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Heritability of Health Behaviours

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Laatija:
Matilda Suontama

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Tekijä: Matilda Suontama
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Ohjaajat: LT Säde Stenlund ja dosentti, FT, Hanna Lagström
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Health behavior significantly impacts life expectancy, the number of healthy life years, and overall life satisfaction. Health behaviours include any actions taken to promote, protect, or maintain health. Public health education has effectively increased awareness of the fundamentals of healthy behaviour among the general population. However, the practical implementation of these behaviours remains insufficient. A key underlying factor is that, similar to other traits, health behaviours are also heritable.

This literature review summarizes the evidence on the heritability of four major health behaviours: dietary patterns, smoking behaviour, alcohol consumption, and physical activity. The primary focus is on prospective parent-child studies and twin studies. Twin studies are particularly valuable as they allow researchers to distinguish between traits influenced by shared environmental factors and those attributable to genetic heritability. Additionally, this review includes findings from twin, family, and genetic association studies. The key findings of these studies are compiled and presented as summaries and in tables.

The review points to significant heritability of health behaviours persisting into middle age. Approximately half of the variance in these behaviours can be attributed to genetic factors. Of the four reviewed health behaviours, smoking behaviour and alcohol consumption exhibit higher heritability compared to dietary patterns and physical activity. Unexpectedly, the influence of shared environment, such as parental upbringing during childhood, showed minimal impact on behaviours in adulthood.

The findings suggest that the influence of health education received during childhood might diminish into adulthood. On the other hand, the substantial genetic contribution to health behaviours in adults fosters empathy towards individuals affected by prevalent public health diseases largely influenced by one’s behaviour. However, while genetics significantly affect health behaviours, an individual’s environment also plays a crucial role. This offers hope: While inherited attributes are permanent, environment, such as residence and social networks, can be actively affected with possible positive health behaviour consequences.

Avainsanat: Health behaviours, heritability, prospective parent-child association studies, twin studies

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1 Introduction

Health behaviours substantially influence our life expectancy, healthy years of life and life satisfaction. Health behaviour is any activity, that is undertaken for the purpose of promoting, protecting and maintaining health, regardless of one's perceived health status or the objective effectiveness of the behaviour (WHO, 1998). They can be intentional or unintentional and include behaviours such as smoking, substance use, diet, physical activity, sleep and adherence to prescribed medical treatments. Generally, people have some level of knowledge of how they should live; they know what health behaviours they should practice. The issue raised here is that, even with knowledge about healthy behaviour, habits are not easy to change. For example, though people know that they personally have a genetic risk for a disease, that isn't enough to prompt a change in health behaviour (Hollands et al., 2016). One underlying explanation is that, likewise to other traits, health behaviours are also heritable (Polderman et al., 2015). We inherit traits socially and genetically from our parents and they are also influenced by our environment.

This literature review aims to examine and summarize the evidence for heritability of the four major health behaviours: dietary patterns, smoking behaviour, alcohol consumption and physical activity. Following questions will be examined: How heritable are health behaviours actually and at what phase in life would there be the greatest opportunity to support a change in behaviour trajectory? How much of these behaviours can be explained by genes and what is the impact of health education? The main focus is on parent-child prospective studies, but they are also accompanied by twin-, family- and genetic studies.

2 Methods

Practically, the articles for the literature review were found through Google Scholar and PubMed. Heritability and eating behaviours were the terms used to find the first studies. From those articles citations and references led to new articles exploring the same theme. Literature search was done between May 2023 and June 2023. All studies were published in the 21st century and the majority later than 2010. Moreover, studies that had large sample sizes were prioritized. As previously stated, the focus was attempted to be in prospective parent-child association studies. Yet, there were numerous parent-child association and follow-up studies when children were underaged but only a few studies where offspring was grown-up and living independent from their parents. For that reason, other family study types and twin studies are greatly discussed here to get a comprehensive picture of heritability of health behaviours. The study selection process is shown in Figure 1.

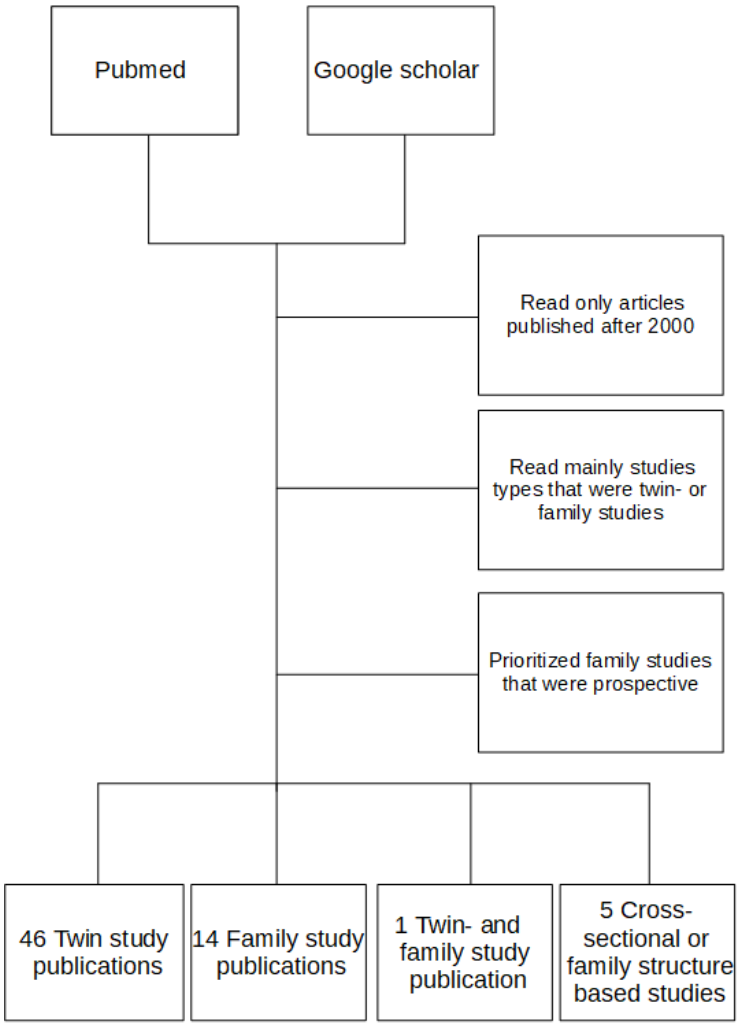

Figure 1. Article selection

In general, all four health behaviours showed similar heritability changes through the lifespan. In childhood and teenage years, parents influence greatly children's health behaviours through shared environment and genes. On the other hand, in adulthood similarities between parents and children are nearly all due to genetics and children's own non-shared environment. Estimates of inheritance are organized for each behaviour by age starting from children and concluding to adults, to facilitate comparisons between estimates in different age groups and life situations. All the studies, that gave an estimate of heritability, and their main results found for this literature review are presented in tables in the supplementary.

In the charts presented in the supplementary studies were categorized based on health behaviour. Dietary patterns, smoking behaviour, and alcohol consumption were categorized by study type as well, including twin studies and family studies. Twin studies examining eating behaviours were further divided into separate charts for children and adults, with adult data separated into charts for eating behaviours and food intake.

3 Results

Study methods of heritability are divided into quantitative methods and molecular genetics. Twin studies as well as family- and adoption studies are quantitative methods and genome-wide association studies, and single nucleotide polymorphism-based heritability estimates belong to molecular genomics. Family studies include parent-child association studies but typically involve more family members e.g. siblings and grandparents. Most known heritability studies are done on twins, but heritability can be estimated in other practices too. In this literature review quantitative methods are the main focus when estimating heritability, because even advanced genomic based-estimates tend to give lower estimates compared to family- and twin studies (Jang et al., 2022).

