



# Family history and perceived risk of diabetes, cardiovascular disease, cancer, and depression



Marleena Vornanen<sup>a,\*</sup>, Hanna Konttinen<sup>a</sup>, Helena Kääriäinen<sup>b</sup>, Satu Männistö<sup>b</sup>, Veikko Salomaa<sup>b</sup>, Markus Perola<sup>b</sup>, Ari Haukkala<sup>a</sup>

<sup>a</sup> Department of Social Research, University of Helsinki, Unioninkatu 37, P.O. Box 54, 00014 Helsinki, Finland

<sup>b</sup> National Institute for Health and Welfare, Mannerheimintie 166, P.O. Box 30, 00271 Helsinki, Finland

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## ABSTRACT

**Background.** Family history is a useful and inexpensive tool to assess risks of multifactorial diseases. Family history enables individualized disease prevention, but its effects on perceived risks of various diseases need to be understood in more detail. We examined how family history relates to perceived risk of diabetes mellitus, cardiovascular disease (CVD), cancer, and depression, and whether these associations are independent of or moderated by sociodemographic factors, health behavior/weight status (smoking, alcohol consumption, physical activity, BMI [kg/m<sup>2</sup>]), or depressive symptoms.

**Methods.** Participants were Finnish 25–74-year-olds ( $N = 6258$ ) from a population-based FINRISK 2007 study. Perceived absolute lifetime risks (Brewer et al., 2004; Becker, 1974; Weinstein and Nicolich, 1993; Guttmacher et al., 2004; Yoon et al., 2002) and first-degree family history of CVD, diabetes, cancer and depression, and health behaviors were self-reported. Weight and height were measured in a health examination.

**Results.** Family history was most prevalent for cancer (36.7%), least for depression (19.6%). Perceived risk mean was highest for CVD (2.8), lowest for depression (2.0). Association between family history and perceived risk was strongest for diabetes ( $\beta = 0.34, P < 0.001$ ), weakest for depression ( $\beta = 0.19, P < 0.001$ ). Adjusting for sociodemographics, health behavior, and depressive symptoms did not change these associations. The association between family history and perceived risk tended to be stronger among younger than among older adults, but similar regardless of health behaviors or depressive symptoms.

**Discussion.** Association between family history and perceived risk varies across diseases. People's current understandings on heritability need to be acknowledged in risk communication practices. Future research should seek to identify effective strategies to combine familial and genetic risk communication in disease prevention.

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## 1. Introduction

The general public is frequently reminded of health risks of certain lifestyle choices – such as smoking, unhealthy diet and lack of exercise – by mass media and health care professionals. The aim of risk communication is to increase risk perception and motivate preventive behavior (Brewer et al., 2004). Several health behavior theories, including the Health Belief Model (Becker, 1974), assume perceived risk to be a key motivator of preventive behavior. In addition, people supposedly adjust their risk perceptions according to their current behavioral and other risk factors (Weinstein and Nicolich, 1993).

A form of risk information indicating inherited risk is family history, which is a useful and inexpensive tool to assess individual risks of multifactorial diseases (Guttmacher et al., 2004; Yoon et al., 2002). Nordic twin studies suggest heritability to be 20% for type 2 diabetes (Poulsen et al., 1999) and 18–33% for cancer (Verkasalo et al., 1999; Mucci et al., 2016), whereas heritability of depression appears to be 37–50% (Levinson, 2006; Sullivan et al., 2000). Early onset indicates familiarity of cardiovascular diseases (CVD) (Jousilahti et al., 1996), diabetes (Almgren et al., 2011), cancer (Risch, 2001), and depression (Levinson, 2006). Family history can serve as the cornerstone for individualized disease prevention, but its effects on perceived risks of various diseases need to be understood in more detail.

