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Spousal resemblance for smoking: Underlying mechanisms and effects of cohort and age☆

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ABSTRACT

Background: In this study we ask why spouses resemble each other in smoking behaviour and assess if such resemblance depends on period of data collection or age. Spousal similarity may reflect different, not mutually exclusive, processes. These include phenotypic assortment (choice of spouse is based on phenotype) or social homogamy at the time spouses first meet, and marital interaction during the relationship.
Methods: Ever and current smoking were assessed between 1991 and 2013 in surveys of the Netherlands Twin Register for 14,230 twins and 1,949 of their spouses (mean age 31.4 [SD = 14.0]), and 11,536 parents of twins (53.4 [SD = 8.6]). Phenotypic assortment and social homogamy were examined cross-sectionally by calculating the probability of agreement between twins and their spouses, twins and their co-twin's spouse and spouses of both twins as a function of zygosity. Marital interaction was tested by investigating the association between relationship duration and spousal resemblance.
Results: Between 1991 and 2013 smoking declined in all age groups for both genders. Spousal resemblance for ever and current smoking was higher when data were more recent. For ever smoking, a higher age of men was associated with lower spousal resemblance. Phenotypic assortment was supported for both smoking measures, but social homogamy could not be excluded. No effect of marital interaction was found.
Conclusions: Differences in smoking prevalence across time and age influence spousal similarity. Individuals more often choose a spouse with similar smoking behaviour (phenotypic assortment) causing higher genotypic similarity between them. Given the heritability of smoking this increases genetic risk of smoking in offspring.

1. Introduction

Many risk factors contribute to human smoking behaviour, including environmental (Freedman et al., 2012) and genetic factors (Li et al., 2003). From previous work in a Dutch sample, we know that spouses often show similar smoking behaviour (Vink et al., 2003). In fact, a systematic review of the literature on spousal resemblance for traits associated with coronary heart disease found that smoking was one of the most strongly correlated traits between spouses (Di Castelnuovo et al., 2009). High spousal correlations (0.19 to 0.55) were also reported by Kuo et al. (2007) and by Clark and Etilé (2006), who showed that the chance of a smoker having a smoking partner is approximately 50%. Boomsma et al. (1994) found that the correlation between husband and wife for 'currently smoking' ($r = 0.43$) was larger than for 'ever smoked' ($r = 0.18$).

There are different, not mutually exclusive, explanations as to how spousal resemblance arises. The three most frequently investigated mechanisms are phenotypic assortment, social homogamy and marital interaction. In case of primary, or *phenotypic assortment*, individuals tend to choose a spouse that is phenotypically

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similar (Falconer and Mackay, 1996). If a trait is heritable, phenotypic assortment is associated with a higher genotypic similarity between spouses, causing a greater phenotypic and genotypic similarity between them and their offspring (Crow and Felsenstein, 1986; Eaves, 1977; Falconer and Mackay, 1996). Alternatively, spouses may be more similar to each other due to *social homogamy* (Heath and Eaves, 1985). Then, individuals are more likely to meet and pair up because they are from similar (social) surroundings. It can also be described as a "passive" influence on mate selection, as opposed to the "active" influence which occurs with phenotypic assortment (Mascie-Taylor and Vandenberg, 1988). Under social homogamy, the genetic resemblance between parents and offspring or between siblings is not expected to increase (Falconer and Mackay, 1996). Lastly, spousal resemblance may be due to *marital interaction* reflecting that two individuals start to resemble each other because they influence each other while being in a relationship together. Here, a longer relationship is associated with more similar behaviour of spouses due to their interaction. Increasing similarity with marriage duration in cross-sectional data could also result from selection: those who are more similar to each other are more likely to remain together. Marital interaction does not have consequences regarding genetic similarity in the next generation.

As phenotypic assortment can be associated with a higher genotypic resemblance between spouses, spousal resemblance can influence the genetic resemblance between relatives. In addition, it shapes the environment to which the offspring of smoking parents is exposed. Data from twins and their spouses can inform on the underlying mechanisms of spousal resemblance (Eaves, 1979; Heath and Eaves, 1985), but studies employing this design for smoking are scarce. In a Swedish sample of 507 twin pairs and 273 twin-spouse couples, Reynolds et al. (2006) found support for phenotypic assortment for quantity of tobacco, while social homogamy explained spousal resemblance for current tobacco use (yes/no). Phenotypic assortment was demonstrated for ever regular smoking in 914 Australian twin-spouse couples (Agrawal et al., 2006), while evidence for both phenotypic assortment and social homogamy was found in a larger US-based study of 14,756 twins and 4390 spouses (Maes et al., 2006). After initial mate selection, a person's smoking status was not influenced by their spouse (Agrawal et al., 2006), arguing against marital interaction for the initiation of smoking. These studies give an indication of the factors behind spousal resemblance for smoking, but only one addressed phenotypic assortment, social homogamy and marital interaction simultaneously (Maes et al., 2006).

