traits.

Daily Internet Use and Low Well-Being

In a one-year longitudinal study, Kraut et al. (1998) found that people who spent more time on the Internet subsequently developed higher levels of depression and loneliness. However, in a follow-up study among participants from the same sample, Kraut et al. (2002) found that the association between daily Internet use and loneliness and depression disappeared. Moreover, they found that for extraverted individuals daily Internet use was positively associated with well-being, whereas negative relationships were found for introverted individuals. In a two-wave longitudinal study with a six-month time interval, Van den Eijnden et al. (2008) found a positive relationship between daily Internet use and subsequent depressive symptomatology. No relationship was found between participants' daily Internet use and loneliness. These findings are supported by several cross-sectional studies. For instance, Ybarra et al. (2005) found a positive relationship between daily Internet use and feelings of depression. In addition, Weiser (2001) found a negative association between daily Internet use and general well-being when the participants' Internet use had a 'social orientation'. In contrast, however, several studies found no relationship between daily Internet use and well-being (Gross et al., 2002; Sanders et al., 2000; Wastlund et al., 2001). Overall, the results with regard to the direct associations

between daily Internet use and well-being may be classified as inconclusive. In addition, whenever relationships were found they were of a rather small magnitude (typically with betas ranging from $-.10$ to $-.19$). Like Davis (2001), we suggest that daily Internet use is inappropriate to identify adolescents' Internet use as problematic. Rather, individual characteristics will affect whether or not individuals use the Internet in a maladaptive way, and develop low well-being as a consequence.

Diathesis-Stress: From Daily to Compulsive Internet Use

This paper adopts Davis' (2001) cognitive-behavioral model of pathological Internet use, proposing that compulsive Internet use (CIU) is the result of problematic cognitive or behavioral patterns that evolve around daily Internet use, which lead to several behaviors that intensify or maintain the maladaptive responses. According to Meerkerk et al. (2009), CIU can be generally defined as the inability to control online activity and is characterized by five main features: (1) continuation of Internet use despite the intention to stop, (2) Internet use dominating the adolescent's cognitions and behaviors, (3) the experience of unpleasant emotions when Internet use is impossible, (4) using the Internet to escape from negative feelings, and (5) Internet use is resulting in conflict with others or with oneself. According to Davis (2001), both daily Internet use and CIU are related to people's social contexts. That is, a lack of social support from one's family members or peers, and/or experiencing social isolation in the peer context may facilitate the development from daily Internet use to CIU, because the social contacts and reinforcement which are obtained on the Internet may result in an increased desire to maintain a 'virtual' social life. When adolescents would develop CIU, however, they would have little time and energy left to actively maintain social contacts in daily life, and this would increase their risk for the onset of feelings of loneliness, depressive moods, and low self-esteem. Thus, we hypothesize that for some subgroups of adolescents there would be a developmental progression from daily Internet use to CIU, which eventually would lead to the development of low well-being or mental health problems in adolescents.

To our knowledge, no previous study has examined the hypothesis that CIU mediates the relationship between daily Internet use and well-being. Nevertheless, preliminary evidence comes from a study by Caplan (2003) and by another from Van den Eijnden et al. (2008), who found that CIU was a much stronger predictor of negative outcomes (i.e., on both professional and well-being domains) than daily Internet use as such. Furthermore, some studies reported a strong association between CIU and low well-being (Meerkerk et al., submitted for publication; Morahan-Martin & Schumacher, 2000). Typically, adolescents who used the Internet in a compulsive way reported to be more

lonely and more depressed than their peers who reported less compulsive Internet use behaviors. In addition, several studies report a positive relationship between daily Internet use and CIU (Van den Eijnden et al., 2008; Morahan-Martin & Schumacher, 2000; Caplan, 2003). On the basis of the results reviewed above, one may put forward that CIU mediates the relationship between daily Internet use and low well-being.

Diathesis-Stress: Personality-Based Vulnerabilities

In line with McKenna and Bargh (1998), we put forward that the Internet does not affect all adolescents in exactly the same way. More specifically, youths differ in what goals they have or what needs they want to be met on the Internet. For example, for socially marginalized or vulnerable adolescents it would be relatively difficult to develop social relations in their daily lives, thereby being particularly vulnerable to develop more intense or even compulsive levels of Internet use by being focused on maintaining virtual social relations in chatrooms or through instant messengers. But who are the ‘socially vulnerable’ adolescents? According to Kraut et al. (2002), people who differ in certain personality traits are likely to use the Internet in different ways. In the present study, personality is defined as a person’s tendency to behave in a certain way across different situations over longer periods of time (Gazzinga & Heatherton, 2003). According to the Big Five description of personality (Goldberg, 1992), five broad dimensions of personality exist: emotional stability, extraversion, openness, agreeableness, and conscientiousness. McKenna and Bargh (1998) identify introversion, emotional instability, and low agreeableness as personality markers of a vulnerability for developing CIU. Introverted adolescents can be described as non-outgoing, silent, and reserved, emotionally unstable adolescents as anxious, tense, and unstable, and adolescents low in agreeableness as antagonistic, unkind, and dishonest (Goldberg, 1990; McCrae & John, 1992). In accordance, adolescents low in emotional stability, extraversion, and agreeableness are more likely to have relatively low competence in the interpersonal domain, and therefore have fewer social resources in their daily lives (Caplan, 2003). Thus, these adolescents can also be expected to use the Internet to avoid being alone and turn to people online who are disconnected from their daily life (Gross et al., 2002; Caplan, 2003; McKenna & Bargh, 1998). These adolescents are more likely to lose control over their Internet use than other adolescents, because an important part of their social lives is on the Internet, whereas in their daily life they may feel socially isolated (Davis, 2001). The personality traits of conscientiousness and openness are thought to affect adolescents’ (compulsive) Internet use to a lesser extent than the other personality traits, since these traits are generally not strongly associated with individuals’

social behavior (McCrae & John, 1992). Individuals high in conscientiousness can be described as orderly, organized, and self-disciplined, and individuals high in openness as curious, insightful, and original (Goldberg, 1990; McCrae & John, 1992).

The Present Study

We used data from a large-scale sample of 7,888 Dutch adolescents and young adults aged 11-21, in order to test two hypotheses that were drawn from a diathesis-stress perspective on the linkages between daily Internet use and well-being. The first hypothesis that was examined was that CIU would mediate the relationship between adolescents' daily Internet use and low well-being (operationalized in terms of loneliness, low self-esteem, and depressive moods). The second hypothesis was that adolescents' extraversion, agreeableness, and emotional stability would moderate the links between daily Internet use and CIU, and between CIU and low well-being. More specifically, we tested the assumption that for adolescents low in emotional stability, extraversion, and agreeableness these associations would be of greater strength than for their peers who reported high levels of these personality traits. Adolescents' openness and conscientiousness were thought not to moderate the links between daily Internet use and CIU, and between CIU and low well-being.

Methods

Procedure

The present study was conducted in The Netherlands. An online questionnaire in the Dutch language, which took about 20 minutes to fill out, was put on the website of the Addiction Research Institute (IVO) by the end of 2003. Participants were recruited by advertisements in daily newspapers, on the radio, and on the television. Participants who completed the online questionnaire received feedback about the extent of compulsivity of their Internet use (CIU) when they had completed the questionnaire. About 17,500 respondents had filled out the questionnaire by the end of 2004. Considerable effort was put into cleaning the data - see also Birnbaum (2004) and Wood et al. (2004) for a discussion on the quality of web-based data collection. Respondents who did not completely finish the questionnaire or provided obviously erroneous answers were removed from the database. When respondents provided identical answers on all items

of the different measures of the questionnaire, their answering pattern was thoroughly scrutinized and, in case of doubt, removed from the database. After this procedure, a total of 16,925 respondents were retained in the sample.

Subjects

As we were interested in explaining the associations between Internet use and low well-being during adolescence, we selected 7,888 adolescents aged 11 to 21 years out of the total sample. This subsample consisted of 6,081 boys (77.1%) and 1,807 girls (22.9%). With regard to age, 653 respondents (8.3%) could be classified as early adolescents (11-14 years), 1,603 respondents (20.3%) as middle adolescents (15-16 years), 2,356 respondents (29.9%) as late adolescents (17-18 years), and 3,276 respondents (41.5%) as young adults (19-21 years). Mean age was 17.79 years ($SD = 2.22$ years). Most respondents (64.0%) were living with their parents. A total of 3,284 respondents (41.6%) were high-school students (including students in lower vocational education, higher-level secondary education, and preparatory scientific education programs), while 3,698 respondents (46.9%) were enrolled in either university or higher-level vocational education, 734 respondents (9.3%) were currently working, and 172 (2.2%) were unemployed.

Measures

Daily Internet use. We defined daily Internet use as the amount of time adolescents spent on the Internet on a typical day when they are online. Accordingly, participants were asked to indicate how much time they spent on the Internet on a daily basis (on average) by answering two items. The first item pertained to the amount of time one spent on average on the Internet on a typical day when online. The answer could be provided on an 8-point scale ranging from 1 = 'less than one hour' to 8 = 'seven hours or more'. Second, participants were asked to report if and how often they spent five hours or more on the Internet on a single day in the last three months. The answer could be provided on a 7-point scale ranging from 1 = 'no, not a single time in the past three months' to 7 = 'yes, every day'.

Compulsive Internet use. The Compulsive Internet Use Scale (Meerkerk et al., 2009) was used to assess the extent to which respondents were making use of the Internet in a compulsive manner. The CIUS consists of 17 items which had to be answered on a 5-point Likert scale ranging from 1 = 'never' to 5 = 'very often'. In Table 6.1 the mean scores and standard deviations of the scale are presented. The scale taps into the dimensions of loss of control, preoccupation, withdrawal symptoms, coping, and conflicts

about Internet use. Examples of items are ‘How often do you find it difficult to stop using the Internet?’ and ‘How often do you think you should use the Internet less often?’. The internal consistency of the scale (Chronbach’s alpha) was .90. Scores could range from 17 to 85 with high scores indicating high levels of compulsive Internet use.

Table 6.1

Mean Scores and Standard Deviations of the CIUS-Scale

Item	M	SD
Difficult to stop using the Internet	2.82	1.11
Continue to use the Internet despite intention to stop	3.10	1.12
Others say you should use the Internet less	2.65	1.29
Prefer to use the Internet instead of spending time with others	2.30	1.08
Short of sleep because of the Internet	2.28	1.22
Thinking about the Internet, even while not online	2.34	1.07
Looking forward to the next Internet session	2.19	1.12
Think you should use the Internet less often	2.35	1.13
Unsuccessfully tried to spend less time on the Internet	1.75	.98
Feel depressed or irritated when can not use the Internet	1.88	1.07
Physical complaints because of using the Internet	1.74	.92
Rush through (home)work in order to go on the Internet	2.34	1.19
Neglect daily obligations because prefer to go on the Internet	1.95	1.05
Using the Internet on the sly	1.46	.87
Hiding the time spent on the Internet for other people	1.51	.89
Go on the Internet when feeling down	2.33	1.20
Use the Internet to escape sorrows or negative feelings	1.93	1.13

Loneliness. To assess respondents’ perceptions of loneliness the revised UCLA loneliness scale was used (Russell et al., 1980). The UCLA Loneliness Scale consists of 10 items which can be answered on a 5-point Likert scale ranging from 1 = ‘totally disagree’ to 5 = ‘totally agree’. Examples of items are ‘I am feeling alone’ and ‘I do not have real friends’. Cronbach’s alpha was .87. Scores could range from 10 to 50 with higher scores indicating higher levels of loneliness.

Self-esteem. Self-esteem was assessed with the Rosenberg self-esteem scale (Rosenberg, 1989) consisting of 10 items with response categories ranging from 1 = ‘strongly disagree’ to 4 = ‘strongly agree’. Examples of items are ‘I take a positive attitude

toward myself' and 'In general I am content with myself'. The internal consistency was .88. Scores could range from 10 to 40 with higher scores indicating higher levels of self-esteem.

Depressive mood. To assess depressive moods the Depressive Mood List was used (Kandel & Davies, 1986). The items were answered on a 5-point Likert scale ranging from 1 = 'never' to 5 = 'always'. Examples of items are 'Feeling too tired to do something' and 'Feeling nervous and tense'. The internal consistency of the scale was .81. Scores could range from 5 to 30 with higher scores indicating higher levels of depressive moods.

Personality Traits. To assess respondents' personality traits the Quick Big Five (QBF; Vermulst & Gerris, 2005) was used. This is a shortened Dutch translation of Goldberg's original 100 item-adjective list (Goldberg, 1992), and assesses the 'Big Five' personality dimensions of extraversion, conscientiousness, agreeableness, emotional stability, and openness. The scale consisted of 30 adjectives (i.e., 6 items for each personality trait), that respondents could react to on a 7-point Likert scale ranging from 1 = 'completely incorrect' to 7 = 'completely correct'. Scores on each personality dimension could range from 6 to 42, with high scores indicating high levels of that personality dimension. Examples of items are 'reserved' (extraversion), 'careful' (conscientiousness), 'helpful' (agreeableness), 'nervous' (emotional stability), and 'creative' (openness). The internal consistency of the subscales extraversion (Cronbach's alpha = .88), conscientiousness (Cronbach's alpha = .86), agreeableness (Cronbach's alpha = .83), emotional stability (Cronbach's alpha = .82), and openness (Cronbach's alpha = .74) were adequate or high. The test-retest reliability of the scale was found to be acceptable (Vermulst & Gerris, 2005). Furthermore, the personality traits appeared to correlate with several criterium variables (e.g. depressive feelings, self-esteem and loneliness), which suggests that the validity of the scale is good (Vermulst & Gerris, 2005).

Statistical Analyses

First of all, with a series of one-way ANOVAs, we examined possible sex and age differences in adolescents' well-being, daily Internet use, and CIU. Next, we calculated Pearson correlations in order to examine the bivariate associations between the concepts in our study, and these correlational findings were subsequently used as input for structural equation modeling analyses (SEM), which we carried out using the LISREL 8.30 program (Jöreskog & Sörböm, 1993).

Covariance matrices were estimated using the default Maximum Likelihood estimation technique. We tested a mediation hypothesis by comparing the fit and path coefficients of two different models: one model in which three direct paths from daily

Internet use to low well-being (i.e., loneliness, low self-esteem, and depressive moods) were specified, and one other model in which daily Internet use was specified to predict CIU, which in turn was specified to predict low well-being. We tested whether a mediation model would fit the data better than a direct linkages model based on Sobel tests (Preacher & Hayes, 2004). Next, we conducted multigroup analyses to examine a moderation hypothesis - that introverted, low-agreeable, or emotionally less-stable adolescents would be more likely to report higher CIU with higher levels of daily Internet use, and to report lower well-being with higher levels of CIU. These moderator effects were estimated using chi-square difference tests.

Model fit was examined with the Non-Normed Fit Index (NNFI), and Root Mean Square Error of Approximation (RMSEA). The NNFI index is usually considered to show adequate fit when attaining values of .95 or higher, and the RMSEA is taken to reflect a good fit with values of .05 or lower (Browne & Cudeck, 1993). The Non-Normed Fit Index is a measure comparing the χ^2/df ratio of the proposed model versus that of a 'null model' in which all covariances are set to zero.

Table 6.2

Factor Loadings (Lambdas) and Number of Items in Indicator Variables (Total Sample-Model)

Indicator Variables	Number of Items	Standardized Lambda-values
Frequency > 5 Hours/Day - Internet use	1	.85
Average Number of Hours - Internet Use	1	.93
Compulsive Internet Use _a	4	.80
Compulsive Internet Use _b	4	.83
Compulsive Internet Use _c	4	.82
Compulsive Internet Use _d	5	.77
Loneliness _a	3	.85
Loneliness _b	3	.81
Loneliness _c	4	.86
Self-Esteem _a	3	.86
Self-Esteem _b	3	.84
Self-Esteem _c	4	.88
Depressive Mood _a	3	.77
Depressive Mood _b	3	.84

Note: ^a First parcel; ^b Second parcel; ^c Third parcel; ^d Fourth parcel

The NNFI is a measure of parsimony, accounting for the ‘complexity’ of a structural model. The Root Mean Square Error of Approximation (RMSEA) is another measure suitable for assessing the fit of a model per df used. Notably, in order to acquire multiple indicator variables for each latent variable, we employed a parcelling method (cf. Bandalos & Finney, 2001). The parcels were formed by ‘splitting’ one specific measure into two, three, or four multiple-item indicators that have an adequate Cronbach’s alpha, ranging from .63 to .78. Table 6.2 shows the measurement model of our total sample LISREL analysis. In constructing the parcels for a specific latent variable, we examined whether they all loaded on one underlying factor. The factor loadings attained values of .77 and higher, indicating that the latent variables in the structural model were reliably estimated by the parcels we used. In addition, we examined the correlations among the parcels. All correlations reached values of $r \geq .38$, indicating that all parcels were positively associated to a moderately or strong degree with each other.

Because the sample size in the present study was very large we had very high levels of statistical power for our statistical analyses. In order to reduce the chance at type one and type two errors, we performed a statistical power analysis (cf. Cohen, 1977). Before performing the analysis statistical power was 100%. The results of the statistical power analysis justified statistical testing at a $p \leq .001$ significance level, with statistical power still reaching a level of 100%. In this way, we avoided very small effects to reach statistical significance.

Results

Table 6.3 shows the results of a first set of one-way ANOVAs, indicating that adolescents spent between two and three hours a day on the Internet, and spent five hours or more per day on the Internet once or twice a week. Clear sex differences were observed with regard to low well-being and daily Internet use. More specifically, boys reported higher self-esteem [$F(1,7886) = 263.84, p < .001$] and less depressive moods [$F(1,7886) = 207.15, p < .001$], and reported higher levels of daily Internet use than girls [$F(1,7885) = 126.95, p < .001$] as well as a higher level of being online for five hours or more per day [$F(1,7885) = 173.35, p < .001$]. In contrast, however, girls indicated to have higher levels of compulsive Internet use than boys [$F(1,7886) = 50.17, p < .001$]. Several age differences also emerged from the ANOVAs, with older adolescents reporting higher levels of loneliness [$F(3,7884) = 9.24, p < .001$], and lower levels of self-esteem [$F(3,7884) = 8.82, p < .001$]. Moreover, a strong age difference was found with regard to CIU [$F(3,7884) = 80.91, p < .001$] - younger adolescents reported higher levels of CIU than older adolescents.

Table 6.3

Mean Levels and Standard Deviations of Well-being, Internet Use, CIU, and Personality: ANOVA for Gender and Age

	Gender			Age			
	Total (N=7888)	Boys (N=6081)	Girls (N=1807)	11-14 (N=653)	15-16 (N=1603)	17-18 (N=2356)	19-21 (N=3276)
Loneliness (10-50)	17.75 (6.33)	17.73 (6.26)	17.83 (6.55)	17.24 (6.52) ^d	17.26 (6.36) ^d	17.67 (6.16) ^d	18.16 (6.38) ^{a,b,c}
Self esteem (10-40)	32.28 (5.77)	32.84 (5.47)	30.37 (6.32) [*]	31.32 (6.35) ^{b,c,d}	32.06 (5.93) ^a	32.37 (5.57) ^a	32.51 (5.68) ^a
Depression (6-30)	13.89 (4.51)	13.49 (4.40)	15.21 (4.61) [*]	13.59 (4.75)	13.74 (4.64)	13.83 (4.46)	14.06 (4.42)
Number of hours (1-8)	3.64 (1.82)	3.76 (1.81)	3.22 (1.79) [*]	3.63 (1.92)	3.64 (1.85)	3.64 (1.79)	3.63 (1.80)
Freq. 5 hours a day (1-7)	3.95 (1.81)	4.10 (1.77)	3.47 (1.86) [*]	3.94 (1.99)	3.96 (1.87)	3.98 (1.79)	3.93 (1.75)
CIU (14-70)	32.30 (10.24)	31.86 (9.90)	33.80 (11.18) [*]	35.98 (11.14) ^{b,c,d}	34.29 (10.79) ^{a,c,d}	32.30 (10.02) ^{a,b,d}	30.60 (9.55) ^{a,b,c}
Extraversion (6-42)	26.97 (7.85)	26.78 (7.77)	27.62 (8.10) [*]	28.81 (7.98) ^{b,c,d}	27.82 (7.64) ^{a,c,d}	26.95 (7.73) ^{a,b,d}	26.21 (7.92) ^{a,b,c}
Agreeableness (6-42)	33.18 (4.90)	33.08 (4.90)	33.51 (4.86) [*]	32.70 (5.63) ^d	32.77 (5.10) ^d	32.98 (4.82) ^d	33.61 (4.65) ^{a,b,c}
Conscientiousness (6-42)	25.22 (7.27)	25.21 (7.29)	25.27 (7.23)	23.65 (7.33) ^{b,c,d}	24.78 (7.30) ^{a,d}	24.94 (7.25) ^{a,d}	25.95 (7.18) ^{a,b,c}
Emotional Stability (6-42)	26.93 (6.82)	27.58 (6.62)	24.74 (7.05) [*]	26.79 (7.22)	27.03 (6.88)	26.91 (6.69)	26.93 (6.81)
Openness (6-42)	29.55 (5.74)	29.59 (5.70)	29.40 (5.88)	28.85 (6.19) ^d	28.99 (5.85) ^d	29.46 (5.69) ^d	30.03 (5.59) ^{a,b,c}

Note. Numbers between parentheses indicate the minimum and maximum of a scale; Superscript letters refer to significant age differences as observed with Bonferroni post-hoc tests; Number of hours = number of hours an adolescent is on the Internet a day; Freq. 5 hours a day = frequency of more than 5 hours a day on the Internet; CIU = compulsive Internet use

^a Significant age difference with age group 11-14 year olds ($p < .001$)

^b Significant age difference with age group 15-16 year olds ($p < .001$)

^c Significant age difference with age group 17-18 year olds ($p < .001$)

^d Significant age difference with age group 19-21 year olds ($p < .001$)

* $p < .001$

Table 6.4
 Pearson Correlations Between Adolescents' Well-Being, Daily Internet Use, CIU, and Personality Traits (n = 7,887)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
(1) Loneliness	--										
(2) Low Self-esteem	.58*	--									
(3) Depressive Moods	.50*	.60*	--								
(4) No. Hours	.11*	.09*	.10*	--							
(5) Freq. > 5 Hours/Day	.12*	.11*	.12*	.77*	--						
(6) CIU	.34*	.35*	.46*	.37*	.40*	--					
(7) Extraversion	-.59*	-.47*	-.40*	-.10*	-.11*	-.22*	--				
(8) Agreeableness	-.50*	-.42*	-.30*	-.09*	-.10*	-.26*	.37*	--			
(9) Emotional Stability	-.38*	-.50*	-.57*	-.04*	-.05*	-.29*	.47*	.20*	--		
(10) Conscientiousness	-.09*	-.19*	-.15*	-.08*	-.09*	-.17*	.00	.25*	.04*	--	
(11) Openness	-.26*	-.31*	-.15*	-.01	.00	-.12*	.25*	.46*	.08*	.20*	--

Note. No. Hours = Number of hours adolescents spend time on the Internet per day; Freq. > 5 Hours/Day = Frequency of more than 5 hours a day on the Internet; CIU = Compulsive Internet Use

* $p < .001$

Table 6.4 presents the Pearson correlations between adolescents' perceptions of their well-being, daily Internet use, CIU, and personality traits. Moderately strong bivariate associations were observed between CIU and loneliness, depressive moods, and low self-esteem (correlations ranging from .34 to .46, $p < .001$). These results suggest that adolescents with higher levels of CIU also report to feel more lonely and depressed and report lower self-esteem. In contrast, daily Internet use was only marginally (positively) associated with low well-being in adolescents. However, daily Internet use was more strongly linked to CIU, making clear that adolescents who spent more time on the Internet were also more compulsively using the Internet. CIU was found to correlate with lower levels of extraversion, agreeableness, emotional stability, conscientiousness, and openness in this sample of adolescents. Finally, loneliness, low self-esteem, and depressive moods were correlated with lower levels of extraversion, agreeableness, emotional stability, and to a lesser extent, with conscientiousness and openness.

Daily Internet Use and Well-Being: The Mediating Role of CIU

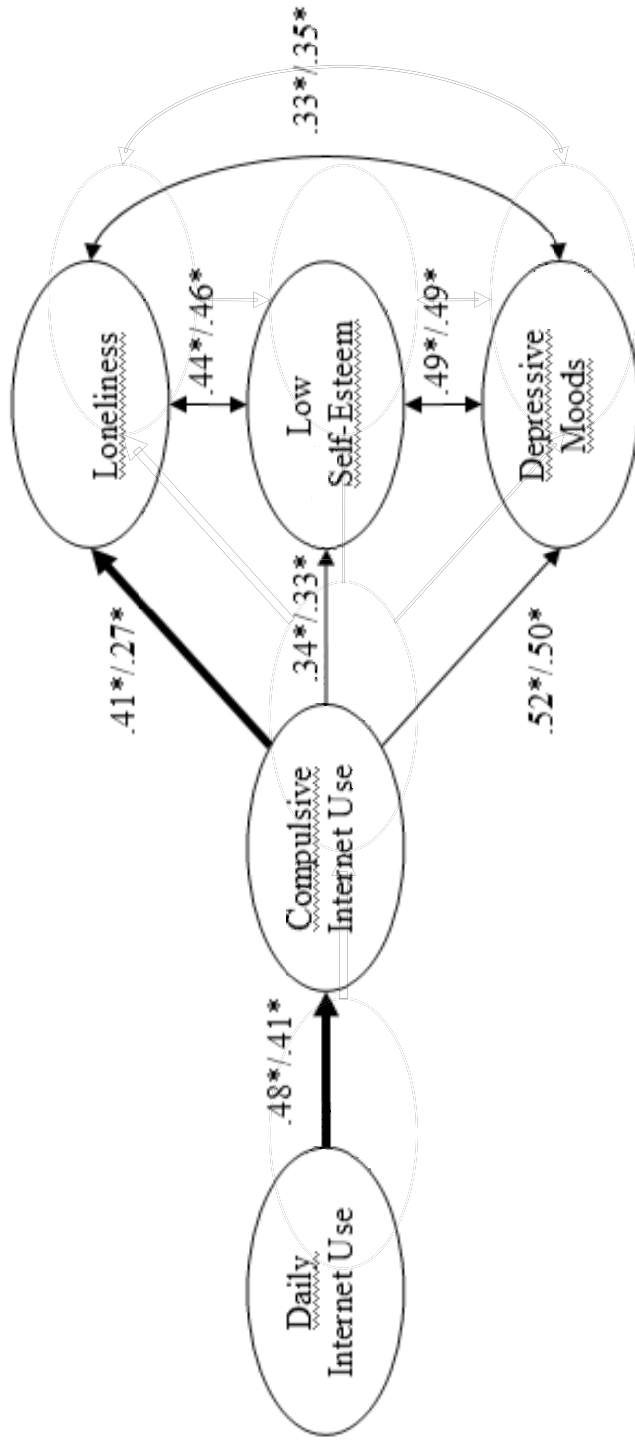
The results from the SEM analysis for the total sample clearly indicated that a mediation model, in which daily Internet use predicted low well-being through CIU, fit the data best. The path of daily Internet use to CIU was significant ($\beta = .46$, $p < .001$), as well as the other paths from CIU to loneliness ($\beta = .39$, $p < .001$), low self-esteem ($\beta = .38$, $p < .001$), and depressive moods ($\beta = .53$, $p < .001$). Fit indices indicated that the model had a good fit to the data: NNFI = .98, RMSEA = .05. Most importantly, CIU indeed mediated the relationship between daily Internet use and low well-being. The fit of the mediation model was far better in comparison with the direct linkages model, as was indicated by a great improvement in χ^2 -values [direct linkages: $\chi^2(67) = 3,089.45$; CIU mediation model: $\chi^2(70) = 1,585.50$] as indicated by a chi-square difference test [$\chi^2(3) = 1,503.95$, $p < .001$]. In the direct linkages model only direct linkages between daily Internet use and well-being, and between CIU and well-being were estimated. Accordingly, an examination of modification indices for the CIU mediation model did not indicate that any direct linkages from daily Internet use to low well-being would significantly improve the fit of the model. In addition, Sobel tests indicated that CIU mediated the relationship between daily Internet use and low well-being (z -scores ranged between 17.48 and 20.51, $p < .001$). Furthermore, we tested the mediation model for boys and girls separately. In both cases the mediation model, in which daily Internet use predicted low well-being through CIU, fit the data best.

The Moderating Role of Personality Traits

We performed LISREL multigroup analyses for adolescents who scored low versus high on the personality traits of extraversion, agreeableness, emotional stability, conscientiousness, and openness. For each of these traits, the distribution of scores was dichotomized by means of a median split. The structural models for the low versus high groups were compared in two different ways, so as to examine two different possible moderator effects. First of all, 'low' versus 'high' personality groups were compared by freeing the path from daily Internet use to CIU, after which the χ^2 for this model with one parameter freed was compared with the χ^2 of a fully restrained model. Secondly, low versus high personality groups were compared by successively freeing the paths from CIU to loneliness, low self-esteem, and depressive moods, following the same χ^2 comparison procedures. Figures 6.1, 6.2, and 6.3 demonstrate that although for all adolescents higher levels of daily Internet use were associated with elevated levels of CIU, the most introverted ($\Delta\chi^2(1) = 22.52$, $p < .001$), least emotionally stable ($\Delta\chi^2(1) = 37.54$, $p < .001$), and least agreeable adolescents ($\Delta\chi^2(1) = 17.44$, $p < .001$) reported higher levels of CIU when using the Internet intensively on a daily basis. No significant moderator effects of conscientiousness and openness were found. In addition, the multigroup tests made clear that whenever adolescents reported higher levels of CIU, it was the group of adolescents who were most introverted ($\Delta\chi^2(1) = 41.61$, $p < .001$), least emotionally stable ($\Delta\chi^2(1) = 18.01$, $p < .001$), and least agreeable adolescents ($\Delta\chi^2(1) = 27.45$, $p < .001$) that were reporting especially high levels of loneliness. Again, no significant effects emerged for the personality traits of conscientiousness and openness. Adolescents' personality traits were not found to moderate the relationship between CIU and low self-esteem or depressive moods.