Most people understand genetics in terms of traits and diseases ‘running in the family,’ instead of in terms of the structural and functional nature of genes (Condit, 2010; Jallinoja and Aro, 1999). Family history has shown to be strongly linked to personal risk perceptions of CVD, type 2 diabetes, and cancers (DiLorenzo et al., 2006; Montgomery et al., 2003; Acheson et al., 2010; Wang et al., 2012), but to our knowledge,

\* Corresponding author.

E-mail addresses: [marleena.vornanen@helsinki.fi](mailto:marleena.vornanen@helsinki.fi) (M. Vornanen), [hanna.konttinen@helsinki.fi](mailto:hanna.konttinen@helsinki.fi) (H. Konttinen), [helena.kaariainen@thl.fi](mailto:helena.kaariainen@thl.fi) (H. Kääriäinen), [satu.mannisto@thl.fi](mailto:satu.mannisto@thl.fi) (S. Männistö), [veikko.salomaa@thl.fi](mailto:veikko.salomaa@thl.fi) (V. Salomaa), [markus.perola@thl.fi](mailto:markus.perola@thl.fi) (M. Perola), [ari.haukkala@helsinki.fi](mailto:ari.haukkala@helsinki.fi) (A. Haukkala).

no previous study has explicitly compared this association across different diseases in the general population. Some diseases may be perceived more threatening than others, therefore perceived and actual risks could differ. Awareness of a familial risk may increase sense of self-control (Pijl et al., 2009) and motivate preventive action, such as seeking information, attending screenings, or attempting lifestyle changes (Hariri et al., 2006). However, if family history leads to greater perceived risk for some diseases than others, preventive motivation may vary accordingly.

Research on people's understanding on etiology suggests that people know that familial diseases may be caused by both genetics and/or shared health behavior (Condit, 2010). Those who acknowledge the influence of genetics are also more aware of the role of lifestyle (Sanderson et al., 2011). There is evidence that most people consider genetic and behavioral causes of multifactorial diseases separately, one adding to the other (Condit and Shen, 2011). That is, most people ignore the interactive nature of genes and behavior. However, it is unknown whether this is reflected in personal risk perceptions, for example, whether family history elevates smokers' risk perceptions to the same degree as non-smokers'.

In addition to actual risk factors like family history and health behavior, risk perceptions may reflect cognitive tendencies. Di Lorenzo et al. (2006) found perceived risks of different diseases to be interrelated. Personality traits (e.g. neuroticism) or depressive symptoms may partly explain this. Depressive symptoms may cause a pessimistic bias and thus increase risk perceptions. Moreover, this bias might contribute to genetic fatalism; depressive symptoms might amplify the association between family history and perceived risk.

The aim of this study was to examine whether family history was related to perceived risk of CVD, diabetes, cancer, and depression, and whether this association varied across diseases in the Finnish population. Furthermore, we explored whether associations between family history and perceived risk were modified by respondent's own health behavior/weight status (smoking, alcohol consumption, physical activity, body mass index [BMI kg/m<sup>2</sup>]) or sociodemographics (gender, age, education). Finally, we examined whether respondent's current depressive symptoms were related to perceived risks of CVD, diabetes, and cancer, and whether depressive symptoms moderated the relationships between family history and perceived risks of these diseases. The study extends previous literature by using a large population-based sample that enables exploration of several different possible modifiers of the association between family history and perceived risk.

## 2. Methods

### 2.1. Participants

The participants were 25–74-year-old Finnish men and women from the National FINRISK 2007 Survey conducted in January–March 2007 (Vartiainen et al., 2010). A random sample of 10,000 people, stratified by gender and 10-year age groups, was derived from a population register in five geographical regions ( $N = 6258$ , response rate: 63%). The participants got a mailed invitation to a health examination and a questionnaire on medical history, health behavior, and family history and personal risk perceptions of CVD, diabetes, cancer, and severe depression. They filled it in at home and returned it at the municipal health center where they attended the health examination. Research protocols were designed and conducted in accordance with the guidelines of the Declaration of Helsinki, and approved by the Ethics Committee of the Hospital District of Helsinki and Uusimaa. All participants gave their written informed consent.