Since smoking behaviour is often measured as a dichotomous variable (current smoking yes/no or ever smoking yes/no), resemblance between spouses will depend on smoking prevalence (Falconer and Mackay, 1996). In the Netherlands, smoking prevalence has dropped considerably in the past decades, partly due to nationwide (media) campaigns and tobacco control policies (Nagelhout et al., 2012; Schaap et al., 2008). This decrease has been observed in countries worldwide (Centers for Disease Control and Prevention (CDC) 2009; Giskes et al., 2005). Age is also associated with smoking behaviour, such that ever smoking initially increases with age (Kuo et al., 2007) while older age groups show lower rates of current smoking (Midlöv et al., 2014). Age differences can be due to effects of age itself or differences in birth cohort. Trends across time and age and their effect on spousal similarity must be assessed when studying spousal resemblance for smoking.

The current study explores trends in ever and current smoking in a large sample of 27,715 Dutch twins, spouses of twins and parents of twins, and investigates spousal resemblance for ever and current smoking conditional on period of data collection (1991–1997, 2000–2004, 2009–2013) and age. Phenotypic assortment, social homogamy and marital interaction are investigated as causes of spousal resemblance.

2. Materials and methods

2.1. Subjects

This study is part of ongoing longitudinal survey studies of the Netherlands Twin Register (NTR) (Willemsen et al., 2013). The NTR consists of adolescent and adult twins and their family members who have completed surveys since 1991. For the current study, cross-sectional data on smoking behaviour were available for 27,715 individuals (40.5% male, originating from 10,905 families), consisting of 14,230 twins and 1,949 spouses of twins (mean age 31.3 [SD 13.9]) as well as 11,536 parents of twins (mean age of 53.4 [SD 8.6]). Fig. 1 depicts a flowchart of all included subjects and corresponding analyses.

Data were retrieved from surveys completed in 1991, 1993, 1995, 1997, 2000, 2002, 2004, 2009, 2011 and 2013. Three research cohorts based on time of data collection were created: 1991–1997, 2000–2004 and 2009–2013. Surveys were sent at the family level to the 1991–1997 cohort, while participants were approached individually in the 2000–2004 and 2009–2013 cohorts. If smoking data were available from more than one survey, preference was given to the survey that was completed by most members of a family to increase the number of complete pairs of relatives available for analysis. Recently collected data were preferred over earlier data to ensure the inclusion of as many spouses of twins as possible. Spouses of twins were not invited to participate until the 2000-survey, with recruitment continuing till the year 2013. In total, 1949 spouses were included for 14,553 twins (13.4%). This rather low percentage of participating spouses is not due to twins being in a steady relationship less often. This was shown by a previous study comparing twins with siblings (Middeldorp et al., 2005) and was confirmed by self-reported data on marital status in 9247 twins indicating that the proportion of twins with a spouse was 61.7%. In the final data set, 16.1% of smoking data came from surveys sent in 1991 to 1997, 28.9% from 2000 to 2004 and 55.0% from 2009 to 2013.

Spousal pairs were excluded from analysis when the duration of relationship that spouses reported differed for more than 2 years between them, since this could indicate that the spouses are separated and report on the relationship duration with a new romantic partner (see Fig. 1). Only parents of twins aged 17+ were invited to participate in NTR surveys. Parents who stated to be in a steady relationship for <17 years were excluded since these were presumably reporting on the relationship duration with a new romantic partner. The final number of pairs with complete data on smoking was 5,537 for twin pairs, 1,734 for twin-spouse pairs, 1,346 for pairs consisting of twins and their co-twin's spouse, 325 for pairs consisting of spouses of both twins and 3,725 for parents of twins (father–mother) pairs.

2.2. Measures

Participants were classified as current smokers, former smokers or never smokers, based on the questions "Have you ever smoked?" ("No", "A few times just to try", and "Yes") and "How often do you smoke now?" ("I don't smoke regularly", "I've quit smoking", "Once a week or less", "A few times a week", and "Once a day or more"). Those who said "Yes" to the first question and subsequently stated to smoke once a week or more were classified as current smokers. Answering "I've quit smoking" to the second question resulted in a classification as former smoker. In case of contradictory answers, or when answers to one of the two main questions were missing, additional questions were used to determine classification. Additional questions were, for example, "How many cigarettes a day/a week do you smoke on average?" (for smokers; when a valid answer was given, current smoking was assumed) or "At what age