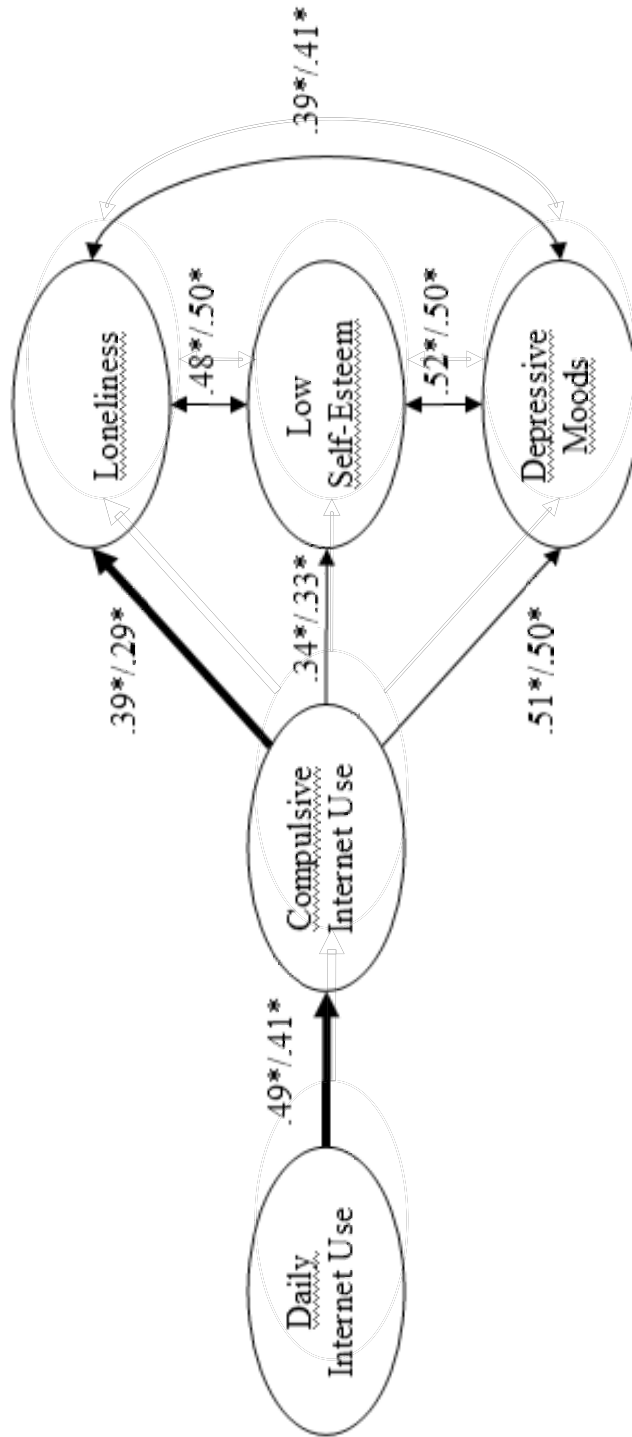
In order to examine whether this pattern of findings could be replicated in both sex subgroups, multigroup analyses for adolescents who scored low versus high on the personality traits were performed separately for boys and girls. For boys, the results were similar to the results of the total group adolescents as ascribed above. However, the results for girls were slightly different compared to the total group of adolescents. More specifically, the association between daily Internet use and CIU was not moderated by agreeableness and extraversion, and the association between CIU and loneliness was not moderated by emotional stability. Further, the multigroup analyses showed the same results for girls compared to the total group of adolescents.

Figure 6.1. Multigroup SEM analyses: Maximum Likelihood Parameters for Introverted versus Extraverted Adolescents



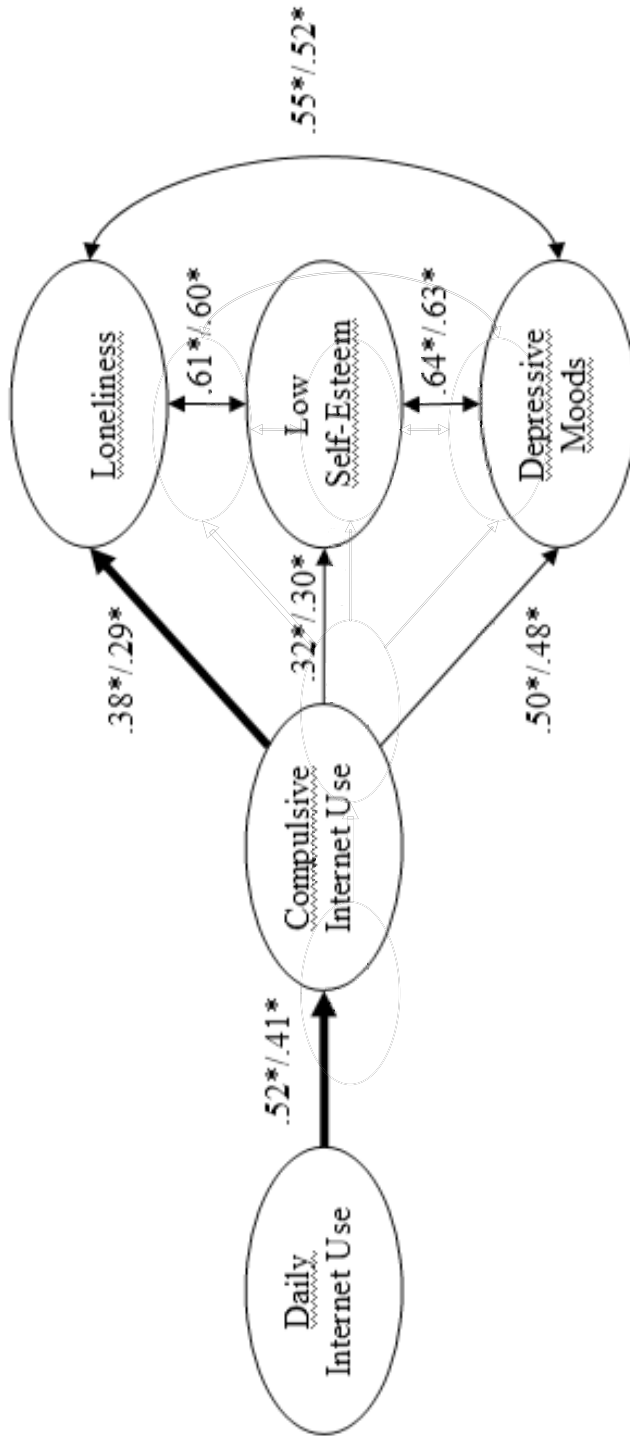
Note. Model fit Introverted Adolescents: NNFI = .92; Model fit Extraverted Adolescents: NNFI = .96. (* $p < .001$)

Figure 6.2. Multigroup SEM analyses: Maximum Likelihood Parameters for Low-Agreeable versus High-Agreeable Adolescents.



Note. Model fit Low-Agreeable Adolescents: NNFI = .97; Model fit High-Agreeable Adolescents: NNFI = .96. (* $p < .001$)

Figure 6.3. Multigroup SEM analyses: Maximum Likelihood Parameters for Emotionally Less-Stable versus Emotionally Highly Stable Adolescents.



Note. Model fit Emotionally Less-Stable Adolescents: NNFI = .97; Model fit Emotionally High-Stables: NNFI = .96. (* p < .001)

Discussion

The present study demonstrated that daily Internet use, in itself, is only marginally and not directly associated with low well-being in adolescence and young adulthood. The ‘risks of Internet use’ are clearly dependent on individuals’ tendencies toward compulsive use of the Internet, such as not being able to stop using the Internet, Internet use interfering with other duties or social contacts, and a constant preoccupation with Internet use. These findings were replicated separately for boys and girls. Moreover, the present study showed that possible negative consequences associated with adolescents’ daily Internet use are in part dependent on their personality constellation. For introverted, low-agreeable, and emotionally less-stable youths, daily Internet use is more strongly associated with CIU and, in turn, CIU more strongly linked to feelings of loneliness.

The results of this study conform with the results of prior examining adolescents’ Internet use, in which CIU was found to be a stronger predictor of negative outcomes than the amount of time spent on the Internet (Caplan, 2003; Van den Eijnden, 2008), and in which a strong positive relationship was found between CIU and loneliness (Morahan-Martin & Schumacher, 2000). Clearly, the results underline the importance of a diathesis-stress mechanism underlying the linkages between adolescents’ Internet use and low well-being: Internet use does not affect everyone in the same way (McKenna & Bargh, 1998). More generally, a specific behavior or event does not necessarily have to lead to the same outcome in every individual. Individuals may start on the same developmental pathway, but due to subsequent choices, vulnerabilities, and characteristics they may exhibit different adaptive or maladaptive patterns (Cicchetti & Tucker, 1994; Sroufe et al., 1990). To meet this diversity in processes and outcomes, research should place emphasis on analysing specific subgroups (Cicchetti & Rogosch, 1996). However, with regard to the association between daily Internet use and well-being, popular media and many studies in the past proceeded from the point of view that use of the Internet affects everyone in the same way. A strength of the present study is that it emphasized the role of individual characteristics (i.e. CIU), and personality-based vulnerabilities with regard to the association between daily Internet use and well-being.

The present results may be explained in terms of the social status youth have in offline contexts. It is very likely that introverted, low-agreeable, and emotionally unstable adolescents experience less social benefits in offline contexts, and are characterized by a relatively marginal position in the peer group (Newcomb & Bagwell, 1995; Rubin et al., 1990). According to Kraut et al. (2002), people who differ in certain personality traits are likely to use the Internet in different ways. For example, for introverted adolescents it will be more difficult to establish and maintain satisfying peer contacts in everyday life.

Especially for these youths, Internet use may be highly rewarding because many of the factors that make it difficult to communicate in 'real life' interactions (i.e. ambiguity of non-verbal cues, the need to instantly react and assert oneself) are not present in online interactions (Rabiner et al., 1990; Spears & Lea, 1994). These adolescents will therefore be more motivated to develop social relationships online (McKenna & Bargh, 1998). The development and maintenance of these online social relationships may increase the likelihood of excessive Internet use, and subsequently CIU, which, in turn, is more likely to lead to a further decrease in social off-line resources and thus lead to loneliness, or a low self-esteem and depressive moods (McKenna & Bargh, 1998; Caplan, 2003). In accordance with Davis (2001) and Caplan (2003), we think that introverted, low-agreeable, and emotional unstable adolescents are likely to end up in a vicious cycle in which adolescents who developed CIU use the Internet in a more and more excessive manner that leads to even higher levels of CIU which, in turn, worsens their problems more and more. This point becomes even stronger since no moderator effect was found for the personality traits of conscientiousness and openness, which affect the individual's interpersonal domain to a lesser extent than the other personality traits (McCrae & John, 1992), suggesting that a vulnerability for CIU indeed may be found in the social status of adolescents. Future longitudinal research designs should examine if this is indeed the case.

In our examination of the moderator effects of adolescents' personality traits on the linkages between CIU and low well-being, we found that only the CIU-loneliness link was moderated by extraversion, agreeableness, and emotional stability - but not the relationships from CIU to low self-esteem and depressive moods. This is probably because the UCLA loneliness measure (Russell et al., 1980) that we employed in the present study taps directly into individuals' perceptions of 'themselves in relation to others'. Especially on this inherently social dimension, the differences between for instance introverted and extraverted individuals or between, for instance, emotionally stable and unstable individuals is most pronounced. Another possible explanation why the links between CIU and depressive moods and CIU and low self-esteem were not moderated by extraversion, agreeableness, and emotional stability is that it is likely that decreasing levels of social off-line resources lead to higher levels of loneliness. A decreasing level of social resources need not necessarily result in lower self-esteem and higher levels of depressive moods. It is important to note that although the SEM multigroup analyses provided evidence for moderator effects, for both 'low' and 'high' personality groups we observed relatively strong associations between daily Internet use and CIU, and between CIU and low well-being. Thus, we did not observe any 'buffering effects' with regard to adolescents' personality traits. Extraverted, agreeable, and emotionally stable adolescents were also reporting lower well-being when reporting more CIU – albeit to a lesser extent. In

our examination of whether the overall pattern of findings could be replicated for boys and girls separately, we found differences in the subgroups of low versus high agreeable, extraverted, and emotional unstable girls. We think these differences are due to the relatively small number of girls compared to the number of boys.

The present study contributes new insights into the linkages between adolescents' Internet use and well-being, by applying a diathesis-stress perspective that emphasizes individual differences in vulnerabilities when using the Internet. Another strength of the present study is that we used a large-scale sample of Dutch adolescents and young adults from a relatively broad age range. However, several limitations should be mentioned as well. First of all, due to the cross-sectional design of this study it is impossible to address in any decisive manner the direction of causality between adolescents' daily Internet use, CIU, and low well-being. Although it is logical to examine a 'natural' sequence of everyday behavior (i.e., daily Internet use) to more compulsive behavior (i.e., CIU) that might have negative consequences for adolescents' mental health, one may consider that loneliness or depressive moods in adolescents could trigger an increasing amount of time spent on the Internet and, as a consequence, more compulsive Internet use (Meerkerk et al., submitted for publication; Armstrong et al., 2000). Future longitudinal studies should therefore explicitly address bidirectional causal models.

Secondly, for assessing personality traits in the present study we used the Big Five description of personality (Goldberg, 1992) which has received a considerable level of criticism. For example, according to Paunonen and Jackson (2000), there are more than five dimensions of personality underlying human behavior. Furthermore, the Big Five describes personality on the basis of individual differences in traits and, according to McAdams (1992), personality cannot be adequately described on the basis of these traits alone. However, in the present study we investigated whether there were differences in the progression from daily Internet use to CIU and from CIU to low well-being between adolescents who score high versus low on certain personality dimensions of the Big Five. This can be done adequately on the basis of the Big Five personality description. Thirdly, this study exclusively assessed adolescents' perceptions of their 'online time' and compulsive behavior, but did not take into account - similar to what has been common practice in the field (but for exceptions see Van den Eijnden et al., 2008; Meerkerk et al., submitted for publication) - what adolescents do when they are online. This is of great importance, however, in future attempts to explain why for some adolescents Internet use may be more rewarding than for others. For instance, introverted or emotionally less-stable youths might be especially inclined to use chatrooms or instant messenger software because of the advantages it gives them in communicating with peers, such as the lower emphasis on non-verbal cues and the possibility to step down from an

interaction (McKenna & Bargh, 1998). Fourthly, the sample of the present study was not representative for the Dutch population of adolescents. Because participants were recruited by advertisements in newspapers, on the radio, and on the television without specific selection criteria, the sample of the present study became an opportunity sample without knowing which backgrounds the participants had. Therefore, care has to be taken in generalizing the present results.

Overall, on the basis of the present data the conclusion seems justified that daily Internet use, in itself, is only marginally associated with low well-being in adolescents. In employing a diathesis-stress perspective on behavior-adjustment linkages this study clearly demonstrated that there might be a progression from daily Internet use to CIU which eventually might lead up to low well-being. Moreover, the results showed that adolescents with low levels of extraversion, agreeableness, and emotional stability are at increased risk for the development of CIU and low well-being, when using the Internet on a daily basis. Future longitudinal studies will need to establish whether the present results still hold when explicitly modeling the bidirectional associations between Internet use, CIU, and emotional maladjustment over time in adolescence.

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Section IV: Psychopathology and subjective wellbeing

7

Psychopathology and Subjective Wellbeing; Opposite ends of a mental health continuum?

This chapter is submitted as:

Bartels, M., Van der Aa, N., Van Beijsterveldt, T.C.E.M, & Boomsma, D.I. Psychopathology and subjective wellbeing; opposite ends of a mental health continuum?

Abstract

Interest in subjective wellbeing (SWB) is increasing and a major force driving this is its assumed association with physical and mental health. SWB has been found to be strongly negatively associated with psychopathology, but there is also evidence that SWB and psychopathology are distinct dimensions of mental functioning. In the current study we investigate the presence or absence of a mental health continuum. Survey data on psychopathology (the Youth Self Report) and SWB (satisfaction with life, subjective happiness, and quality of life) collected in a large sample of Dutch adolescent twins and their non-twin siblings (6381 twins and 1195 siblings from 3511 families) were used. Mean levels of SWB are compared for high versus low scoring individuals on psychopathology and the genetically informative design is used to estimate phenotypic, genetic, and environmental correlations between the SWB and psychopathology measures. High negative phenotypic correlations are found between SWB and the YSR scales. Individuals that score high on psychopathology report significant lower levels of SWB than individuals that score low on psychopathology. It is found that the observed phenotypic correlations are mainly driven by significant genetic correlations. Finally, 85% of the sample jeopardize the two group division of a mental health continuum. It can be conclude that SWB and Psychopathology cannot be considered the opposite ends of a mental health continuum. On the other hand the two traits are not fully independent. Results are discussed in light of future studies and intervention and prevention strategies.

Introduction

Within several disciplines that study human behavior, there is a growing interest in positive aspects of psychological functioning, such as subjective wellbeing (SWB) and happiness. An important question which has been raised is whether SWB and psychopathology, defined as behavioral and emotional problems, constitute the opposite ends of a mental health continuum or whether SWB and psychopathology constitute distinct dimensions of mental functioning. If psychopathology and SWB constitute opposite ends of the same mental health continuum one would expect high negative correlations between both phenotypes. Mean levels of SWB in individuals affected with some form of psychopathology should be significantly lower than mean levels of SWB in unaffected individuals. In other words two groups of individuals would be distinguishable; a first group that score low on psychopathology and high on SWB and a second group that score high on psychopathology and low on SWB.

Although SWB has been found to be strongly negatively associated with different types of psychopathology and especially with depression (e.g. Proctor et al., 2009), there is also evidence that SWB and psychopathology are distinct dimensions of mental functioning (e.g. Greenspoon & Saklofske, 2001; Ryff et al., 2006). Causes and correlates applying to psychopathology and SWB are, according to this view, likely to be, at least partly, specific to both dimensions. For example, Ryff et al. (2006) reported that significant correlations between wellbeing and several biological correlates were not accompanied by significant correlations between ill-being and the same biological correlates. Furthermore, a recent study in a large Dutch populations sample reported that 68.4% of individuals with a mental disorder reported that they felt often happy (Bergsma et al., 2010). If SWB and Psychopathology are considered to be interrelated yet distinct constructs, as has been proposed by Greenspoon and Saklofske (2001), less strong negative correlations are expected and four groups of individuals would be distinguishable. The first two groups would represent the continuum and are similar to the two groups mentioned above (low SWB – High Psych and High SWB – low Psych). A third group, that scores high on SWB and high on Psychopathology, and a fourth group, that scores low on SWB and low on Psychopathology would be identified.

To gain insight in the etiology of the associations of SWB and psychopathology a genetically informative design can be used. A decomposition of the observed association between SWB and psychopathology into genetic and environmental correlations will reveal whether the often found association is accounted for by overlapping genetic factors

or overlapping environmental influences. If one underlying continuum is assumed the genetic correlation between psychopathology and SWB should be significant and approaching one.

Assuming a simple genetic association between psychopathology in general and SWB might not be representative for the complexity of human mental functioning. Psychopathology is an umbrella term for both Internalizing (INT) as well as Externalizing (EXT) behavioral and emotional problems. Internalizing problems are generally considered to belong to the subgroup of psychopathology that involves disturbances in emotion or mood (e.g. Anxious/Depressed and Withdrawn/Depressed behavior), whereas Externalizing problems tend to refer to dysregulation in behavior, for instance Aggressive and Rule-Breaking behavior. Theoretically it is expected that SWB is highly overlapping to Internalizing problems and an underlying continuum, quantified in a significant genetic correlation is expected. Externalizing problems, though, are less obviously related to SWB and significance of genetic overlap is doubtful.

The presence or absence of a significant genetic correlation between SWB and INT or EXT has implications for prevention and intervention strategies. If the hypothesis that overlap between SWB and INT will be genetically mediated is confirmed in the current study, it is to be expected that factors that cause individual differences in SWB could also be used to help the ones who suffer from Internalizing problems. This also fits recent evidence that indicated the potential role of SWB as a buffer against the development of psychopathology (Proctor et al., 2009; Sin & Lyubomirsky, 2009). Furthermore, information from genetic studies on SWB can broaden the horizon for the gene hunting in the field of Internalizing psychopathology such as anxiety and depression. On the other hand if it is confirmed that the overlap between SWB and EXT is less genetically mediated, prevention and intervention for EXT should focus more on environmental factors that also mediate individual differences in SWB.

In order to study the presence or absence of a mental health continuum and to unravel the underlying sources of a possible association, three measures of SWB (i.e. Quality of life, Satisfaction with life, and Subjective happiness), and the syndrome scales of the Youth Self-Report (YSR; Achenbach & Rescorla, 2001) measuring different dimensions of emotional and behavioral problems, were studied in a large sample of Dutch adolescent twins and their non-twin siblings. We examined whether psychopathology and SWB are opposite ends of a mental health continuum using a three step approach. First, mean levels of SWB are compared for high (T-score of psychopathology above clinical threshold) versus low scoring individuals on the YSR syndrome scales and the higher order Internalizing and Externalizing dimensions. Secondly, it is investigated if the two groups (High SWB-Low Psychopathology and Low SWB and High Psychopathology) are

present or whether the sample should be divided into multiple groups. If the latter is the case a mental health continuum is challenged. Finally, a genetically informative design is used to estimate phenotypic, genetic, and environmental correlations between the SWB and psychopathology measures. We hypothesize that the phenotypic and genetic overlap between SWB and Internalizing problems is higher than the phenotypic and genetic overlap between SWB and Externalizing problems. Since the study is based on a large sample of adolescent twins and their non-twin siblings we will be able to test for sex-differences in mean levels as well as phenotypic and genetic correlations.

Methods

Subjects

Participants were registered at birth with the Netherlands Twin Registry (NTR), established by the Department of Biological Psychology at the VU University in Amsterdam (Boomsma et al., 2006; Bartels et al., 2007). For the current study, data from surveys collected in adolescent twins and their nontwin siblings were analyzed. The sample consisted of 7616 individuals (6381 twins and 1195 siblings from 3511 families) with a 45%–55% boy–girl distribution as described in Table 7.1.

Table 7.1.

Constitution and Zygosity Distribution of the sample

Family Constitution	Number of Families	Zygosity	Number of Families
One of the twin, no sibling	417	MZM	551
Twin Pair, no sibling	1864	DZM	476
One of the twin with a brother	21	MZF	792
One of the twin with a sister	60	DZF	571
One of the twin with a brother and a sister	3	DOSmf	576
Twin pair with a brother	499	DOSfm	545
Twin pair with a sister	577		
Brother only (no twin)	24	In total 3511 families Cohort: 1984-1997 Mean age 16.47 (1.66)	
Sister only (no twin)	40		
Brother and Sister only (no twin)	4		

Mean age of the sample is 16.47 (1.66), with age ranging from 12 to 20 and born in cohorts 1984-1997. Siblings were included in the analyses with a maximum of 2 siblings (1 brother and 1 sister) per family. Some families (n=30 families) had data of more than one sibling of the same sex. From these families data from the sibling closest in age to the twin was selected for analyses. Zygosity was determined for 960 same-sex twin pairs by DNA analysis or blood group polymorphisms. For all other same-sex twin pairs, zygosity was determined by discriminant analysis, using longitudinal questionnaire items from the previously collected parental report. Agreement between zygosity assignment by the replies to the longitudinal questionnaire and zygosity determined by DNA markers/blood typing was around 93% (Rietveld et al., 2000).

Measures

The Dutch Health Behavior Questionnaire (DHBQ) is a self-report instrument containing a range of measures on health, lifestyle, and behavior. It includes the Youth Self Report (Achenbach and Rescorla, 2001) to assess adolescent behavioural and emotional problems and three measures of Subjective Wellbeing.

The Youth Self-Report is a screening tool for behavioral and emotional problems in adolescents that comprise the Achenbach System of Empirically Based Assessments (ASEBA). Adolescents are asked to fill out 118 items (112 items of the 2001 version supplemented with 6 items for the older version of the YSR) on a 3-point scale based on the occurrence of the behavior during the preceding 6 months: 0 if the problem item was not true, 1 if the item was somewhat or sometimes true, and 2 if it was very true or often true. The syndrome scales (Anxious/Depressed, Somatic Complaints, Withdrawn/Depressed, Social Problems, Thought Problems, Attention Problems, Rule-Breaking, Aggressive Behavior) were composed according to the 2001 profile (Achenbach and Rescorla, 2001). Furthermore the broadband scales Internalizing behavior (Anxious/Depressed, Somatic Complaints and Withdrawn/Depressed) and Externalizing behavior (Rule-Breaking and Aggressive Behavior) were computed. Dutch syndrome scales and comparability with the syndrome scales developed by Achenbach are reported in Verhulst, Van der Ende, and Koot (1997).

Three measures of self-reported evaluations of subjective wellbeing (SWB) were used in the present study: (1) satisfaction with life (SAT) was assessed with the Satisfaction with Life Scale (Diener et al., 1985). The scale consists of 5 items which had to be answered on a 7-point scale ranging from 1 = 'strongly disagree' to 7 = 'strongly agree'. Example items are "My life is going more or less as I wished" and "I'm satisfied with my life". Internal consistency of the scale was good with a Chronbach's Alpha of

.86. (2) Subjective happiness (HAP) was assessed with the Subjective Happiness Scale (Lyubomirsky & Lepper, 1999). The scale consists of 4 items which had to be answered on 7-point scale ranging from 1 = ‘strongly disagree’ to 7 = ‘strongly agree’. Example items are “On the whole I’m a happy person” and “On the whole, I’m not very happy”. Internal consistency of the scale was good with a Chronbach’s Alpha of .84. (3) Quality of life in general (QLg) as assessed with the Cantril Ladder (Cantril, 1965). The ladder has 10 steps: the top indicated the best possible life, and the bottom the worst possible life. Participants had to indicate the step of the ladder at which they place their lives in general.

In our previous work (Bartels and Boomsma, 2009) we showed that underlying sets of genetic factors are responsible for the moderate to high phenotypic associations between SAT, HAP, and QLg and therefore it is assumed that the phenotypic overlap between the observed measures of SWB is determined by a single ‘phenotypic’ latent variable (SWB). In order to reduce the data of SWB from three observed measures (i.e. SAT, HAP, and QLg) to one single score for SWB, a latent factor score for SWB was estimated for each individual in the dataset. This was done by specifying a common pathway model with the three observed measures of SWB loading on a single phenotypic latent factor (SWB) to the data, using Mplus (Muthén & Muthén, 2007). Given this factor solution and individuals’ scores on the observed measures of SWB, a latent factor score can be estimated and provided by Mplus. This factor score has a mean of zero and ranges in the current sample from -4.297 to 1.491 (the distribution is slightly skewed), with a low (negative) score meaning low levels of SWB.

To investigate differences in mean levels of SWB between high and low scoring individuals the YSR syndrome and broadband scales were dichotomized using sex-specific T-scores. These scores were derived by saving sex-specific z-scores for each syndrome, and calculating T score with a mean of 50 and a standard deviation of 10 ($T_{score} = 10 * z\text{-score} + 50$). Individuals with a T-score of 67 and higher were assigned to the high scoring group (Achenbach 1991; Verhulst et al., 1997). In order to be able to identify the two groups that fit a continuum (High Psychopathology- Low SWB and Low Psychopathology-High SWB) and other groups that challenge this continuum (e.g. High Psychopathology-High SWB and Low Psychopathology-Low SWB) the SWB factor score is trichotomized on the 10th and 90th percentile, with the lower 10% being the low SWB group, the middle 80% being the middle SWB group, and the upper 10% being the high SWB group.

Analyses

Sex-differences in mean levels of SWB and YSR scales and mean differences in SWB between the low psychopathology and high psychopathology group were tested by mixed-

model analyses of variance in SPSS18, with family as a random factor to account for the within-family dependence of the outcome variables. Cross-tabulation was used to identify distinct groups of individuals that either fit or jeopardize a mental health continuum. Cross-tabulations were calculated for all combinations of SWB (low, middle, high) and psychopathology (low, high).

Phenotypic correlations between the Psychopathology syndrome and broadband scales and the SWB measures were estimated in Mx (Neale et al., 2006). A small but significant effect of age on mean levels have been reported for SWB (Bartels and Boomsma, 2009) and the YSR (e.g. Lamb et al., 2010), so age was included as covariate on the means. The different degree of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs and their non-twin siblings was used in a genetic structural equation model to obtain estimates of genetic and environmental correlations. Genetic bivariate models were fit to the data so that genetic and environmental correlations between SWB and the YSR scales could be estimated. Standardization of the genetic covariance between YSR syndrome scores and SWB reveals the genetic correlations and the strength of this correlation indicates the overlap in genes influencing both traits. Standardization of the environmental covariance between YSR syndrome scores and SWB reveals the environmental correlations and the strength of this correlation indicates the overlap in environmental factors influencing both traits. Based on our previous work (Bartels and Boomsma, 2009) a genetic model was fitted that specified additive genetic (A) and nonshared environmental (E) influences. The influence of A and E was allowed to be sex-specific. By constraining the genetic and environmental influences on the covariance to zero it was tested whether the genetic and environmental correlations were significant. Genetic structural equation modeling in Mx (Neale et al., 2006) was used with the raw-data ML procedure for estimation of parameters. Nested submodels were compared by hierarchic χ^2 tests. The χ^2 statistic is computed by subtracting $-2LL$ (log-likelihood) for a reduced model from that for the full model ($\chi^2 = -2LL_0 - (-2LL_1)$). Given that the reduced model is correct, this statistic is χ^2 distributed with degrees of freedom (df) equal to the difference in the number of parameters estimated in the two models ($\Delta df = df_0 - df_1$).

Results

Means and standard deviations of SWB and the YSR syndrome and broadband scales are presented in Table 7.2 for males and females separately. Males report significant higher levels of SWB ($p < .05$) than females. In the current sample females also report significant more Internalizing problems while males report significant higher levels of Externalizing problems ($p < .05$).

Table 7.2.

Number of individuals, Means, and standard deviations for the SWB factor score and the YSR scales

	Males			Females		
	N	M	sd	N	M	sd
SWB	3335	.062	.89	4256	-.050*	.96
Withdrawn	3049	2.11	1.96	3749	2.58*	2.20
Somatic complaints	2979	1.62	1.93	3663	2.71*	2.51
Anxious/ depressed	3029	3.10	3.35	3726	4.97*	4.48
Internalizing	2952	6.70	5.67	3631	9.99*	7.45
Delinquent behavior	3058	2.87*	1.99	3757	2.43*	1.87
Aggressive behavior	3045	6.40*	4.47	3738	6.25*	3.99
Externalizing	3045	9.28*	5.85	3736	8.69*	5.27
Social problems	3042	2.10	1.84	3742	2.01*	1.81
Thought problems	3042	1.42	1.64	3748	1.63	1.72
Attention problems	3039	4.62	2.69	3731	4.84	2.78

Note. * females score significantly different than males ($p < .05$); SWB = subjective wellbeing factor score.

The latter is based on a significant higher score on Rule-Breaking for males, while no differences in mean levels are found for Aggressive Behavior between males and females.

Individuals who score high on psychopathology (both males and females) rate their SWB significant lower, than individuals that score below the sex-specific clinical cut-off for the syndrome scales of the YSR (see Table 7.3). This is an indication that SWB and psychopathology are not completely independent. Cross tabulation (see Table 7.4), though, indicate that a mental health continuum from psychopathology to SWB would be too simplistic to describe the association between both traits. About 15% of the sample fits a continuum, since 1.8-5.0% of the individuals score high on psychopathology and score

low on SWB and 10.6-11.9% of the individuals score low on psychopathology and score high on SWB. The remaining 85% of the sample does not fit in a mental health continuum framework.