Almost in all cases analyses of twin samples are displayed in ACE-models where the letter "A" stands for phenotype variation explained by genetic components. "C" is variation by shared environment. It means the environment twins share when they are living with their parents for example, home, parenting behaviours, school, diet, neighbourhood, and peer group attitudes and behaviour. "E" means unique environment variance, also referred to as non-shared environment, explaining the proportion of phenotype that is explained by unique experiences of the world for example circumstances and events they experience and what people an individual meets. (Vainik et al., 2019.)

While twin studies are the most popular study type in the field of heritability, parent-offspring association studies give great insight of heritability, where genetic and shared environment go almost inevitably together. They identify clearly the intra- and extra-familial risk. As it is seen, twin studies allow to differentiate between shared environment and genetics in one generation, the same is possible with parent-child studies when they are compiled with information from many generations and siblings. Most importantly, even though twin- and family studies make different assumptions in heritability estimate calculations the results are still usually outstandingly similar (Stallings et al., 2022).

3.1 Heritability of dietary patterns

3.1.1 Various type studies

The effects of age and life situation are seen prominent in family studies. The child's young age and living at home increases heritability estimates. On the other hand, studies indicate that children who have left their home do not show a strong positive correlation with their parents in dietary variety (Hosseini-Esfahani et al., 2022; Lahmann et al., 2017; Mirmiran et al., 2022). The specific details of the studies included are presented in table S1 in the supplementary.

3.1.1.1 Cross-sectional studies in children

A meta-analysis investigating resemblance in child and parental dietary intake, had data from 15 published articles from 1980-2009 from different continents, though mostly from the US (Y. Wang et al., 2011) . Offspring's age range was 1-30, but most children were under 18. The parent-child correlation was 0.17 (95% CI: 0.18-0.24) for energy intake and 0.19 (95% CI: 0.13-0.28) for fat intake. Resemblance was found to be stronger in younger children (< 10 years). An Australian cross-sectional study provided similar results for dietary intake of mother and 18–23-year offspring (n=2,017 pairs) (Lahmann et al., 2017). Most food groups had correlation of r=0.12-0.29 ($p<0.0001$) and correlation also got lower when children no longer lived with their parents. Correlation was seen stronger in daughters. These results support weak resemblance between parents and children as well as the large effect of non-shared environment.

On the other hand, some traits have shown remarkably strong correlation. A relatively small parent-child study (n=109 pairs) showed that self-reported food neophobia and picky eating were found to correlate significantly in university-aged students in the United States by 0.60 (95%CI; 0.46-0.71) and 0.65 (95%CI; 0.51-0.75)(Elkins, 2018). Most of the children partly lived with their parents and the trait of interest is different, thus the results are not entirely comparable to previously mentioned studies.

3.1.1.2 Parent-child association studies

A three-generational follow-up study explored parent-child correlations in dietary intakes of children, some of whom were adults. The study included 1,286 families from Iran (n=6,949) with ages ranging 8–77 (Hosseini-Esfahani et al., 2022). The study measured the correlation in 15 food components including for example fruit, whole grains, dairy, protein foods,

seafood, vegetable, added sugar and sodium intake. A healthy eating index (HEI) score was computed from these measures. (Krebs-Smith et al., 2018), which is based on American dietary guidelines.

In the same study (Hosseini-Esfahani et al., 2022), in mother-daughter measurements 11 out of 15 food components had a significant correlation ($r>0.10$ and $p<0.01$) and for mother-son pairs all 15 correlated. For father-daughter pairs 4 out of 15 food components correlated significantly and for boys it was 8. The strongest correlation for father-offspring pairs living in the same household was found for whole grain consumption ($r=0.38$ sons and $r=0.24$ daughters, both $p<0.01$). When children lived independently for daughters 5 components still correlated with their mothers and 2 with their fathers. For boys only sodium intake correlated with their mothers ($r=0.11$, $p<0.01$) and total energy with their fathers ($r=0.13$, $p<0.01$).

When children were living at home mother-child correlation in healthy eating was up to 0.44 ($p<0.01$) for girls and boys had a moderate correlation of 0.37 ($p<0,01$). It was noteworthy, that in the same life situation the total healthy eating index correlated significantly with father-son pairs ($r=0.34$, $p<0.01$) but not in father-daughter pairs. When children moved away healthy eating correlated only between mother and daughter ($r=0.22$, $p<0.01$). In addition, there was only a weak correlation between grandparents and grandchildren, meaning that the influence of parents had eventually disappeared. (Hosseini-Esfahani et al., 2022.)

Another study used the same data but examined parent-offspring correlation instead in nutrient intake (Mirmiran et al., 2022). The results were highly similar. Measured nutrients included e.g. total energy, carbohydrate, starch, protein, fat, fibre, vitamin C and mineral intake. When daughters were living with their parent's mother-daughter correlation varied from 0.10-0.34 ($p<0.01$) and 0.02-0.18 ($p<0.01$) when moving out. Correspondable father-daughter correlations were in both situations very low. The sons' correlation with their mother when living at home were from 0.09-0.23 ($p<0.01$) and -0.003-0.18 ($p<0.01$) when moving out. And with father-son pairs in nutrient intake when living at home was 0.08-0.21 ($p<0.01$) and -0.004-0.11 ($p<0.01$) when living independently.

3.1.1.3 Family structure study

A Dutch study investigating heritability of healthy eating patterns explored heredity by both genes and shared environment creating a family structure estimate (n=1,690, age 19–92). The heredity for healthy dietary intake pattern was 0.32 ($p<0,001$) and unhealthy behaviour 0.27

(p<0,001). (van den Berg et al., 2013.) They demonstrated that one-third of participants' health choices had an inherited genetic basis. This is evident in traits that are passed down through generations within families in the study. However, the authors noted that heritability estimates can be affected by shared environmental factor, but they attempt to mitigate this influence through various calculation methods.

3.1.1.4 Summary of the correlations

These results show that the influence of mothers on eating behaviours appears stronger than fathers. Also, daughters' eating behaviours are more persistently influenced by parents than sons, showing up in some traits when daughters have moved away. Most importantly, the correlation between parents and children in eating behaviours in adult years is very modest. Still, these parent-child association not to mention prospective association studies are rare, and evidence in different populations is insufficient.

3.1.2 Twin studies

Heritability of dietary habits can be examined for certain behaviours such as speed of eating, emotional eating and food fussiness or by food intake i.e. what foods people tend to consume. Twin studies in children explored mainly eating behaviours but for adults there were studies on both areas of interest. The detailed information of the studies can be found in table S2 (children) and table S3 and S4 (adults) within the supplementary section.

3.1.2.1 Children

Genomics appeared to explain strongly children's eating behaviours with estimates ranging between 0.24-0.87 (Dubois et al., 2013; Faith et al., 2013; Fildes et al., 2016; Kan et al., 2020; C. H. Llewellyn et al., 2008; Warkentin et al., 2022). Lower correlations were however, found in heritability of emotional eating of 0,07 in 5-year olds (95% CI, 0.06-0.09)(Herle et al., 2017), 2,5 year olds food fussiness and 2,5- and 9-year olds eating a different meal, where there wasn´t genetic component at all (Dubois et al., 2013). In these traits, the shared environment had instead a significant effect, indicating that the attributes are transmitted to children but are not genetically heritable.

A sample of 5-year-old twins (n=2,402) from UK were examined to define heritability of external eating and food responsiveness. External eating means child's desire to eat for pleasure in response to a food cue (smell, sight and taste) and food responsiveness means the

compulsion to eat the food in response to the cues. These traits can be the underlaying cause for a child's BMI. Heritability estimate for external eating was 0.50 (95%CIM 0.43-0.58) and for food responsiveness 0.60 (95%CI; 0.51-0.69). The shared environment had also a large effect on these traits; 0.42 (95%CI; 0.34-0.39) to external eating and 0.29 (95%CI; 0.20-0.38) to food responsiveness. (Kan et al., 2020) A smaller Portuguese twin study (n=172) on 10-year-old children found that food responsiveness was in the same range (0.69, 95%CI; 0.41-0.93)(Warkentin et al., 2022).