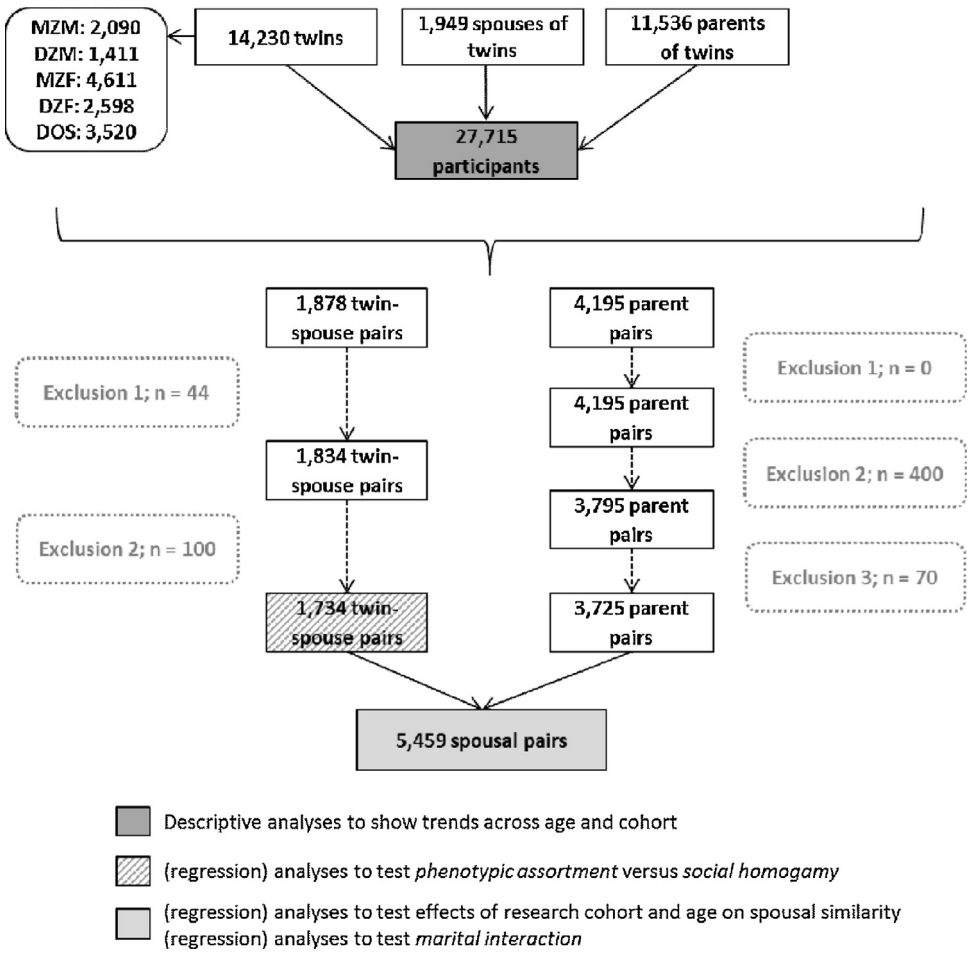

Fig. 1. Flowchart of the groups of subjects and their corresponding analyses. MZM = monozygotic male twins; DZM = dizygotic male twins; MZF = monozygotic female twins; DZF = dizygotic female twins; DOS = dizygotic opposite sex twins; exclusion 1 = exclusion of same-sex spousal pairs; exclusion 2 = exclusion of pairs for which the answer regarding duration of relationship differed for more than 2 years between spouses; exclusion 3 = exclusion of parent pairs which said to have been in a steady relationship or marriage for less than 17 years (given that only parents of twins aged 17+ were invited to participate, these were presumably reporting on the relationship duration with a new romantic partner).

did you quit smoking" (when an age was given, former smoking was assumed). When participants said "Yes" to the question "Have you ever smoked?", but no additional information on their current smoking status was available, smoking status was coded as unknown. These individuals were not included in the analyses or the flowchart in Fig. 1.

Two variables were created reflecting *ever* and *current* smoking. For ever smoking, all participants classified as never smokers were assigned the value "0", while all others (current and former smokers) were assigned the value "1". For current smoking, both never and former smokers were assigned "0", and only current smokers were assigned "1". Covariates were age (in years, continuous), gender (0 = male, 1 = female) and duration of relationship (in years, continuous). Duration of relationship was determined by asking participants "For how long have you had a steady relationship with/have you been married to your spouse?". Information on relationship duration was available for data collected between 2000 and 2013.

2.3. Statistical analyses

Data management and statistical analyses were conducted in SPSS (version 21). To describe age and cohort differences, ever and current smoking rates were stratified by research cohort (1991–1997, 2000–2004, 2009–2013), age in categories (18–24, 25–34, 35–44, 45–54, 55–64 and ≥65 years) and gender. The effects of research cohort and age on smoking rates were determined through logistic regression analysis with smoking as the outcome variable and both research cohort and age as the independent variables. For age, the resulting odds ratio (OR) reflected the odds of being a(n) ever/current smoker compared to a non-smoker at a particular age compared with being one year younger. For research cohort the OR reflected the odds of smoking for a particular research cohort compared with an older research cohort.

The probability of agreement (PA) between two individuals of a pair determined their resemblance and was obtained as follows: *n pairs in agreement/(n pairs in agreement + n pairs not in agreement)*. Pairs with both individuals having the same smoking status are in agreement while pairs who differ in their smoking status are not in agreement. Three sets of analyses were performed, which are described below.