Table 7.3.

Mean levels of SWB for low versus high scoring individuals (tested for significance within sex)

	Subjective Wellbeing mean levels			
	MALES		FEMALES	
YSR Syndrome Scales	Low	High	Low	High
Withdrawn	.127	-.926*	.034	-1.309**
Somatic complaints	.095	-.210*	.019	-.925**
Anxious/ depressed	.150	-1.218*	.056	-1.651**
Internalizing	.136	-1.031*	.056	-1.562**
Delinquent behavior	.074	-.194*	-.002	-.729**
Aggressive behavior	.092	-.250*	-.011	-.704**
Externalizing	.086	-.310*	.002	-.750**
Social problems	.101	-.626*	-.010	-.804**
Thought problems	.087	-.411*	.010	-.815**
Attention problems	.088	-.519*	.000	-.890**

Note. * high psychopathology males show significant lower SWB than low psychopathology males ($p < .001$); ** high psychopathology females show significant lower SWB than low psychopathology females ($p < .001$); High = T-score ≥ 67 .

A very low percentages of individuals (.1-.6% of the sample) score high on psychopathology but report high levels of SWB, while 19.7-27.0% of the sample score low on psychopathology but still report low levels of SWB. This indicates that SWB and psychopathology cannot be considered opposite ends of the same continuum solely. The pattern of group division is similar in males and females. A somewhat higher percentage of females are unaffected and report low SWB, while males percentages for unaffected individuals that score middle SWB outnumber females a bit.

Phenotypic, genetic, and environmental correlations are reported in table 7.5. Significant and strong negative correlations (ranging from -.45 to -.57) are observed for SWB and the Internalizing syndrome scales and broadband scale in both males and females. Correlations between EXT and SWB are moderate to strong and negative (ranging from -.24 to -.44), indicating that both Internalizing as well as Externalizing problems are

negatively associated with SWB. The phenotypic correlations between SWB and INT, but also between SWB and EXT are mainly driven by significant genetic correlations. Environmental correlations are moderate for most syndromes in females (except for Rule-Breaking) and for 5 out of 10 of the possible associations in males (see Table 7.5 for details). For males no significant environmental correlations between SWB and EXT are found, indicating that the negative association between SWB and EXT in males (r -ranging from $-.24$ to $-.44$) is fully accounted for by overlapping genes.

Discussion

In the current study it was investigated if Psychopathology and Subjective Wellbeing can be considered to be opposite ends of a mental health continuum. It was hypothesized that a continuum can be assumed to be present if; 1) high negative correlations would be found; 2) mean levels of SWB in individuals scoring high on some form of psychopathology are significantly lower than mean levels of SWB in low scoring individuals; 3) the sample could be divided into two groups of individuals that support a continuum; 4) the expected negative association is mainly driven by genetic influences.

Results of the current study indicate that SWB and Psychopathology cannot be considered the opposite ends of a mental health continuum. On the other hand the two traits are not fully independent. The presence of significant negative correlations between SWB and the syndrome scales of the YSR are indicative for an association between SWB and Psychopathology. Furthermore, it is observed in the current study that individuals that score in the clinical range of any form of psychopathology rate their SWB significantly lower than the remaining of the sample. Finally, the observed negative association between the two traits is mainly accounted for by genetic effect. So genes that influence SWB also influence Psychopathology revealing a biological overlap between the two constructs. Environmental correlations are significant, except for Externalizing problems in males, indicating that the often observed negative association between SWB and psychopathology is also driven by overlapping environmental influences. The fact, though, that the genetic correlations outnumber the environmental correlations is a strong indication that SWB and psychopathology are not completely independent. The less than perfect genetic correlations indicate that it is not a one to one relationship and that there are several factors, both genetic and environmental, that will in the end cause individual differences in one's mental health status.

Table 7.4.

Cross tabulations for SWB (low, middle, high) and Psychopathology (low, high)

YSR Syndrome Scales	Subjective Wellbeing						
	MALES			FEMALES			
	low	middle	high	low	middle	high	
Withdrawn	Low	124 (4.1%)	64 (2.1%)	7 (.2%)	176 (4.7%)	42 (1.1%)	9 (.2%)
	High	624 (20.5%)	1881 (61.7%)	349 (11.4%)	939 (25%)	2184 (58.3%)	399 (10.6%)
Somatic complaints	Low	54 (1.8%)	81 (2.7%)	17 (.6%)	122 (3.4%)	54 (1.5%)	7 (.2%)
	High	610 (21.5%)	1752 (61.7%)	324 (11.4%)	912 (25.8%)	2061 (58.3%)	381 (10.8%)
Anxious/depressed	Low	118 (3.9%)	31 (1.0%)	5 (.2%)	185 (5%)	14 (.4%)	3 (.1%)
	High	586 (19.7%)	1876 (63.2%)	353 (11.9%)	889 (24.2%)	2190 (59.5%)	399 (10.8%)
Internalizing	Low	127 (4.3%)	50 (1.7%)	7 (.2%)	178 (4.9%)	24 (.7%)	5 (.1%)
	High	589 (20.0%)	1837 (62.2%)	342 (11.6%)	880 (24.4%)	2132 (59.2%)	384 (10.7%)
Delinquent behavior	Low	63 (2.1%)	83 (2.7%)	10 (.3%)	130 (3.5%)	94 (2.5%)	8 (.2%)
	High	687 (22.5%)	1864 (61.0%)	351 (11.5%)	988 (26.3%)	2136 (56.9%)	401 (10.7%)

Table 7.4. (CONCLUDED)

Aggressive behavior	Low	69 (2.3%)	86 (2.9%)	10 (.3%)	110 (2.9%)	78 (2.1%)	3 (.1%)
	High	647 (21.7%)	1823 (61.1%)	347 (11.6%)	1001 (26.8%)	2143 (57.3%)	403 (10.8%)
Externalizing	Low	79 (2.6%)	79 (2.6%)	11 (.4%)	105 (2.9%)	75 (2.0%)	4 (.1%)
	High	665 (21.8%)	1861 (61.1%)	350 (11.5%)	977 (26.5%)	2121 (57.6%)	400 (10.9%)
Social problems	Low	88 (2.9%)	76 (2.5%)	5 (.2%)	105 (2.8%)	65 (1.74%)	10 (.3%)
	High	658 (21.6%)	1860 (61.1%)	355 (11.7%)	1011 (27.0%)	2154 (57.6%)	397 (10.6%)
Thought problems	Low	81 (2.7%)	79 (2.6%)	7 (.2%)	150 (4.0%)	92 (2.5%)	14 (.4%)
	High	665 (21.9%)	1857 (61.0%)	353 (11.6%)	965 (25.7%)	2131 (56.9%)	396 (10.6%)
Attention problems	Low	68 (2.2%)	60 (2.0%)	9 (.3%)	137 (3.7%)	67 (1.8%)	6 (.2%)
	High	677 (22.3%)	1876 (61.7%)	349 (11.5%)	976 (26.2%)	2148 (57.6%)	397 (10.6%)

Note: Groups that fit a mental health continuum are bold-faced

Table 7.5.

Phenotypic, Genetic, and Environmental Correlations between SWB and YSR syndrome and Broadband Scales

YSR Syndrome Scales	Subjective Wellbeing						
	r	Males			Females		
		rg	re	r	rg	re	
Withdrawn/ Depressed	-.52	-.67	-.29	-.53	-.67	-.31	
Somatic Complaints	-.47	-.39	-.07*	-.47	-.50	-.17	
Anxious/ Depressed	-.52	-.71	-.32	-.57	-.71	-.42	
Internalizing	-.45	-.68	-.31	-.53	-.70	-.39	
Rule-Breaking Behavior	-.44	-.46	-.01*	-.44	-.49	-.03*	
Aggressive Behavior	-.28	-.48	-.01*	-.36	-.40	-.15	
Externalizing	-.24	-.51	-.02*	-.31	-.46	-.13	
Social Problem	-.48	-.47	-.17	-.47	-.44	-.16	
Thought Problems	-.47	-.59	-.07*	-.47	-.47	-.14	
Attention Problems	-.44	-.47	-.13	-.47	-.45	-.22	

Note. * = non significant; r = phenotypic correlation; rg = genetic correlation; re = environmental correlation.

This study provided evidence that the association between SWB and psychopathology is complex. The cross tabulations show that only about 15% of the individuals fit a continuum. Surprisingly, 19.3-25.8% of the individual score low on psychopathology but also report low levels of SWB. Identification of individuals in the latter group might be of interest since a longitudinal study by Lewinsohn and colleagues (1991), albeit based on an adult sample, found that non-depressed subjects that score low on satisfaction with life were more likely to become depressed two to three years

later as compared to average or high satisfaction with life scorers. This group can thus be considered an at-risk group and would be the major target of a wellbeing intervention program.

The current findings have implications for the prevention and intervention strategies for adolescent behavioral and emotional problems. Founded in the field of epidemiology and somatic medicine, it has been proposed that larger benefits to overall public health and mental capital is to be expected when the bell curve of mental health in the human population is shifted a little to the healthy site, the so-called population strategy (e.g. Rose 1992, 2008). This could only be reasonable and feasible when psychopathology and SBW are at least partly associated, as has been confirmed in the current study. A relative slight increase in the level of subjective wellbeing of the bulk of the population may have a larger preventive effect than targeting the much smaller group of people at high risk. If for example the risk group (low psychopathology and low SWB) can be identified and stimulated to participate in an online prevention program, they might be protected against the development of psychopathology.

To this end knowledge on the causes of individual differences in subjective wellbeing and the factors that jeopardize or promote wellbeing (so-called risk and protective factors) is crucial. Based on the high genetic correlations it can be assumed that information on the causes of individual differences in subjective wellbeing can provide insights into the causes of variation in psychopathology and provide new handles for psychopathology intervention. However, it must be emphasized that, although interventions to increase SWB are being implemented world-wide, studies into the effects of positive psychology interventions (PPIs) provided mixed results. Some studies report PPIs not to be effective (e.g. Froh, Sefick, & Emmons, 2008), while others report that PPI delivered to depressed individuals significantly boosted SWB and decreased depression (e.g. Seligman et al., 2006). A recent meta-analysis (Sin & Lyubomirsky, 2009) of 49 studies revealed that PPIs do significantly enhance SWB. Furthermore, the combined results of 25 studies showed that PPIs also decrease depressive symptoms. Further research is warranted to gain insight into causes of individual differences in intervention effectiveness and the format of the treatment, since one-size-fits all strategy might not be the best strategy for optimal results. This is in line with the finding of Sin & Lyubomirsky (2009), who reported that motivation of participation in PPIs moderates the outcome of the intervention, with the expected larger effects for more motivated individuals.

From a biological perspective the high genetic correlation between SWB and Psychopathology opens new opportunities for gene-hunting studies. The high genetic correlations indicate that part of the genes that influences SWB also influence behavioral and emotional problems. Gene-hunting studies that make use of a continuum from ill to

high SWB might be more informative and powerful than the ones that use the common approach of looking at either cases ('ill') versus controls ('not ill') or a quantitative approach with a score from ill to normal.

The current results are based on a large population based sample of twins and their non-twin siblings. Some limitations should be kept in mind while interpreting the outcome of this study. First, results are based on a population-based sample of which the largest part (94% of the sample) score below the clinical cut-off for psychopathology. It could be the case that results change when analyses are restricted to clinical samples. Next, data are collected in 14, 16, and 18 year old twins and their non-twin siblings aged 12 to 22. This results in a large age range. Correction for possible linear age effects are done by including age as covariate on the mean levels of SWB and Psychopathology. Other influences of age (e.g. quadratic effects) are not taken into account. Finally, information on SWB and Psychopathology is based on self-report. Both for SWB and Psychopathology high reliability has been confirmed and to enable analyses in large sample sizes survey research is the most feasible way of data collection. Furthermore, it has been shown that behavioral problems can reliably be assessed using self-report (Verhulst et al., 1997).

From the current study it can be concluded that SWB and Psychopathology are two overlapping but partly independent constructs. Significant negative associations are found and it is shown that individuals that score high on psychopathology score significantly lower on SWB. A genetic overlap is confirmed with the finding of moderate to high genetic correlations. However, only 15% of the sample fits a mental health continuum. Taken together these results would imply that gaining information on the causes of individual differences in SWB could be helpful for intervention and prevention of psychopathology. On the one hand will the overlap between the two constructs provide power and possibilities for gene-hunting studies. On the other hand will identification of individuals that challenge the mental health continuum provide possibilities to prevent individual at risk for developing psychopathology.

Acknowledgements

Funding was obtained from the following grants: "Spinozapremie" (NWO/SPI 56-464-14192); "Twin-family database for behavior genetics and genomics studies" (NWO 480-04-004); "Genetic and Family influences on Adolescent Psychopathology and Wellness" (NWO 463-06-001); "A twin-sib study of adolescent wellness" (NWO-VENI 451-04-034). M. Bartels is financially supported by a senior fellowship of the EMGO+ Institute for Health and Care . Niels van de Aa is financially supported by a WCRF International research grant.

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8

Frequency of Truancy at High School: Evidence for Genetic and Twin Specific Environmental Influences

This chapter is published as:

Van der Aa, N., Rebollo-Mesa, I., Willemsen, G., & Boomsma, D.I. (2009). Frequency of truancy at high school: evidence for genetic and twin specific shared environmental influences. *Journal of Adolescent Health*, 45, 579-586.

Abstract

Purpose. The purpose of this study was to examine the relative influence of genetic and environmental factors on variation in truancy during high-school. We examined the significance of genetic and shared and non-shared environmental influences. In addition, we tested for the presence of environmental factors specifically shared by twins, but not by their siblings.

Methods. A threshold model was used to analyze data from 4,835 twins and their non-twin siblings.

Results. Results showed a higher prevalence of truancy for males (38% vs. 29%) and an increase in prevalence with age ($\beta = -.53$). Individual differences in frequency of truancy were partly explained by genetic influences ($h^2 = 45\%$); for twins, the environmental variance was partitioned into 25% shared by twins, and 30% nonshared, whereas for singletons all environmental variance was nonshared (i.e. 55%).

Conclusions. Prevalence of truancy higher for boys than for girls and it increased with age. Genetic and environmental influences accounted for the variance in frequency of truancy. Part of the shared environment represents influences that are shared by twins but not by non-twin siblings.

Introduction

Truancy, defined as the illicit absence from school during one or more hours or days, is frequent from early adolescence onwards. Thirteen percent of Dutch high-school students report skipping one or more hours of school during the past 2 months (CBS, 2004). Among adolescents from the United States 11% of 8th-grade students, 16% of 10th-grade students, and 35% of 12th-grade students reported skipping 1 or more days of school during the past month (Johnston et al., 2004). Truancy has been identified as one of the 10 most serious educational problems in the United States (Zhang et al., 2004). It has been linked to a range of negative outcomes such as substance use (Henry & Huizinga, 2007; Best et al., 2006; Hallfors et al., 2002), poor performance at school (Petrides et al., 2005; Bosworth, 1994), drop out of school (Alexander et al., 1997; Kaplan et al., 1995), and psychiatric disorders (Egger et al., 2003; Fergusson et al., 1995). For example, adolescents who skipped school were 3 to 6 times more likely to use substances than adolescents who did not skip school (Hallfors et al., 2002). Frequency of truancy has found to be negatively correlated with performance at school, even after controlling for verbal ability and personality (Petrides et al., 2005). Egger et al. (2003) found that a quarter of children who skipped classes had at least one psychiatric disorder compared to 6.8% of children who never skipped classes. They found independent relationships between truancy and conduct disorder, oppositional defiant disorder, and depression. These findings clearly make truancy a topic of major concern.

Truancy is not an isolated behavior as it occurs in a broader context of antisocial behaviors (ASB) (Achenbach, 1991). Research in the field of ASB has started to focus on aggressive versus non-aggressive ASB (Bartels et al., 2003; Gelhorn et al., 2005; Eley et al., 1999), with truancy being a part of the latter (Achenbach, 1991). In the present study, we focused on truancy because ASB is especially likely to occur in situations of unsupervised and unstructured time with peers (Osgood & Anderson, 2004; Osgood et al., 1996). Truancy is distinct from other non-aggressive ASB because it provides such a context in which adolescents are especially likely to initiate various kinds of ASB (Henry & Huizinga, 2007). Indeed, truancy was found to be a first sign and powerful predictor of both aggressive and non-aggressive ASB with truants being more likely further penetrating the juvenile justice system (Zhang et al., 2004), and with truancy increasing the odds of initiation of drug use (Henry & Huizinga, 2007). Since truancy provides a context for the onset of various kinds of ASB, preventing high-school students from truancy might prevent them from engaging in other kinds of ASB as well. From a clinical perspective it is therefore important to understand the etiology of truancy. The causes of individual

differences in frequency of truancy, however, are understudied. Therefore, the aim of the present study was to examine the relative influence of genetic and environmental factors on frequency of truancy.

There are sex differences in prevalence of truancy. In the Netherlands, 44% of adolescent boys and 35% of girls reported to skip school during lifetime (CBS, 2004). The same pattern of sex differences has been found for other types of non-aggressive ASB with higher prevalence in boys than in girls (Bartels et al., 2003; Eley et al., 1999; Ligthart et al., 2005).

One behavior genetic study examined genetic influence on individual differences in truancy and found that the variation could be completely accounted for by shared (73%) and nonshared (27%) environmental influences (Gelhorn et al., 2005). However, the authors stated to interpret this result with caution, because the parameter estimates were based on a small prevalence of truancy. Genetic and shared environmental factors have been found to account for a substantial part of variation in non-aggressive ASB (Gelhorn et al., 2005; Eley et al., 1999; Eley et al., 2003; Ligthart et al., 2005).

Truancy and other types of non-aggressive ASB are often performed together with peers (Kendler et al., 2007; Moffitt, 1993), and twins are likely to grow up within the same peer group (Harris, 1995; Loehlin, 1997). Rowe (Rowe, 1995) reported that non-aggressive ASB is often performed together with a co-twin. He found that 61% of female twins and 79% of male twins reported committing one or more delinquent acts together with their co-twin. These high levels of co-action in twin pairs may be explained by environmental influences that are shared by twin pairs and not by other members of the family. In other words, the existence of a specific twin environment is plausible for frequency of truancy. However, the possible influence of environmental factors specifically shared by twins on variation in frequency of truancy or other types of ASB has not been studied before.

The relative influence of genetic, shared and nonshared environmental influences explaining variation in non-aggressive ASB differs between boys and girls. For example, variation in rule breaking behavior in boys was explained by genetic influences (79%), whereas in girls, both genetic (56%) and shared environmental factors (23%) contributed to variation in rule breaking behavior (Bartels et al., 2003). This finding may indicate that genetic influences are more penetrant in truancy behavior in males as well.

In the present study, we investigated self-reported, current or retrospective, truancy in a large sample of twins and their non-twin siblings. We examined the relative influence of genetic and environmental factors on frequency of truancy and whether the relative influence of these factors differed between males and females. Due to the large sample size this was the first study able to reliably estimate genetic and environmental

influences on frequency of truancy. The addition of non-twin siblings to the classical MZ-DZ twin design, offers several advantages: i.e. it increases statistical power to distinguish between additive genetic effects and shared environmental effects (Posthuma & Boomsma, 2000) and allows for testing of differences in prevalence between twins and singletons. Moreover, due to the addition of non-twin siblings it was possible to distinguish between environmental factors especially shared by twins and those shared by non-twin siblings. The classical twin design assumes that MZ twins are not treated more equal than DZ twins.

Methods

Subjects

Participants were registered with the Netherlands Twin Registry (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam. The data collection was part of an ongoing, large scale longitudinal study. Since 1991, families with adolescent and young adult twins were assessed every 2-3 years. In 1995, data on frequency of truancy were collected in twins and 1 or 2 additional non-twin siblings. Questionnaires were sent to participating families by mail, including 2 questionnaires for non-twin siblings. If there were more than 2 non-twin siblings in the family, those siblings who were closest in age to the twins were asked to participate. The response rate was 64%. Part of the non-response was due to the possibility that the addresses in our database were not up to date for all participants. Twins and siblings who were newly and voluntarily registered, who had participated in other data collection waves, and who were older were more likely to complete the questionnaire. In total, 4,889 twins and their non-twin siblings participated in the 1995 survey. The sample was representative of the general Dutch population with regard to educational level of the parents: 13.9% of fathers and 15.3% of mothers had a basic education at elementary school, 61.8% of fathers and 72.5% of mothers had a high-school education, and 24.3% of fathers and 12.1% of mothers had a college or university education (Koopmans et al., 1995). In the same age group of the general population, these percentages were 16.9, 60.6, and 22.5% for men respectively, and 21.5, 64.4, and 13.9% for women (CBS, 1994). Participants came from all regions of the Netherlands, including both rural and urban areas. The exact procedures have been described in detail elsewhere (Boomsma et al., 2002; Boomsma et al., 2000).

One twin pair was excluded from the analyses because of unknown zygosity and one triplet was excluded. All participating non-twin siblings were included in the analyses regardless of age except for 32 non-biological siblings and another 17 siblings

who had not reached high-school age. This resulted in a total sample of 4,835 twins and non-twin siblings. The characteristics of the sample are presented in Table 8.1. At the time of data collection 24% of the sample was in high-school. A possible explanation for the overrepresentation of female MZ twin pairs is that MZ twins and females are generally more inclined to participate in research of the NTR. For 477 twin pairs (27.7%) zygosity was determined based on blood group or DNA typing. Blood group analysis was based on 13 red cell blood group antigens. When a twin pair had one or more differences in the blood grouping profile it was designated to be a dizygotic twin pair. This is an accurate method to assess zygosity based on blood group analysis (Van Dijk et al., 1996). Zygosity for the remaining same sex twin pairs was determined on the basis of questionnaire items. The correspondence between questionnaire and DNA based zygosity was 97% (Willemsen et al., 2005).

Table 8.1

Sample characteristics			
	N		N
Total sample	4,835	MZM	283
Twins	3,406 (44% m)	DZM	232
Non-twin siblings	1,429 (49% m)	MZF	438
Total families	1,722	DZF	279
Families with twins only	441 (26%)	DOSMF	263
Families with twins + 1 sibling	1,133 (66%)	DOSFM	227
Families with twins + 2 siblings	148 (8%)	Incomplete pairs	38
	Range	M	SD
age twins	14.27 – 28.25	19.78	3.12
age siblings	12.01 – 39.47	22.26	4.97
age difference twins and siblings	--	3.84	2.34

Note. N = number of; m = male; MZM = Monozygotic male twin pairs; DZM = Dizygotic male twin pairs; MZF = Monozygotic female twin pairs; DZF = Dizygotic female twin pairs; DOSMF = opposite-sex twin pairs with males born first; DOSFM = opposite-sex twin pairs with females born first; M = mean; SD = standard deviation

Measures

To assess frequency of truancy, subjects were asked how often they skipped lessons during a whole day while in high-school. Answers could be provided on a 6-point scale. The distribution of the scale was: never (67%), 1-2 times (14%), 3-4 times (7%), 5-10 times (5%), 11-20 times (3%), and more than 20 times (4%). Because the distribution of the data was very skewed and few subjects indicated to skip lessons during a whole day more than 10 times, scores on frequency of truancy were classified into two distinct categories: 0 = never, 1 = 1 or more times.

Statistical analyses

Structural equation modeling was used to examine the relative contributions of genetic and environmental influences to frequency of truancy. A threshold model with one threshold was fitted to the raw ordinal data using the software package Mx (Neale et al., 2006). The threshold model which is a special application of structural equation modeling, assumes that a trait, measured as dichotomous, has an underlying liability with a continuous and normal distribution with a unit variance. The threshold (expressed in z-values), which is based on the prevalence of the two categories in the sample, represents the value in the latent distribution above which a given individual will endorse the next category. In the latent truancy distribution this means that a lower value of the threshold represents higher prevalence of truancy.

The amount of variance in the underlying liability was modeled as a function of additive genetic (A), shared environmental (C), and nonshared environmental effects (E) which can be estimated by considering the different level of genetic relatedness between MZ and DZ twin pairs and non-twin siblings. MZ twin pairs are genetically identical, whereas DZ twin pairs and non-twin siblings share on average 50% of their segregating genes. The amount of variance due to environmental effects shared by twins (T) is obtained by allowing the environmental variances to be more highly correlated among twins than among non-twin siblings (Eaves et al., 1999). The presence of environmental influences shared specifically by twins implies that twin pairs share more of their environment than non-twin siblings.

Mx was used to estimate thresholds and tetrachoric correlations in a saturated model. When the MZ correlation is higher than the DZ and the non-twin sibling correlation, it is inferred that genetic variation influences individual differences in liability. A DZ correlation higher than half the MZ correlation implies shared environmental effects. When the DZ correlation is higher than the sibling correlation a specific environment

might exist which is shared by twins but not by non-twin siblings. The remaining variation is attributed to environmental effects which are not shared by family members. The nonshared environmental variance component also includes measurement error variance.

The data were structured into entire family units consisting of 2-4 individuals (i.e. 2 twins and 1 or 2 additional siblings) with missing sibling data for families without siblings. As a first step, in a saturated model the threshold for the twins and the non-twin siblings was estimated and the correlations between twin-twin pairs, twin-sibling pairs, and sibling-sibling pairs. Because of the broad age range in our sample we adjusted for possible age effects on the prevalence of truancy by modeling age as a covariate on the threshold. The model for the prevalence of truancy consists of an intercept independent of age, which we call the threshold, plus the effect of age as in a regression equation. Consequently, the threshold can be different between groups (e.g. between twins and siblings), although the actual prevalence might be the same as a consequence of age differences between the groups. The threshold could differ as a function of sex. Within a series of nested models we first tested the significance of the age effect on the threshold by testing whether fixing the beta-weight to zero led to a significant deterioration of model fit. Second, we tested whether the threshold is equal across zygosity groups, twins and siblings, and sex by constraining the corresponding thresholds to be equal. Finally, we tested whether twin correlations were equal across MZ and DZ twins, and DZ twins and siblings by constraining the corresponding twin correlations to be equal.

Next, genetic models were fitted to the data. We started with testing a full ACTE model (see figure 8.1) against the saturated model (i.e. model without any constraints). In the ACTE model the shared environment (represented by C) is perfectly correlated in both twins and their non-twin siblings, whereas the environment shared specifically by twins (represented by T) is perfectly correlated only by twins (Eaves et al., 1999). Because twins and their non-twin siblings are differently influenced by their shared environment (i.e. twins share more environment than their non-twin siblings) the model assumes that twins and siblings will differ in correlation, but not in variance (Eaves et al., 1999). Modeling the variance component T this way implies that the magnitude of T will contribute to the variance of siblings too. The model implies that the environmental factors comprised within T are part of the nonshared environmental variance for non-twin siblings. Sex differences in the relative influence of the variance components A, C, T, and E were assessed by testing whether constraining all parameter estimates of males and females to be equal resulted in a significant deterioration of model fit. Subsequently, the statistical significance of the variance components A, C, and T was assessed by testing whether fixing the corresponding parameter estimate to zero resulted in a significant deterioration of model fit.

calculated the power based on the sample size and different family constellations in our study. The simulated power was based on the estimates of the variance components in the full ACTE model. The estimated power is supplied by Mx.

Table 8.2

Model fitting results for the saturated model of frequency of truancy

Model	vs	-2LL	df	χ^2	Δdf	p
1. Saturated model	-	5598.00	4762	-	-	-
2. Significance of age effect	1	5717.51	4763	119.5	1	< .01
3. Threshold MZM = DZM and MZF = DZF	1	5599.43	4764	1.428	2	.490
4. Threshold MZM = DZM = DOSM and MZF = DZF = DOSF	3	5599.58	4766	0.148	2	.929
5. Threshold MZM = DZM = DOSM = brother and MZF = DZF = DOSF = sister	4	5616.17	4768	16.59	2	< .01
6. Threshold equal for male and female twins	4	5626.67	4767	27.09	1	< .01
7. Threshold equal for brothers and sisters	4	5609.19	4767	9.61	1	< .01
8. correlation MZM = DZM and MZF = DZF	4	5605.74	4768	6.162	2	.046
9. correlation DZM = brothers; DZF = sisters and DOS = brother-sister	4	5621.41	4769	21.83	3	< .01

Note. vs = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; Δdf = degrees of freedom of χ^2 test; p = p-value; MZM = Monozygotic male twin pairs; DZM = Dizygotic male twin pairs; MZF = Monozygotic female twin pairs; DZF = Dizygotic female twin pairs; DOS = opposite-sex twin pairs; DOSM = Male twins of opposite-sex twin pairs; DOSF = Female twins of opposite-sex twin pairs; brothers = male twin-brother pair and brother-brother pair; Sisters = female twin-sister pair and sister-sister pair; Brother-sister = Male twin-sister pair, female twin-brother pair, brother-sister pair.

Results

The results of the saturated model are presented in Table 8.2. There was a significant negative effect of age on the threshold ($\beta = -.53$), meaning that the prevalence of truancy increased with age (model 2). The threshold was equal for, MZ and DZ (model 3), and

DOS twins (model 4). The threshold was different for twins and non-twin siblings (model 5) and for male and female twins (model 6) and siblings (model 7). The prevalence of truancy was 38% for male twins, 36% for male siblings and 29% for female twins and siblings. This prevalence is similar to the prevalence of individuals of the same age group in the general Dutch population (CBS, 2004) and to what was reported in other studies (Henry & Huizinga, 2007; Fergusson et al., 1995).

Table 8.3

Twin Correlations and Confidence Intervals for Frequency of Truancy		
	r	CI
MZM	.64	.49-.76
DZM	.42	.23-.60
MZF	.73	.62-.78
DZF	.59	.41-.73
DOS	.46	.32-.58
Brothers	.28	.14-.41
Sisters	.24	.11-.35
Brother-sister	.21	.12-.30

Note. r = correlation coefficient; CI = 95% confidence interval; MZM = Monozygotic male twin pairs; DZM = Dizygotic male twin pairs; MZF = Monozygotic female twin pairs; DZF = Dizygotic female twin pairs; DOS = Opposite-sex twin pairs; Brothers = male twin-brother pair and brother-brother pair; Sisters = female twin-sister pair and sister-sister pair; Brother-sister = Male twin-sister pair, female twin-brother pair, brother-sister pair.

Twin correlations are presented in Table 8.3. MZ twin correlations were higher than DZ correlations suggesting that individual differences in liability to truancy are influenced by genes (model 8). MZ twin correlations were less than twice the DZ twin correlations, suggesting that the shared environment also has an influence. Sibling correlations were lower than DZ twin correlations (model 9). This suggests a specific twin environment for liability to truancy and therefore we started our genetic modeling with an ACTE model in which T represents a specific twin environment.