A Canadian longitudinal twin study (n=692) assessed children's eating behaviours at the age of 2,5 and 9. The genetic and shared environment component decreased between age groups and the pattern was similar for different behaviours. For certain eating behaviours i.e. not eating enough, eating too much and eating too fast the genetic component decreased when children aged. Furthermore, for eating between meals and eating a different meal the combined effect of genetic and shared environment decreased. Only the genetic component for being fussy about food increased from 0 to 0.81 (95%CI; 0.66-0.91) to the 9-year olds. (Dubois et al., 2013) Overall, it seems like with increased age the effect of one's unique environment increases. Other studies did not find that shared environment had an effect on children over 10-years old (C. H. Llewellyn et al., 2008; Warkentin et al., 2022).

3.1.2.2 Summary of eating behaviours amongst children

Otherwise, all these studies were in line with the observation, that the impact of shared environment in early childhood is crucial, but when children reach the age of 10 years, the effect is gone or decreased greatly. Genetic influence is strong through the entire childhood with the heritability estimates ranging about 0.0-0.87 (but being generally about 0.70).

3.1.2.3 Adults

There was only one study sample was used to investigate the similarity of dietary patterns between parents and their adult children. Therefore, combining twin studies, we can obtain a more accurate estimate of heritability in adults.

In general, eating behaviours in adult twin studies seem to correlate between 0 and 0.64 (Herle et al., 2020; Keskitalo et al., 2008; Leeming et al., 2022; Lopez-Minguez et al., 2019; Masip et al., 2020; Song et al., 2013; Tholin et al., 2005). The study that had biggest sample size (n= 3,977) was a Finnish study with 31-37 old participants. The study presented heritability estimates for emotional and external eating (0.36, 95%CI; 0.29-0.43), snacking (0.39, 95%CI; 0.32-0.45)

infrequent and unhealthy eating (0.48, 95%CI; 0.42-0.54) and avoidant eating (0.36, 95%CI; 0.29-0.42). None of these were explained by the shared environment. (Masip et al., 2020.) Shared environment showed a minor effect on eating behaviours in only two studies with estimates ranging from 0.10 (95%CI; 0.0-0.35) to 0.39 (95%CI; 0.14-0.59) (Herle et al., 2020; Lopez-Minguez et al., 2019). The timing of dinner was most strongly affected by shared environment, with a contribution of 0,39 (95%CI; 0,14-0,59)(Lopez-Minguez et al., 2019).

Food intake included e.g. vegetable, fruit, coffee, sugar and bread consumption along with eating window and dietary variety. Food intake heritability estimates varied between 0.04-0.55 (Hasselbalch et al., 2010; Leeming et al., 2022; Matison et al., 2023; Scheibehenne et al., 2014; Teucher et al., 2007; Treur et al., 2016, 2017) being almost as high as in eating behaviours. Perhaps one of the descriptive studies on food intake explored dietary variety. The study, conducted in the in the USA, involved 5,543 participants from the Mid-Atlantic Twin Registry, who returned the survey they sent. Amongst females' dietary variety was 0.27 heritable in females and 0.30 in males. Shared environment explained 0.14 and 0.15 of the variances. (Scheibehenne et al., 2014.) Confidence intervals can be found in a figure in the original article. Similarly, to eating behaviours only one more study (Teucher et al., 2007) showed any effect of shared environment .

Younger adults (18-48 year old) had heritability estimates of dietary patterns in twin studies between 0.36 and 0.60 (Hasselbalch et al., 2010; Masip et al., 2020; Tholin et al., 2005; Treur et al., 2016, 2017). Though in twin studies, where population was all-aged adults (17-92 old), estimate range was 0-0.64 (Hasselbalch et al., 2010; Keskitalo et al., 2008; Lopez-Minguez et al., 2019; Matison et al., 2023; Scheibehenne et al., 2014; Song et al., 2013; Sung et al., 2010; Teucher et al., 2007; van den Berg et al., 2013). The studies explored eating behaviours and food intake. These may support the direction that there is genetic influence on dietary behaviours, but it decreases through lifespan. Elderly people are known to eat healthier in general(van den Berg et al., 2013), which can be one cause of these results.

Interestingly some studies found a difference between sexes in hereditability of dietary patterns. Females had higher heritability rates in restrained eating (females 0.41 and males 0.21) and emotional eating (0.30 females and 0.21 males) (Song et al., 2013). Similarly in another study cognitive restraint eating, uncontrolled eating and emotional eating had also higher heritability estimates amongst females (Keskitalo et al., 2008). Moreover, vegetable and fruit intake were also analysed separately for females and males. In these traits females

showed higher hereditability estimates: 0.55 for vegetable intake in females and 0.23 in males and 0.28 and 0.04 fruit intake respectively . (Matison et al., 2023).

3.1.3 Summary of dietary patterns

To sum up, the evidence supports that children's dietary behaviours are affected by a mixed effect of genetics and shared environment. In adults, dietary patterns appear also heritable but almost all from genes and not, as it was believed, from shared environment. In the majority of adult studies, the AE-model, excluding the C-component for greater accuracy, provided the best fit for data. This implies, that the factors explaining behaviour are mainly from genes and unique environment. Therefore, non-shared environment has great if not supreme effect in adulthood supported by parent-child association studies, family heritability estimate, GWAS-study and twin-studies.

3.2 Heritability of smoking behaviours

Smoking has been studied greatly in twins and genome-based studies. To the extent of this literature review there was only one longitudinal family study found where offspring were adults (Brook et al., 2013). Other family studies were cross-sectional where causality cannot be demonstrated. Heritability estimates of smoking behaviour were mostly done using twin pairs and whole genome-sequences and in addition supported by parent-child association studies. These showed that shared environment contributed primarily to smoking initiation and smoking in adolescents and genetic factors to nicotine dependence and smoking persistence (Madden et al., 2004; Öncel et al., 2014). There were also SNP-based heritability studies, but the estimates were significantly lower than from other study types. The specific details of the studies included are presented in table S5 in the supplementary.

3.2.1 Various type studies

One family study was longitudinal where the offspring was adult (Brook et al., 2013). The study showed that mothers' smoking will affect offspring's smoking in their late 30s. The children were 14.1 and 36.6 years old when the data was collected from them and their mothers. Study population was from U.S. consisting of 404 participants. Heritability was demonstrated and it was mostly linked to the mother's maladaptive attributes and mother-adolescent relationship. These both affect child's smoking directly and through child's education. Maladaptive attributes were assessed by asking about depressed mood, ego control, impulsivity, and self-esteem. Mothers' cigarette smoking in early 40s as an individual component didn't significantly explain child's cigarette smoking as an adult and mother smoking in mid-60s explained only 0.08 ($p<0.05$) of child's smoking behaviour. The study focused more on what components cause heritability of smoking and didn't give exact rate of heritability.

An earlier study calculated risk ratios for smoking when one parent or both parents smoked. Data was from Dutch twin registry (n=6,501-8,155).They compared adolescents (12–15 years, 16–20 years) and adults (21–40). When 12–15-year-olds had one smoking parent their risk of smoking was 1.38-2.46 (95% CI; 0.85-3.90), non-significant for father-daughter pairs. When both parents smoked risk raised to 2.16-3.06. (95%CI, 1.19-5.64). When offspring was 21–40 years old the risk declined to 1.18-1.41 (95% CI; 0.19-1.74) when one parent smoked and for

both parents smoking it was 1.51-1.62 (95%CI, 1.05-2.16). These suggest that that parent's influence will decrease through age. Risk for adolescent to start smoking if parents smoked was still lower compared to having a smoking sibling. (Vink et al., 2003.)