- PA was calculated for all spousal pairs (twin-spouse pairs and parents of twins). To test whether research cohort or age affected spousal resemblance, a logistic regression analysis was then performed with the following regression formula: $Y = \beta_0 + \beta_1 x_{\text{cohort}} + \beta_2 x_{\text{age\_person1}} + \beta_3 x_{\text{age\_person2}} + \varepsilon$. Y represents the resemblance between spouses (either ever or current smoking; 0 = not in agreement 1 = in agreement), β_0 is the intercept (i.e. the value of Y when independent variables are 0), independent variable are x_{cohort} (regression coefficient for cohort;

1991–1997, 2000–2004 or 2009–2013, treated as a continuous variable), $x_{age\_person1}$ (regression coefficient age person 1 in years [continuous]; person 1 is always the man), $x_{age\_person2}$ (regression coefficient age person 2 in years [continuous]; person 2 is always the woman), and ε is the error term.

- To explore phenotypic assortment and social homogamy as causes of spousal resemblance, PA was calculated between twins ($PA_{tw1-tw2}$), twins and their spouses (PA_{tw-sp}), twins and their co-twin's spouses ($PA_{cotw-sp}$) and spouses of both twins ($PA_{sp1-sp2}$). The pattern of resemblance between these different pairs of (extended) family members provides the information to determine the mechanism(s) of assortment (Heath and Eaves, 1985; van Grootheest et al., 2008). If the following is found; $PA_{tw-sp} > PA_{cotw-sp} > PA_{sp1-sp2}$, phenotypic assortment (mate choice is based on phenotype), is likely to be the exclusive mechanism. If genetic influences on the phenotype are present, as in smoking, it is also expected that $PA_{cotw-sp}$ and $PA_{sp1-sp2}$ are higher in families of MZ (monozygotic) twins compared to families of DZ (dizygotic) twins. MZ twins are genetically (almost) 100% identical while DZ twins share only 50% of their segregating genes. Any predisposition for smoking and preference for a spouse with the same smoking status will thus be more similar in MZ versus DZ twins. In contrast, when PA_{tw-sp}, $PA_{cotw-sp}$ and $PA_{sp1-sp2}$ are almost equal and there are no MZ-DZ differences, social homogamy is more likely. It was tested in a logistic regression analysis whether zygosity had a significant effect on the PA, while correcting for differences due to research cohort and age. These analyses included participants of the 2000–2004 and 2009–2013 research cohorts (data on spouses of twins were not available for the 1991–1997 cohort). The formula was as follows: $Y = \beta_0 + \beta_1 x_{zygosity} + \beta_2 x_{cohort} + \beta_3 x_{age\_person1} + \beta_4 x_{age\_person2} + \varepsilon$, with Y representing the resemblance between two individuals (either ever or current smoking; 0 = not in agreement 1 = in agreement) and independent variables being $x_{zygosity}$ (regression coefficient for twin zygosity; 0 = MZ 1 = DZ), x_{cohort} (regression coefficient for cohort; 0 = 2000–2004 1 = 2009–2013), $x_{age\_person1}$ (regression coefficient age person 1 in years [continuous]; person 1 is twin1 in twin1–twin2 pairs, twin in twin-spouse pairs, co-twin in co-twin-spouse pairs and spouse1 in spouse1–spouse2 pairs) and $x_{age\_person2}$ (regression coefficient age person 2 in years [continuous]; person 2 is twin2 in twin1–twin2 pairs, spouse in twin-spouse pairs, spouse in co-twin-spouse pairs and spouse2 in spouse1–spouse2 pairs).
- In the case of marital interaction, spouses who have been together for longer will be more similar. A logistic regression analysis similar to those described above was carried out to test whether duration of relationship has a significant effect on spousal resemblance: $Y = \beta_0 + \beta_1 x_{duration} + \beta_2 x_{cohort} + \beta_3 x_{age\_person1} + \beta_4 x_{age\_person2} + \varepsilon$. Independent variables were $x_{duration}$ (regression coefficient for duration of relationship in years [continuous]), x_{cohort} (regression coefficient for cohort; 0 = 2000–2004 1 = 2009–2013), $x_{age\_person1}$ and $x_{age\_person2}$.

The likelihood that spouses have the same smoking status (PA) depends heavily on how many individuals smoke. When smoking prevalence is for example very low, many spousal pairs will consist of two non-smokers, resulting in high spousal resemblance. To address this issue, Cohen's kappa (k) was reported alongside all probabilities of agreement. k is also a measure of similarity, but takes into account agreement occurring by chance (the proportion of spousal pairs in agreement when mating occurs at random). The first step in calculating k is to subtract all agreement arising by chance from the PA. After it is "normed", k equals 1 if there is full agreement and 0 when there is no agreement at all above that expected by chance (Kwiecien et al., 2011). For all statistical tests, a p-value of <0.05 was considered statistically significant.

3. Results

3.1. Trends in ever and current smoking

In the total sample of 27,715 subjects, the prevalence of ever smoking was 47.1% while for current smoking it was 21.3%. Table 1 gives ever and current smoking rates for different age and gender groups, stratified by research cohort. Additional smoking characteristics such as age at first cigarette and total number of years smoked are shown in Table S1[1]. Men smoked more often than women, but these differences were not large in most groups. A decrease in both ever and current smoking was seen over time, with the lowest rates for the most recently collected data (2009–2013). This was confirmed with regression analyses including both research cohort and age as independent variables. An increasing age was associated with a higher prevalence of ever smoking and a lower prevalence of current smoking, but effect sizes were small (see last two columns of Table 1).

