Summary

According to the principles of psychobiology (Zuckerman, 1991), personality psychology is still far from the establishment of a commonly accepted paradigm, analogous to what is found in other fields, such as, say, the study of cognitive abilities (Carrol, 1993). Apart from the two generally accepted traits, common to the majority of personality theories, namely Neuroticism (N), and Extraversion (E), little agreement and great variability exist with respect to the number and the nature of the basic dimensions of personality, which are considered necessary to explain individual differences in human behavioral tendencies.

The present thesis represents a contribution to the identification of the basic dimensions of personality from a psychobiological perspective (Eysenck, 1992a; Zuckerman, 1992). To this end, we made use of the personality data from the ongoing longitudinal study of the Netherlands Twin Register (Boomsma et al., 2002). Data from close to twenty thousand individuals from twin families have been collected in six survey studies over twelve years from 1991 to 2002. The sample size, the composition, and the longitudinal character of these data make of them an invaluable resource for the study the structure of personality and its sources of variance.

Chapter 1 provides an introduction to the field of Behavioral Genetics, the methods of twin and family studies, and the general findings concerning the genetic and environmental sources of individual differences in personality. Chapter 2 contains a simulation study that gauges the degree of bias produced by the dependency of family data on the estimates of standard errors and chi-squared, when they are treated as independent observations in a phenotypic model. In addition chapter 2 assesses the efficiency of an estimator, which corrects for dependency. The results showed that when family-clustered data are used for phenotypic analysis, in treating individuals as independent, and using standard Maximum Likelihood estimation, there is a tendency for the chi-square statistic to be overestimated, and the standard errors of the parameters to be underestimated. The bias increases with family resemblance, due to heritability or shared environment. The source of family resemblance –either heritability (h^2) and/or shared environment (c^2)- interacts with the composition of the sample. In the absence of c^2, samples with twins, parents and spouses show the lowest bias, whereas in the presence of c^2 samples with only twins show the lowest bias. In all conditions the bias remained below 15%. The use of the 'complex option' available in Mplus (clustering corrected Robust Maximum Likelihood estimation) reduces the bias to the levels observed when only independent cases are considered. Thus with the use of robust estimates the bias due to family dependency becomes practically negligible in all conditions of dependency. In conclusion, chapter 2 shows that the bias

due to dependency in family data does not form a serious obstacle to phenotypic data analysis. Therefore Mplus Robust Maximum Likelihood estimation was applied in subsequent chapters.

In Chapter 3, following an overview of the leading psychobiological theories of personality (the theories of Eysenck, Gray, Cloninger and Zukerman) contains an empirical study of the structure of personality. The aim of this study was to shed light on the composition and characteristics of the third factor (or factors) beyond Extroversion and Neuroticism, by making use of a longitudinal design to control for inter and intraindividual differences in personality due to age. This method led to the extraction of a third factor composed by the variables Aggression, Anger, Type A Behavior, Extroversion and Rule Breaking Behavior, identified with Gray's Fight Flight System and Zukerman's Aggressive-Hostility factor, and labeled in this study Aggressive Emotionality. The reminder of this thesis addressed the study of the genetics of three of the components of the Aggressive Emotionality factor, namely Type A Behavior, Anger and Aggression. These studies provide the beginning of the study of the characteristics of this third factor as a possible basic dimension of personality.

In chapter 4 the genetic and environmental influences of Type A Behavior (TABP) were studied using an extended twins design (twins and their parents) in an attempt to identify the presence of non-additive genetic effects and sibling interaction effects. The results showed that 45% of the variance in TABP is due to genetic factors, (28% were additive and 17% were non-additive). The remaining 55% of the variance was explained by environmental factors not shared by the members of the same family. Competitive sibling interaction effects were not significant and there was no evidence of sex differences either in variances or means. Chapter 5 addressed the same issues as chapter 4 with respect to the trait anger, by incorporating a repeated measures design that increased the power to detect replicable effects. Results showed that the sources of variance differ across sexes. For males 23% of the variance is due to additive genetic effects, and 26% to dominance genetic effects. For females 34% of the variance is due to additive genetic effects, and no dominance effects are found. There was no consistent evidence to confirm the presence of competitive sibling interaction as an alternative explanation for the low correlations in DZ males. Finally, in chapter 6, we used the analysis of individual growth curves to study individual changes in aggression as well as the genetics of aggression at age 18, in a sample of twins from 11 to 40 years old who participated in four survey studies between 1991 and 2000. The results showed that mean scores on aggressive behavior decrease

during adolescence. By the age 18, aggressive behavior stabilizes. The genetic analysis showed that most of the variance on the slope between 11 and 18 years old was explained by additive genetic effects. The results also showed that 26% of the variance on aggression at the age 18 is explained by additive genetic effects (A), 40% by non-additive genetic effects (D), and 34% by the non-shared environment (E). There were no sex differences in the amount of variance explained by A, D and E.

In summary, this thesis in conjunction with previous evidence show that Aggressive Emotionality should be considered a basic dimension of personality, which is factorially distinct from the other basic dimensions considered in this study, namely Neuroticism, or Impulsivity. Variability in this dimension is influenced by non-additive genetic variation in adulthood, and the effective environmental factors are not shared by the members of the same family. Those personality traits that form this dimension have been consistently related with several social phenomena, but especially with cardiovascular health problems. Behavior genetic studies of the proportion of variance explained by genes and environment constitute only a first step towards the understanding of the process through which genetic and environmental variation act and interact to give rise to variation in a specific phenotype (Kendler, 2005). A detailed understanding of this process, through molecular genetic studies, or the study of gene environment interaction and correlation processes may be instrumental in the prevention of the coronary heart disease and cardiovascular disease associated with high levels of Aggressive Emotionality.