A more recent study of heritability of smoking initiation was done using a kinship-model, applying data not only from twins, but also from parents, siblings, and offspring. Data was large consisting of 50,318 of U.S. and Australian adult participants. Their heritability estimate from genetic factors was 0.55 for smoking initiation. They suggested that 0.30-0.35 of the variance could be accounted for all sources of shared environment. From the shared environment, the proportion which parents influenced called "special twin environment" accounted for 0.09 of smoking initiation in males and 0.15 amongst females. Rest of the variation was explained by non-parental shared environment, cultural transmission and individual specific environment. (Maes et al., 2018.) This was the almost the only parent-child using data which gave exact heritability estimates.

3.2.2 Twin studies

3.2.2.1 Children

In the teenage years genetics appeared to influence smoking behaviour. In a twin study executed in Virginia with a sample size of 2,804 participants, smoking behaviour was examined, including smoking initiation and current quantity smoked (Do et al., 2015). The study found that smoking initiation during adolescence was influenced by genetics, but the quantity of smoking was not. Significant influence of genetics on smoking quantity started when participants were 22–32 –year old and explained 0.79 (95% CI: 0,66-0.88) of variation. On the other hand, the proportions of genetics in smoking initiation of 14–15-year-olds was 0.54 (95%CI; 0.22-0.86) and in 16–17-year-olds 0.85 (95% CI; 0.77-0.90).

In the previously mentioned Virginian twin study (Do et al., 2015), the shared environment had an effect on 14–15-year-olds on smoking initiation but not anymore for ages 16–17 and 22–32. Furthermore, in a meta-analysis, which had participants (n=19,313) from United States, Europe and Australia, the effect of the shared environment was no less than 0.70 (CI 95%; 0.30-0.95) on smoking initiation at 13 years (Maes et al., 2017). For lifetime smoking, a study using Norwegian twins and added information from parents (n=1,394 families) 0.56 (95% CI; 0.53-0.64) of variance was explained by the shared environment in 12–18-year-olds (Seglem et

al., 2015). For 14–17 year olds 0,85-0,89 (95%CI; 0.68-0.95) of smoking quantity was influenced by shared environment(Do et al., 2015). The shared environment appears to be an important factor for adolescent smoking behaviour.

In conclusion, the shared environment had a major role during childhood for smoking initiation and behaviour likely because the majority start smoking before they become adults (Edwards et al., 2013). Median starting age in a Spanish study (n= 4,570) was 17 years old and 83,7% of future smokers smoked already at 20 years old (Mezquita et al., 2018). Genetics are also important for smoking initiation but to smoking quantity genetics seem to have more significant contribution only after 20s.

3.2.2.2 Adults

Smoking behaviour is largely due to genetics in adult population. Studies suggest that being a smoker is by half accounted by inherited traits. Genetic estimates for smoking from adult twin studies were between 0.23-0.76 (Bao et al., 2016; Dongmeng et al., 2022; Lessov-Schlaggar et al., 2006; Madden et al., 2004; Mezquita et al., 2018; Treur et al., 2017; Vink & Boomsma, 2011; Zhang et al., 2012). The lowest estimate came from a Chinese male twin population (n= 11,625 pairs) where the heritability of cigarette smoking was found to be 0.23 (95% CI; 0.16-0.29) (Dongmeng et al., 2022). The highest heritability was found in a Dutch Twin register of 10,368 twins. For current smoking the heritability was 0.76 (95% CI 0.70-0.79). (Treur et al., 2017.)

For all smoking behaviours including amount smoked, smoking cessation and smoking initiation, genetic hereditability from twin and genome sequence studies were between 0.05-0.88 (Bao et al., 2016; Broms et al., 2006; Do et al., 2015; Domingue et al., 2016; Dongmeng et al., 2022; Hamilton et al., 2006; Lessov-Schlaggar et al., 2006; Li et al., 2003; Madden et al., 2004; Maes et al., 2017; Mezquita et al., 2018; Öncel et al., 2014; Treur et al., 2017; Vink et al., 2003; Vink & Boomsma, 2011; Zhang et al., 2012) . Not surprisingly, lowest estimate was for onset age of smoking 0.05 (95% CI; 0.0-0.14), which was an estimate from 6,458 Chinese male twins (Bao et al., 2016). Highest genetic heritability estimate was number of years smoked done in Spain (n=2,285 twins). In that study, heritability of number of years smoked to 47 years old was 0.79 (95% CI 0.64-0.89) for males and 0.88 (95% CI, 0.77-0.94) for females. (Mezquita et al., 2018.) Both studies confirm the earlier conclusion that smoking persistence is highly heritable whereas initiation and early smoking are more affected by the shared environment.

The genetic risk of expressing smoking behaviour has appeared larger in men (Broms et al., 2006; Hamilton et al., 2006; Li et al., 2003; Madden et al., 2004; Mezquita et al., 2018). For example, in the aforementioned Spanish investigation, lifetime smoking variance was 0.87 (CI 95% 0.73-0.95) explained by genetics in males and 0.49 (95% CI 0.17-0.87) for females showing a considerable difference. The study proposed that the society in the during the 1900s, when the data was collected, was more permissive for men smoking than for females and hence shared-environment factors didn't influence male's smoking behaviour as much as females'. (Mezquita et al., 2018.) Due to a different approach, another study came to a distinct conclusion from similar results. They suggested that genotype explaining the variation in both sexes has increased with decades , where smoking is being more banned and for that reason the ones who can stop smoking (from genetic point of view) will do so. (Domingue et al., 2016.) Their implication was that banning will result in genetic hereditability while the previous study proposed that freedom was the cause of increase in genetic hereditability.

But the evidence is not that clear for the difference between sexes. Other studies have found genetics explaining a little more of females' amount of smoking (Broms et al., 2006), number of years smoked (Mezquita et al., 2018) and smoking initiation (Hamilton et al., 2006; M. D. Li et al., 2003).Furthermore, in smoking persistence there wasn't found difference between groups (Hamilton et al., 2006).

In line with studies done in children were shared environment explained largely smoking behaviours also adults are affected by the shared environment. For adult smokers the shared environment explained 0-0.54 in twin studies (Dongmeng et al., 2022; Lessov-Schlaggar et al., 2006; Madden et al., 2004; Mezquita et al., 2018; Vink & Boomsma, 2011). For example, in 18-25-year-old Dutch twins (n= 2,669 pairs) 0.23 (95% CI 0.07-0.39) of variation was explained by shared environment (Vink & Boomsma, 2011). Other smoking behaviours, including the amount smoked (Broms et al., 2006) and smoking persistence (Li et al., 2003) were influenced significantly by the shared environment in adult twins. A Chinese study further identified the factors that had a significant effect on cigarette smoking (Dongmeng et al., 2022). These were family habits, cognition and attitude towards smoking hazards and family income.

However, there is evidence that the effect of the shared environment could decrease during life. In Scandinavian and Australian twin pairs (n = 21,883), the effect of genetics stayed the same while that of the shared environment in smoking decreased during the life course. The groups composed of 18-25-, 26-35- and 36–46-year-olds and in females the shared

environment accounted for 0.45 (95% CI 0.40-0.53), 0.35 (95% CI; 0.29-41) and 0.26 (95% CI; 0.19-0.32) of variance respectively. Similarly, among Scandinavian men the shared environment explained 0.33 (95% CI; 0.26-0.40), 0.29 (95% CI; 0.22-0.35) and 0.19 (95% CI; 0.12-27) of the variance in the same age-groups. The comparable estimates for Australian men were 0.26 (95% CI; 0.14-0.36), 0.09 (95% CI; 0.0-0.23), 0.11 (95% CI; 0.0-0.28) . (Madden et al., 2004.) Also in 19,000 adolescent twin pairs, additive genetic factors appeared to increase by age while the effect of the shared environment decreased (Maes et al., 2017).

3.2.3 Summary of smoking behaviours

The role of genes increases by aging, while the role of shared environment seems to decrease. In conclusion, for smoking behaviours what you receive from your parents is profound. Both genetics and upbringing are important. As an estimate, additive genetic effect and shared environment account more than half of one's smoking behaviours, which is large.