3.2. Effect of research cohort and age on spousal resemblance

In all 5,459 spousal pairs, resemblance between spouses, reflected by the probability of agreement and by Cohen's kappa (k), was higher for current (79.7%, $k = 0.39$ $p < 0.001$) than for ever smoking (65.3%, $k = 0.30$ $p < 0.001$). Spousal resemblance differed across research cohort (1991–1997, 2000–2004 and 2009–2013). The odds of a couple being in agreement versus not being in agreement for ever smoking was 1.13 (95% CI 1.05–1.22) for a more recent compared to a less recent cohort. For current smoking this was 1.85 (CI 1.70–2.02). The age of men showed a significant, negative association with spousal resemblance for ever smoking (OR 0.97 [CI 0.96–0.99]), but not for current smoking (OR 0.99 [CI 0.97–1.01]). The age of women was not significantly associated with spousal resemblance for either ever or current smoking. Duration of relationship and age difference within spousal pairs, stratified by research cohort, are given in Table S2[2].

3.3. Phenotypic assortment versus social homogamy

The probability of agreement was calculated for pairs of family members to test phenotypic assortment versus social homogamy as mechanisms explaining spousal resemblance for smoking (see Table 2). The probability of agreement and Cohen's kappa were higher in MZ families compared with DZ families in twin1–twin2, co-twin-spouse and spouse1–spouse2 pairs for both ever and current smoking. This finding, showing that twins and their co-twin's spouse and spouses of both twins are more similar in MZ twins compared to DZ twins, is supportive of phenotypic assortment (Heath and Eaves, 1985; van Grootheest et al., 2008). In most zygosity groups, twin-spouse probability of agreement was higher than co-twin-spouse and spouse1–spouse2 probability of agreement, again implying phenotypic assortment. However, similarity within spouse1–spouse2 pairs was relatively high (in some cases equal to twin-spouse similarity), which could indicate an influence of social homogamy. Under social homogamy, one would

[1] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering http://dx.doi.org/10.1016/j.drugalcdep.2015.05.018.

[2] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering http://dx.doi.org/10.1016/j.drugalcdep.2015.05.018.

Table 1
Rates of ever smoking and current smoking, stratified by research cohort (1991–1997, 2000–2004 or 2009–2013), age (18–24, 25–34, 35–44, 45–54, 55–64 or ≥65 years when completing the survey) and gender.

	1991–1997		2000–2004		2009–2013		Total		Effect research cohort	Effect age
	% smk	*N*	% smk	*N*	% smk	*N*	% smk	*N*	OR (95% CI)	OR (95% CI)
Ever smoking										
Men										
18–24 years	44.8	764	38.1	488	23.4	1,498	32.0	2,750	0.59 (0.56–0.62)	1.045 (1.043–1.048)
25–34 years	50.7	67	42.6	1,321	36.1	368	41.6	1,756		
35–44 years	75.9	348	39.0	354	37.3	585	48.2	1,287		
45–54 years	78.4	811	65.9	428	50.5	1,446	61.4	2,685		
55–64 years	81.1	169	68.9	499	69.6	1,022	70.5	1,690		
≥65 years	90.0	30	70.2	198	72.6	817	72.6	1,045		
Total	65.8	2,189	50.2	3,288	47.7	5,736	52.0	11,213		
Women										
18–24 years	37.7	697	34.9	855	21.5	2,814	26.7	4,366	0.73 (0.70–0.77)	1.029 (1.027–1.031)
25–34 years	46.0	63	35.5	1,748	30.7	841	34.2	2,652		
35–44 years	72.0	600	44.6	619	42.9	1,162	50.7	2,381		
45–54 years	64.4	779	61.9	730	56.6	2,634	59.0	4,143		
55–64 years	48.5	134	50.6	545	62.6	1,164	58.1	1,843		
≥65 years	n.a.	1	27.4	223	43.9	891	40.5	1,115		
Total	56.8	2,274	42.0	4,720	41.8	9,506	43.9	16,500		
Current smoking										
Men										
18–24 years	38.0	764	29.5	488	19.0	1,498	26.1	2,750	0.56 (0.53–0.60)	0.990 (0.987–0.993)
25–34 years	44.8	67	29.6	1,321	19.6	368	28.1	1,756		
35–44 years	40.8	348	26.6	354	15.4	585	25.3	1,287		
45–54 years	37.4	811	27.6	428	16.0	1,446	24.3	2,675		
55–64 years	35.5	169	21.2	499	13.9	1,022	18.2	1,690		
≥65 years	20.0	30	18.2	198	9.3	817	11.3	1,045		
Total	38.0	2,189	27.0	3,288	15.6	5,736	23.3	11,213		
Women										
18–24 years	31.0	697	27.0	855	15.8	2,814	20.4	4,366	0.61 (0.58–0.64)	0.994 (0.992–0.997)
25–34 years	28.6	63	23.1	1748	12.2	841	19.8	2,652		
35–44 years	40.0	600	21.2	619	16.8	1,162	23.8	2,381		
45–54 years	31.3	779	27.1	730	17.8	2,634	22.0	4,143		
55–64 years	26.9	134	18.5	545	15.7	1,164	17.4	1,843		
≥65 years	n.a.	1	8.1	223	7.1	891	7.3	1,115		
Total	33.2	2,274	22.9	4,720	15.3	9,506	20.0	16,500		