Results of genetic model fitting are presented in Table 8.4. In model 2, we tested a full ACTE model in which the variance components were estimated for males and females separately. The full genetic model provided a good fit compared to the saturated model. In model 3, we tested whether there were sex differences in the magnitude of the variance components by constraining the ACTE model of males and females to be equal.

Table 8.4
Univariate model fitting results for frequency of truancy

Model	Vs1	-2LL	df	χ^2	Δdf	p	AIC 2	A	C	T	E
1. Sat.	-	5598.00	4762	-	-	-	-	-	-	-	-
2. ACTE	Males	1	5599.71	4766	5.539	.699	-6.29	.48 (.01-.66)	.02 (.00-.31)	.15 (.03-.32)	.35 (.24-.46)
	Females							.25 (.00-.58)	.11 (.00-.31)	.36 (.17-.54)	.28 (.19-.38)
3. ACTE		2	5603.28	4770	3.568	.468	-10.72	.41 (.18-.57)	.03 (.00-.17)	.25 (.14-.36)	.31 (.24-.39)
4. ACE		3	5625.20	4771	21.92	<.01	9.20	.67 (.55-.74)	.00 (.00-.07)	-	.33 (.26-.40)
5. ATE		3	5603.43	4771	0.148	.700	-12.57	.45 (.34-.57)	-	.25 (.14-.35)	.30 (.24-.37)
6. CTE		3	5614.63	4771	11.35	<.01	-1.37	-	.24 (.16-.30)	.34 (.26-.43)	.42 (.36-.49)

Note. Applies only to chi-square tests; 2 every model was compared to the saturated model; Bold: best fitting model; vs = versus; -2LL = -2 log likelihood; df = degrees of freedom; χ^2 = chi-square test statistic; Δdf = degrees of freedom of χ^2 test; p = p-value; AIC = Akaike's Information Criterion; A = genetic variance component; C = shared environmental variance component; T = specific twin environmental variance component; E = nonshared environmental variance component; Sat. = Saturated model

There appeared to be no significant sex differences in the magnitude of the variance components explaining variation in liability to truancy.

In models 4 till 6, we tested whether fixing the specific twin environmental, the shared environmental, or the genetic parameter estimate to zero would lead to a significant deterioration in model fit. The specific twin environmental variance component was statistically significant. The effect of the shared environment was not statistically significant, and additive genetic effects were significant. The LRT tests and the AIC pointed to the ATE model as the best fitting model in which 45% of the variance in liability to truancy could be explained by genetic influences and 55% by nonshared environmental influences. 25% of the environmental influences are specifically shared by twins.

Results from the statistical power analysis indicated that there was not sufficient power to detect sex differences in the magnitude of the variance components as given in Table 8.4. Based on the sample size and different family constellations we had a power of .49 to detect these sex differences.

Discussion

In a large sample of Dutch twins and their non-twin siblings we found that the prevalence of truancy was higher for boys than for girls and that it increases with age indicating that older subjects in our sample reported to skip classes to a larger extent than younger subjects. Overall, 45% of the variance in liability to truancy was accounted for by genetic influences and 55% by environmental influences. Part of the shared environment represents influences that are shared by twins but not by non-twin siblings. In twins 45% of the variance could be accounted for by genetic influences, 25% by environmental influences shared specifically by twins and 30% by nonshared environmental influences. Environmental influences shared by all members of the family were insignificant. Because of a lack of power no sex differences in the magnitude of variance components were found.

The results link partly back to the outcomes of previous research on frequency of truancy and non-aggressive ASB in which truancy represents a distinct behavior (Zhang et al., 2004). Our finding that boys are more often truants is in line with what we expected based on previous research. It has previously been found that the prevalence of truancy (CBS, 2004) and non-aggressive ASB (Bartels et al., 2003; Eley et al., 1999; Ligthart et al., 2005) is higher in males.

Our results suggest that both genetic and environmental variation play an important role in the etiology of truancy. The magnitude of genetic influences on frequency of truancy appeared to be similar to the heritabilities found for non-aggressive

ASB in other studies (e.g. Gelhorn et al., 2005; Eley et al., 2003), which is in line with our expectation because truancy occurs in a broader context of non-aggressive ASB (Achenbach, 1991). The influence of environment shared by all family members (i.e. C) was insignificant which is not in line with other studies regarding truancy (Gelhorn et al., 2005) and non-aggressive ASB (Bartels et al., 2003; Eley et al., 1999; Eley et al., 2003; Ligthart et al., 2005). However, we found that a significant proportion of the environmental factors that explain variation in frequency of truancy are specifically shared by twins. These environmental factors can not be explained by age differences between twins and siblings, because they remained significant after adjusting for age effects on the prevalence of truancy. Given that twins share classrooms in many cases they are more likely to skip classes together than regular siblings who do not share classrooms or at least less often than twins. More generally, individuals being in the same situation show similar behaviors. Twins are more likely to be in the same situation than regular siblings. This is likely not only to hold for truancy but for many other behaviors as well. For example, it has previously been demonstrated that delinquent acts are often performed by twins together with a co-twin (Rowe, 1995). It is plausible that previous findings of environmental influences shared by all family members explaining variation in non-aggressive ASB in fact represent environmental influences specifically shared by individuals in the same situation (in our case twins). Because previous studies did not include non-twin siblings they were not able to detect this specific environmental effect for other environmental effects. An important implication for future research with regard to frequency of truancy and related behaviors is that environmental influences shared specifically by twins should be assessed by adding non-twin siblings to the classical twin study.

Participants ranged between 12 and 39 years, so many of the twins and siblings already reached an age at which they left high-school. As a consequence, reports of frequency of truancy may be biased because participants who were still in high-school may have had less chance for truancy and those who already left high-school may not remember exactly how often they skipped school while in high-school (Collins et al., 1985). To overcome this bias, we adjusted for age effects on the prevalence of truancy in the genetic models. Additional analyses indicated that there was no specific effect of being in high-school versus being not in high-school on the prevalence of truancy in addition to the age effect. Moreover, genetic and environmental influences on frequency of truancy were found not to differ between participants who were still in high school compared to those who already left high school (i.e. current vs. retrospective reports).

A couple of limitations of the present study should be mentioned. First, frequency of truancy was assessed by self-report. This may lead to a bias because participants may

be more reluctant to provide true information about frequency of truancy because it is deviant behavior. The prevalence of truancy may therefore be underestimated (Kazemian & Farrington, 2005; Ensminger et al., 2007). Future research with regard to truancy should therefore use other informants like teachers or other school agents who may have a more objective view on students' frequency of truancy. Second, frequency of truancy was analyzed as a dichotomous variable, whereas analyzing it as an ordinal variable with 3 or more categories may have provided more information. The genetic and environmental architecture of frequency of truancy might be different between participants skipping classes occasionally and those skipping classes frequently. Making use of larger samples with twins and non-twin siblings, future studies should investigate whether this is the case with regard to truancy and other ASB.

On the basis of the present data the conclusion seems justified that genetic, nonshared environmental, and environmental influences shared specifically by twins play a role in the etiology of truancy. Because truancy occurs in a broader context of non-aggressive ASB, it is plausible that a genetic liability towards truancy applies to other non-aggressive ASB as well. Since truancy is a first sign and powerful predictor of ASB (Zhang et al., 2004) and truancy provides a context of unsupervised and unstructured time with peers in which ASB is likely to be initiated (Henry & Huizinga, 2007), an important implication of these results is that preventing high-school students from truancy is also likely to prevent them from the onset of other kinds of ASB. The influence of environmental factors specifically shared by twins on frequency of truancy emphasizes that truancy is performed together with classmates and other peers. Therefore, another implication of the present study is that prevention of truancy should focus on high-school students skipping classes together rather than on the individual truant.

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9

Data Collection Procedures And Study Sample

This thesis is based on data from an ongoing longitudinal study on behavior, wellbeing, lifestyle, and health in adolescent twins and their family members registered at the Netherlands Twin Register (NTR). The NTR is maintained by the Department of Biological Psychology at the Vrije Universiteit in Amsterdam, and it was established in 1987 (Boomsma et al., 2006; Bartels et al., 2007). The twin register can be divided into two parts: (1) the Young Netherlands Twin Register (Y-NTR) which focuses on twins (and their family members) from birth until twins reach the age of 18, and (2) the Adult Netherlands Twin Register (A-NTR), which focuses on adult twins (from 18 years onwards) and their family members and spouses. Data collection procedure strategies of both parts of the twin register follow different procedures. The sample of adolescent twins and their non-twin siblings, where this thesis is primarily based on is part of the Y-NTR. In this chapter I will give a detailed description of the data collection procedures.

In the Y-NTR, young twins and multiples are recruited within the first few months after birth with the help of an organization that visits parents of newborns (Felicitas B.V.) and the ‘Dutch association for parents of multiples’ (NVOM; Nederlandse Vereniging voor Ouders van Meerlingen) which provide contact information of families with newborn twins and multiples. The Y-NTR contacts these families with the request for registration in the Y-NTR and sends them a registration form to complete the initially obtained address information, a TWINFO (the yearly news bulletin of the NTR), and the first questionnaire, containing questions about the pregnancy, delivery and first weeks of the twin.

Data collection procedures in the Y-NTR are cohort driven. Parents of twins in the Y-NTR receive questionnaires when their twins are 1, 2, 3, 5, 7, 9, and 12 years of age. In addition, at ages 7, 9, and 12, teachers also receive a questionnaire, after written permission is given by the parents. In 2005 the Y-NTR started to collect data among adolescent twins and their non-twin siblings. During adolescence, twins and their non-twin siblings are invited to complete a self-report survey when the twins are 14 and 16 years. Before 2007, 18-year old twins and their non-twin siblings were also invited to complete the self-report questionnaire. Thereafter, twins registered with the Y-NTR enroll into the longitudinal survey collection of the A-NTR when they reach the age of 18.

PARENTAL CONSENT

Before inviting twins and their non-twin siblings to provide self-report data, parents are contacted to ask for written permission to send their children a self-report survey and to register non-twin siblings of the twins. From the onset of data collection among adolescents in 2005, different procedures have been followed to collect informed consent

(IC) from the parents and to register non-twin siblings. These procedures are described in detail in this section. In Table 9.1, an overview of the different parental consent mailings (categorized by birth cohort of the twins) and the response rates is presented.

Table 9.1

Overview and response rates of parental consent collection

Mailing	Cohort	Invited	Response
2005 – 2006	1986 – 1992	7,665	43%
2007	1989 – 1994	7,241	40%
2009	1994 – 1996	3,204	35% ^a

Note. ^a This mailing is still ongoing; Mailing = year(s) of the mailing; Cohort = birth cohorts that were selected for a mailing; Invited = number of families to which a parental consent form was sent; Response = response rate of a mailing.

IC Procedure I

In 2005 and 2006, three questionnaires along with a parental consent form (see Appendix I) attached to them were sent to the parents of all twins registered with the Y-NTR when they were 14, 16 or 18 years old. In the accompanying letter (see Appendix II), parents were asked if they allowed their children to fill in the self-report questionnaire. If parents gave permission they had to sign the parental consent form attached to the questionnaires before handing over the booklet to their offspring. In this way, parental consent forms were collected when the completed surveys were returned by the twins. Parents were also asked to sign the parental consent form attached to the additional questionnaire if the twins had a non-twin sibling who was willing to participate. A non-twin sibling was registered with the Y-NTR when he/she returned the survey and if he or she was not already in the database. In Table 9.1 it can be seen that in 2005 and 2006, parental consent forms were collected and non-twin siblings were registered among a total of 7,665 families with twins born between 1986 and 1992. The overall family-wise response rate was 43%.

IC Procedure II

In 2007, in parallel with the release of a new sophisticated twin-family database (Boomsma et al., 2008), a new procedure was introduced to ask parents for written permission. From this moment onwards, parental consent was not obtained simultaneously with the questionnaires. Instead, written permission from the parents was obtained and non-twin siblings were registered before twins and non-twin siblings were invited to participate in

self-report questionnaire research, according to the following procedure. Parents of twins aged 13 or 15 years, registered with the Y-NTR, were contacted by a letter (see Appendix III) in which they were asked to return the completed parental consent form to the Y-NTR regardless of whether or not they consented. At the accompanying parental consent form (see Appendix IV) parents could indicate whether or not they allowed the Y-NTR to invite their children to participate in self-report questionnaire research. If the twins had any siblings who were also willing to participate in this research, their contact information could be provided on this form which was subsequently used to register the siblings to the Y-NTR. Parents could indicate if one of the twins was not able to fill out a questionnaire by themselves, because of illness or a handicap. In this case, this member of the twin pair was not invited to participate in self-report questionnaire research. If parents did not return the consent form within one or two months, they were contacted by mail for a first reminder. If, one month after the first reminder, they still did not return the consent form, the parents were contacted by phone for a second reminder. With the introduction of this procedure in 2007, parents of all adolescent twins born between 1989 and 1994 were invited to complete the parental consent form, resulting in a total of 7,241 families (see Table 9.1). By the end of 2008, after the two reminders, the response rate was 40%. Of the parents who returned the parental consent form 90% permitted the Y-NTR to send their children a self-report survey. For 46 individual twins, their parents indicated that they were not able to fill out a questionnaire by themselves because of illness or handicap. These twins were not invited to participate in self-report questionnaire research.

IC Procedure III

In 2009 and 2010 the protocol to ask parents for written permission was integrated with parental consent for contacting the teachers of the twins and siblings. Parents of 12-year old twins registered with the Y-NTR were contacted by a letter (see Appendix V) in which they were asked to return the completed parental consent form to the Y-NTR regardless of whether or not they consented. At the accompanying parental consent form (see Appendix VI) parents could indicate whether or not they permitted the Y-NTR to send the teachers of the twins a survey and to invite their children to participate in self-report questionnaire research from the age of 14 onwards. If the twins had any siblings who were also willing to participate in this research, their contact information could be provided on this form which was subsequently used to register the siblings to the Y-NTR. Parents could indicate if one of the twins was not able to fill out a questionnaire by themselves because of illness or a handicap. If parents did not return the consent form within one or two months, they were contacted by mail with a first reminder. If parents did not return

the consent form at the time the twins were 13-years old, they were reminded by a second letter (see Appendix VII) in which they were asked to complete the consent form online. The letter contained a hyperlink to the online consent form in combination with a log-in code and an identification number which was used by the Y-NTR to link the consent form to the corresponding family. At the end of 2009 parents of adolescent twins born in 1995 and 1996 were invited to complete the parental consent form (see Table 9.1). In addition, parents of twins born in 1994 who did not return the parental consent form in the 2007 mailing were also invited. This resulted in a total selection of 3,204 families. At the time of writing this thesis, the procedure was still ongoing. At the end of August 2010, the response rate was 35%. Of the parents who returned the parental consent form 90% permitted the Y-NTR to send their children a self-report survey. For 3 individual twins, their parents indicated that they were not able to fill out a questionnaire by themselves because of illness or handicap.

Table 9.2

Overview of available data and response rate of self-report survey collection at September 1st 2010

Survey	Cohort	Paper & pencil	Online	Twins	Siblings	Response
Pilot DHBQ	1987 – 1988	724	--	583	141	70%
Age 14	1990	1,232	--	999	233	49%
	1991 – 1992	2,233	--	1,855	378	42%
	1993 – 1994	803	1,281	1,617	467	70%
Age 16	1988	831	--	673	158	40%
	1989 – 1990	1,733	--	1,392	341	37%
	1991 – 1992	234	1,204	1,063	375	55%
Age 18	1986	162	--	132	30	44%
	1987 – 1988	1,814	--	1,422	392	33%
Total	1986 – 1994	9,766	2,485	9,736	2,515	47%

Note. Cohort = birth cohorts that were selected; Paper & Pencil = number of individuals who completed the paper & pencil version of the DHBQ; Online = number of individuals who completed the online version of DHBQ; Twins = number of twins who completed the DHBQ; Siblings = number of non-twin siblings who completed the DHBQ; Response = response rate.

SELF-REPORT SURVEY: DHBQ

The Dutch Health and Behavior Questionnaire (DHBQ) was developed at the department of Biological Psychology of the VU University. It is a collection of standardized questions

targeting multiple domains. The survey focuses on emotional and behavioural problems, subjective wellbeing, lifestyle, exercise behavior and family functioning. In addition, demographic characteristics and age-related information (pubertal development) is collected in the DHBQ. A detailed overview and description of all items and scales in the DHBQ is presented in Appendix VIII. In 2005 a subsample of twins born in 1986 – 1987 and their non-twin siblings was selected for a pilot study of the DHBQ. After two reminders the response rate of the pilot study was 70% (see Table 9.2). Based on the positive response rate and positive feedback of the pilot sample, the DHBQ was integrated in the longitudinal survey study of the Y-NTR. Upon parents consent, 14 and 16 year old twins and their non-twin siblings received the self-report survey. Before 2009, 18-year old twins and their non-twin siblings were also invited to complete this survey. Two different procedures have been followed to collect the self-report surveys from the adolescent twins and their non-twin siblings. Both procedures are described in detail below. In Table 9.2, an overview of the survey collection (categorized by birth cohort of the twins) and the response rates is presented.

DHBQ Procedure I

Following the procedure which was used in 2005 and 2006, three questionnaires with a parental consent form attached were sent to the parents of all twins registered with the Y-NTR. This was done when twins were 14, 16 or 18-years of age. If parents consented, they had to sign the parental consent form attached to the questionnaire and to hand the booklet over to the twins and/or to a non-twin sibling of the twins if they had any. The sibling had to be over 12 years of age. Families with more than one sibling were invited to contact the Y-NTR for additional surveys. A letter (see Appendix IX) accompanied each questionnaire in which the twins and/or sibling were invited to complete the self-report survey and return it by mail to the Y-NTR. In addition, a flyer (Appendix X) was also included in which information about the Y-NTR and the survey collection among adolescents was provided. If twins did not return the survey within one or two months, their parents were contacted by mail for a first reminder. If the survey was not returned one month after the first reminder, parents were contacted by phone and they were asked to remind their children to complete the survey. In two subsequent mailings (2005 & 2006) the procedure was repeated for the survey collection among twins born between 1986 and 1992 and their non-twin siblings. As can be seen in Table 9.2 response rates for these mailing ranged between 33 – 49%.

DHBQ Procedure II

A new procedure for the survey collection among adolescents was introduced in 2007. Parental permission was obtained and non-twin siblings were registered before the actual survey collection. Twins, for whom parental consent was obtained, were personally invited to participate in our self-report survey research when they were 14 and 16 years, as well as their siblings who had been registered with the Y-NTR. Siblings were not invited if they were younger than 12 years. The content of the DHBQ was also slightly changed and twins (and their siblings) received different versions of the DHBQ at ages 14 and 16. In the version for the 16-year olds the NEO Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992) was included. In order not to make the survey too long, items regarding religion, stuttering, type of transportation to school, and hair/eye color were excluded in the survey for 16-year olds. These items remain in the survey for 14 year olds.

In 2007, both versions of the DHBQ became available online. A personal invitation letter (see Appendix XI) was sent to the participating twins and their siblings with an accompanying information flyer (Appendix XII). The invitation letter contained a hyperlink to the online questionnaire in combination with a personal log-in code and a password which could be used to log-in the online survey. If participants preferred a paper and pencil version of the DHBQ they could contact the Y-NTR. All participants who did not complete the online survey after two months were reminded by mail. If, one month after the reminder, participants still did not complete the online survey a paper and pencil version of the DHBQ was sent to them by mail. This procedure in which the online survey was combined with the paper and pencil survey was used for survey collection among twins born between 1991 and 1994. In Table 9.2 can be seen that the response rate for 14-year old twins and their non-twin siblings was 70%, and for 16-year old twin and their non-twin siblings it was 55%.

Sample

Using these ongoing survey collection procedures, a total of 12,251 surveys among adolescent twins ($N = 9,736$) born between 1986 and 1994 and their non-twin siblings ($N = 2,515$) have been collected at the time of writing this thesis. The overall response rate was 47%. This response rate is likely to be an underestimation of the true response rate, as we do not know exactly the number of potential participants who never received the surveys because it was not sent to the correct address. Data on at least one measurement occasion have become available for 8,212 individuals, i.e. 6,465 twins and 1,747 non-twin siblings. Data on 2 or 3 measurement occasions were available for 29% of the individuals

in the sample. As the data collection is an ongoing process and the sample has become larger during the past years, different studies which are presented in this thesis are based on different subsamples.

NONRESPONSE ANALYSES

Moderate to low response rates of the parental consent collection (see Table 9.1) and the survey collection (see Table 9.2) are a serious matter of concern. If nonresponse is not a random process it may seriously limit the validity of the findings. To provide insight into the magnitude of possible response bias, the group of nonrespondents was compared to the respondents with respect to survey information that was provided by parents of the twins throughout childhood. Respondents and nonrespondents were compared on a number of different characteristics, i.e. the number of childhood surveys that were returned by parents, written permission for contacting the teachers of the twins, socioeconomic status (SES), parental educational level, religiousness, smoking and drinking behavior during pregnancy, emotional and behavioral problems of the twins at 3 ages (7, 10, 12), unhappiness at 3 ages, educational achievement of the twins, and participation in survey research of the A-NTR. Response analyses were conducted in two different ways. First, these characteristics were compared between families who returned the parental consent form versus those who did not return the form. Second, the same characteristics were compared between twins who completed the self-report survey versus those who did not.

Nonresponse parental consent

Table 9.3 presents the results of the comparisons between families who returned the parental consent form versus those who did not. Parents who returned the parental consent form participated more often in previous childhood survey research of the Y-NTR compared to those who did not return the form: 74% of parents who returned the informed consent form participated in at least six out of seven childhood surveys versus 31% of the parents who did not. The majority of nonrespondents participated in the early childhood surveys and dropped out in subsequent surveys, whereas the majority of respondents completed all childhood surveys. Informed consent forms for contacting the teachers of the twins at ages 7, 10, and 12 were less often returned to the Y-NTR by nonresponding families (75 – 83%) compared to responding families (38 – 49%), indicating that these parents do not specifically object against adolescent self-report but object against other raters than themselves in general. Again, we do not know exactly how many families received surveys and how many surveys may have been sent to the wrong address.

Table 9.3

Prevalence and Means (SD) of childhood survey history, family, parental, and childhood variables for families who returned the parental consent form compared to those who did not return the parental consent form

	Nonrespondents	Respondents
Childhood survey history		
% Returned \geq 6 childhood surveys	31	74
% Not Returned informed consent for teacher report at age 7 / 10 / 12	75 / 71 / 83	38 / 33 / 49
Family characteristics		
% Religious	67	69
% Low / Average / High SES	4 / 68 / 27	3 / 58 / 39
Parental characteristics		
% Lower vocational education (mothers/fathers)	49 / 42	33 / 32
% Intermediate vocational education (mothers/fathers)	37 / 35	43 / 34
% Higher/scientific education (mothers/fathers)	14 / 23	24 / 34
% Smoking during pregnancy (mothers/fathers)	33 / 42	22 / 34
% alcohol consumption during pregnancy (mothers)	18	22
Twin characteristics during childhood		
Mean level (standard deviation) of Internalizing at age 3 ^a	4.9 (4.0)	4.7 (3.9)
Mean level (standard deviation) of Internalizing at age 7 ^a	4.9 (4.7)	4.9 (4.7)
Mean level (standard deviation) of Internalizing at age 12 ^a	4.8 (5.8)	4.8 (5.4)
Mean level (standard deviation) of Externalizing at age 3 ^a	17.3 (10.1)	16.5 (9.9) [*]
Mean level (standard deviation) of Externalizing at age 7 ^a	8.6 (7.3)	7.8 (6.9) [*]
Mean level (standard deviation) of Externalizing at age 12 ^a	6.8 (7.1)	6.2 (6.5)
% little unhappy or unhappy at age 3 / 7 / 12	5 / 9 / 14	4 / 8 / 11
Mean (standard deviation) CITO score ^a	536.7 (8.8)	537.8 (8.7) [*]
% Language / arithmetic: insufficient or weak	10 / 8	7 / 6
% Language / arithmetic: sufficient	37 / 36	35 / 34
% Language / arithmetic: good or very good	53 / 56	57 / 60

Note. ^{*} Significant difference in mean score between nonrespondents and respondents ($p < .01$); ^a Because results of ANOVAs did not differ between first- and second born twins, means and standard deviation are only presented for first born twins.

Table 9.4

Prevalence and Means (SD) of childhood survey history, family, parental, and childhood variables for twins who completed the self-report survey compared to those who did not complete the self-report survey

	Nonrespondents	Respondents
Childhood survey history		
% Returned \geq 6 childhood surveys	59	78
Family characteristics		
% Religious	67	67
% Low / Average / High SES	4 / 65 / 31	3 / 54 / 43
Parental characteristics		
% Lower vocational education (mothers/fathers)	38 / 35	31 / 32
% Intermediate vocational education (mothers/fathers)	39 / 39	42 / 31
% Higher/scientific education (mothers/fathers)	22 / 26	27 / 37
% Smoking during pregnancy (mothers/fathers)	27 / 41	20 / 30
% alcohol consumption during pregnancy (mothers)	22	21
Twin characteristics during childhood		
Mean level (standard deviation) of Internalizing at age 3 ^a	4.8 (3.9)	4.6 (3.8)
Mean level (standard deviation) of Internalizing at age 7 ^a	5.4 (4.8)	4.8 (4.8)
Mean level (standard deviation) of Internalizing at age 12 ^a	4.6 (5.4)	4.2 (4.9)
Mean level (standard deviation) of Externalizing at age 3 ^a	17.4 (10.2)	16.4 (9.8)
Mean level (standard deviation) of Externalizing at age 7 ^a	8.6 (7.3)	7.4 (6.6) [*]
Mean level (standard deviation) of Externalizing at age 12 ^a	6.5 (6.8)	5.0 (5.3) [*]
% little unhappy or unhappy at age 3 / 7 / 12	3 / 9 / 3	4 / 7 / 4
Mean (standard deviation) CITO score ^a	536.4 (8.8)	538.3 (8.5) [*]
% Language / arithmetic: insufficient or weak	9 / 8	8 / 5
% Language / arithmetic: sufficient	35 / 34	34 / 34
% Language / arithmetic: good or very good	56 / 58	58 / 61

Note. ^{*} Significant difference in mean score between nonrespondents and respondents ($p < .01$); ^a Because results of ANOVAs did not differ between first- and second born twins, means and standard deviation are only presented for first born twins.

Religiousness was similar for nonresponding and responding families, i.e. ~30% of the families were nonreligious whereas ~70% reported to be actively or non-actively engaged in religion. With regard to SES, families were classified as having low, average or high SES based on survey information. Small differences were observed with responding families being better represented in the highest SES class compared to nonresponding families, and nonresponding families being better represented in the average SES class. In the table can be seen that each of the three classes of parental educational level was well represented in respondents and nonrespondents. Small differences can also be observed with respondents being better represented in the highest class of parental educational level compared to nonrespondents.

With regard to smoking and drinking behavior during pregnancy, the prevalence of smoking cigarettes during pregnancy differed for mothers (33% versus 22%) and fathers (42% versus 34%) from nonresponding families compared to those from responding families. However, the prevalence of consuming alcohol during pregnancy was nearly similar for nonrespondents and respondents (18% versus 22% respectively).

Information on emotional and behavioral problems in twins was obtained with the Child Behavior Checklist (CBCL; Achenbach, 1991). For responding and nonresponding families, mean levels and standard deviations of the broadband scales Internalizing and Externalizing obtained with the CBCL at ages 3, 7, and 12 are presented in the table. ANOVAs did not find differences between first- and second born twins, thus means and standard deviations were only presented for first-born twins. At ages 3, 7, and 12, mean levels of Internalizing were equal between twins from nonresponding families compared to those from responding families. Mean levels of Externalizing at ages 3 and 7 were higher for twins from nonresponding families compared to those from responding families, whereas no significant differences were found between responding and nonresponding families at age 12. Although differences in mean levels of Externalizing were significant at age 3 and 7, the differences were relatively small, with mean differences of less than 1 point on a scale ranging between from 0 to 62 (age 3) or 66 (age 7). One item of the CBCL assesses unhappiness (i.e. 'unhappy, sad or depressed'). At ages 3, 7, and 12, nonrespondents and respondents reported similar prevalence of unhappiness in twins.

Educational achievement was measured by means of CITO scores that were available for a large number of twins in the Y-NTR. The CITO is a national test of educational achievement in The Netherlands, administered to the majority of children in the last class of elementary school around age 12. The CITO assesses four different intellectual skills: language, mathematics, information processing, and world orientation. Together these performance scales result in a standardized score between 501 and 550. Although the difference in mean CITO score was significant the difference was small,

i.e. 1.1 point on a total range of 50 points. Besides the CITO score, information on achievement in language and arithmetic was rated by parents of twins on a five-point scale ranging from 'insufficient' to 'very good'. Achievement on language and arithmetic was similar for twins from responding families and those from nonresponding families.

Finally, an important piece of information comes from the longitudinal survey collection of the A-NTR. Since 2009 Y-NTR twin families are approached for participation in A-NTR surveys when the twins reach the age of 18. This invitation is sent out regardless of whether or not the adolescent twins returned the DHBQ. Thirteen percent of twins from nonresponding families completed the A-NTR survey (i.e. survey 8), compared to 44% of twins from responding families. Thus, even non-responders do not necessarily remain non-responders.

Nonresponse adolescent self-report

Response analyses were repeated among twins whose parents gave permission to invite their children to complete a self-report survey. Within this group, twins who completed the self-report survey were compared to those who were invited but did not complete the self-report survey. Table 9.4 presents the results of these comparisons. Parents of twins who completed the self-report survey participated more often in the childhood surveys compared to the parents of twins who did not complete the self-report survey: 78% of parents of responding twins participated in at least six out of seven childhood surveys versus 59% of parents of nonresponding twins. A relatively larger number of parents from nonresponding twins dropped out survey research during middle and late childhood compared to parents from responding twins.

Religiousness was similar for families with nonresponding twins and those with responding twins, i.e. 33% of the families were nonreligious whereas 67% reported to be actively or non-actively engaged in religion. With regard to SES, small differences were observed between respondents and nonrespondents, i.e. families with responding twins were better represented in the highest SES class, whereas families with nonresponding twins were better represented in the average SES class. Small differences can also be observed for parental educational level, i.e. parents of responding twins were somewhat better represented in the highest class of parental educational level compared to nonresponding twins.

With regard to smoking and drinking behavior during pregnancy, the prevalence of smoking cigarettes during pregnancy was higher for mothers and fathers from

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nonresponding twins compared to those from responding twins. However, the prevalence of mothers' alcohol consumption during pregnancy was nearly equal for nonresponding and responding twins.