3.3 Hereditability of alcohol consumption

3.3.1 Family studies

Alcohol use has been studied greatly in parent-child association studies, and specifically for adolescents there is wide literature of cohort studies available. Association of parents' and offspring's alcohol consumption is often found but there has been question about, how well confounding factors are controlled for and how much association studies can say about causality. In a systematic review about children's drinking, Rossow et al. (2016) proposed that false associations could be due to shared local environment, cultural and religious factors and parental comorbidities and temperament. Longitudinal prospective studies can still help us in understanding the heritability of alcohol consumption alongside other study methods. Furthermore, the main result from twin studies is that drinking behaviours seem to be supremely genetically heritable and slightly from shared environment also. Alcohol disorders might also have a tendency to be more heritable than alcohol consumption suggested by few studies (Clarke et al., 2021; Hansell et al., 2008; Heath & Martin, 1994). The details of the studies are found in the supplementary in table S6.

3.3.1.1 Children

An extensive parent-offspring resemblance study of drinking behaviours was implemented using three twin cohorts from Minnesota. Children were assessed at five time points during ages 14–29 while parents were assessed when children participated the study. Study population in total was 3,762 offspring and 3,508 parents. Alcohol use was estimated using drinking quantity and frequency as well as with maximum drinks and number of intoxications. Drinking index, a measure for alcohol consumption, correlated more than dependence symptoms. Offspring's alcohol consumption correlated with their parents at the age of 14 r=0.12 and increased by the age 17 to r=0.25. By the age 29 correlation of alcohol consumption decreased a little to r=0.19. Symptoms of alcohol dependence correlated at the age of 17 r=0.18 and declined at 20 and remained stable to 29 r=0.11. Standard errors can be found in a graph in the original article. There wasn't found statistically significant sex difference in these two traits, although female offspring showed consistently higher correlations with both parents (p=0.08). (Saunders et al., 2017.)

A similar curve and stabilization at least by the age of 30 was found in a later study from the U.S. The study examined parents, some who had history of alcohol use disorder (AUD).

Correlation between parents' and their children's AUD episodes ranged between 0.02-0.25 in those aged 14-30 years, varying depending on whether parents were diagnosed with AUD or not. Due to a small number of offspring (n=739) differences in correlation for parents with and without history of AUD weren't statistically significant. On the other hand, it was observed that between offsprings risk for alcohol dependence and parents who had AUD, there was a significant positive linear association (p=0.013.)(Kosty, 2020.)

Coming back to adolescents, a systematic review of longitudinal studies of adolescent alcohol use, published in 2010, included 77 articles supporting the result that parental drinking is associated with earlier initiation age ($p<0.001$) and increased later alcohol use ($p<0.001$). They also found that there is besides parental modelling an effect of modifiable shared environment that protects offspring from alcohol use such as limiting availability of alcohol, parental monitoring, parent–child relationship quality, parental involvement disapproval of adolescent drinking, general discipline, and general communication. (Ryan et al., 2010.)

A systematic review of prospective cohort studies of children's drinking tried to evaluate the causality of parent and offspring alcohol consumption (Rossow et al., 2016.). Studies were from U.S, Australia, Netherlands, New Zealand, Finland and the UK. Out of 21 studies four studies could show a small causality from parents drinking to children's drinking. The association of parental drinking and alcohol-related outcomes in children was strong, found in 19 out of 21 studies, but from their point of view evidence was still insufficient to indicate definite causality. Their reasoning was that, only some of the studies had taken into account confounding factors and studies weren't adequately theory-based. (Rossow et al., 2016.)

From the four studies that showed some evidence for cause and effect, in three causality varied between $\beta = 0\text{-}0.22$(Latendresse et al., 2008; Mares et al., 2011; Pears et al., 2007) Follow-ups had at least three years between starting point and outcome measure. Causality was linked stronger to fathers' drinking behaviours than mothers'. (Rossow et al., 2016.)

The fourth one, showing small causality, was a prospective study of adolescents. They counted risk ratios for children aged 13.5, 15.5 and 17.5. The study population was from Australia with 715 individuals. They divided offspring into high and low-drinking groups and controlled the influence of parental education, socio-economic status and family stress. Parents' drinking increased the risk of children's high alcohol use: father's drinking by 1.40 (95% CI, 1.04-1.89) and mother's drinking by 2.77 (95% CI; 1.86-4.13). In addition, the amount parents drank influenced the children's drinking amount. If the parents never drank,

high drinker child prevalence was 48.2% for male and 54.2% for female offspring whereas if the parent was a moderate or high drinker prevalence was 92.5% and 94.1 %. (Alati et al., 2014.) The results could point to some heritability of drinking behaviours.

3.3.1.2 Adults

To extend evaluation into adults, a Norwegian prospective study found that parental binge drinking and frequency of alcohol consumption at mid-adolescents predicted the same outcomes in 28-year-old children. They had 2,558 respondents from a population-based sample, and data was collected when children were 15, 17 and 28 years old. Parent's binge drinking predicted 0.13 ($p<0.001$) of children's binge drinking and alcohol consumption predicted 0.09 ($p<0.001$) of children's alcohol consumption. Results were controlled over covariates associated with other parental influences, peers, educational career and emerging marginalization processes. In addition, although amongst males binge drinking was more prevalent, magnitude of parent's influence on alcohol related behaviour was still the same for males and females. (Pedersen & von Soest, 2013.)

A Finnish population population-based cohort study observed participants into the middle age. This enabled studying the question whether there are long-term associations between children's and parents' drinking behaviour. The parents were assessed when children were 16. Fathers' mean age that time was 46.0 and mother's 44.0. The children were tested with the same measure of drinking between ages 21-28 (n = 2,969) and 31-37 (n=2,269). The correlation between fathers and adult children in problem drinking was 0.12-0.18 ($p<0.001$) and between mothers and adult children 0.9-0.14 ($p<0.01$). There wasn't a remarkable change in the correlation between offspring's younger and older assessment. Mid-twenties association was between 0.09-0.18 ($p<0.01$) and in their mid-thirties. 0.11-0.18 ($p<0.001$). Heavy drinking occasions -correlation with parents and children was similar: 0.12-0.19 ($p<0.001$). Their conclusion from these results was that parental problem drinking explained only 0.01-0.03 of variation in offspring's problem drinking. (Sipilä et al., 2023.)

3.3.1.3 Summary of family studies in adults

On the whole, in prospective studies drinking behaviour correlated between 0.09-0.19 (at least $p<0.01$) (Englund et al., 2008; Merline et al., 2008; Pedersen & von Soest, 2013; Saunders et al., 2017; Sipilä et al., 2023) Correlation estimates were between parents and their adult children. As always, there were exceptions and contradictions. For example, two longitudinal studies

from Finland, which both related to problem-drinking. No significant association was found between parents drinking when children were 16 years old and child's alcohol use disorder when they were 28-year-old (n=6,963)(Parra et al., 2020). Association of 0.31 ($p<0.001$) was found between parental drinking when children lived at home and children's problem drinking in an article, about parental drinking association to children's (age mean 42) problem drinking (n=347)(Pitkänen et al., 2008). All in all, the results from family studies support little causality with modest correlation.

3.3.2 Twin studies

In twin studies with mainly adult participants, heritability of alcohol consumption, current drinking and first alcohol use is about 0.31-0.79. Shared environment factor of the same traits has found to have 0-0.39 effect (Hansell et al., 2008; Lessov-Schlaggar et al., 2006; Rose et al., 2001; Sartor et al., 2009; Zhang et al., 2012). In same settings, adolescents genetic component for drinking behaviours was about 0.27-0.47 and shared environment between 0-0.62(Hopfer et al., 2003; Pagan et al., 2006; Zheng et al., 2019). In other words, the considerable difference between adults' and adolescents drinking behaviour is in the effect of shared environment. Adolescent are affected considerably by it, whereas adults are not. The details of twin studies and one family structure estimate examining alcohol consumption are found in the supplementary in table S7.