% smk = percentage of ever or current smokers; n.a. = not applicable, numbers were too low; OR = odds ratio representing the odds of being a(*n*) ever/current smoker compared to a non-smoker at a particular age compared with being one year younger, or the odds of being a(*n*) ever/current smoker compared to a non-smoker in a particular research cohort compared with an older research cohort; 95% CI = 95% confidence interval. Two individuals were excluded from this table due to missing information on gender.

Table 2
Probability of agreement for pairs of (extended) family members, stratified by twin zygosity.

	MZM		MZF		DZM		DZF		DOS		Total		Effect of zygosity on PA
	PA	*k*	PA	*k*	PA	*k*	PA	*k*	PA	*k*	PA	*k*	OR (95% CI)
Ever smoking													
Twin1–twin2	83.1%	0.63***	85.3%	0.67***	69.2%	0.37***	72.6%	0.41***	68.0%	0.33***	77.3%	0.51***	0.41 (0.35–0.46)
Pairs (*N*)	842		1,943		530		1,018		1,204		5,537		
Twin-spouse	79.1%	0.58***	77.4%	0.53***	76.7%	0.54***	73.9%	0.48***	77.9%	0.55***	77.1%	0.53***	0.86 (0.69–1.08)
Pairs (*N*)	273		671		146		287		357		1,734		
Co-twin-spouse	67.2%	0.34***	70.0%	0.38***	59.2%	0.19*	60.6%	0.21**	63.5%	0.26***	66.0%	0.31***	0.69 (0.55–0.87)
Pairs (*N*)	217		573		98		231		227		1,346		
Spouse1–spouse2	75.0%	0.48***	86.2%	0.69***	62.5%	0.25	75.0%	0.50**	62.5%	0.25	76.9%	0.52***	0.40 (0.23–0.68)
Pairs (*N*)	56		145		24		44		56		325		
Current smoking													
Twin1–twin2	85.6%	0.58***	86.7%	0.50***	73.0%	0.33***	77.2%	0.30***	69.9%	0.21***	79.8%	0.39***	0.43 (0.38–0.50)
Pairs (*N*)	842		1,943		530		1,018		1,204		5,537		
Twin-spouse	86.1%	0.60***	87.0%	0.52***	81.5%	0.47***	84.7%	0.50***	84.9%	0.59***	85.5%	0.54***	0.87 (0.66–1.14)
Pairs (*N*)	273		671		146		287		357		1,734		
Co-twin-spouse	78.8%	0.37***	78.3%	0.18***	68.3%	0.08	69.3%	0.06	66.1%	0.05	74.1%	0.16***	0.61 (0.48–0.79)
Pairs (*N*)	217		573		98		231		227		1,346		
Spouse1–spouse2	85.7%	0.51***	86.2%	0.37***	75.0%	0.24	72.7%	0.17	59.0%	−0.06	78.8%	0.26***	0.35 (0.20–0.62)
Pairs (*N*)	56		145		24		44		56		325		

MZM = monozygotic male twins; DZM = dizygotic male twins; MZF = monozygotic female twins; DZF = dizygotic female twins; DOS = dizygotic opposite sex twins; PA = probability of agreement; *k* = Cohen's kappa; OR = odds ratio representing the odds of a pair being in agreement on smoking compared to not being in agreement for families of DZ twins, compared to families of MZ twins, corrected for research cohort (0 = 2000–2004 1 = 2009–2013), age of person 1 in years (continuous) and age of person 2 in years (continuous); 95% CI = 95% confidence interval. The analyses included only participants of the 2000–2004 and 2009–2013 cohorts, because data on spouses of twins were not available for the 1991–1997 cohort.
* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$.