For responding and nonresponding twins, mean levels and standard deviations of the broadband scales Internalizing and Externalizing obtained with the CBCL at ages 3, 7, and 12 are presented in the table. Because results of the ANOVAs did not differ between first- and second-born twins, means and standard deviations were only presented for first-born twins. At ages 3, 7, and 12, mean levels of Internalizing were equal for nonresponding compared to responding twins. Mean levels of Externalizing at ages 7 and 12 were higher for nonresponding twins compared to responding twins, whereas no differences were found between responding and nonresponding twins at age 3. Although differences in mean levels of Externalizing were significant at age 7 and 12, the differences were relatively small, with mean differences of 1.5 point or less on a scale ranging between from 0 to 66. At ages 3, 7, and 12, similar prevalence of unhappiness was reported for nonrespondents compared to respondents.

Mean CITO scores were significantly lower for nonrespondents compared to respondents, although the difference was very small, i.e. 1.9 point on a total range of 50 points. Achievement on language and arithmetic, as was reported by parents, was similar for responding and nonresponding twins.

When twins reached the age of 18, they were invited to participate in A-NTR surveys regardless of whether or not they returned the DHBQ. Eleven percent of nonresponding twins completed the latest survey (i.e. survey 8) of the A-NTR, compared to 51% of responding twins.

Taken together, nonresponse analyses showed that parents who did not return the parental consent form were also less willing to participate in survey research of the Y-NTR during childhood compared to parents who did return the informed consent form. The same holds for parents of adolescent twins who were invited to participate in self-report research but did not complete the self-report survey. Observed differences were relatively small, i.e. respondents and nonrespondents were fairly comparable. In other words, response bias does not constitute a serious threat to the validity of the results in this thesis. It is important to note that a small proportion of the nonrespondents returned to the study when they reached the age of 18.

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10

Summary and General Discussion

Summary

Subjective wellbeing (SWB) is characterized by a positive sense of wellbeing which is highly subjective and non-specific in such a way that it is concerned with abstract feelings that individuals have about themselves in a general sense. Individual levels of SWB show substantial stability over time (Cummins, 2010; Wagner et al., 2007; Lucas, 2007; Huebner, 2004; Lykken & Tellegen, 1996). SWB is a moderately heritable trait with genetic factors typically accounting for 40 – 55% of the individual differences (Caprara et al., 2009; Bartels & Boomsma, 2009; Nes et al., 2006; Stubbe et al., 2005; Røsbjerg et al., 2002; Lykken & Tellegen, 1996; Tellegen et al., 1988). An extensive literature on many aspects of adult SWB has been developed, whereas studies on adolescent SWB have only emerged recently. This thesis aimed to provide insight into the causes of individual differences in adolescent SWB. To this end, data on SWB and on factors that may be associated with SWB, such as family functioning, exercise behavior, sedentary behavior, internet use, psychopathology, and truancy, were analyzed. The genetic architecture of these associated factors was investigated. In addition, it was assessed whether the associations between SWB and these factors reflected causal effects or whether genetic factors accounted for the relationships by genetic pleiotropic effects and / or gene-environment interaction. Data were analyzed in a large sample of Dutch adolescent twins and their non-twin siblings, registered with the Netherlands Twin Registry. Data collection is ongoing and the content of the surveys changed throughout the period this thesis was written. Therefore, the different studies in this thesis were based on different subsamples.

Introductory chapter

The first chapter of this thesis served as an introduction into the concept of SWB and reviews the current knowledge regarding genetic influences on SWB. In addition, factors associated with adolescent SWB which were considered throughout this thesis and the possible mechanisms through which these factors may affect individual differences in adolescent SWB were introduced.

Family environment and SWB

Chapter two focused on the relation between SWB and different aspects from adolescents' family environment, such as parental divorce and family functioning. We investigated the degree to which genetic and environmental factors contributed to individual differences in evaluations of family functioning and SWB, and the association between them. In addition,

we assessed whether genetic and environmental influences differed between adolescents living in intact families compared to those from divorced families. Two measures of family functioning, i.e. general family functioning (McMaster Family Assessment Device; Epstein et al., 1983) and family conflict (Family Environment Scale; Moos, 1974), and a measure of SWB, i.e. quality of life (Cantril Ladder, Cantril, 1965), were studied in a sample of 5,773 adolescent twins and 1,000 non-twin siblings.

Univariate genetic analyses showed that genetic, shared environmental, and nonshared environmental factors accounted for variation in evaluations of general family functioning and family conflict, with genetic factors being relatively more important for girls compared to boys with regard to general family functioning. Heritabilities of general family functioning were estimated at 6% for boys and 35% for girls, and heritability of family conflict at 31%. Genetic and nonshared environmental factors accounted for individual differences in quality of life, with genetic factors being relatively more important for girls (43%) compared to boys (30%). Adolescents' evaluations of family functioning and their SWB are thus partly genetic in origin.

Evidence was found for interaction between genetic factors and parental divorce: genetic influence on general family functioning was larger in participants from divorced families (boys: 14%, girls: 45%) compared to those from intact families (boys: 6%, girls: 35%). Furthermore, girls from divorced families showed larger heterogeneity in their evaluations of quality of life compared to girls from intact families, as was evident from increased variation in SWB for girls from divorced families compared to those from intact families.

Higher levels of general family functioning and lower levels of family conflict were associated with higher levels of quality of life. Bivariate genetic analyses showed that the associations between these measures were primarily accounted for by genetic factors, with nonshared environmental factors accounting for the remaining part. In addition, interaction between latent genetic factors and latent nonshared environmental factors was explored by looking at the correlations between intrapair sum and difference scores in monozygotic twin pairs (Jinks & Fulker, 1970). Variation in intrapair sumscores appears because twins belonging to different families have different genotypes and/or family environments, whereas intrapair difference scores provide an estimate of nonshared environmental influences within families. Significant negative correlations were found for evaluations of general family functioning, family conflict, and quality of life, indicating that genetic factors in adolescents' evaluations of family functioning and SWB interact with factors from their personal environment. Genetic factors are relatively more

important in those adolescents reporting higher levels of family functioning and SWB (since twin pairs reporting higher levels of family functioning and SWB were more similar) compared to those reporting lower levels of family functioning and SWB.

Exercise behavior and SWB

Chapters three and four focused on adolescent exercise behavior and its relation with SWB. We examined the degree to which genetic and environmental factors affect individual differences in exercise behavior throughout adolescence in chapter three. In a large sample of adolescent twins (i.e. 7,195 individuals) we were able to estimate the relative influence of genetic and environmental factors on self-reported leisure time exercise behavior within three different age groups (13 – 14, 15 – 16, and 17 – 19 years) and to assess quantitative as well as qualitative sex differences in the genetic architecture within these age groups. Exercise behavior was assessed with survey items about type of regular leisure time exercise, frequency, and duration of the activities. Using Ainsworth Compendium of physical activity (Ainsworth et al., 2000) a total weekly MET score was computed for each participant. Participants were classified as non-exercisers, moderate exercisers or vigorous exercisers based on their total weekly MET score. The prevalence of moderate exercise behavior declined from age 13 to 19 years with a parallel increase in the prevalence of non-exercise. However, the prevalence of vigorous exercise behavior remained constant throughout adolescence. At all ages, girls were more often non-exercisers than boys. When regularly engaged in exercise, girls more often exercised at a moderate rather than a vigorous level. The genetic analyses indicated that the genetic architecture of exercise behavior changed during adolescence for girls, such that genetic effects increased with age while shared environmental effects diminished. During early adolescence, individual differences in exercise behavior could be accounted for by genetic (boys: 85%, girls: 38%) and nonshared environmental factors (boys: 15%, girls: 16%), whereas for girls shared environmental factors accounted for a substantial part of the individual differences as well (46%). Genetic factors accounted for the largest part of the variation in exercise behavior during middle (80%) and late adolescence (72%), while the remaining variation was accounted for by nonshared environmental factors. No evidence was found for qualitative sex differences in the genetic factors, indicating that the same genetic variants appear to influence exercise behavior in boys and girls.

In chapter four, it was investigated whether exercise behavior causally influenced SWB and internalizing problems or whether the association reflected the effects of underlying genetic factors. Data on exercise behavior, internalizing problems, and SWB were available in a sample of 6,317 adolescent twins and 1,180 non-twin siblings. The

majority of the sample had longitudinal data with 2-year follow-up. Exercise behavior was assessed using the same procedure as described above and it was treated as a continuous measure. Self-reported internalizing problems were collected with the Youth Self Report (Achenbach & Rescorla, 2001). Satisfaction with life (Satisfaction with Life Scale; Diener et al., 1985), subjective happiness (Subjective Happiness Scale; Lyubomirsky & Lepper, 1999), and quality of life (Cantril Ladder; Cantril, 1965) were used as measures for SWB and these were summarized into a single factor score. In keeping with population based studies investigating adult samples (Stubbe et al., 2007; De Moor et al., 2006; Norris et al., 1992; De Moor et al., 2008; Wise et al., 2006; Camacho et al., 1991), we found that exercise behavior was cross-sectionally and longitudinally associated with increased levels of SWB and fewer internalizing problems across the total sample of adolescents. However, within genetically identical twin pairs, a twin who exercised more did not have higher levels of SWB or fewer internalizing problems than his or her less exercising co-twin. In addition, a twin showing increased frequency and intensity of exercise behavior over time did not show higher levels of SWB or lower levels of internalizing problems over time than his or her co-twin with unchanged or decreased levels of exercise behavior. The cross-sectional and longitudinal associations between exercise behavior and SWB and internalizing problems were entirely accounted for by genetic factors in boys and genetic and shared environmental factors in girls, whereas nonshared environmental factors influencing exercise participation did not affect individual differences in SWB and internalizing problems, currently or 2 years later. Based on these results, the hypothesis that increased levels of SWB and lower levels of internalizing problems found in adolescent exercisers reflect a causal effect of exercise, has to be rejected. Instead, these results are compatible with the hypothesis that the associations between exercise behavior and SWB and internalizing problems in adolescence are mainly due to underlying pleiotropic genes.

Sedentary behavior and SWB

Sedentary behavior has often been addressed as the opposite of physical activity, but research has shown this to be incorrect (e.g. Biddle, 2007; Te Velde et al. 2007). Sedentary behavior may therefore contribute to adolescents' SWB independently of their exercise participation level. Chapters five and six addressed adolescent sedentary behavior and its relation to SWB. In chapter five, a study was described in which the genetic and environmental contribution to individual differences in self-reported screen-viewing sedentary behavior during adolescence was estimated, in a sample of 5,074 adolescent twins and 937 of their non-twin siblings aged between 12 – 20 years. We assessed whether genetic and environmental influences on variation in sedentary behavior were moderated

by age. Screen-viewing sedentary behavior was assessed with survey items about weekly frequency of TV viewing, playing electronic games, and engaging in personal computer / internet activities. The results indicated that adolescents become less frequently engaged in sedentary behavior throughout adolescence and that boys were more often sedentary than girls. We observed that the overall decline in frequency of sedentary behavior during adolescence was due to a lower frequency of TV viewing. Sex differences in frequency of sedentary behavior were mainly due to a higher frequency of playing electronic games in boys. The genetic analyses indicated that genetic and environmental influences on individual differences in sedentary behavior were moderated by age, such that genetic and nonshared environmental effects increased with age while shared environmental effects diminished. Individual differences in sedentary behavior among the youngest participants in our sample (i.e. age 12) were accounted for by genetic (boys: 35%, girls: 19%), shared environmental (boys: 29%, girls: 48%), and nonshared environmental factors (boys: 36%, girls: 34%). Variation in sedentary behavior among the oldest participants in our sample (i.e. age 20) was accounted for by genetic (boys: 48%, girls: 34%) and nonshared environmental factors (boys: 52%, girls: 66%). No evidence was found for qualitative sex differences in genetic factors, indicating that the same genetic variants appear to influence sedentary behavior in boys and girls. The substantial genetic influence on sedentary behavior suggests that there is genetic liability towards sedentary activities such as TV viewing, gaming, and engagement in personal computer and internet activities.

To increase our knowledge about the impact of sedentary activities on adolescents' wellbeing, we investigated the association between internet use and low wellbeing at the phenotypic level in chapter six. It was hypothesized that (a) linkages between high levels of daily internet use and low wellbeing would be mediated by compulsive internet use, and (b) that adolescents with low levels of neuroticism, extraversion, and agreeableness would be more likely to develop compulsive internet use and lower wellbeing. In a sample of 7,888 Dutch adolescents, daily and compulsive internet use was assessed by survey items about the time spent on the internet on a daily basis and by means of the Compulsive Internet Use Scale (Meerkerk et al., 2009). Low wellbeing was operationalized in terms of loneliness (revised UCLA Loneliness Scale; Russell et al., 1980), self-esteem (Rosenberg Self-Esteem Scale; Rosenberg, 1989), and depressive moods (Depressive Mood List, Kandel & Davies, 1986). Big Five personality traits were assessed by using the Quick Big Five (Vermulst & Gerris, 2005). Results indicated that daily internet use was indirectly related to low wellbeing through compulsive internet use. In addition, daily internet use was found to be more strongly related to compulsive internet use, and compulsive internet use was more strongly linked to loneliness in emotionally less-stable, introverted, and low-agreeable adolescents.

Psychopathology and SWB

Chapter seven addressed the question whether SWB and psychopathology constitute the opposite ends of a mental health continuum or whether SWB and psychopathology constitute distinct dimensions of mental health. Although SWB has been found to be strongly negatively associated with different types of psychopathology (e.g. Proctor et al., 2009), there is also evidence that SWB and psychopathology are distinct dimensions of mental health with separate determinants (Ryff et al., 2006; Greenspoon & Saklofske, 2001; Bergsma et al., 2010). Three measures of SWB captured in one factor score, eight syndrome scales of the Youth Self-Report (YSR; Achenbach & Rescorla, 2001), measuring eight different forms of emotional and behavioral problems, and the broadband scales internalizing and externalizing behavior problems were studied in a sample of 6,381 adolescent twins and 1,195 of their non-twin siblings. Results indicated that affected individuals rate their SWB significantly lower than individuals scoring below a clinical cut-off for the syndrome scales of the YSR. If psychopathology and SWB were opposite ends of a mental health continuum, two groups should have been distinguishable: one group with participants scoring low on psychopathology and high on SWB, and another group with participants scoring high on psychopathology and low on SWB. Only 15% of our sample fit these groups, whereas the remaining 85% did not. On the phenotypic level, negative correlations ranging between $-.24$ and $-.57$ were observed between SWB and the syndrome scales. These phenotypic correlations were mainly driven by significant genetic correlations ($-.31$ to $-.71$) and to a smaller extent by overlapping environmental factors ($-.13$ to $-.42$). No significant environmental correlations between SWB and externalizing problems were found for boys. These results indicate that SWB and psychopathology are not simply opposite ends of a mental health continuum, but that they are partly independent dimensions of mental health.

Besides the association between psychopathology and SWB in general, assessing specific types of psychopathology might provide useful information as well. To this end, chapter eight examined the relative importance of genetic and environmental factors on frequency of truancy while in high school. In addition, the presence of environmental influences specifically shared by twins but not by their siblings was assessed. Data on frequency of truancy were analyzed in a sample of 3,406 twins and 1,429 non-twin siblings. Results showed that the prevalence of truancy increased with age and that boys skipped class more often than girls. Overall, individual differences in liability to truancy could be accounted for by genetic (45%) and nonshared environmental factors

(55%). For twins, the environmental variance could be partitioned into twin specific environmental (25%) and nonshared environmental factors (30%), whereas for singletons all environmental variance was nonshared (i.e. 55%).

Data collection procedures and study sample

In chapter nine, an overview of the data collection procedures and the study sample was presented. Because response rates were moderate to low, non-response analyses were conducted in which respondents and nonrespondents were compared on several characteristics, such as previous participation in survey research of the NTR, socioeconomic status (SES), parental education level, religion, smoking and alcohol consumption during pregnancy, emotional and behavioral problems of the twins at 3 ages during childhood, unhappiness, CITO scores, and educational achievement in language and arithmetic as reported by parents. Results showed that nonrespondents were less willing to participate in survey research of the Y-NTR during childhood compared to respondents. Differences between respondents and nonrespondents were found with regard to SES, parental educational level, prevalence of smoking during pregnancy, externalizing problems at ages 3, 7 and 12, and CITO scores, whereas no differences were found on religion, internalizing problems and unhappiness at ages 3, 7, and 12 and educational achievement on language and arithmetic as was reported by parents. It is important to note that whenever differences between respondents and nonrespondents were observed they were rather small.

General discussion

Research literature in the field of positive psychology has focused on many aspects of subjective wellbeing (SWB) among adults (see for reviews Diener, 1984; Diener et al., 1999). Based on this literature four main characteristics of SWB have been described: 1) it represents a general positive sense of wellbeing (e.g. Cummins, 2010); 2) it shows substantial stability over time (Cummins, 2010; Wagner et al., 2007; Lucas, 2007; Lykken & Tellegen, 1996), 3) it is strongly associated with stable personality traits (DeNeve & Cooper, 1998; Diener et al., 1999); and 4) individual differences in SWB are substantially accounted for by genetic and nonshared environmental factors with heritabilities typically ranging between 40 – 55% (Tellegen et al., 1988; Lykken & Tellegen, 1996; Røysamb et al., 2002; Stubbe et al., 2005; Nes et al., 2006; Caprara et al., 2009). Studies on adolescent SWB have only emerged recently, although studying SWB during adolescence is of particular interest because adolescence is a period of important social, neurobiological, and behavioral changes (Paus et al., 2008).

Individual differences in adolescent subjective wellbeing (SWB)

Throughout this thesis individual differences in adolescent SWB were reported to be accounted for by genetic and nonshared environmental factors. Sex differences in the genetic architecture of SWB were observed, with higher heritabilities for girls compared to boys. Different measurements of SWB resulted in similar heritabilities which were estimated at ~35% and ~45% for boys and girls respectively. No evidence was found for shared environmental effects on individual differences in SWB. These findings are consistent with the only study that also investigated causes of individual differences in SWB among adolescents (Bartels & Boomsma, 2009). In this study, using a subsample of the same dataset available for this thesis, heritabilities of four different measures of SWB (i.e. satisfaction with life, subjective happiness, quality of life in general and at the moment) ranged between 36 – 47%, which is consistent with the findings in this thesis. Although no sex differences in the genetic architecture of SWB were reported, evidence for such sex differences has been reported among adults by others (Røysamb et al., 2002; Nes et al., 2006). Heritability of SWB may be larger in girls because heritabilities of traits that are likely to influence one's evaluation of SWB, such as personality traits and depression have also been found to be larger for girls (Boomsma et al., 2000; Kendler et al., 2001; Jardine et al., 1984). In addition, Weiss et al. (2008) reported that the associations between SWB and the personality traits neuroticism, extraversion, and conscientiousness were largely accounted for by genetic factors, suggesting that genetic effects on these personality traits may affect individual differences in how soon SWB returns to its baseline level after disturbance, and the extent to which the baseline levels change in response to challenging factors.

According to the set-point theory of SWB, genetic factors affect SWB by maintaining stable baseline levels of SWB that differ between individuals (Nes, 2010; Lykken, 1999; Lykken & Tellegen, 1996; Hamer, 1996). The level of SWB is assumed to fluctuate around these baseline levels as a reaction to challenging factors that occur in people's lives, but neurological systems acting in a homeostatic fashion will maintain the stable baseline level of SWB (see Cummins, 2010). Consistent with findings among adults, our data showed adolescent SWB to be moderately stable over time. Summarizing the three measures of SWB into a single factor score, a correlation of .40 was observed between adolescent SWB assessed at two time points with a 2-year time interval. In a review of the SWB literature among children and adolescents, Huebner (2004) also reported child and adolescent life satisfaction to show moderate levels of stability. For example, Huebner et al. (2000) reported a one-year stability coefficient of .53 in a sample of high school students. Individuals' baseline levels of SWB may change when the influence

of challenging factors becomes too strong and overwhelm the homeostatic system that maintains the baseline level. As a consequence, the level of SWB will change according to the strength of the challenging factors (Cummins, 2010).

To increase our knowledge of the etiology of SWB, insight into the factors that may cause long term stability and changes in adolescent SWB should be obtained. Throughout this thesis, adolescent SWB has been found to be positively associated with evaluations of general family functioning and exercise behavior, and negatively associated with evaluations of the level of family conflict, parental divorce, daily and compulsive internet use, and different types of psychopathology (i.e. withdrawn behavior, somatic complaints, anxious/depressed, delinquent behavior, aggressive behavior, social problems, thought problems, and attention problems). Little is known, however, about the mechanisms through which these factors affect individual differences in adolescent SWB. The main aim of this thesis was to provide insight into the causes of individual differences in adolescent SWB. Three mechanisms which may explain individual differences in SWB were addressed, i.e. genetic pleiotropic effects, gene-environment interaction, and causality. Initial insight into the role of genetic factors in these associations was obtained by investigating the genetic architecture of those factors that have been associated with adolescent SWB. If individual differences in these factors are (partly) accounted for by genetic factors it is plausible that genetic effects are also responsible for at least part of the associations between SWB and these factors.

Family environment and SWB

Individual differences have been found with respect to adolescents' evaluations of family functioning and the association between family functioning and SWB is presumed to be less due to true family experiences but more to the ways adolescents perceive and interpret these events and hence their family functioning (Millikan et al., 2002; Neiderhiser et al., 1998; Harold, 1997). Results presented in this thesis showed that the way adolescents perceive and interpret family functioning is partly influenced by genetic factors. Our results were consistent with other studies investigating the genetic architecture of adolescents' evaluations of family functioning (Herndon et al., 2005; Jacobson & Rowe, 1999; Neiderhiser et al., 1998), that also reported individual differences to be partly accounted for by genetic factors.

Increased levels of quality of life were found in adolescents perceiving higher levels of general family functioning and lower levels of family conflict and vice versa. These associations were largely accounted for by genetic factors influencing adolescents' evaluations of family functioning as well as their level of SWB, whereas the contribution

of environmental factors to these associations was only marginal. The high genetic overlap might be explained by adolescents' evaluations of family functioning and their SWB being distinct components of a general way in which individuals evaluate their lives, which is partly under genetic control. The association between evaluations of family functioning and SWB may therefore be less due to true family experiences but rather to a genetically predisposed way of evaluating one's life in general. Consistent with this hypothesis, Neiderhiser et al. (1998) reported genetic factors to contribute to individual differences in adolescent perceptions of parenting and, in addition, that adolescent perceptions of parenting mediate the association between actual observed parental behavior and adolescent depressive symptoms and antisocial behavior. Although this thesis presented the first study addressing the role of genetic factors in the association between adolescents' evaluations of family functioning and SWB, effects of pleiotropic genes have also been found to be largely responsible for the association between adolescents' evaluations of family functioning and depressive symptoms (Jacobson & Rowe, 1999; Pike et al., 1996).

More evidence that events in the family do not have to be directly associated with adolescents' SWB comes from the finding that individual differences in SWB were larger for girls residing in families with divorced parents compared to those living in intact families. If parental divorce had a general negative effect on SWB, residing in divorced families would lead to more homogeneous effects with regard to SWB. However, these results suggested that negative family experiences, such as parental divorce, do not have the same effect on SWB for all adolescents, but rather that there is heterogeneity in the effects of such events on SWB. This is likely to be due to adolescents' subjective evaluations of such experiences which are partly under genetic control. This is a plausible explanation for general small effect sizes found in research investigating direct effects of negative family experiences on adolescent SWB, such as parental divorce (e.g. Amato & Keith, 1991; McFarlane et al., 1995).

Exercise behavior and SWB

The conclusion that has often been drawn from studies investigating the association between exercise behavior and wellbeing is that increased levels of subjective and psychological wellbeing found in exercisers are the reflection of causal effects of exercise. However, there is also evidence that the association between exercise and psychological wellbeing (De Moor et al., 2008, 2007) and between exercise and SWB (Stubbe et al., 2007) in the adult population is due to genetic factors influencing exercise as well as psychological and subjective wellbeing. The association between exercise behavior and SWB, however, is understudied, especially among adolescents.

To get a first insight into the role of genetic factors in the association between exercise behavior and SWB in adolescents, the genetic architecture of exercise behavior during adolescence was examined. For boys, individual differences in exercise behavior were largely accounted for by genetic factors during adolescence. In contrast, shared environmental factors accounted for a major part of individual differences in young adolescent girls, whereas from 15 years onwards the influence of these shared environmental factors completely disappeared in favor of genetic factors. These results are consistent with other studies investigating the genetic architecture of adolescent exercise behavior in which large genetic influence and diminishing shared environmental influence was reported (Stubbe et al., 2005; Maia et al., 2002; Beunen & Thomis, 1999).

In keeping with population based studies investigating adult samples (De Moor et al., 2008, 2006; Stubbe et al., 2007; Wise et al., 2006; Norris et al., 1992; Camacho et al., 1991), we found that adolescent exercise behavior was cross-sectionally and longitudinally associated with increased levels of SWB and fewer internalizing problems. The results did not support the hypothesis that increased levels of SWB and decreased levels of internalizing problems found in adolescent exercisers reflect a causal effect of exercise. Instead, the association was largely accounted for by genetic factors influencing a tendency towards exercise as well as a disposition towards high levels of SWB and low levels of internalizing problems.

It is important to note, that we do not rule out the possibility that exercise might have a causal effect on SWB for specific subgroups of adolescents. De Geus and De Moor (2008) proposed a model that may explain genetic pleiotropic effects and still allows causal effects of exercise on SWB in specific subgroups. They hypothesized that to maintain regular exercise participation, appetitive effects of exercise (e.g. mood enhancement) need to outweigh the aversive effects (e.g. fatigue). Individuals for who the aversive effects of exercise are stronger than the rewarding effects will eventually cease their exercise participation. In contrast, individuals for who the appetitive effects are stronger than the aversive effects will maintain exercise participation and become regular exercisers. It has indeed been shown that regular exercisers report greater acute exercise-induced mood enhancement than non-exercisers (Hoffman & Hoffman, 2008; Gauvin, 1990). These positive mood experiences after exercise activities may be a positive reward and therefore an important component of the higher levels of SWB reported by regular exercisers. Genetic factors tipping the balance between appetitive and aversive effects may be a major source of individual differences in the effects of exercise on these acute mood responses. These genetic factors may not only keep individuals maintaining regular exercise

participation, but the repeated exercise-induced increases in mood may also influence their overall SWB in a positive way. It is important to note that this causal effect is limited to a genetically sensitive subgroup and does not apply to the total adolescent population.

In addition, De Geus and De Moor (2008) proposed a social-psychological mechanism through which the association between exercise behavior and SWB may be explained by genetic pleiotropy and which is particularly plausible in the adolescent population. It has been suggested that the heritability of exercise behavior may partly represent genetic influences on exercise ability (Stubbe et al., 2006; De Geus & De Moor, 2008). The basic idea is that people seek out the activities that they are good in. This is particularly true for adolescent boys, because being good in sports is an important source of self-esteem for adolescent boys and the athletic role model is continuously reinforced by the media (Field et al., 1999; Pope et al., 2001). Therefore, genes coding for exercise ability may well become genes for adolescent exercise behavior. These genetic factors may further influence SWB because higher exercise ability will coincide with the experience of strong feelings of competence and mastery as well as positive feedback from peers, leading to an increased sense of self-esteem and SWB. In short, genetic variants influencing exercise ability will act to maintain exercise behavior and to increase SWB, but only for those adolescents with high exercise ability.

Sedentary behavior and SWB

Evidence from previous research has shown that sedentary behavior and physical activity are largely independent (e.g. Biddle, 2007; Te Velde et al., 2007). Thus, sedentary behavior may contribute to adolescents' SWB independently of their exercise participation level. To increase our knowledge about adolescent screen-based sedentary behavior, measured as weekly frequency of watching TV, playing electronic games, and engaging in personal computer and internet activities, and the possible mechanisms through which it may affect SWB we assessed the importance of genetic and environmental factors as determinants of sedentary behavior throughout adolescence. The genetic architecture of sedentary behavior changed during adolescence. During early adolescence, individual differences in sedentary behavior were accounted for by genetic, shared environmental, and nonshared environmental factors. Shared environmental effects on sedentary behavior diminished during middle and late adolescence, suggesting that as adolescents grow older and become more independent, their sedentary behavior increasingly depends on their genetic make-up and on factors from their personal environment. The substantial influence of genetic factors on sedentary activities such as TV viewing, gaming, and engaging in personal computer and internet activities suggests that there is a genetic liability towards such

sedentary activities. Consistent with these results, Nelson et al. (2006) reported variation in adolescent sedentary behavior to be accounted for by genetic, shared environmental, and nonshared environmental factors, whereas during the transition from adolescence to young adulthood shared environmental effects disappeared.

Some studies have investigated the association between sedentary activities and psychological and subjective wellbeing. Especially the association between daily internet use, which is one of the most popular leisure time activities among adolescents, and wellbeing has received considerable attention (e.g. Van den Eijnden et al., 2008; Ybarra et al., 2005; Wastlund et al., 2001; Weiser, 2001; Gross et al., 2002; Kraut et al., 2002; Sanders et al., 2000; Kraut et al., 1998). Some of these studies found a negative association between daily internet use and wellbeing (Van den Eijnden et al., 2008; Ybarra et al., 2005; Weiser, 2001; Kraut et al., 1998), whereas other studies found no association (Gross et al., 2002; Kraut et al., 2002; Sanders et al., 2000; Wastlund et al., 2001). Overall, results with regard to the direct association between daily internet use and adolescent wellbeing may be classified as inconclusive. The analyses showed that daily internet use, in itself, is only marginally and not directly associated with low wellbeing in adolescence, suggesting that sedentary activities such as daily internet use may not have the same effects on SWB for all adolescents. Alternatively, the impact of sedentary activities on SWB may depend on individual characteristics. Indeed, whether or not daily internet use had a negative effect on adolescents' wellbeing was clearly dependent on individuals' tendencies toward compulsive use of the internet, such as not being able to stop using the internet, internet use interfering with other duties or social contacts, and a constant preoccupation with internet use. In addition, possible negative consequences associated with adolescents' daily internet use were in part dependent on their personality constellation. For introverted, low-agreeable, and emotionally less-stable adolescents, daily internet use is more strongly associated with compulsive internet use and, in turn, compulsive internet use is more strongly linked to feelings of loneliness. These results are consistent with the results of prior research examining adolescents' internet use, in which compulsive internet use was also found to be a stronger predictor of negative outcomes than daily internet use (Van den Eijnden et al., 2008; Caplan, 2003), and in which a strong positive association was found between compulsive internet use and loneliness (Morahan-Martin & Schumacher, 2000). These results clearly suggest that internet does not affect everyone in the same way.

It has been suggested that people who differ in certain personality traits are likely to use the internet in different ways (Kraut et al., 2002). Introverted, low-agreeable, and emotionally less stable adolescents are characterized by a relatively marginal position in the peer group (Newcomb & Bagwell, 1995; Rubin et al., 1990). These personality

traits are also substantially heritable (Distel et al., 2009; Vernon et al., 2008; Loehlin et al., 1998; Riemann et al., 1997). Especially for these youth, internet use and playing online video games may be highly rewarding because many factors that make it difficult to communicate in “real life” interactions are not present in online interactions (Rabiner et al., 1990; Spears & Lea, 1994). The development and maintenance of these online interactions may increase the likelihood of excessive and compulsive use of these online sedentary resources, which in turn may lead to a further decrease in social resources in everyday life and, hence, to low psychological and subjective wellbeing (McKenna & Bargh, 1998; Caplan, 2003). In this way, genetic factors influencing personality characteristics such as neuroticism, extraversion, and agreeableness, may increase the likelihood for sedentary activities and low SWB for some subgroups of adolescents.