A review of twin and adoption studies of adolescent substance use had eight studies that had examined alcohol use. Heritability estimates from genetic and shared environment were 0.34 and 0.58 in 15–16-year-olds and 0.43 and 0.47 in 17 and older. The effect of genetics for alcohol use increased even in a few years of time. Genetic factors were observed more in boys than girls. (Hopfer et al., 2003.)

Furthermore, a study of adolescent's alcohol drinking identified three drinking trajectories from 877 Canadian twins: early, normative and low. Study was based on twins' reports of their alcohol use when they were 13, 14, 15 and 17 years old. Hereditability estimates were between 0.27-0.38 (95% CI, 0.01-0.70) from genetic sources. Shared environment accounted for 0.22 (95%CI; 0.0-0.53) and 0.42 (95%CI; 0.20-.62) of variance in the early and low drinking groups. Interestingly, adolescents in the normative trajectory, which meant that children basically never drank, weren't influenced by shared environment at all, but by unique environment 0.62 (95%CI; 0.50-0.77). (Zheng et al., 2019.)

As we have seen with other traits also, initiation of alcohol use was most strongly affected by shared environment, and frequency of drinking is affected more by genetics and unique environment. In a Finnish twin study, the initiation age of alcohol use was mostly explained by shared environment 0.59 (95%CI; 0.51-0.66) and additive genetic factors came second with 0.29 (95%CI; 0.22-0.37). For frequency of alcohol use the corresponding estimates were 0.34 (95%CI; 0.25-0.42) and 0.39 (95%CI; 0.30-0.49). Data was from two independent longitudinal twin cohorts (n=3,009 twin pairs), where alcohol initiation and frequency were measured at the age of 14, 17 and 25. (Pagan et al., 2006.) Generally speaking, at adolescence genetics and shared environment are approximately equal in value and most influential to drinking behaviours. When children start their independent lives, the proportion of shared environment decreases to mostly insignificancy as it is shown in the following section.

The effect of age was observed in an Australian twin study investigating heritability and stability of alcohol consumption and dependence. Alcohol consumption was measured from the quantity and frequency of use whilst dependence from the symptoms. Study included 12,045 individuals from a younger (age 23–39) and an older cohort (age 28–90). In the younger cohort heritability for alcohol consumption was 0.31 (95%CI, 0.23-0.36) and in the older cohort 0.47 (95%CI; 0.43-0.51). In the fully saturated model, only 0-0.006 of alcohol consumption could be accounted for shared environment, which meant that unique environment was the best explanation for the remaining similarity. In contrast, heritability of alcohol dependence in the younger and older cohort were 0.46 (95%CI; 0.37-0.49) and 0.46 (0.42-0.51) respectively, being much more similar regardless of age. They also found that genetic influence was highly stable (r = 0.96) in the 5.5. and 11 years intervals when data was collected, whereas nonshared environmental influence varied more (r=0.33-0.34). (Hansell et al., 2008.)

To back up evidence for problematic drinking, in a meta-analysis alcohol use disorders were found to be to be 0.49 (CI 95%; 0.43-0.53) heritable. (Verhulst et al., 2015). Meta-analysis consisted of 12 twin studies and 5 adoption studies. Shared environment explained 0.10 (95% CI 0.03–0.16) of alcohol use disorders. In this sample, they found no evidence for sex-differences in heritability.

3.3.3 Summary of alcohol consumption

The effect of genetics was observed to be stronger in adults whereas the shared environment during adolescence. Heritability was observed stronger in twin studies than in family studies.

In addition, there wasn't clear evidence for any sex difference for heritability of drinking behaviours. Dependence and problematic drinking didn't have significantly higher heritability or association estimates in the studies included in this literature review. Shared environment had a minor influence on drinking in adulthood and can't be completely neglected in later life because parents are a part of children's shared environment. For long-lasting changes in alcohol use, the focus should be turned to parents' behaviour regardless of its genetic roots, because it is the most modifiable component in the chain of generations.

3.4 Heritability of physical activity

Just recently, in 2023, a review article was published with a meta-analysis investigating the genetic pathways underlying individuals' regular physical activity. It reviewed 219 twin and family studies from articles published up to 2021 and data was from 1952-2018. Meta-analysis had in total 70,200 members from family studies and 83,694 twin pairs. Because estimates of physical activity were accomplished with various methods and study designs as well as observing different physical activity traits, results were diverse. Most remarkably, parent-offspring studies gave systematically lower results. Nevertheless, when all the data was compiled, results for heritability of physical activity were 0.26 for females and 0.35 and males in childhood. In adolescence heritability was 0.42 for females and males and 0.45 through adulthood for both genders as well. The shared environment influence during childhood and adolescence was 0.23-0.62 and in adulthood mostly not significant. (de Geus, 2023) This meta-analysis is presented below in more detail along with evidence from a few other studies. The detailed information of studies discussed related to physical activity are found in table S8 in the supplementary.

3.4.1 Family studies

3.4.1.1 Children

In the De Geus' meta-analysis, parent-offspring correlation ranged for different types of physical activity between r= 0.05-0.19. The age of the children in the data wasn't specified. Device-based total physical activity (TPA) and device-based moderate to vigorous activity (MVPA) correlated least (n = 5,098 parent-offspring pairs). On the contrary, self-reported leisure time physical activity (LTPA) and voluntary exercise behaviour (VEB) got the highest estimate for heritability (n = 137,695 pairs). The estimates for self-reported TPA and MVPA were between the aforementioned groups (n = 29,147). Mother-offspring correlation was seen stronger in these examined traits.

Interestingly, sibling-correlation was seen greatly more prominent, which point out the importance of siblings' shared environment to physical activity, rather than the environment shared by parents. Also, the reason why correlation with parents wasn't so strong could be because of age-specific expression of genetic factors and non-additive genetic effects. This means that different genes are active at different ages. A non-additive genetic effect instead

refers to genes interacting with each other and influence each other's activity. Sibling correlations ranged from 0.19-0.33 (n = 4,342-33,605 pairs). (de Geus, 2023.)

From family studies, heritability estimates were calculated using variance-weighted meta-analysis, which adjusted for age and sex. The heritability estimates for device-based TPA and MVPA were 0.48 (95%CI; 0.30-0.66) and that for self-reported TPA and MVPA was 0,21 (95%CI; 0,14-0,28). The heritability estimate for self-reported LTPA and VEB was 0.29 (95%CI; 0.22-0.36). Only three studies out of 61 family studies detected significant contribution of shared environment to physical activity 0.04 –0.25(Choh et al., 2009; Pérusse et al., 1989; van der Zee et al., 2020). (de Geus, 2023.)

3.4.1.2 Adults

Two new studies that weren't included in the meta-analysis explored how LTPA correlates in the long run with parents. The earlier publication was a prospective study, with a duration of 30 years. At the study baseline, children (n=3,596) were 9–18 years old. When the study ended the participants were 34–49 years old. Parents were assessed in the early phases. Parents' and their children's physical activity correlated significantly until the age of 24 from 0.10 to 0.20 ($p<0.01$), except fathers and daughters at age 24, where correlation was 0.07 ($p<0.005$). To the age 49 significant correlations remained low, 0.13 or under, if significant at all. However, a notable exception was observed in father-son pairs, which exhibited an association of 0.21 at the age of 46 and significant associations were observed in many years leading up to that. These correlations were independent of important health-related covariates (e.g. living area, socioeconomic status, BMI). In this study, contrary to the meta-analysis fathers' physical activity was a stronger predictor of offspring physical activity. (Kaseva et al., 2017)

A recent article reached higher association estimates in the same data as above. Adult leisure time physical activity correlated with their parents by 0.19 in females ($p=0.002$) and 0.22 in men ($p<0.001$). In addition, significant association was found between all parent-child pairs except mother-son. (Yang, Kukko, Hirvensalo, et al., 2022) The study population was divided only to youth and adults, whereas in the previous analysis the study population was divided by age to several groups which most likely is the cause for different conclusions.