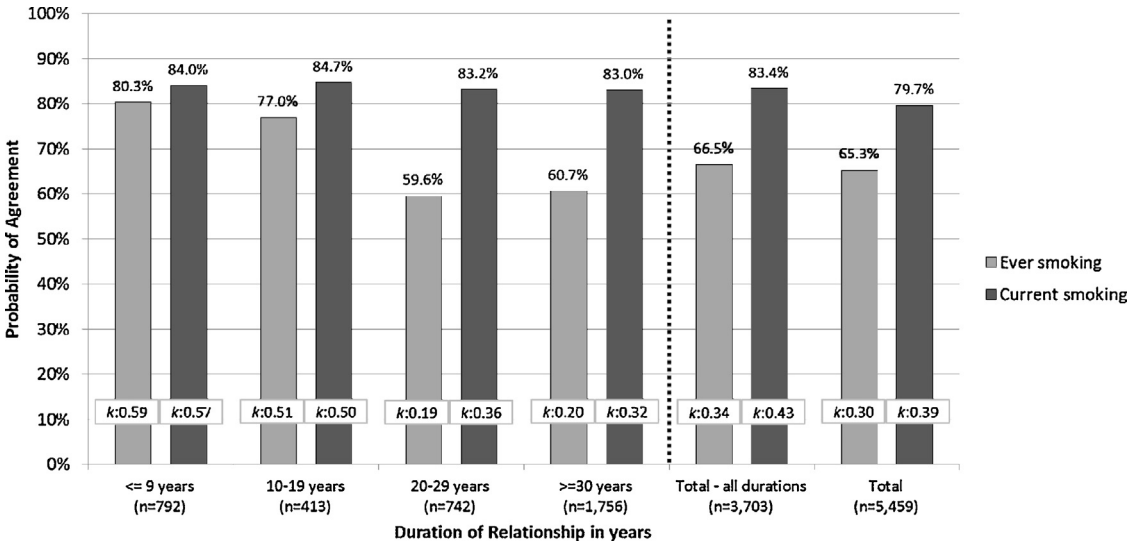

Fig. 2. Similarity within spousal pairs, stratified by duration of relationship in years (≤9, 10–19, 20–29 or ≥30 years). *k* = Cohen's kappa; all *k* statistics were significant at $p < 0.001$; in the group 'Total–all durations', only spousal pairs with information on duration of relationship were included while the group 'Total' contained all available spousal pairs; the spousal pairs comprised both twin-spouse and parent pairs; analyses with information on duration of relationship included only participants of the 2000–2004 and 2009–2013 cohorts, since this information was not available for the 1991–1997 cohort.

expect an equal resemblance within twin-spouse, co-twin-spouse and spouse1–spouse2 pairs.

3.4. Marital interaction

The probability of agreement and Cohen's kappa were calculated for differing durations of relationship (≤9 years, 10–19 years, 20–29 years and ≥30 years) in Fig. 2. When marital interaction is the reason that spouses are similar on smoking behaviour, a higher resemblance is expected for couples with a longer duration of relationship. Though the probability of agreement and Cohen's kappa decreased somewhat when comparing categories ≤9 years and 10–19 years to 20–29 years and ≥30 years, logistic regression analysis (corrected for age and research cohort) showed no significant effect of duration of relationship on spousal resemblance (OR 1.00 [CI 0.99–1.02]). The same was true for current smoking, where logistic regression analysis found no significant effect of relationship duration on spousal resemblance either (OR 1.02 [0.99–1.04]).

4. Discussion

In this large, population based, twin-family study, we studied spousal resemblance for smoking, investigated effects of research cohort and age on this resemblance, and explored the underlying mechanisms. For both measures of smoking (ever and current), spousal resemblance increased significantly in the more recent research cohorts. This increase in resemblance was mostly driven by an increase in the number of nonsmoking couples, while the number of couples in which both had ever smoked or currently smoked decreased across research cohort (data not shown). These findings are consistent with the decrease in both ever and current smoking rates in the more recent research cohorts. Cobb et al. (2014) reported similar results for current smoking, showing a sharp decrease in number of couples where both husband and wife were currently smoking over the course of the study (1986–1998).

Spousal resemblance for ever smoking was lower when men were older, while the age of women did not matter. This difference between genders can be explained by the fact that the prevalence of ever smoking in men increased greatly over age, while in women this increase was more modest and followed by a decrease. Kuo et al. (2007) reported a similar finding, with higher spousal correlations for lifetime smoking in a younger group of twins and their spouses compared to their parents and grandparents, but it was not investigated if this effect was specific for the age of men only. A previous study by our own research group reported the risk of current smoking when having a smoking spouse to be higher compared with having a non-smoking spouse, and this risk decreased with age. There were gender differences too, with a somewhat stronger influence of men on their (female) spouse compared to the influence of women on their (male) spouse (Vink et al., 2003). Resemblance between spouses for smoking may be due to the fact that spouses are usually of a similar age, and age is strongly associated with smoking prevalence. The current study implies that not taking age and time period into account when measuring spousal resemblance for smoking or when making comparisons between different populations, could lead to incorrect conclusions.