For analyses concerning depressive symptoms, we used a subsample of the same participants who attended the Dietary, Lifestyle and Genetic Determinants of Obesity and Metabolic syndrome (DILGOM) substudy in April–June 2007 ( $N = 5024$ , response rate: 80%) (Konntinen et al., 2010). All the FINRISK 2007 participants were invited to this second

study phase, which contained a health examination and various questionnaires, including one on depressive symptoms (Radloff, 1977).

### 2.2. Measures

Perceived lifetime risks of CVD, diabetes, cancer, and severe depression were measured with single items: *How do you perceive your own risk of developing [disease] in your lifetime?* 0 = I have [disease], 1 = very low, 2 = low, 3 = moderate, 4 = high, 5 = very high. Those who reported currently having CVD ( $N = 292$ ), diabetes ( $N = 191$ ), severe depression ( $N = 61$ ), or having/having had cancer ( $N = 184$ ) were excluded from analyses concerning that disease. In a recent study, a similar five-point scale correlated highly with a more continuous measure of perceived absolute risk, and moderately with perceived comparative risk (Godino et al., 2014).

Family history of CVD was assessed with questions on whether a) father, b) mother, c) one or more brothers, d) one or more sisters of the participant had encountered a myocardial infarction prior the age of 60 (in case of mother, 65). Family histories of diabetes, cancer, and depression were assessed with items on whether a) father, b) mother, c) one or more brothers, d) one or more sisters of the participant had been diagnosed with the disease. These items were summed to form a 'family history' variable (scale 0–4) for each disease.

Age group comparisons were made between younger adults (25–39 years, 25.1%), middle-aged (40–59, 42.6%) and older adults (60–74, 32.3%).

Education years were measured with a single item: *How many years have you attended school or studied full time altogether?* For group comparisons, education years were divided into tertiles (indicating low, middle, and high level of education) according to birth year.

Smoking was divided into three categories, 1 = never smokers (53.9%), 2 = former smokers (25.3%), and 3 = current smokers (20.3%). Current smokers reported smoking regularly more than once a day for at least 1 year, including the preceding month.

Average weekly alcohol consumption (grams of pure alcohol per week) during the last 12 months was measured and calculated by asking respondents to describe their usual frequency and quantity of consuming different alcoholic beverages (Dufy and Alankoz, 1992). To reduce skewness (4.55) and kurtosis (28.07) of the distribution, we used square root transformation before conducting correlative and regression analyses. Group comparisons were made by tertiles.

Leisure time physical activity was assessed with a single item, which has shown good criterion validity against morbidity and mortality and moderate correlation against accelerometer counts among the working age population (Fagt et al., 2011): *How much do you exercise and strain yourself physically in your free time?* Response choices were 1 = reading, television, or physically non-exhausting work at home (sedentary, 20.3%); 2 = walking, cycling, or similar at least 4 h/week excluding travel to work (moderately active, 53.2%); 3 = vigorous exercise or work at least 3 h/week; and 4 = competitive training of strenuous sports several times a week. Since few participants ( $N = 97$ ) responded 4, we combined 3 and 4 for group comparisons (active, 26.0%).

BMI was calculated as weight (kg) divided by squared height (m), which were measured by trained research nurses in the health examination. For group comparisons, categories 1 = normal weight (BMI = 18.50–24.99, 36.0%), 2 = overweight (BMI = 25.00–29.99, 40.4%), and 3 = obese (BMI  $\geq 30.00$ , 22.8%) were created (underweight participants were excluded from group comparisons,  $N = 37$ ), based on the World Health Organization classification (Organization WH, 2000). Even though BMI is no actual measure of behavior, for simplicity, we refer to BMI, physical activity, alcohol consumption, and smoking by 'health behaviors.'

Depressive symptoms were measured with the Center for Epidemiological Studies Depression Scale (Radloff, 1977) in the DILGOM substudy.

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