3.4.2 Twin studies

In the De Geus's meta-analysis, genetics accounted for 0.19 to 0.36 (95%CI; 0.10-0.43) of the variance in different types of physical activity (MVPA, TPA, LTPA and VEB) amongst children under 12 years old (n=42,879). In Adolescence (n=39,710) the variance was 0.42-0.47 (95%CI; 0.35-0.55) and in adulthood (n=148,830) estimates rose even a little to 0.37-0.54 (95%CI; 0.30-0.59). Similar to the family studies, the objective device-reported physical activity resulted in remarkably higher heritability estimates. In adult study population, the heritability of device-based total physical activity was 0.54 (95%CI; 0.48-0.59) compared to that of self-reported total physical activity 0.37 (95%CI; 0.30-0.44).

The heritability estimates of the meta-analysis extended to middle-age. There weren't studies found in the elderly population about heritability of physical activity. However, there was one master's thesis based on the Finnish Twin Study from material from which contained data from 63–76-year-old females (n=434). The thesis estimated the heritability of physical activity in leisure time and its stability in the elderly females. In their calculation genetics explained 0.28 of the self-reported physical activity and 0.13 came from the shared environment. (Laine, 2007.) This was in line with the meta-analysis and suggested that genetic proportion will decrease slightly in later life. Anyhow, more certified and controlled research in this area is needed.

As it is seen in other health behaviours, in De Geus' meta-analysis there was a dramatic decrease in variation due to shared environment when reaching adulthood. Estimates for physical activity were 0.51-0.62 (95%CI; 0.42-0.67) in small children and decreased to non-significance (0.02-0.03) when reaching adulthood. Presumably, shared environment effect during adolescence was in the middle ranging from 0.23-0.28 (95%CI; 0.13-0.38).(de Geus, 2023.) Even so, authors of meta-analysis found some contradicting results for the non-significant common environment component in adulthood. A Chinese (n = 19,308) study found even more contradicting results. In the study twin and sibling correlation were almost equal (r= 0.87 and r= 0.85) suggesting that large proportion of physical activity would be determined by the shared environment (B. Wang et al., 2016). De Geus's et al. suggested that the discrepancy of results for shared environment might be because of the use of binary phenotype for physical activity.

In childhood the heritability estimate for voluntary physical activity (LTPA and VEB) from twin studies was 0.36 (95%CI; 0.28-0.43) for boys and for girls a little lower 0.24 (95%CI;

0.18-0.30). The difference is seen also in adolescence, though the CIs overlap: 0.47 (95%CI; 0.39-0.55) for males and 0.42 (95%CI; 0.35-0.50) for females. In other physical exercise types, sex-difference wasn't seen. Likewise, the shared environment in voluntary physical exercise differentiated slightly in underaged children. The shared environment, on the other hand, is seen stronger in girls under 12 years 0.62 (95%CI; 0.57-0.67) and 0.28 (95%CI; 0.19-0.38) in adolescence. For boys the comparable estimates were 0.51 (95%CI; 0.42-0.60) and 0.23 (95%CI; 0.13-0.33). The results indicate that girls at a young age are more influenced by shared environment than boys and then consequently boys are more driven by genetics. (de Geus, 2023.)

De Geus's Meta-analysis came to the conclusion that the mother-child correlation in physical activity was stronger. However, other studies not included in their data came to opposite conclusion (Kaseva et al., 2017; Yang, Kukko, Hirvensalo, et al., 2022; Yang, Kukko, Kaseva, et al., 2022) so there isn't clear evidence for either side, though meta-analysis has more statistical significance.

3.4.3 Summary of physical activity

Physical activity in childhood can be encouraged greatly by parents but in adulthood one's environment should be changed in a way that would promote an active lifestyle if an individual wants to be physically active. Good genes are also a major blessing. In adulthood genetics explain half of physical activity behaviours and in childhood over one quarter. There is not really a real sex difference between heritability of physical activity, but some evidence exists for girls being more influenced by social environment in childhood.

3.5 Summary of heritability of all health behaviours

The range for the four studied health behaviours are presented in table 1.

Table 1. Range of heritability estimates included in the literature review by each health behaviour

Health behaviour	Main results – Heritability estimate	Additional effect of shared environment
Dietary patterns	0,0-0,64	0,0-0,39
Smoking behaviour	0,0-0,88	0,0-0,57
Alcohol consumption	0,02-0,79	0,0-0,45
Physical activity	0,0-0,54	0,0-0,28

4 Discussion

4.1 Heritable health behaviours

The aim of this literature review was to compile the evidence for heritability of health behaviours. The heritability of dietary patterns, smoking behaviour, alcohol consumption and physical activity are explored to demonstrate the magnitude of their heredity. All these behavioural traits have been researched using twin studies, while alcohol consumption and physical activity have been additionally investigated through multiple prospective parent-child association studies. The review suggests that health behaviours are substantially heritable which can be observed even until the middle age. In contrast, the effect of the environment, through for example parental teaching during childhood years, has ultimately almost a non-observable effect in adulthood. Nonetheless, it must be kept in mind that even if adults would not demonstrate learned health behaviours from their childhood, a healthy childhood is an asset for adulthood health.

In a large meta-analysis, the heritability of all traits was found to be 0.49 (Polderman et al., 2015). The heritability estimates for the four examined health behaviours are comparable in magnitude to those of other traits. All four health behaviours showed similar pattern that the major effect of childhood shared environment weakened towards adult, independent life. However, the shared environment prevailed as a significant factor explaining smoking behaviours and alcohol drinking in adult years. These traits also had higher estimates of genetic heritability, thus meaning that out of these four important health behaviours they are most likely inherited.

The genetic component of dietary habits could be explained through eating behaviours, with a significant portion of them attributed to genetics (Masip et al., 2020). However, this implies that even when adults live in the same country and environment, for some it is easier to eat nourishing food and follow dietary recommendations. According to the behavioural susceptibility theory, genetic factors influence appetite regulation, which is expressed in different eating behaviours that contribute to weight gain (C. Llewellyn & Wardle, 2015). This theory is supported by findings on significant common genetic influence on eating behaviours (Herle et al., 2020). Additionally, unhealthy eating behaviours have been associated with higher BMI (Livingstone & McNaughton, 2016) and they have been shown directly to mediate obesity in adult population (Masip et al., 2020). This suggests that eating behaviours

are one answer as well as a solution to the global obesity problem. Interestingly, a common genetic background explaining obesity isn't yet present during childhood, when shared environment is the determinant (Kan et al., 2020). In childhood there is an opportunity for at least a temporary change in behaviours because genetics have less of an effect at this stage. Children have always been and continue to be the promise of the new generation that can live better than the previous one.

Out of the four health behaviours, smoking got the highest estimates of attribution of both genetics and shared environment. In other words, the likelihood of inheriting smoking is the highest for these behaviours. Data from 10,368 twins from Netherlands register showed that current smoking was 0.76 heritable (Treur et al., 2017) derived only from genetics. Other studies found similar genetic contributions but also a higher shared environment proportion explaining smoking behaviour (Dongmeng et al., 2022; Mezquita et al., 2018; Vink & Boomsma, 2011). The primary driver of genetic heritability arises from variations within the genes encoding nicotinic receptor subunits (Loukola et al., 2014). From the social side of heritability, a study focusing on mothers-child relationship, the mothers' maladaptive attributes (depressed mood, ego control, impulsivity and bad self-esteem) and offspring's education were associated with each other and further with smoking. On the positive side, strong and healthy attachment protected the children until their thirties. (Brook et al., 2013.) All in all, smoking and other intoxicants appear to have a high risk of being inherited both through genetics and parents' behaviour.

Moreover, in the framework of the heritability of smoking and substance use, an interaction between the genes and the environment (G x E) has been suggested. In other words, environmental risk could enhance the effect of genetic risk. The interaction has been found for some attributes but overall the evidence for the phenomenon is not particularly strong (Pasman et al., 2019). This interaction could still be one explanation for the high heritability rates of smoking.