Psychopathology and SWB

Although SWB has been found to be strongly negatively associated with different types of psychopathology and especially with depression (see Proctor et al., 2009 for a review), there is also evidence that SWB and psychopathology are distinct dimensions of mental health with separate determinants (Ryff et al., 2006; Greenspoon & Saklofske, 2001; Bergsma et al., 2010). For example, Ryff et al. (2006) reported SWB and ill-being to be associated with different biomarkers, such as cortisol, norepinephrine, waist-hip ratio, systolic blood pressure, and HDL cholesterol, which is consistent with the hypothesis that SWB and psychopathology are distinct dimensions of mental health. Greenspoon and Saklofske (2001) reported that if SWB and psychopathology are on the same mental health continuum two groups of participants should be identified: those low on psychopathology and high on SWB, and those high on psychopathology and low on SWB. However, two additional groups were identified, i.e. those low on psychopathology and low on SWB, and those high on psychopathology and high on SWB, suggesting that psychopathology and SWB are distinct dimensions of mental health. We added to this by examining whether the association between psychopathology and SWB was driven by overlapping genetic factors, and showed that the association was indeed mainly accounted for by genetic effects. The association was also driven by overlapping environmental factors, except for externalizing problems in boys. Genetic correlations, though, outnumbered the environmental correlations which is an indication that SWB and psychopathology are not completely independent. The less than perfect genetic overlap, however, indicated that SWB and psychopathology are partly independent and not simply the opposite ends of a mental health continuum.

As was mentioned before, genetic factors are thought to affect SWB by maintaining stable baseline levels of SWB which differ between individuals. Genetic factors influencing individual differences in liability towards psychopathology are plausible challenging factors to the system protecting the baseline level of SWB. If the negative challenge becomes chronic and strong, which is the case when genetic factors predispose individuals towards psychopathology, the maintenance of baseline levels of SWB may fail, resulting in decreased levels of SWB for these individuals. Evidence for such a mechanism has been reported with regard to depression by Cummins (2010), showing that SWB was maintained at a relatively stable level with increasing depression scores. However, only at very high depression scores the system that maintains the baseline level of SWB gets overwhelmed, represented by a sharp fall in SWB scores. Such a mechanism might explain the association between genetic effects on variation in psychopathology and SWB. Furthermore, such a mechanism might also explain why some individuals with relatively high levels of emotional and behavioral problems still report relatively high levels of SWB, whereas others report decreased levels of SWB. Individuals differ in the extent to which adversities are navigated. Those who successfully stand aversive events in their lives and report little mental health consequences are often characterized as resilient. Genetic factors maintaining stable levels of SWB may explain individual differences in resilience. Boardman et al. (2008) indeed reported individual differences in resilience to be substantially accounted for by genetic factors. Whether or not genetic factors accounting for individual differences in SWB and resilience overlap should be elucidated in future research.

Besides the association between psychopathology and SWB in general, we also assessed the etiology of a specific type of psychopathology, i.e. truancy. We found that genetic, nonshared environmental, and twin specific environmental factors were important in the etiology of frequency of truancy. The substantial influence of genetic factors on individual differences in frequency of truancy suggests that there is a genetic liability towards truancy. In addition, twin specific environmental effects emphasize that truancy is performed together with classmates and other peers of the same age since twins are of similar age and often share classrooms. Insight into the etiology of truancy may have important implications for SWB research. Truancy is very prevalent among adolescents (CBS, 2004; Johnston et al., 2004; Zhang et al., 2004) and it occurs in a broader context of externalizing problem behavior (Achenbach, 2001) which has been found to be negatively associated with SWB as was mentioned throughout this thesis. Preliminary findings in our data also showed frequency of truancy to be modestly associated with lower levels of SWB ($r = -.15$) among adolescents. Moreover, it has been reported that truancy provides a context in which adolescents are likely to initiate more severe types of

psychopathology (Henry et al., 2007; Osgood & Anderson, 2004; Osgood et al., 1996). For example, truancy has been linked to substance abuse (Henry & Huizinga, 2007; Best et al., 2006; Halfors et al., 2002) and psychiatric disorders (Egger et al., 2003; Fergusson et al., 1995), and truants have been found to be more likely to further penetrate the juvenile justice system (Zhang et al., 2004).

Gene-environment interaction in SWB

Interaction between genetic factors influencing adolescent SWB and latent nonshared environmental factors was explored by examining the association between intrapair sum and absolute difference scores in monozygotic twin pairs (Jinks & Fulker, 1970). Each absolute intrapair difference score provides an estimate of the magnitude of nonshared environmental influences on SWB within families. Variation in these intrapair differences appears, because twins in some families are likely to react differently from those in other families when exposed to environmental influences, or because twins in some families are exposed to different environments than in other families. On the other hand, individual differences in the sum of intrapair twin scores appear because twins belonging to different families have different genotypes and/or family environments. Negative correlations of moderate magnitude were found between intrapair sum and absolute difference scores on SWB. This means that adolescents reporting higher levels of SWB are less different compared to those reporting lower levels of SWB, suggesting that genetic factors are relatively more important in those adolescents reporting higher levels of SWB (since twin pairs reporting higher levels of SWB were more similar) compared to those reporting lower levels of SWB. Adolescents with a genetic tendency towards higher baseline levels of SWB may therefore be disproportionately more likely to experience higher levels of SWB when certain influences from their personal environment are present. However, which particular factors are involved needs to be elucidated.

Nonresponse bias

If nonresponse is not a random process, i.e. when respondents and nonrespondents differ on a trait under study, this may seriously limit the validity of the findings. To investigate the magnitude of possible response bias, nonresponse analyses were performed in which respondents and nonrespondents were compared on several parental and family characteristics (i.e. participation in childhood survey research of the Y-NTR, socioeconomic status, parental educational level, religion, smoking and alcohol consumption during pregnancy) and childhood characteristics of the twins (i.e. emotional and behavioral

problems at 3 ages during childhood, unhappiness, CITO scores, educational achievement in language and arithmetic as reported by parents). Whenever differences between respondents and nonrespondents were found, these appeared to be relatively small. For example, differences in mean levels of childhood externalizing problems (scale ranges from 0 to 66) and CITO scores (scale ranges from 501 to 550) were less than two points.

Because SWB was not assessed during childhood, respondents and nonrespondents could not be compared on this. However, internalizing problems are strongly inversely associated with SWB. No differences were found between respondents and nonrespondents in mean levels of internalizing problems at three time points during childhood (age 3, 7, and 12). In addition, one item in the childhood surveys assessed unhappiness and no differences were found between respondents and nonrespondents with regard to childhood unhappiness measured at three time points. These findings suggest that nonresponse bias is unlikely to heavily influence the results presented in this thesis since respondents and respondents were largely similar on several different characteristics, including some that are closely linked to SWB.

General conclusion

Throughout this thesis insight into the factors that cause individual differences in adolescent SWB has been gained. In addition, insight into possible mechanisms through which these factors may affect individual differences in SWB was obtained. Genetic factors accounted for a substantial part of the variation in adolescent SWB. Subjective evaluations of family functioning, parental divorce, exercise behavior, internet use, psychopathology, and truancy have been found to be associated with SWB among adolescents. The associations between SWB and evaluations of family functioning, exercise behavior, and psychopathology appeared to be largely accounted for by genetic factors. In addition, evidence was found that factors from adolescents' personal environment interact with genetic factors influencing SWB. It has often been assumed that influences from adolescents' family and personal environment directly affect their SWB, but these findings clearly indicate that genetic factors mediate the associations between SWB and factors from their family and personal environment in such a way that similar experiences and situations have different effects on adolescents' SWB.

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NEDERLANDSE SAMENVATTING

Oorzaken van Variatie in Welbevinden tijdens de Adolescentie

Subjectief welbevinden (SWB) kan worden gekarakteriseerd als een subjectief en aspectief positief gevoel van welbevinden. Het verwijst naar abstracte en algemene gevoelens die mensen over zichzelf hebben. Een belangrijk kenmerk van SWB is de hoge mate van stabiliteit over tijd (Cummins, 2010; Wagner et al., 2007; Lucas, 2007; Huebner, 2004; Lykken & Tellegen, 1996). Verder is SWB een erfelijke eigenschap: individuele verschillen in SWB kunnen voor 40 – 55% verklaard worden door genetische factoren (Caprara et al., 2009; Bartels & Boomsma, 2009; Nes et al., 2006; Stubbe et al., 2005; Røsbjerg et al., 2002; Lykken & Tellegen, 1996; Tellegen et al., 1988). Veel studies naar SWB hebben zich op volwassenen gericht, terwijl onderzoek naar SWB bij adolescenten pas recentelijk van de grond is gekomen. Het doel van dit proefschrift is het verkrijgen van meer inzicht in de oorzaken van individuele verschillen in SWB bij adolescenten. Daartoe zijn gegevens geanalyseerd met betrekking tot SWB en eigenschappen die samenhangen met SWB, zoals gezinsfunctioneren, sportgedrag, sedentair gedrag, internetgebruik, psychopathologie en spijbelen. Hierdoor kon worden nagegaan hoeveel van de variatie in deze eigenschappen wordt verklaard door genetische en omgevingsfactoren. Tevens is gekeken of er interactie bestaat tussen genetische factoren die SWB beïnvloeden en omgevingsfactoren. Daarnaast is onderzocht of er een causale samenhang bestaat tussen SWB en bovengenoemde eigenschappen. Deze causale hypothese is vergeleken met een model waarin genetische factoren de samenhang tussen SWB en deze eigenschappen verklaren door pleiotropische effecten. Pleiotropische effecten verwijzen naar genetische invloeden die meerdere eigenschappen beïnvloeden.

Mijn onderzoek is gebaseerd op gegevens die afkomstig zijn van een groot cohort adolescente tweelingen en hun broers en zussen die staan ingeschreven bij het Nederlands Tweelingen Register (NTR) van de afdeling Biologische Psychologie van de Vrije Universiteit te Amsterdam. De datacollectie van het NTR is een continu proces dat eind jaren '80 van de vorige eeuw is gestart. In de loop van de jaren is de inhoud van de vragenlijsten die aan tweelingen en hun familieleden worden gestuurd, een aantal keren verbeterd. De onderzoeken gepresenteerd in dit proefschrift zijn daardoor gebaseerd op verschillende subgroepen van het totale cohort.

Gezinsomgeving en SWB

In hoofdstuk twee is ingegaan op de samenhang tussen SWB en verschillende aspecten van de gezinsomgeving, zoals het meemaken van een echtscheiding en perceptie van gezinsfunctioneren. De mate waarin genetische en omgevingsinvloeden individuele verschillen verklaren in de wijze waarop adolescenten hun gezinsfunctioneren en SWB evalueren, en de samenhang tussen beide, is onderzocht. Tevens is er gekeken of de mate

waarin genetische en omgevingsfactoren variatie in perceptie van gezinsfunctioneren en SWB beïnvloeden, verschilt tussen adolescenten die opgroeien in intacte gezinnen en adolescenten uit gezinnen met gescheiden ouders. Perceptie van gezinsfunctioneren is gemeten met een Nederlandse vertaling van de subschaal “Algemeen functioneren” van de McMaster Family Assessment Device (FAD-N; Wenniger et al., 1993) en de subschaal “Conflict” van de Gezinsklimaatschaal (GKS; De Coole & Jansma, 1983). Kwaliteit van leven, gemeten met de zogenaamde “Cantril Ladder” (Cantril, 1965), is gebruikt als maat voor SWB. Voor dit onderzoek zijn gegevens geanalyseerd van 5.773 tweelingen en 1.000 broers of zussen.

Uit de genetische analyses bleek dat individuele verschillen in evaluaties van algemeen gezinsfunctioneren en de mate van conflict binnen het gezin verklaard kunnen worden door genetische factoren, gedeelde en unieke omgevingsfactoren. Genetische factoren zijn relatief belangrijker voor meisjes dan voor jongens met betrekking tot evaluaties van algemeen gezinsfunctioneren. Genetische invloed op variatie in de perceptie van algemeen gezinsfunctioneren is geschat op 6% voor jongen en 35% voor meisjes. Genetische invloed op individuele verschillen in de perceptie van de mate van conflict binnen het gezin is geschat op 31% voor zowel jongens als meisjes. Individuele verschillen in kwaliteit van leven konden verklaard worden door genetische en unieke omgevingsfactoren, waarbij de relatieve invloed van genetische factoren belangrijker bleek te zijn voor meisjes (43%) dan voor jongens (30%). Hoe adolescenten hun gezinsfunctioneren en SWB evalueren, wordt dus deels beïnvloed door genetische factoren.

Er bleek interactie te bestaan tussen genetische factoren die de perceptie van algemeen gezinsfunctioneren beïnvloeden en het meemaken van een echtscheiding. De relatieve invloed van genetische factoren op individuele verschillen in de perceptie van algemeen gezinsfunctioneren was groter voor adolescenten met gescheiden ouders (jongens: 14%, meisjes: 45%) dan voor hen die opgroeien in intacte gezinnen (jongens: 6%, meisjes: 35%). Er werd geen interactie gevonden tussen genetische factoren die de perceptie van de mate van conflict binnen het gezin beïnvloeden en echtscheiding. Met betrekking tot SWB is gebleken dat individuele verschillen in SWB groter zijn voor meisjes met gescheiden ouders vergeleken met meisjes uit intacte gezinnen.

Hogere scores op perceptie van algemeen gezinsfunctioneren en lagere scores op perceptie van de mate van conflict binnen het gezin bleken in dit onderzoek samen te hangen met hogere scores op kwaliteit van leven. Resultaten van bivariate genetische analyses lieten zien dat de samenhang tussen perceptie van gezinsfunctioneren en SWB voornamelijk verklaard wordt door genetische factoren. Het overige deel van de samenhang wordt verklaard door unieke omgevingsfactoren.

Interactie tussen genetische factoren die de perceptie van gezinsfunctioneren en SWB beïnvloeden en unieke omgevingsinvloeden is onderzocht door de samenhang tussen som- en verschillcores binnen eenzelfde (genetisch identieke) tweelingparen te bestuderen. Een significante correlatie tussen som- en verschillcores op een bepaalde eigenschap is een indicatie voor interactie tussen genetische en unieke omgevingsfactoren (Jinks & Fulker, 1970). Verschillen in somcores *tussen* tweelingparen ontstaan doordat tweelingen uit verschillende gezinnen verschillen in genotype en/of gezinsomgeving. Verschillen *binnen* een tweelingpaar ontstaan door unieke omgevingsinvloeden. Significante negatieve correlaties tussen som- en verschillcores bij eenzelfde tweelingparen zijn gevonden voor evaluaties van algemeen gezinsfunctioneren, de mate van conflict binnen het gezin en kwaliteit van leven. Dit impliceert een samenspel tussen genetische factoren die evaluaties van gezinsfunctioneren en SWB beïnvloeden en unieke omgevingsfactoren: de relatieve invloed van genetische factoren is groter voor adolescenten met hogere scores op perceptie van gezinsfunctioneren en SWB. Eenzelfde tweelingparen met hogere scores op gezinsfunctioneren en SWB bleken namelijk meer gelijkenis te vertonen wat betreft scores op gezinsfunctioneren en SWB ten opzichte van hen met lagere scores voor gezinsfunctioneren en SWB. Genetische factoren blijken dus belangrijker te zijn voor adolescenten die zich goed voelen over zichzelf en hun gezin dan voor hen die minder gelukkig zijn over zichzelf en hun gezin.

Sportgedrag en SWB

De hoofdstukken drie en vier gaan over sportgedrag bij adolescenten en de samenhang tussen sportgedrag en SWB. In hoofdstuk drie is onderzocht in welke mate individuele verschillen in sportgedrag gedurende de adolescentie verklaard kunnen worden door genetische en omgevingsfactoren. Gegevens over sportgedrag waren beschikbaar voor 7.195 tweelingen. Omdat er aanwijzingen zijn dat de genetische architectuur van sportgedrag verandert gedurende de adolescentie, is de relatieve invloed van genetische en omgevingsfactoren op variatie in sportgedrag geschat voor drie leeftijdsgroepen: 13 – 14, 15 – 16 en 17 – 19 jarigen. Tevens zijn kwantitatieve en kwalitatieve verschillen in genetische invloeden tussen jongens en meisjes bestudeerd binnen deze leeftijdsgroepen. Sportgedrag is gemeten door deelnemers te vragen welke sport(en) ze beoefenen en aan te geven hoe vaak per week en de gemiddelde tijd per keer ze deze sport(en) beoefenen. Op basis van deze vragenlijstgegevens en met behulp van Ainsworth Compendium of Physical Activity (Ainsworth et al., 2000) is voor iedere deelnemer een score voor de intensiteit van sportgedrag berekend. Op grond van deze score zijn de respondenten ingedeeld in drie categorieën: niet-sporters, gematigde sporters en fanatieke sporters. Tussen 13- en

19-jarige leeftijd nam de prevalentie van gematigd sportgedrag af. De prevalentie van fanatiek sportgedrag was constant gedurende de adolescentie. Op alle leeftijden bleken meisjes vaker niet-sporters dan jongens. Binnen de groep van sportende deelnemers bleken meisjes vaker gematigde sporters en jongens vaker fanatieke sporters. De genetische analyses laten zien dat de genetische architectuur van sportgedrag verandert gedurende de adolescentie. Individuele verschillen in sportgedrag bij de jonge adolescenten worden verklaard door genetische (jongens: 85%, meisjes: 38%) en unieke omgevingsfactoren (jongens: 15%, meisjes: 16%), terwijl bij meisjes gedeelde omgevingsfactoren 46% van de variatie verklaren. Genetische factoren verklaren het grootste gedeelte van de variatie in sportgedrag bij de 15 – 16 (80%) en 17 – 19 jarigen (72%). Unieke omgevingsfactoren verklaren het overige gedeelte van de variatie in sportgedrag voor deze leeftijdsgroepen. Er is geen indicatie voor kwalitatieve sekseverschillen in genetische invloeden op sportgedrag gedurende de adolescentie. Dit betekent dat het sportgedrag van jongens en meisjes door dezelfde genetische varianten wordt beïnvloed.

In hoofdstuk vier is bestudeerd of SWB en internaliserende problemen causaal worden beïnvloed door sportgedrag of dat deze samenhang wordt verklaard door onderliggende genetische factoren. Gegevens voor deze studie waren beschikbaar voor 6.317 tweelingen en 1.180 broers of zussen. Longitudinale gegevens waren voor een groot gedeelte van het cohort beschikbaar op 2 tijdstippen met een interval van 2 jaar. Sportgedrag is op dezelfde manier gemeten als in hoofdstuk 3. De mate van internaliserende problemen is gemeten met de Nederlandse vertaling van de Youth Self Report (YSR; Verhulst et al., 1997). SWB is gemeten met de schalen “Satisfaction with Life” (Diener et al., 1985), “Subjective Happiness” (Lyubomirsky & Lepper, 1999) en “Quality of Life” (Cantril, 1965). Op basis van de scores op deze schalen is voor alle deelnemers een factorscore voor SWB geschat. Zowel cross-sectioneel als longitudinaal is een positief verband vastgesteld tussen sportgedrag, internaliserende problemen en SWB. Adolescenten die regelmatig sporten bleken gemiddeld hogere niveaus van SWB en minder last van internaliserende problemen te hebben. Deze bevindingen komen overeen met eerdere studies bij volwassenen (Stubbe et al., 2007; De Moor et al., 2006; Norris et al., 1992; De Moor et al., 2008; Wise et al., 2006; Camacho et al., 1991). Door de samenhang tussen sportgedrag, internaliserende problemen en SWB te bestuderen bij eenige tweelingparen (genetisch identiek) kan worden onderzocht wat de oorzaak van deze samenhang is. Als de tweeling die meer aan sport doet minder internaliserende problemen en hogere niveaus van SWB heeft dan de niet of minder sportende tweelingbroer of -zus, kan dit niet verklaard worden door genetische verschillen tussen deze personen. Internaliserende problemen en SWB worden in dit geval mogelijk causaal beïnvloed door sportgedrag. Als echter de tweeling die meer aan sport doet *niet* minder internaliserende

problematiek en hogere niveaus van SWB vertoont dan de niet of minder sportende tweelingbroer of -zus, dan wijst dit erop dat genetische factoren verantwoordelijk zijn voor de samenhang tussen sportgedrag, internaliserende problemen en SWB. De tweeling die meer aan sport doet bleek *niet* minder internaliserende problemen of hogere niveaus van SWB te hebben dan de tweelingbroer of -zus die minder of niet aan sport doet. De tweeling die meer is gaan sporten over een periode van 2 jaar bleek tevens *geen* grotere afname in internaliserende problematiek of toename in het niveau van SWB te laten zien over dezelfde periode van 2 jaar ten opzichte van de tweelingbroer of -zus die geen verandering of een afname in sportgedrag vertoont over tijd. Beide bevindingen wijzen erop dat de samenhang tussen sportgedrag, internaliserende problemen en SWB verklaard kan worden door onderliggende gedeelde genetische factoren.

Als er een causaal verband bestaat tussen sportgedrag, internaliserende problemen en SWB moeten alle genetische en omgevingsfactoren die sportgedrag beïnvloeden tevens van invloed zijn op internaliserende problemen en SWB. Uit bivariate genetische analyses bleek dat de cross-sectionele en de longitudinale samenhang tussen sportgedrag, internaliserende problemen en SWB verklaard kan worden, doordat genetische factoren die een rol spelen bij sportgedrag deels overlappen met genetische factoren voor internaliserende problemen en SWB. Bij meisjes kan de samenhang tussen sportgedrag en internaliserende problemen tevens verklaard worden doordat gedeelde omgevingsfactoren die sportgedrag beïnvloeden deels overlappen met gedeelde omgevingsfactoren voor internaliserende problemen. Unieke omgevingsfactoren die sportgedrag beïnvloeden bleken echter niet te overlappen met unieke omgevingsfactoren die internaliserende problemen en SWB beïnvloeden. Ook deze bevindingen leveren geen bewijs voor een causaal verband tussen sportgedrag, internaliserende problemen en SWB. De bevindingen zijn wel verenigbaar met de hypothese dat genetische factoren zowel de behoefte van een jongere om regelmatig te sporten beïnvloeden, als de aanleg voor SWB en de afwezigheid van internaliserende problemen.

Sedentair gedrag en SWB

Sedentair gedrag (of: inactief gedrag) wordt vaak ten onrechte gezien als het tegenovergestelde van lichaamsbeweging en/of sportgedrag. Uit eerder onderzoek is bijvoorbeeld gebleken dat sedentair gedrag een risicofactor vormt voor overgewicht en metabolische aandoeningen onafhankelijk van de mate van lichaamsbeweging en sport (Martínez-Gómez et al., 2010; Mark & Jansen, 2008; Biddle, 2007; Te Velde et al., 2007; Wong et al., 1992). Daarnaast hoeft intensief sportgedrag een hoge mate van sedentair gedrag niet uit te sluiten. De hoofdstukken vijf en zes richten zich op sedentair gedrag

tijdens de adolescentie en de samenhang tussen sedentair gedrag en SWB. In hoofdstuk vijf is onderzocht in hoeverre individuele verschillen in sedentair gedrag verklaard kunnen worden door genetische en omgevingsfactoren gedurende de adolescentie. Hierbij is bestudeerd of de invloed van genetische en omgevingsfactoren op variatie in sedentair gedrag verandert als een functie van leeftijd in een cohort van 5.074 adolescente tweelingen en 937 broers of zussen tussen 12 en 20 jaar. Sedentair gedrag is gemeten met behulp van drie items waarin deelnemers werd gevraagd hoeveel tijd ze per week besteden aan TV kijken, gamen en computeren/internetten. Gedurende de adolescentie bleken jongeren steeds minder tijd aan sedentaire activiteiten te besteden. Verder bleken jongens meer tijd te besteden aan sedentaire activiteiten ten opzichte van meisjes. De afname in de frequentie van sedentair gedrag gedurende de adolescentie kan worden toegeschreven aan het feit dat jongeren minder tijd besteden aan TV kijken. Sekseverschillen in de prevalentie van sedentair gedrag kunnen worden toegeschreven aan de hogere frequentie van gamen bij jongens. Uit de genetische analyses bleek dat de relatieve invloed van genetische en omgevingsfactoren veranderd gedurende de adolescentie. De relatieve invloed van genetische en unieke omgevingsfactoren nam toe, terwijl de invloed van gedeelde omgevingsfactoren verdween in de loop van de adolescentie. Individuele verschillen in sedentair gedrag onder de jongste deelnemers (12 jaar) worden verklaard door genetische factoren (jongens: 35%, meisjes: 19%), gedeelde omgevingsfactoren (jongens: 29%, meisjes: 48%) en unieke omgevingsfactoren (jongens: 36%, meisjes: 34%). Variatie in sedentair gedrag onder de oudste deelnemers (20 jaar) wordt verklaard door genetische (jongens: 48%, meisjes: 34%) en unieke omgevingsfactoren (jongens: 52%, meisjes: 66%). Er is geen indicatie voor kwalitatieve sekseverschillen in de genetische factoren. Dit betekent dat sedentair gedrag van jongens en meisjes wordt beïnvloed door dezelfde genetische varianten. De substantiële rol die genetische factoren blijken te spelen bij sedentair gedrag impliceert een genetische aanleg voor sedentaire activiteiten, zoals TV kijken, gamen en internetten.

Om meer inzicht te krijgen in het verband tussen sedentaire activiteiten en welbevinden bij adolescenten is in hoofdstuk zes de samenhang bestudeerd tussen dagelijks internetgebruik, een van de populairste sedentaire activiteiten onder jongeren, compulsief internetgebruik en welbevinden. Tevens is gekeken of de mate waarin internetgebruik en welbevinden samenhangen, verschilt voor adolescenten met verschillende persoonlijkheidseigenschappen. Dagelijks internetgebruik is gemeten door deelnemers te vragen hoeveel tijd ze dagelijks aan internetten besteden. Compulsief internetgebruik is gemeten met de Compulsive Internet Use Scale (Meerkerk et al., 2007). Welbevinden is geoperationaliseerd door eenzaamheid (revised UCLA Loneliness Scale; Russell et al., 1980), gevoel van eigenwaarde (Rosenberg Self-Esteem Scale; Rosenberg, 1989) en

depressieve gevoelens (Depressive Mood List; Kandel & Davies, 1986). De “Big Five” persoonlijkheidsdimensies zijn gemeten met de Quick Big Five (Vermulst & Gerris, 2005). Er werd een indirecte samenhang gevonden tussen dagelijks internetgebruik en welbevinden. Deze samenhang wordt gemedieerd door compulsief internetgebruik. Met andere woorden, er blijkt geen directe samenhang tussen dagelijks internetgebruik en welbevinden te zijn, maar wel tussen dagelijks internetgebruik en compulsief internetgebruik en tussen compulsief internetgebruik en welbevinden. Het verband tussen dagelijks internetgebruik en compulsief internetgebruik is sterker voor adolescenten die hoog scoren op neuroticisme en laag op extraversie en altruïsme. Hetzelfde geldt voor het verband tussen compulsief internetgebruik en eenzaamheid.

Psychopathologie en SWB

Hoofdstuk zeven is gericht op de vraag of SWB kan worden gedefinieerd als de afwezigheid van psychopathologie of dat SWB en psychopathologie onafhankelijke dimensies van geestelijke gezondheid zijn. Uit verschillende studies is naar voren gekomen dat SWB negatief is geassocieerd met verschillende soorten psychopathologie (zie Proctor et al., 2009). Andere bevindingen zijn verenigbaar met de hypothese dat SWB en psychopathologie deels verschillende oorzaken hebben en daarmee als onafhankelijke dimensies van geestelijke gezondheid gezien kunnen worden (Ryff et al., 2006; Greenspoon & Saklofske, 2001; Bergsma et al., 2010). Op basis van drie schalen is voor iedere deelnemer een factorscore voor SWB geschat. Psychopathologie is gemeten met de Nederlandse vertaling van de Youth Self-Report (YSR; Verhulst et al., 1997), waarmee verschillende gedrags- en emotionele problemen worden gemeten aan de hand van acht syndroomschalen en twee zogenoemde brede-band syndroomschalen (Internaliseren en Externaliseren). De gegevens zijn afkomstig van 6.381 adolescenten tweelingen en 1.195 broers of zussen. Adolescenten die boven een seksespecifieke klinische grenswaarde scoorden voor een of meer van de syndroomschalen (T-score groter of gelijk aan 67; zie Verhulst et al., 1997), rapporteerden lagere niveaus van SWB dan diegenen die onder de klinische grenswaarde voor de syndroomschalen scoorden. Als SWB gedefinieerd kan worden als de afwezigheid van psychopathologie zouden er 2 groepen geïdentificeerd moeten worden: a) adolescenten die laag scoren op psychopathologie en hoog op SWB en b) adolescenten die hoog scoren op psychopathologie en laag op SWB. Slechts 15% van het cohort voldeed aan deze classificatie. Negatieve correlaties, variërend tussen $-.24$ en $-.57$, werden gevonden tussen SWB en de verschillende syndroomschalen. De samenhang tussen SWB en de verschillende syndroomschalen werd voornamelijk verklaard door deels overlappende genetische factoren (genetisch correlaties varieerden

tussen $-.31$ en $-.71$) en in mindere mate door overlappende unieke omgevingsfactoren (unieke omgevingscorrelaties varieerden tussen $-.13$ en $-.42$). Het verband tussen SWB en externaliserend probleemgedrag werd alleen verklaard door deels overlappende genetische factoren. De conclusie die uit deze resultaten getrokken kan worden is dat SWB niet uitsluitend gedefinieerd kan worden als de afwezigheid van psychopathologie. SWB en psychopathologie zijn deels onafhankelijke dimensies van geestelijke gezondheid.

In hoofdstuk acht is de relatieve invloed van genetische en omgevingsfactoren op individuele verschillen in spijbelgedrag op de middelbare school onderzocht. Tevens is er gekeken of variatie in spijbelgedrag verklaard kan worden door tweelingspecifieke omgevingsfactoren. Gegevens voor spijbelgedrag zijn afkomstig van 3.406 tweelingen en 1.429 broers of zussen. De prevalentie van spijbelen nam toe met het vorderen van de leeftijd en jongens bleken vaker spijbelaars te zijn dan meisjes. Variatie in spijbelgedrag kon worden verklaard door genetische (45%) en unieke omgevingsinvloeden (55%). Variatie verklaard door omgevingsfactoren kon voor tweelingen worden opgesplitst in tweelingspecifieke omgevingsfactoren (25%) en unieke omgevingsfactoren (30%). De substantiële rol die genetische factoren spelen bij spijbelgedrag impliceert een genetische aanleg voor spijbelen. De invloed van tweelingspecifieke omgevingsfactoren op spijbelgedrag van adolescenten wijst erop dat jongeren samen met klas- en/of leeftijdgenoten spijbelen.