Most of the evidence pointed to phenotypic assortment explaining spousal resemblance for smoking behaviour. The main indicator was the fact that co-twin-spouse pairs and spouse1–spouse2 pairs were significantly more similar in MZ families compared with DZ families. The underlying assumption being that the degree of social homogamy is similar in MZ compared with DZ twin families. For ever smoking, these findings comply to the conclusions of Agrawal et al. (2006) and those of an earlier study investigating the three main mechanisms of assortative mating simultaneously (Maes et al., 2006). Our findings on current smoking do not corroborate with the only previous study employing twins and spouses to study spousal resemblance for current smoking (Reynolds et al., 2006), which reported that social homogamy was the most probable underlying mechanism. This discrepancy might have to do with the time of data collection. Reynolds et al. (2006) analysed data from a Swedish sample collected in 1977. Since then, major changes have taken place. Public opinion about smoking has changed, smoking rates have decreased and gender differences in smoking have all but disappeared (Bayer and Stuber, 2006). One could speculate that in the sample of Reynolds et al. (2006), individuals were not specifically rejected by (or attracted to) a person's smoking status because the social 'stigma' on smoking was not as large as it is today (Bayer and Stuber, 2006). Thus, people may have been less concerned about smoking behaviour when choosing a spouse. In such a situation, similarity in smoking status may well be a cause of social homogamy. It demonstrates how time of data

collection can lead to different conclusions about the mechanism(s) through which spousal resemblance for smoking arises. Since the spouse1–spouse2 resemblance for ever and current smoking was relatively high, social homogamy could not be entirely excluded in the current study either. Etcheverry and Agnew (2009) conducted a prospective, multi-wave study in young adults and concluded that spousal similarity on smoking is due to the selection of a spouse more similar to oneself. As one of the very few studies employing twin-family data to explore mechanisms of spousal resemblance for smoking, we have provided further support for phenotypic assortment.

Duration of relationship was not associated with spousal similarity for ever or current smoking, indicating that marital interaction is not of influence. This is in contrast to previous studies reporting that smoking behaviour of one's romantic partner significantly influences smoking initiation in adolescents aged 11–14 years (Aikins et al., 2010) and young adult women (mean age of 26.8 [SD 5.8]) (Dollar et al., 2009). The specific samples of young participants might be the reason that we couldn't replicate these findings. In our sample, with a mean age of 48.1 [SD 15.1] for women and 50.4 [15.1] for men, spousal influence on ever smoking is not very likely given that smoking is usually initiated in adolescence or early adulthood (Freedman et al., 2012). Another study finding evidence for marital interaction (Homish and Leonard, 2005), suggested that the influence between spouses is more prominent in the early phase/years of a relationship. The spousal couples in the current sample have a mean duration of relationship of 26.2 years (SD 14.7) with only 21.3% reporting to be together $\leq$9 years and 5.1% $\leq$2 years, which could explain why we found no evidence for this process. Our findings are in agreement with other reports, showing that spouses do not become more similar for smoking across time (Agrawal et al., 2006; Maes et al., 2006; Clark and Etilé, 2006; Ask et al., 2012). In a smaller sample of NTR participants we previously found a decrease in spousal similarity for current smoking with relationship duration (Willemsen et al., 2003). This was not replicated in the present (larger) study, but both results suggest that marital interaction is not the main mechanism causing spousal resemblance for smoking.

There are some limitations to consider. Since mechanisms underlying spousal resemblance can differ across time, population and/or country, our findings may not be generalizable to all populations. The relatively small number of spouses of twins also made it difficult to disentangle effects of phenotypic assortment from those of social homogamy. Yet, we still found significant effects of zygosity on similarity in co-twin-spouse and spouse1–spouse2 pairs, pointing to phenotypic assortment. Sample sizes were too small to investigate the interaction between research cohort/age and the mechanisms underlying spousal resemblance (phenotypic assortment, social homogamy and marital interaction).

Phenotypic similarity caused by phenotypic assortment also reflects a higher genotypic similarity. It can therefore have important implications for smoking susceptibility in offspring. Children receive both their (family) environment and their genetic material from their parents. Under phenotypic assortment, offspring of two smoking parents are at an increased risk of smoking by receiving the 'risk' genes from both parents. Despite the genetic influences on smoking behaviour, measures taken by the government to discourage smoking have been highly effective in reducing smoking prevalence (Nagelhout et al., 2012; Schaap et al., 2008). This makes sense considering that individual differences in both the initiation of smoking and nicotine dependence can be explained by genetic influences (respectively 44% and 75%) but also for a considerable part by environmental influences (remaining 56% and 25%) (Vink et al., 2005). Especially for smoking initiation, there is a lot to be gained from preventive measures. These prevention programs have so far focused on the general (smoking) population. It might also be beneficial to develop prevention programs focusing on individuals who are the most susceptible. This has been shown to be more effective than applying the same programs to the general population (Sherman and Primack, 2009). Given the importance of genetic factors, these prevention programs should select individuals that are at high genetic risk. Our findings suggest that high risk groups are best identified by selecting children from families where both parents smoke (or have smoked).

Spousal resemblance for both ever and current smoking was associated with research cohort (with a higher resemblance for more recent research cohorts), while only for ever smoking spousal resemblance was associated with the age of men (with a lower resemblance for a higher age). Spousal resemblance for smoking is most likely the result of phenotypic assortment, where spouses select each other directly on their phenotype, but a small influence of social homogamy could not be ruled out. Spousal resemblance was not associated with duration of relationship, arguing against marital interaction.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.drugalcdep.2015.05.018

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