Procedures van dataverzameling

In hoofdstuk negen zijn de procedures van dataverzameling beschreven. Om inzicht te krijgen in verschillen tussen respondenten en niet-respondenten is er een responsanalyse uitgevoerd. In de responsanalyse zijn respondenten en niet-respondenten vergeleken aan de hand van de volgende eigenschappen: participatie in vragenlijstonderzoek van het Nederlands Tweelingen Register (NTR) in het verleden, sociaal-economische status (SES), opleidingsniveau van de ouders, religie, roken en alcoholgebruik tijdens de zwangerschap door de moeder, emotionele problemen, gedragsproblemen en welbevinden van de tweelingen op drie verschillende leeftijden, CITO scores van de tweelingen en taal- en rekenprestaties van de tweelingen zoals gerapporteerd door de ouders. Niet-respondenten deden in het verleden minder vaak mee aan vragenlijstonderzoek van het NTR dan respondenten. Niet-respondenten rapporteerden lagere SES en opleidingsniveau van de ouders, hogere prevalentie van roken en alcoholgebruik tijdens de zwangerschap door de moeder, meer gedragsproblemen op leeftijd 3, 7 en 12 en lagere CITO scores. Met betrekking tot religie, emotionele problemen en welbevinden op leeftijd 3, 7 en 12 en taal- en rekenprestaties zoals gerapporteerd door de ouders werden geen verschillen gevonden

tussen respondenten en niet-respondenten. Het is belangrijk om te vermelden dat de verschillen die gevonden werden tussen respondenten en niet-respondenten gering waren. Met andere woorden, respondenten en niet-respondenten blijken grotendeels vergelijkbaar.

Algemene conclusie

De bevindingen in dit proefschrift dragen bij aan het inzicht in factoren die individuele verschillen in SWB tijdens de adolescentie verklaren. Tevens levert dit proefschrift een bijdrage aan de kennis omtrent mogelijke mechanismen waardoor deze factoren individuele verschillen in SWB kunnen verklaren. Variatie in SWB tijdens de adolescentie kan voor een gedeelte verklaard worden door genetische factoren. Evaluaties van gezinsfunctioneren, het meemaken van een echtscheiding, sportgedrag, internetgebruik en psychopathologie blijken samen te hangen met SWB bij adolescenten. Het verband tussen SWB en evaluaties van gezinsfunctioneren, sportgedrag en psychopathologie kan voor een belangrijk deel verklaard worden door genetische factoren. De bevindingen wijzen tevens op de aanwezigheid van een complexe wisselwerking tussen genetische factoren die SWB beïnvloeden en factoren uit de persoonlijke omgeving van adolescenten. In het verleden is vaak aangenomen dat factoren vanuit de gezins- en persoonlijke omgeving van adolescenten een directe of causale invloed hebben op SWB. De bevindingen in dit proefschrift laten echter duidelijk zien dat het verband tussen deze factoren en SWB gemedieerd wordt door genetische factoren, waardoor dezelfde ervaringen en situaties SWB op verschillende manier kunnen beïnvloeden afhankelijk van het genotype van een individu.

APPENDICES

- Appendix I: Parental consent form (version 2005 – 2006)
- Appendix II: Invitation letter in which parents were asked if they permitted their children to fill in a self-report survey (version 2005 – 2006)
- Appendix III: Invitation letter in which parents were asked to complete the parental consent form (version 2007)
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- Appendix V: Invitation letter in which parents were asked to complete the parental consent form (version 2009)
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- Appendix VIII: Overview of items and scales in the Dutch Health and Behavior Questionnaire (DHBQ)
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- Appendix X: Twin flyer DHBQ (version 2005 – 2006)
- Appendix XI: Invitation letter in which twins and/or siblings were invited to complete the self-report survey (version 2007)
- Appendix XII: Twin flyer DHBQ (version 2007 – 2009)

Appendix I: Parental consent form (version 2005 – 2006)

registratienr. NTR:.....-...../.....

Toestemmingsverklaring voor het Familieonderzoek naar Gedrag en Leefgewoonten in de Adolescentie

In te vullen door de ouder/verzorger

Ik stem toe met het invullen van de vragenlijst over gedrag en leefgewoonten door mijn zoon/dochter
(vult u per kind een toestemmingsformulier in a.u.b.)

Voornaam zoon/dochter :

Achternaam :

Geboortedatum zoon/dochter :-.....-.....

Geslacht 1 jongen 2 meisje

Geboortegewicht zoon/dochter :gram

Zwangerschapsduur (in weken) :weken

Naam ouder/verzorger :

Dit formulier is ingevuld door : 1 biologische vader 2 biologische moeder 3 ander, nl:.....

Datum: Plaats:..... Handtekening:

Appendix II: Invitation letter in which parents were asked if they permitted their children to fill in a self-report survey (version 2005 – 2006)

Nederlands Tweelingen Register (NTR)

Datum	Uw brief van	Telefax 020-5988832	Bijlage(n) informatiefolder; brief voor kinderen.
Ons kenmerk NTR/DHBQ 0207	Uw kenmerk	Telefoon 020-5988827	E-mail DHBQ@psy.vu.nl

Postadres: Van der Boechorststraat 1, 1081 BT Amsterdam

vrije Universiteit amsterdam



Geachte heer, mevrouw,

U heeft vele jaren meegedaan aan tweelingonderzoek naar de ontwikkeling van het gedrag van kinderen. Uw tweeling heeft inmiddels de tienerleeftijd bereikt. Om de veranderingen in gedrag en leefgewoonten op deze leeftijd in kaart te brengen willen we dit keer graag uw kinderen zelf een vragenlijst laten invullen. Als de tweeling broers en/of zussen van 12 jaar of ouder heeft, willen we die graag in het onderzoek betrekken. In de informatiefolder kunnen u en uw kinderen meer lezen over dit onderzoek.

Indien uw kinderen minderjarig zijn, willen we uw toestemming voor hun deelname vragen. Als u toestemt, kunt u, **nadat u de toestemmingsverklaring op de voorkant van de vragenlijst hebt ondertekend**, de vragenlijst en bijbehorende brief aan uw tweeling en eventuele broers of zussen geven. Uw kinderen kunnen de ingevulde lijst retourneren in bijbehorende antwoordenvolp (geen postzegel nodig). Als er binnen uw gezin meerdere broers/zussen willen meewerken, kunt u contact met ons opnemen. Wij sturen u dan extra vragenlijsten (NTR: 020-5988827).

Mocht u na het lezen van deze brief en de informatiefolder nog vragen hebben over het onderzoek dan kunt u contact opnemen met het Nederlands Tweelingen Register (*tel:* 020-5988827, *email:* DHBQ@psy.vu.nl). Indien u vragen heeft die u liever niet aan de onderzoeker zelf stelt, dan is het mogelijk om een onafhankelijke onderzoeker te raadplegen (prof. dr. J.M. Koot, *tel:* 020-5988901).

Bij het Nederlands Tweelingen Register staan gezinnen ingeschreven met twee of meerdere tweelingen binnen één gezin. Deze vragenlijst is bedoeld voor tweelingen van 12 jaar en ouder. Mochten er binnen uw gezin meerdere tweelingen van 12 jaar of ouder zijn wilt u dan zo vriendelijk zijn om contact op te nemen met het Nederlands Tweelingen Register, *tel:* 020-5988827; *e-mail:* DHBQ@psy.vu.nl

Met het invullen van deze vragenlijst leveren uw kinderen een belangrijke bijdrage aan het wetenschappelijk onderzoek van het NTR. Deelname is geheel vrijwillig. Wij willen u en uw kinderen bij voorbaat hartelijk danken voor de medewerking.

Met vriendelijke groet,
mede namens mevr. prof. dr. D.I. Boomsma,

mevr. dr. M. Bartels

Meer informatie over het NTR kunt u vinden op onze website: www.tweelingenregister.org

Afdeling Biologische Psychologie

Bezoekadres: Van der Boechorststraat 1
Transitorium

Appendix III: Invitation letter in which parents were asked to complete the parental consent form (version 2007)

Nederlands Tweelingen Register (NTR)

Datum	Uw brief van	Telefax	Bijlage(n) toestemmingsverklaring, informatiefolder, antwoordenvolpette
Ons kenmerk NTR / PC-1 DHBQ	Uw kenmerk	Telefoon 020-5982532 020-5982533	E-mail DHBQ@psy.vu.nl

Postadres: Van der Boechorststraat 1, 1081 BT Amsterdam

vrije Universiteit *amsterdam*



Geachte heer/mevrouw,

U staat met uw tweeling ingeschreven bij het Nederlands Tweelingen Register (NTR). Al vele jaren werkt u mee aan onderzoek van het NTR door vragenlijsten over uw kinderen voor ons in te vullen. Hiervoor willen wij u heel hartelijk danken. Zonder uw inzet zou dit onderzoek niet mogelijk zijn!

Uw tweeling heeft inmiddels de tienerleeftijd bereikt. Om de veranderingen in gedrag en leefgewoonten gedurende deze periode in kaart te brengen, willen wij uw tweeling graag persoonlijk benaderen om te vragen of ze een vragenlijst over zichzelf willen invullen. Als de tweeling broers en/of zussen heeft, zouden wij die ook graag bij het onderzoek willen betrekken: wij willen ze een vragenlijst sturen op hetzelfde moment dat we de tweeling een vragenlijst sturen. Broers en/of zussen van de tweeling die mee willen doen moeten bij het NTR worden ingeschreven.

Op de bijgevoegde toestemmingsverklaring kunt u aangeven of u het NTR toestemming geeft uw kinderen te vragen mee te doen aan vragenlijstonderzoek. Ook kunt u op dit formulier de namen en adresgegevens invullen. Nadat u de toestemmingsverklaring heeft ingevuld en ondertekend, kunt u het formulier in de antwoordenvolpette naar ons terugsturen (geen postzegel nodig). Wanneer u géén toestemming geeft, vernemen wij dat ook graag van u.

Meer informatie over het onderzoek en de vragenlijst wordt gegeven in de informatiefolder. Mocht u na het lezen van deze brief en de folder nog vragen hebben over het onderzoek of over de bijgevoegde toestemmingsverklaring dan kunt u contact opnemen met Niels van der Aa (*tel:* 020-5982532, *email:* DHBQ@psy.vu.nl) of met een andere medewerker van het NTR (*tel:* 020-5982533). Als u er prijs op stelt informatie over dit onderzoek in te winnen bij iemand die niet bij de uitvoering van het onderzoek betrokken is, dan is prof. dr. H.A. Delemarre (*tel:* 020-4440896) bereid uw vragen te beantwoorden. Voor andere vragen verwijzen wij u graag naar de website van het Nederlands Tweelingen Register (www.tweelingenregister.org).

Met het invullen van de vragenlijst leveren uw kinderen een belangrijke bijdrage aan het wetenschappelijk onderzoek van het Nederlands Tweelingen Register. Deelname is geheel vrijwillig. Wij willen u en uw kinderen bij voorbaat hartelijk danken voor de medewerking.

Met vriendelijke groet,

Mevr. dr. M. Bartels
Mevr. prof. dr. D.I. Boomsma

Afdeling Biologische Psychologie

Bezoekadres: Van der Boechorststraat 1

Toestemmingsverklaring voor het Familieonderzoek naar Gedrag en Leefgewoonten in de Adolescentie

In te vullen door de ouder/verzorger

Let bij het invullen van deze toestemmingsverklaring op de volgende zaken:

1. Wanneer u toestemming geeft voor broers en/of zussen van de tweeling die op dit moment jonger zijn dan 12 jaar worden zij pas door ons benaderd wanneer zij 12 jaar of ouder zijn. Wij vragen echter nu al om gegevens voor registratie van broers en/of zussen, omdat deze toestemmingsverklaring éénmalig naar ouders/verzorgers verstuurd wordt.
2. Wanneer één van de tweeling niet in staat is vragenlijsten in te vullen (bijvoorbeeld door ziekte of handicap), dan zouden wij toch erg blij zijn met de gegevens van zijn/haar tweelingbroer/zus en eventuele broers/zussen. Wanneer u hieronder aangeeft dat één van de *tweeling* niet in staat is vragenlijsten in te vullen, dan zal hij/zij daarvoor ook nooit door ons benaderd worden.

Naam: is niet in staat om vragenlijsten in te vullen

3. Op dit moment zijn wij bezig met het digitaliseren van de vragenlijst voor adolescente tweelingen en hun broers/zussen. Op termijn zal het mogelijk zijn dat uw kinderen de vragenlijst óók via het internet kunnen invullen. Om uw kinderen ook daadwerkelijk via het internet te kunnen benaderen willen we u vragen om bij de adresgegevens op de achterzijde van dit formulier ook het emailadres van uw kinderen te vermelden.

₁ Ja, ik geef *wel* toestemming om mijn kinderen persoonlijk te benaderen met de vraag of ze een vragenlijst over zichzelf willen invullen

₂ Nee, ik geef *geen* toestemming om mijn kinderen persoonlijk te benaderen met de vraag of ze een vragenlijst over zichzelf willen invullen

Dit formulier is ingevuld door:

₁ biologische vader ₂ biologische moeder ₃ anders, nl:

Naam en voorletter(s) ouder/verzorger:

Datum: Plaats: Handtekening:.....

Indien u toestemt, vult u dan alstublieft de gegevens van uw kinderen op de achterzijde van dit formulier in

Z.O.Z.

Gegevens van de tweeling	
oudste van de tweeling (eerstgeborene)	jongste van de tweeling (laatstgeborene)
Geboortedatum kind:	Geboortedatum kind:
Voornamen:	Voornamen:
Roepnaam:	Roepnaam:
Achternaam:	Achternaam:
Adres: *	Adres: *
Postcode: *	Postcode: *
Woonplaats: *	Woonplaats: *
Telefoonnummer: *	Telefoonnummer: *
Email:@.....	Email:@.....
Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje	Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje

Gegevens van broers en/of zussen van de tweeling	
Geboortedatum kind:	Geboortedatum kind:
Voornamen:	Voornamen:
Roepnaam:	Roepnaam:
Achternaam:	Achternaam:
Adres: *	Adres: *
Postcode: *	Postcode: *
Woonplaats: *	Woonplaats: *
Telefoonnummer: *	Telefoonnummer: *
Email:@.....	Email:@.....
Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje	Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje
Geboortedatum kind:	Geboortedatum kind:
Voornamen:	Voornamen:
Roepnaam:	Roepnaam:
Achternaam:	Achternaam:
Adres: *	Adres: *
Postcode: *	Postcode: *
Woonplaats: *	Woonplaats: *
Telefoonnummer: *	Telefoonnummer: *
Email:@.....	Email:@.....
Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje	Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje
Geboortedatum kind:	Geboortedatum kind:
Voornamen:	Voornamen:
Roepnaam:	Roepnaam:
Achternaam:	Achternaam:
Adres: *	Adres: *
Postcode: *	Postcode: *
Woonplaats: *	Woonplaats: *
Telefoonnummer: *	Telefoonnummer: *
Email:@.....	Email:@.....
Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje	Geslacht: <input type="checkbox"/> jongen <input type="checkbox"/> meisje

* alleen invullen indien uw kind op een ander adres woont dan uw thuisadres

Appendix V: Invitation letter in which parents were asked to complete the parental consent form (version 2009)

Nederlands Tweelingen Register (NTR)

Datum	Uw brief van	Telefax	Bijlage(n) toestemmingsverklaring, antwoordenvolp
Ons kenmerk PC-1 TRF12/DHBQ 09	Uw kenmerk	Telefoon 020-598 8948 / 598 2533	E-mail ntr@psy.vu.nl

Postadres: Van der Boechorststraat 1, 1081 BT Amsterdam

vrije Universiteit amsterdam



Geachte ouder/verzorger,

U staat met uw tweeling ingeschreven bij het Nederlands Tweelingen Register (NTR) van de Vrije Universiteit. Door het invullen van vragenlijsten levert u een belangrijke bijdrage aan wetenschappelijk onderzoek naar de gedragsontwikkeling van opgroeiende kinderen. Aan het einde van dit schooljaar zult u de laatste vragenlijst over uw tweeling ontvangen. Daarna willen wij uw kinderen zélf graag vragenlijsten sturen. Hiervoor hebben wij uw toestemming nodig en daarom ontvangt u deze toestemmingsverklaring.

Dit schooljaar willen wij ook weer graag de leerkracht van uw kinderen betrekken bij het onderzoek door hem of haar een vragenlijst te sturen. Met informatie van zowel de ouders/verzorgers als de leerkracht van de kinderen, kan een zo breed en compleet mogelijk beeld van de gedragsontwikkeling van een kind worden gevormd.

Als u buiten de tweeling nog andere kinderen heeft die op de basisschool zitten, dan willen wij hen én hun leerkracht ook graag bij ons onderzoek betrekken. Op deze manier kan onderzocht worden in hoeverre tweelingkinderen verschillen van kinderen die geen tweeling zijn.

De deelname van de leerkracht en uw kinderen is geheel vrijblijvend. Uw toestemming geeft hen geen verplichting om de vragenlijst in te vullen. Alle gegevens worden door ons vertrouwelijk behandeld. Dit betekent onder andere dat ouders, kinderen en de leerkracht van de kinderen geen inzage krijgen in elkaars antwoorden.

Op de bijgevoegde toestemmingsverklaring kunt u aangeven of wij uw kinderen en/of hun leerkracht een vragenlijst mogen sturen. U kunt het formulier in de antwoordenvolp naar ons terugsturen zonder een postzegel te plakken. Wilt u óók wanneer u geen toestemming geeft zo vriendelijk zijn om dit op het formulier aan te geven en naar ons terug te sturen? Op die manier zult u geen herinnering ontvangen.

Mocht u vragen hebben of meer informatie willen dan kunt u tijdens kantooruren contact met ons opnemen (telefoon: 020-598 8948 of 598 2533; email: ntr@psy.vu.nl).

Wij willen u en uw kinderen bij voorbaat hartelijk danken voor de medewerking!

Met vriendelijke groet,

mevr. prof.dr. D.I. Boomsma

Meer informatie over het NTR kunt u vinden op onze website: www.tweelingenregister.org

Afdeling Biologische Psychologie

Bezoekadres: Transitorium, Van der Boechorststraat 1

Appendix VI: Parental consent form (version 2009)



Toestemmingsverklaring

Let bij het invullen van deze toestemmingsverklaring op het volgende:

1. Op dit formulier kunt u aangeven of u ons toestemming geeft om uw tweeling, eventuele broers en zussen van de tweeling en hun leerkracht een vragenlijst te mogen sturen.
2. Alléén leerkrachten van broers en/of zussen die op de basisschool zitten zullen benaderd worden. De kinderen zélf zullen pas door ons benaderd worden wanneer zij 12 jaar of ouder zijn.
3. Wanneer één van uw tweelingkinderen niet in staat is vragenlijsten in te vullen (bijvoorbeeld door ziekte of handicap), dan stellen wij toch prijs op de gegevens van zijn of haar tweelingbroer/zus en eventuele andere broers en zussen. Wanneer u hieronder aangeeft dat één van de tweelingkinderen niet in staat is vragenlijsten in te vullen, dan zal dit kind niet door ons benaderd worden.

Naam: is niet in staat om vragenlijsten in te vullen.

Dit formulier is ingevuld door:

₁ biologische vader ₂ biologische moeder ₃ anders, nl: _____

Naam en voorletter(s): _____

Postcode: _____ Huisnummer: _____

Datum: _____ Plaats: _____

Handtekening: _____

Toestemming voor het benaderen van de TWEELING

<p>OUDSTE (eerstgeborene) van de tweeling</p> <p><input type="checkbox"/> Ik geef toestemming om mijn (tweeling)kind te benaderen</p> <p><input type="checkbox"/> Ik geef <i>geen</i> toestemming om mijn (tweeling)kind te benaderen</p>	<p>JONGSTE (laatstgeborene) van de tweeling</p> <p><input type="checkbox"/> Ik geef toestemming om mijn (tweeling)kind te benaderen</p> <p><input type="checkbox"/> Ik geef <i>geen</i> toestemming om mijn (tweeling)kind te benaderen</p>
<p>Voornamen kind: _____</p> <p>Roepnaam: _____</p> <p>Achternaam: _____</p> <p>Geboortedatum: _____ - _____ - _____</p> <p>Geslacht: jongen/ meisje*</p> <p>Email: _____ @ _____</p>	<p>Voornamen kind: _____</p> <p>Roepnaam: _____</p> <p>Achternaam: _____</p> <p>Geboortedatum: _____ - _____ - _____</p> <p>Geslacht: jongen/ meisje*</p> <p>Email: _____ @ _____</p>

Toestemming voor het benaderen van de LEERKRACHT van de tweeling

<p>OUDSTE (eerstgeborene) van de tweeling</p> <p><input type="checkbox"/> Dit kind zit niet op de basisschool</p> <p><input type="checkbox"/> Ik geef toestemming om de leerkracht te benaderen</p> <p><input type="checkbox"/> Ik geef <i>geen</i> toestemming om de leerkracht te benaderen</p>	<p>JONGSTE (laatstgeborene) van de tweeling</p> <p><input type="checkbox"/> Dit kind zit niet op de basisschool</p> <p><input type="checkbox"/> Ik geef toestemming om de leerkracht te benaderen</p> <p><input type="checkbox"/> Ik geef <i>geen</i> toestemming om de leerkracht te benaderen</p>
<p>Voornaam leerkracht: _____</p> <p>Achternaam leerkracht: _____</p> <p>Geslacht: man/ vrouw*</p> <p>Naam school: _____</p> <p>Postbusnummer school: _____ postcode: _____</p> <p>Adres school: _____</p> <p>Postcode school: _____</p> <p>Plaatsnaam school: _____</p> <p>Telefoonnummer school: _____</p> <p>E-mailadres school: _____</p> <p>Type Basisonderwijs: regulier/speciaal* Groep: _____</p>	<p>Voornaam leerkracht: _____</p> <p>Achternaam leerkracht: _____</p> <p>Geslacht: man/ vrouw*</p> <p>Naam school: _____</p> <p>Postbusnummer school: _____ postcode: _____</p> <p>Adres school: _____</p> <p>Postcode school: _____</p> <p>Plaatsnaam school: _____</p> <p>Telefoonnummer school: _____</p> <p>E-mailadres school: _____</p> <p>Type Basisonderwijs: regulier/speciaal* Groep: _____</p>

** graag doorhalen wat niet van toepassing is*

Appendix VII: Invitation letter in which parents were asked to complete the parental consent form online (version 2009)

Nederlands Tweelingenregister (NTR)

Datum	Uw brief van	Telefax 020-5988832	Bijlage(n) informatiefolder
Ons kenmerk PC-1 DHBQ	Uw kenmerk	Telefoon 020-5988948	E-mail DHBQ@psy.vu.nl

Postadres: Van der Boechorststraat 1, 1081 BT Amsterdam

vrije Universiteit amsterdam



Geachte heer/mevrouw,

U staat met uw tweeling ingeschreven bij het Nederlands Tweelingen Register (NTR). Al vele jaren werkt u mee aan onderzoek van het NTR door vragenlijsten over uw kinderen voor ons in te vullen. Hiervoor willen wij u heel hartelijk danken. Zonder uw inzet zou dit onderzoek niet mogelijk zijn!

Uw tweeling heeft inmiddels de tienerleeftijd bereikt. Om de veranderingen in gedrag en leefgewoonten gedurende deze periode in kaart te brengen, willen wij uw tweeling graag persoonlijk benaderen om te vragen of ze een vragenlijst over zichzelf willen invullen. Als de tweeling broers en/of zussen heeft, zouden wij hen ook graag bij het onderzoek willen betrekken door hen een vragenlijst te sturen op hetzelfde moment dat we de tweeling een vragenlijst sturen. Broers en/of zussen van de tweeling die mee willen doen moeten bij het NTR worden ingeschreven.

Toestemming voor het benaderen van uw kinderen met de vraag of zij een vragenlijst over zichzelf in willen vullen, kunt u geven via onze website:

http://www.tweelingenregister.org/pc_dhbq

Uw *inlogcode* is:

Uw *ID nummer* is:

Wilt u ook wanneer u géén toestemming geeft zo vriendelijk zijn om de verklaring in te vullen? Uw toestemming en de deelname van uw kinderen is geheel vrijwillig en alle gegevens worden vertrouwelijk behandeld. Meer informatie over het onderzoek en de vragenlijst wordt gegeven in de informatiefolder. Als u de verklaring liever op papier wilt invullen of als u meer informatie wenst, dan kunt u tijdens kantooruren contact met ons opnemen (*tel:* 020-5988948, *email:* DHBQ@psy.vu.nl). Als u er prijs op stelt informatie over dit onderzoek in te winnen bij iemand die niet bij de uitvoering van het onderzoek betrokken is, dan is prof. dr. H.A. Delemarre (*tel:* 071-5262824) bereid uw vragen te beantwoorden. Voor andere vragen verwijzen wij u graag naar de website van het Nederlands Tweelingen Register: www.tweelingenregister.org.

Wij willen u en uw kinderen bij voorbaat hartelijk danken voor de medewerking.

Met vriendelijke groet,

Mevr. dr. M. Bartels

Mevr. prof. dr. D.I. Boomsma

Afdeling Biologische Psychologie

Bezoekadres: Van der Boechorststraat 1
Transitorium

Appendix VIII: Overview of items and scales in the Dutch Health and Behavior Questionnaire (DHBQ)

Section	Description	Age
Demographics	Information on the following demographic variables is collected: 1) response date, 2) first name, 3) gender, 4) birth date, ans 5) and zip code	14, 16
	Participant are asked if they are a twin (including birth order) or a non-twin sibling of a twin pair (full sibling, half-sibling with the same biological mother, half-sibling with the same biological father or adoption-, foster-, or stepsibling)	14, 16
	Natural hair and eye color	14
Sport	Respondents are asked whether they exercise on a regular basis	14, 16
	Frequency of participation in specific sports was assessed. Participation is quantified in number of years, times a year, times a week, and average minutes per occasion for a fixed list of sports. In the new version of the DHBQ, level of participation was also added (only for fun, in a league, club selection, regional selection, national selection)	14, 16
	Respondents were asked how good they are in sports compared to their peers.	14, 16
Religion	Respondents were asked whether 1) they had a religious upbringing, 2) whether or not they are religious at the time of assessment, and 3) what religion they support.	14
Attitude toward self	Self-esteem was assessed with the Rosenberg Self-Esteem Scale (Rosenberg, 1965, 1979). Respondents were asked whether they strongly agree, agree, disagree, or strongly disagree with 5 positive and 5 negative statements dealing with a favorable or unfavorable attitude toward oneself.	14, 16
	Optimism was assessed with the Life Orientation Test – Revised (LOT-R; Scheier et al., 1994). Respondents were asked to strongly agree, agree, disagree, or strongly disagree with 3 positive and 3 negative statements dealing with optimistic or pessimistic attitude toward oneself.	14, 16

Physical activity	Information (number of days, average duration per day) on physical activity was measured by questions on frequency of cycling and dancing in clubs during the week and the weekend (number of days, average time per occasion).	14, 16
Life events	Life event questions as they were used in FinnTwin studies (Kaprio et al., 2002) were used to assess the occurrence of 13 life events.	14, 16
Loneliness	Loneliness was assessed using the Shortened version of the R-UCLA (Russell et al., 1980; Hughes et al., 2004). Respondents were asked to indicate how often (almost never, sometimes or often) they were dealing with 3 statements about feelings of loneliness.	14, 16
Friendship	Respondents were asked how many good friends they have ('0 = I have no good friends' – '7 = more than 15'); how satisfied they are with their friends ('0 = very unsatisfied' – '4 = very satisfied'); how important their friends are for them ('0 = very unimportant' – '4 = very important'). Questions about friends were separated into male and female friends.	14, 16
Psychopathology	Emotional and behavioral problems were measured using the Youth Self Report (YSR; Achenbach & Rescorla, 2001). The YSR is for children between 11 and 18 years of age to describe their own functioning. It consists of 112 items from the 2001 version supplemented with 6 items from older versions of the YSR, that are scored on 3-point scales based on the occurrence of the behavior during the preceding 6 months: 0 if the problem item was not true of the child, 1 if the item was somewhat or sometimes true, and 2 if it was very true or often true. In addition to the total problem score (TP), eight syndromes (Anxious Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, Aggressive Behavior) and two broad band scales (Internalizing and Externalizing behavior) are scored from the YSR.	14, 16
Physical health and development	Self-reported health was measured by asking participants to describe their health as excellent, very good, good, fairly good or bad.	14, 16

	Respondents were asked whether one or more illnesses / disorders out of a list of 25 were ever diagnosed. Furthermore, they were asked whether and what medication they used medication for these illnesses / disorders.	14, 16
	Left / right handedness	14
	Respondents were asked about their height and to compare their height with their classmates in primary and high school. Also with regard to height, respondents were asked about the age of their growth spurt.	14, 16
	Respondents were asked about their weight and whether other people ever worried about their weight since primary school because it was either to high or low.	14, 16
	Pubertal development was assessed by asking respondents about hair growth, how old other people estimated them, and whether they have spots.	14, 16
	Sleep pattern was assessed by asking respondents how long they slept on average every night in the past 4 weeks (1 = 10 hours or more – 6 = 5 hours or less)	14, 16
	Treatment for mental problems in the past 4 years	14, 16
	Under treatment of a medical specialist and for what complaints.	14, 16
	Presence of disability or illness reducing daily activities	14, 16
	Presence of a sport injury	14, 16
	Questions about pubertal development for girls: breast development; onset, regularity, and frequency of menstruation; use of some form of anti-conception.	14, 16
	Questions about pubertal development for boys: onset of lowering voice; comparing onset with classmates	14, 16
Personality	Big Five personality traits were assessed with the NEO Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992). Respondents were asked whether they strongly agree, agree, neutral, disagree, or strongly disagree with 74 statements dealing with the personality traits neuroticism, extraversion, agreeableness, openness, conscientiousness.	16

Migraine	Presence of headache not caused as a result of cold, fever, alcohol or drugs	14, 16
	Frequency of migraine (1 = less than once a year – 7 = several times a week)	14, 16
	Duration of the migraine (1 = less than 4 hours – 4 = more than 3 days; 5 = unknown, because of differing duration; 6 = unknown, because of continuous migraine)	14, 16
	Frequency of modest or heavy migraine (1 = 0-4 times – 3 = 11 times or more)	14, 16
	Respondents had to indicate whether they have aversion of light, sound, or smell; sickness / vomiting; loss of sight; twinkling in arm or mouth / speech defect; worsening of migraine through physical effort	14, 16
	Intensity of migraine (1 = light – 5 = heavy)	14, 16
	Whether or not migraine is located at one side of the head	14, 16
	Whether migraine is often pounding / pulsating or different	14, 16
Eating habits	Dieting: respondents were asked whether and how often they ever dieted (1 = never – 5 = I'm always dieting) and how old they were when they started dieting.	14, 16
	Body image: respondents were asked how afraid they are to gain weight or to grow fat (1 = not afraid – 5 = extremely afraid) and which role their appearance / weight plays in how they think about themselves (1 = the most important role – 5 = no role at all). Items are derived from the Growing Up Today Study (Berkey et al., 2000; Rich-Edwards et al., 1994) and the Virginia Twin Survey (Anderson et al., 2002)	14, 16
	Binge eating: respondents were asked whether and how often they binged (1 = never – 5 = more than once a week), whether they felt losing control during binge eating, how long they already binge / had binged (1 = more than 1 year – 5 = less than 1 month), and how old they were when they started binge eating. Items are derived from the Growing Up Today Study (Berkey et al., 2000; Rich-Edwards et al., 1994) and the Virginia Twin Survey (Anderson et al., 2002)	14, 16

Smoking	Smoking: Questions regarding whether respondents ever smoked; age of onset; frequency of smoking (1 = I never smoked on a regular basis – 5 = smoking once or more times a day); quantity of cigarettes (1 = less than 1 a day – 6 = more than 30 a day); expectancy of smoking behavior in one year (1 = no, for sure – 5 = yes, for sure); number of peers who smoke (none, a few, about half, most, all).	14, 16
	Marihuana use: Questions regarding whether respondents ever used marihuana, age of onset; frequency of use during entire life and during the past 12 months; the number of peers who use marihuana (none, a few, about half, most, all).	14, 16
Alcohol	Alcohol use: Questions regarding whether respondents ever drunk alcohol; age of onset; quantity during entire life and during the past 12 months; frequency of alcohol use ('1 = once a year or less' – '7 = daily use'); frequency of getting drunk during entire life and during the past 12 months; average amount of glasses during days in the week and weekend ('1 = less than one' – '7 = more than 20'); the number of peers who drink alcohol (none, a few, about half, most, all).	14, 16
	To asses alcohol problems among adolescents the CRAFFT (Knight et al., 1999; 2002) was used. The 6 items form the acronym CRAFFT: Have you ever ridden in a car driven by someone (including yourself) who had been using alcohol?"; "Do you ever use alcohol to relax, feel better about yourself, or fit in?"; "Do you ever use alcohol while you are by yourself (alone)?"; "do you ever forget things you did while using alcohol?"; Do your family or friends ever tell you that you should cut down on your drinking?"; Have you ever gotten into trouble while you were using alcohol?";	14, 16
Stuttering	Respondents were asked whether they ever stuttered (no; yes, as a child but not anymore; yes, as a child and currently; not as a child, but now). If respondents indicated that they ever stuttered they were asked whether the stuttering troubled them.	14

Subjective wellbeing	Satisfaction with life was assessed with the Satisfaction with life scale (SWLS; Diener et al., 1985). Respondents were asked whether they strongly disagree, disagree, little disagree, neutral, little agree, agree, or strongly agree with 5 statements about satisfaction with life.	14, 16
	Subjective happiness was assessed with the Subjective happiness scale (Lyubomirsky & Lepper, 1997). Respondents were asked whether they strongly disagree, disagree, little disagree, neutral, little agree, agree, or strongly agree with 2 positive and 2 negative items about happiness.	14, 16
	Respondents are asked to rate their quality of life on the 10-step Cantril ladder (Cantril, 1965). The top of the ladder indicates the best possible life, and the bottom, the worst possible life. Participants will be asked to indicate the step of the ladder at which they would place their lives in general.	14, 16
School, Work and Leisure Time	Respondents were asked which situation(s) applied best to them: school, work, unemployed, disabled, parttime work and school, volunteer work, other.	14, 16
	Academic achievement will be assessed by the standardized score of a Dutch National Test (CITO).	14
	Education: respondents were asked which type of education they attend or attended; which profile they attended during secondary school; whether and what diplomas they obtained.	14, 16
	Foreign languages: respondents were asked which foreign languages they attended in secondary school; what their last term result was for these languages; how good they were in these languages compared to their peers ('1 = much worse' - '5 = much better').	14, 16
	Work: respondents were asked what type of work they did: No work; fulltime, more than 32 hours; parttime, 12-32 hours; parttime, less than 12 hours; volunteer work, more than 32 hours; volunteer work, 12-32 hours; volunteer work, less than 12 hours.	14, 16
	Truancy: respondents were asked whether they ever skipped classes; frequency of skipping classes ('0 = never' - '6 = several times a week'); and duration (1 = 1 hour - 4 = whole day).	14, 16

	Leisure time: Respondents were asked how many time they spend on the following activities during a typical day: watching television/ movie, gaming, engaging in pc / internet activities, listening music, joining friends, going out (club, bar) on a seven-point scale ('1 = never' – '7 = more than 8 hours'). The seven-point scale was introduced in the 2007 version of the DHBQ.	14, 16
	Leisure time: Respondents were asked how many spent time they spend on the following activities: making music; reading; drawing/painting; tinkering; joining friends at home; joining friends at their home; joining friends at the street; sport club; brain teasers; going out (club, bar) on a seven-point scale (1 = never – 7 = every day). Before 2007, all items regarding leisure time had to be answered on this seven-point scale.	14, 16
	Charity: respondents were asked how much money they spent on charity on an 8-point scale (1 = no money – 8 = more than 50 euros).	14, 16
Quality of life at present	Respondents are asked to rate how they feel at the moment on a 10-step ladder based on the Cantril Ladder (Cantril, 1965).	14, 16
Family	Family composition: information is obtained on whether respondents live (1) with both biological parents; (2) with their biological mother; (3) with their biological father; (4) with their biological mother and her new partner; (5) with their biological father and his new partner; (6) on their own; (7) together with their partner; (8) other	14, 16
	Family conflict was assessed with the subscale 'Conflict' from the Family Environment Scale (FES; Moos, 1974). Respondents were asked whether or not 11 statements dealing with conflicts in the family applied to their own family.	14, 16
	Number of siblings besides co-twin: dates of birth; sex; relation towards respondent (full sibling, half sibling with same mother, half sibling with same father, step sibling, adoptive or foster sibling); resident or non-resident.	14
	General family functioning was assess with the subscale 'General functioning' from the Family Assessment Device (FAD; Epstein et al., 1983). Respondents were asked whether they strongly agree, agree, disagree or strongly disagree with 12-items measuring the overall	14, 16
	Number of siblings besides co-twin health/pathology of the family, with 6 items worded to describe healthy functioning and 6	16
	items worded to describe unhealthy functioning. The items assess the areas of problem solving, communication, roles, affective responsiveness, affective involvement, and behavior control.	
Twins	Questions were asked about twin similarity (no; sometimes; yes) in the past and currently with regard to clothes preferences; spending time together; friends. In addition, female twins were asked whether they had their first period before, at the same time, or after their female co-twin.	14
	Questions were asked about zygosity; zygosity determination (DNA-test or not); sex of co-twin; birth weight; similarity with co-twin on face, hair color, skin color, and eye color; confusion by family members and strangers.	16
	Twins were asked whether they are in the same school and class.	14, 16
Siblings	Questions were asked about twin-sibling similarity (no; sometimes; yes) in the past and currently with regard to clothes preferences and spending time together	14

Appendix IX: Invitation letter in which twins and/or siblings were invited to complete the self-report survey (version 2005 – 2006)

Nederlands Tweelingen Register (NTR)

Datum	Uw brief van	Telefax 020-5988832	Bijlage(n) informatiefolder, antwoordenvlop.
Ons kenmerk NTR/DHBQ/0207	Uw kenmerk	Telefoon 020-5988827	E-mail DHBQ@psy.vu.nl

Postadres: Van der Boechorststraat 1, 1081 BT Amsterdam

vrije Universiteit amsterdam



Beste tweeling of broer/zus van een tweeling,

Zoals je misschien weet hebben je ouders in het verleden een aantal keer een vragenlijst ingevuld voor onderzoek van het Nederlands Tweelingen Register (NTR). Inmiddels ben je oud genoeg om zelf een vragenlijst in te vullen. We willen je vragen mee te werken aan het grootschalige familieonderzoek naar Gedrag en Leefgewoonten in de Adolescentie. Voor dit onderzoek nodigen we graag de tweeling en hun broers/zussen tussen de 12 en 18 jaar uit.

We willen je vragen om thuis een vragenlijst over gedrag en leefgewoonten in te vullen. De lijst kun je naar ons terugsturen in de antwoordenvlop die bij deze brief is gevoegd. Bij deze brief zit ook een informatiefolder waarin je meer kunt lezen over het onderzoek. Mocht je na het lezen van deze brief en de informatiefolder nog vragen hebben dan kun je contact opnemen met het Nederlands Tweelingen Register (*tel*: 020-5988827, *email*: DHBQ@psy.vu.nl). Voor meer informatie over het tweelingenonderzoek van het NTR kun je ook kijken op www.tweelingenregister.org.

Wij hopen dat je bereid bent om aan dit onderzoek mee te werken. Met het invullen van deze vragenlijst lever je een belangrijke bijdrage aan het wetenschappelijk onderzoek van het NTR. Je deelname is vrijwillig. Als je vragen te indringend of te vervelend vindt, hoef je ze niet in te vullen.

Met vriendelijke groet,
mede namens mevr. prof. dr. D.I. Boomsma,

mevr dr. Meike Bartels

Afdeling Biologische Psychologie

Bezoekadres: Van der Boechorststraat 1
Transitorium

Appendix X: Twin flyer DHBQ (version 2005 – 2006)

ten slotte

We hopen van harte dat je aan dit onderzoek wilt meedoen. Alle informatie wordt strikt vertrouwelijk behandeld. Persoonlijke gegevens, zoals naam en adres, worden losgekoppeld van beantwoorde vragen. Ook in wetenschappelijke publicaties zullen geen namen terug te vinden zijn.

Door een vragenlijst over jezelf in te vullen lever je een belangrijke bijdrage aan het onderzoek van het Nederlands Tweelingen Register. De medewerking van zoveel mogelijk tweelingen en hun familieleden is van essentieel belang voor de voortgang van het onderzoek. Met jouw medewerking kunnen we onder andere meer te weten komen over de factoren die sportgedrag, roken en alcoholgebruik bij jongeren beïnvloeden en over het ontstaan van mentale en lichamelijke problemen bij Nederlandse jongeren.

deelname

Wanneer je ouders daarvoor toestemming geven, zullen we je ongeveer eens in de twee jaar benaderen om te vragen of je een vragenlijst voor ons wilt invullen. Je kunt per keer besluiten wel of niet mee te doen. Broers en zussen van tweelingen die willen deelnemen aan dit onderzoek worden door ons benaderd vanaf het moment dat zij 12 jaar of ouder zijn. Broers en zussen jonger dan 12 jaar kunnen wel ingeschreven worden bij het Nederlands Tweelingen Register. Zij worden echter, voordat zij deze leeftijd bereikt hebben, niet zelf door ons benaderd.

extra informatie

Als je na het lezen van deze folder nog vragen hebt willen we je graag meer vertellen. Neem gerust contact op met de uitvoerende onderzoekers:


Niels van der Aa (tel. 020-598 25 32)
Meike Bartels (tel. 020-598 88 12)
of per e-mail: dhbq@psy.vu.nl

Als je er prijs op stelt informatie over dit onderzoek in te winnen bij iemand die niet bij de uitvoering van het onderzoek betrokken is, dan is prof. dr. H.A. Delemarre bereid je vragen te beantwoorden. Zij is te bereiken op telefoonnummer 071-526 28 24.

website


Het Nederlands Tweelingen Register heeft ook een website: www.tweelingenregister.org. Ga er gerust eens kijken en lees er nog meer over dit onderzoek en haar uitkomsten of over ander onderzoek van het NTR.

<http://www.tweelingenregister.org/>



familieonderzoek

naar
gedrag & leefgewoonten
in de adolescentie



onderzoek naar gedrag en leefgewoonten bij jongeren

Waarom vinden sommige jongeren het heerlijk om urenlang een boek te lezen of muziek te maken, terwijl anderen het liefst de hele dag buiten voetballen? Waarom beginnen sommige jongeren al vroeg met roken, terwijl anderen dat hun leven lang niet doen?

Worden dit soort verschillen tussen jongeren bepaald door verschillen in omgeving (zoals sportgedrag van hun ouders, door vrienden, of door leraren op school) of door verschillen in genetische aanleg?

In het huidige onderzoek willen we proberen een antwoord te vinden op dit soort vragen. We nodigen je uit mee te doen aan dit project door een vragenlijst over gezondheid, sport, persoonlijkheid en welbevinden in te vullen.

langlopend onderzoek bij tweelingfamilies

Dit vragenlijstonderzoek maakt deel uit van een langlopende studie naar ontwikkeling, gedrag en gezondheid. Ouders van jonge tweelingen, die ingeschreven staan bij het Nederlands Tweelingen Register (NTR), worden ongeveer eens in de twee jaar benaderd om een vragenlijst in te vullen over de ontwikkeling en het gedrag van hun tweeling. Als vervolg hierop willen we nu de tweelingen en hun broers en zussen (vanaf 12 jaar) persoonlijk benaderen met het



verzoek om zelf informatie te geven over hun gedrag en leefgewoonten.

waarom onderzoek bij tweelingfamilies?

Tweelingfamilies vormen voor de wetenschap een unieke groep. Met onderzoek bij tweelingen en hun broers/zussen kunnen wetenschappers er achter komen in welke mate verschillen in gedrag en gezondheid worden beïnvloed door verschillen in erfelijke aanleg (de genen) of door verschil in leefomgeving. Eeneige tweelingen zijn genetisch 100% identiek. Twee-eige tweelingen en biologische broers en zussen delen gemiddeld de helft van hun erfelijk materiaal.

Stel nu dat erfelijke aanleg van belang is voor het ontstaan van overgewicht. Dan zullen kinderen binnen één gezin, qua gewicht meer op elkaar lijken dan kinderen uit verschillende gezinnen. Tevens zullen leden van een eeneig tweelingpaar meer in gewicht overeenkomen dan leden van een twee-eig tweeling of andere broers en zussen.

Daarom vragen we tweelingen en hun broers en zussen om mee te doen aan dit onderzoek. We nodigen graag alle broers en zussen vanaf 12 jaar uit (ook half-, stief-, adoptie- en pleegbroers en zussen zijn van harte welkom).

de vragenlijst

In de vragenlijst kun je zelf aangeven in hoeverre een bepaalde gedraging of eigenschap op jou van toepassing is. Onderwerpen die in de vragenlijst aan bod komen zijn onder andere: sportgedrag, vrijetijdsbesteding, roken, alcoholgebruik, school, gezondheid, groei en vriendschappen.

Zodra je de vragenlijst hebt ingevuld kun je deze naar ons terugsturen. Je hoeft niet te wachten tot je broers of zussen hun eigen lijst hebben ingevuld. Ook als je broers of zussen onverhoopt niet mee willen werken, zijn we wel erg blij met jouw deelname.



TWEELINGENONDERZOEK

Appendix XI: Invitation letter in which twins and/or siblings were invited to complete the self-report survey (version 2007)

Nederlands Tweelingenregister (NTR)

Datum	Uw contactpersonen Niels van der Aa	Telefoon 020-5982532	Telefax 020-5988832	Bijlagen Informatiefolder
Ons kenmerk DHBQ_online		E-mail DHBQ@psy.vu.nl		Website www.tweelingenregister.org

Postadres: Van der Boechorststraat 1, 1081 BT Amsterdam



vrije Universiteit amsterdam

Beste tweeling of broer/ zus van een tweeling,

Zoals je misschien weet hebben je ouders in het verleden een aantal keer een vragenlijst ingevuld voor onderzoek van het Nederlands Tweelingen Register (NTR). Voor ons onderzoek naar gedrag en leefgewoonten bij jongeren willen we je vragen of je nu zelf een vragenlijst in zou willen vullen. Informatie over dit onderzoek kun je lezen in de folder die bij deze brief zit.

De vragenlijst kun je invullen op internet of op papier. Om de lijst online in te vullen ga je naar de website van het Nederlands Tweelingen Register:

www.tweelingenregister.org/DHBQ

Door op de link te klikken en je persoonlijke inlognaam en wachtwoord in te vullen kom je in de vragenlijst terecht en kun je beginnen met het invullen.

Persoonlijke inloggegevens van:

Inlognaam:

Wachtwoord:

Nadat je ingelogd hebt, kun je eventueel je wachtwoord veranderen. Je kunt op ieder moment stoppen met het invullen van de vragenlijst en er op een later moment weer mee verdergaan.

Als je de vragenlijst liever op papier invult of als je na het lezen van deze brief en de informatiefolder nog vragen hebt, dan kun je contact opnemen met Niels van der Aa (*tel:* 020-5982532, *email:* DHBQ@psy.vu.nl).

Met het invullen van de vragenlijst lever je een belangrijke bijdrage aan het wetenschappelijk onderzoek van het NTR. Je deelname is vrijwillig. Het kan zijn dat je sommige vragen te indringend of te vervelend vindt. Je hoeft deze vragen dan niet in te vullen. Wij hopen dat je bereid bent om aan dit onderzoek mee te werken en willen je daarvoor alvast hartelijk bedanken.

Met vriendelijke groet,
Mede namens mevr. prof. dr. D.I. Boomsma

Mevr. dr. Meike Bartels

Afdeling Biologische Psychologie

Bezoekadres: Van der Boechorststraat 1
Transitorium

Appendix XII: Twin flyer DHBQ (version 2007 – 2009)

ten slotte

WE HOPEN DAT JE AAN DIT ONDERZOEK WILT MEEWERKEN. ALLE INFORMATIE WORDT STRIKT VERTROUWELIJK BEHANDELD. PERSOONLIJKE GEGEVENS, ZOALS NAAM EN ADRES, WORDEN LOSGEKOPPELD VAN BEANTWOORDE VRAGEN. OOK IN WETENSCHAPPELIJKE PUBLICATIES ZULLEN GEEN NAMEN TERUG TE VINDEN ZIJN.

DOOR EEN VRAGENLIJST OVER JEZELF IN TE VULLEN LEVER JE EEN BELANGRIJKE BIJDRAGE AAN HET ONDERZOEK VAN HET NEDERLANDS TWEELINGEN REGISTER. DE MEDEWERKING VAN ZOVEEL MOGELIJK TWEELINGEN EN HUN FAMILIELEDEN IS VAN ESSENTIEEL BELANG VOOR DE VOORTGANG VAN HET ONDERZOEK. MET JOUW MEDEWERKING KUNNEN WE ONDER ANDERE MEER TE WETEN KOMEN OVER FACTOREN DIE SPORTGEDRAG, ROKEN EN ALCOHOLGEBRUIK BIJ JONGEREN BEÏNVLOEDEN EN OVER HET ONTSTAAN VAN MENTALE EN LICHAAMELIJKE PROBLEMEN BIJ NEDERLANDSE JONGEREN.

deelname

WANNEER JE OUDERS DAARVOOR TOESTEMMING GEVEN, ZULLEN WE JE ONGEVEER EENS IN DE TWEE JAAR BENADEREN OM TE VRAGEN OF JE EEN VRAGENLIJST VOOR ONS WILT INVULLEN. JE KUNT PER KEER BESLUITEN WEL OF NIET MEE TE DOEN. BROERS EN ZUSSEN VAN TWEELINGEN DIE WILLEN DEELNEMEN AAN DIT ONDERZOEK WORDEN DOOR ONS BENADERD VANAF HET MOMENT DAT ZIJ 12 JAAR OF OUDER ZIJN. BROERS EN ZUSSEN JONGER DAN 12 JAAR KUNNEN WEL INGESCHREVEN WORDEN BIJ HET NEDERLANDS TWEELINGEN REGISTER. ZIJ WORDEN ECHTER, VOORDAT ZIJ DEZE LEEFTIJD BEREIKT HEBBEN, NIET ZELF DOOR ONS BENADERD.

extra informatie

ALS JE NA HET LEZEN VAN DEZE FOLDER NOG VRAGEN HEBT WILLEN WE JE GRAAG MEER VERTELLEN. NEEM GERUST CONTACT OP MET DE UITVOERENDE ONDERZOEKERS: NIELS VAN DER AA (TEL. 020-598 25 32) MEIKE BARTELS (TEL. 020-598 88 12) OF PER E-MAIL: DHBQ@psy.vu.nl

ALS JE ER PRIJS OP STELT INFORMATIE OVER DIT ONDERZOEK IN TE WINNEN BIJ IEMAND DIE NIET BIJ DE UITVOERING VAN HET ONDERZOEK BETROKKEN IS, DAN IS PROF. DR. H.A. DELEMARRE BEREID JE VRAGEN TE BEANTWOORDEN. ZIJ IS TE BEREIKEN OP TELEFOONNUMMER 071-526 28 24.



website

HET NEDERLANDS TWEELINGEN REGISTER HEeft OOK EEN WEBSITE: www.tweelingenregister.org. GA ER GERUST EENS KIJKEN EN LEES ER NOG MEER OVER DIT ONDERZOEK EN HAAR UITKOMSTEN OF OVER ANDER ONDERZOEK VAN HET NTR.

familieonderzoek naar gedrag & leefgewoonten in de adolescentie



onderzoek naar gedrag en leefgewoonten bij jongeren

WAAROM VINDEN SOMMIGE JONGEREN HET HEERLIJK OM URENLANG EEN BOEK TE LEZEN OF MUZIEK TE MAKEN, TERWIJL ANDEREN HET LIEFST DE HELE DAG BUITEN VOETBALLen? WAAROM BEGINNEN SOMMIGE JONGEREN AL VROEG MET ROKEN, TERWIJL ANDEREN DAT HUN LEVEN LANG NIET DOEN?

WORDEN DIT SOORT VERSCHILLEN TUSSEN JONGEREN BEPAALD DOOR VERSCHILLEN IN OMGEVING (ZOALS SPORTGEDRAG VAN HUN OUDERS, DOOR VRIENDEN, OF DOOR LERAREN OF SCHOOL) OF DOOR VERSCHILLEN IN GENETISCHE AANLEG?

IN HET HUIDIGE ONDERZOEK WILLEN WE PROBEREN EEN ANTWOORD TE VINDEN OP DIT SOORT VRAGEN. WE NODIGEN JE UIT MEE TE DOEN AAN DIT PROJECT DOOR EEN VRAGENLIJST OVER GEZONDHEID, SPORT, PERSOONLIJKHEID EN WELBEVINDEN IN TE VULLEN.

langlopend onderzoek bij tweelingfamilies

DIT VRAGENLIJSTONDERZOEK MAAKT DEEL UIT VAN EEN LANGLOPENDE STUDIE NAAR ONTWIKKELING, GEDRAG EN GEZONDHEID. OUDERS VAN JONGE TWEELINGEN, DIE INGESCHREVEN STAAN BIJ HET NEDERLANDS TWEELINGEN REGISTER (NTR), WORDEN ONGEVEER EENS IN DE TWEE JAAR BENADERD OM EEN VRAGENLIJST IN TE VULLEN OVER DE ONTWIKKELING EN HET GEDRAG VAN HUN TWEELING. ALS VERVOLG HIEROP WILLEN WE NU DE TWEELINGEN EN HUN BROERS EN ZUSSEN (VANAF 12 JAAR) PERSOONLIJK BENADEREN MET HET VERZOEK OM ZELF INFORMATIE TE GEVEN OVER HUN GEDRAG EN LEEFGEWOONTEN.

waarom onderzoek bij tweelingfamilies?

TWEELINGFAMILIES VORMEN VOOR DE WETENSCHAP EEN UNIEKE GROEP. MET ONDERZOEK BIJ TWEELINGEN EN HUN BROERS/ZUSSEN KUNNEN WETENSCHAPPERS ER ACHTER KOMEN IN WELKE MATE VERSCHILLEN IN GEDRAG EN GEZONDHEID WORDEN BEÏNVLOED DOOR VERSCHILLEN IN ERFELIJKE AANLEG (DE GENEN) OF DOOR VERSCHIL IN LEEFOMGEVING. EENIGE TWEELINGEN ZIJN GENETISCH 100% IDENTIEK. TWEE-EIIGE TWEELINGEN EN BIOLOGISCHE BROERS EN ZUSSEN DELEN GEMIDDELD DE HELFT VAN HUN ERFELIJK MATERIAAL.

STEL NU DAT ERFELIJKE AANLEG VAN BELANG IS VOOR HET ONTSTAAN VAN OVERGEWICHT. DAN ZULLEN KINDEREN BINNEN EEN GEZIN, QUA GEWICHT MEER OP ELKAAR LIJKEN DAN KINDEREN UIT VERSCHILLENDE GEZINNEN. TEVENS ZULLEN LEDEN VAN EEN EENIG TWEELINGPAAR MEER IN GEWICHT OVEREENKOMEN DAN LEDEN VAN EEN TWEE-EIIGE TWEELING OF ANDERE BROERS EN ZUSSEN.

DAAROM VRAGEN WE TWEELINGEN EN HUN BROERS EN ZUSSEN OM MEE TE DOEN AAN DIT ONDERZOEK. WE NODIGEN GRAAG ALLE BROERS EN ZUSSEN VANAF 12 JAAR UIT (OOK HALF-, STEF-, ADOPTIE- EN PLEEGBROERS EN ZUSSEN ZIJN VAN HARTE WELKOM).



de vragenlijst

IN DE VRAGENLIJST KUN JE ZELF AANGEVEN IN HOEVERRE EEN BEPAALDE GEDRAGING OF EIGENSCHAP OP JOU VAN TOEPASSING IS. ONDERWERPEN DIE IN DE VRAGENLIJST AAN BOED KOMEN ZIJN ONDER ANDERE: SPORTGEDRAG, SCHOOL, ROKEN, ALCOHOLGEBRUIK, GEZONDHEID, GROEI, VRIJETIJDSEBETEDING EN VRIENDSCHAPPEN.

ZODRA JE DE VRAGENLIJST HEBT INGEVULD KUN JE DEZE NAAR ONS TERUGSTUREN. JE HOEFT NIET TE WACHTEN TOT JE BROERS OF ZUSSEN HUN EIGEN LIJST HEBBEN INGEVULD. OOK ALS JE BROERS OF ZUSSEN ONVERHOOPT NIET MEE WILLEN WERKEN, ZIJN WE WEL ERG BLIJ MET JOUW DEELNAME.



DANKWOORD

Dit proefschrift is tot stand gekomen met de hulp en steun van vele mensen. De volgende personen wil ik in het bijzonder bedanken.

Op de eerste plaats wil ik graag de vele duizenden tweelingen bedanken die onze vragenlijsten trouw hebben ingevuld en mij daarmee van de gegevens hebben voorzien waarmee mijn proefschrift geschreven kon worden. Zonder jullie medewerking zou dit niet mogelijk zijn geweest!

Dorret en Meike, in de eerste plaats bedankt voor jullie vertrouwen om mij als pedagogische niet-geneticus aan te nemen op dit project. De wereld van de gedragsgenetica boeit me enorm en ik ben blij dat ik erin terecht ben gekomen. Natuurlijk wil ik jullie ook heel erg bedanken voor de energie die jullie in het tot stand komen van mijn proefschrift hebben gestoken en de inzichten en inspiratie die ik vooral dankzij jullie heb opgedaan.

De leden van mijn leescommissie wil ik graag bedanken voor het lezen en beoordelen van mijn proefschrift. Rutger, leuk dat je nu in mijn commissie zit nadat ik tijdens mijn studie stage heb gelopen op jouw afdeling. Hans, bedankt voor de mogelijkheid die je me geboden hebt om als postdoc aan de slag te gaan, ik kijk uit naar onze samenwerking komend jaar.

Alle collega's en ex-collega's van Biologische Psychologie en het Nederlands Tweelingenregister wil ik bedanken voor de leerzame en leuke tijd op de afdeling. Eco, ik heb met plezier met je samengewerkt aan de sportpapers, bedankt voor de inzichten en inspiratie op dit vlak. Lannie, ook mij was het een woest genoeg om de afgelopen 4 jaar een kamer met je gedeeld te hebben, ook al heeft jou dat een koffieverslaving opgeleverd en waren je planten er niet altijd even blij mee. Ik hoop dat we ook de komende tijd roomies zullen blijven! Anouk, onze 'verhitte' discussies over de praktische implicaties van ons werk, maar natuurlijk ook de gezelligheid op en buiten het werk en het 'on you'll light hand side, you see traffic light' zal ik niet snel vergeten. Hannah, je mag dan inmiddels een andere weg zijn ingeslagen maar ik vond het bijzonder fijn dat je hier op de afdeling hebt gewerkt. Ik heb goede herinneringen aan onze gesprekken, de etentjes samen met Sylvana, het klimmen en de overige momenten op en buiten het werk.

Rick en Sylvana, bedankt dat jullie mijn paranimfen willen zijn. Rick, menig weekend hebben we besteed aan het op onvergetelijke wijze inluiden van mijn leven in Amsterdam en onder andere daarmee heb je indirect een belangrijk aandeel gehad in dit boekje. Ik ben er dan ook trots op dat je op de grote dag naast me zit als paranimf. Sylvana,

nadat we tijdens onze stage in Nijmegen al samen op hetzelfde project werkten, vervolgens door onze stagebegeleider gekroond werden tot Jut en Jul en daarna ook nog tegelijkertijd werden aangenomen als AiO, ben ik erg blij dat je, zoals een echte Jul betaamd, mijn paranimf wilt zijn.

Tiuri, bedankt voor de lay-out en het ontwerpen van de cover van mijn proefschrift. De cover is mooier geworden dan ik had durven hopen dankzij jouw verstand van zaken.

Pa, ma en Danja, voor jullie was het misschien niet altijd even goed te volgen waar ik me mee bezig hield de afgelopen 4 jaar; toch wil ik jullie ook graag bedanken voor de interesse die jullie hebben getoond. In mijn proefschrift heb ik laten zien dat zowel genen als een prettige omgeving belangrijk zijn om je goed te voelen. De genen om een gelukkig mens te zijn heb ik niet van een vreemde en iedereen die ooit bij ons thuis is geweest weet dat het aan prettige omgevingsfactoren ook niet heeft geschort. Daarmee zijn jullie het beste praktijkvoorbeeld dat ik me kan bedenken!

En dan ben ik tot slot aangekomen bij de meest speciale persoon in mijn leven: Sandra. Wat betreft mijn proefschrift heb ik het ontzettend fijn gevonden dat je zoveel interesse en betrokkenheid hebt getoond en ben ik dankbaar voor alle steun die je me geboden hebt op de momenten dat ik die nodig had. Jij haalt het beste in mij naar boven en dat is een van de vele redenen waarom ik blij ben dat je er bent!

Niels, november 2010