Alcohol consumption, like smoking, showed high heritability estimates and a minor effect of shared environment (Hansell et al., 2008; Saunders et al., 2017; Verhulst et al., 2015). All parental drinking habits seem to correlate with child's drinking, regardless of whether parental drinking is in control or disordered. The effect of shared environment specifically affected alcohol use initiation similarly to smoking.

Alcohol use initiation is naturally affected by the shared environment. When peers start drinking it is easier to yield with the peer pressure. Shared environment can also be affected by the positive home environment. This was demonstrated in a study where early onset and never drinking groups were compared, both groups had genetically as high tendency to use alcohol but never drinking group was not affected by shared environment (Zheng et al., 2019). Presumably, this points to factors, which protects children from the shared environment. One could be parent-child relationship quality, which was proposed by Ryan et al. (2010) when they reviewed parenting factors associated with reduced alcohol use, a factor also mentioned with smoking behaviours earlier. Good relationships would enable children not to be so vulnerable to the pressure of peers. Nine longitudinal studies supported that the level of warmth, bonding and affection between parents and the child were protective for the age of initiation and with the levels of drinking in later life (Ryan et al., 2010). However, this raises the question of why good parent-child relationship is not included in the shared environment. Potentially it might not show up in twin studies because individuals experience relationships uniquely. Furthermore, other protective factors identified in several studies include parental modelling, which is a fundamental aspect of heritability, and parental monitoring (Ryan et al., 2010).

The heritability of physical activity was comparable to dietary patterns. The genetical impact on physical activity in adulthood appeared independent from the childhood environment. The heritability of physical activity in adult's was 0.45 in a large meta-analysis (de Geus, 2023). There was barely any evidence of a shared environment component shaping how people exercised in the long run. Two biological pathways were mainly found to explain the strong genetic hereditary of physical activity. The first is the genetic ability to exercise, primarily mediated by cardiorespiratory and musculoskeletal system but also the resilience to tolerate sport injuries. In physical performance capability people fall into the normal distribution and typically when individuals notice that they are good at one trait, they enjoy it more and consequently start to practice it regularly. Moreover, for example being overweight, which is also party mediated by genes, can predispose to injuries and by that reduce exercise rates. (de Geus, 2023.) The second biological pathway is the motivational mechanisms in the brain. The positive affective response, i.e. having a positive experience, during and after physical activity has been demonstrated to be heritable and genetically linked with exercise behaviour (Schutte et al., 2017, 2019). In addition, positive affect and general enjoyment predicts participation in

sport activities (Aaltonen et al., 2014; Rhodes & Kates, 2015). Depending on genetics, fortunate or not, some find exercise more pleasurable than others.

Given these considerations, the profound impact of healthy childhood on long-term well-being cannot be overlooked. Although studies may not demonstrate a direct link between upbringing and behaviour in adulthood, providing a healthy foundation in childhood allows individuals to reach their full potential, both mentally and physically. Health during childhood serves as a significant predictor of health in adulthood (Hartiala et al., 2012, Ajala et al., 2017). Furthermore, it is considerably easier to maintain a healthy lifestyle when such habits have been instilled early on, rather than attempting to adopt them later in life. Research shows that most behavioural choices are made unconsciously (Bargh & Chartrand, n.d.). For example, people tend to eat foods they are accustomed without much conscious thought (Wansink & Sobal, 2007). A meta-analysis examining nutrition and physical activity behaviours found that habitual behaviour was one of the strongest predictors, explaining approximately 20% of the variation of these behaviours (Gardner et al., 2011, p. gardner). Building habits and maintaining routine around healthy lifestyle makes practising healthy lifestyle more familiar and accessible, even in the absence of optimal genetic predispositions.

4.2 Strengths

The literature review is based on a rich body of research using a variety of study designs. It included the four most important behavioural factors contributing to chronic diseases and also enabled their comparison. The literature review included samples ranging all age groups, and the expression of heritable traits could be distinguished between children and adults. Because of large number of studies, a diversity of features of health behaviour has been described and most results point in similar direction. In addition, the sample size in a considerable proportion of studies is large and the review covers populations from relatively diverse origins. Articles included in the review were relatively recently published, which demonstrates the current knowledge and scientific interest about heritability of health behaviours.

4.3 Limitations

Although, the study population demonstrated some degree of diversity, most of the studies were conducted in Western societies. Thus, they don’t necessarily represent the heritability in all global regions. Dietary and smoking behaviours also had limited evidence from

prospective parent-child association studies. Therefore, these behaviours lack evidence how they are demonstrated in daily life across generations. While the diversity of study methods is a strength in the review the comparison of heritability estimates across study types is difficult. Therefore, definite overall estimation is challenging. Although the literature review was extensive, it was not systematically done, which meant that some potentially important studies might have been missed. In addition, other health behaviours, such as sleeping behaviour our substance use beyond alcohol, were not reviewed. Hence, we had to rely on this evidence to be comprehensive enough to roughly estimate the overall heritability of health behaviours.

One technical concern with twin studies was that the shared environment factor (C-component) was left out in models estimating heredity, because it was low or not significant. However, De Geus & De Moor (2008) noted that non-significance doesn't actually mean that it cannot be there, but it is just hard to pick up without a very large sample size. This can result in too low shared environment estimates in single studies and this literature review also included a number of small studies. Furthermore, another issue with twin studies has been additive and non-additive genetic contribution. Additive genetic factors, also referred to as narrow sense heritability, mean that phenotype stems from the sum of genes and can be passed on to children. Non-additive variation, however, includes the effect of intra-allelic dominance and cross-allelic interaction. (de Geus, 2023.) The difference is that additive genetic variance is predictable and linear whereas non-additive genetic variance is more unpredictable, because of the interaction between different alleles. Since in non-additive influenced attributes, heredity is not that simple, it can be mixed with the effect of environment. Fortunately, some studies used broad sense heritability, where additive and non-additive factors are presented together. A large-meta-analysis of all human traits estimated that 69% of genetic variation is from additive genetic variation (Polderman et al., 2015), therefore being the cause of largest portion in heritability. This suggests, that even if studies included only additive genetic effects, they still identify most of the genetic contribution to the behaviour.

4.4 Implications

The first implication of this literature review is the increased knowledge of the origin of health behaviours. The understanding of the substantial heritability of health behaviour enables more empathetic and understanding encounter with patients and neighbours. When people are acknowledged and understood in healthcare receptions and hospitals it results in

better clinical outcomes, lower anxiety and stress levels as well as better patient satisfaction (Derksen et al., 2013). Furthermore, these results can be a source of hope at the doctor's appointment. The studies are encouraging since everything is not determined by genes and the results emphasize the opportunities of an individual for change. The environment has a considerable role in adulthood, especially for dietary behaviours and physical activity, but also to alcohol consumption and smoking behaviours. The unique environment such as school, workplace, teachers, friends and culture could be modified to support better health behaviours. Feeling competent to achieve a task e.g. change health behaviour, is one of the three basic psychological needs and according to self-determination theory. This enhances intrinsic motivation (R. M. Ryan & Deci, 2000). Fulfilment of these needs also increases general well-being (León & Núñez, 2013). A meta-analysis showed that interventions grounded in the theory are more effective (Prestwich et al., 2014), thus these results can be used to motivate individuals.

In the context of children, the focus could be turned to the impact of parenting. Parents need to understand their great role for the child's health during the early years. They are the most determinant factor if their children live in a healthy way. Children benefit greatly from healthy behaviours during their childhood; Lower risk for non-communicable diseases and any form of malnutrition, improved cognitive development, normal growth and other advantages are attained just by good health behaviours (Janssen & LeBlanc, 2010; Saeidi et al., 2015). Furthermore, some traits from childhood are associated with characteristics in adulthood as well, as for example obese children are more likely to be obese in their adult years (Simmonds et al., 2016). On the other hand, parents can get relief of the results indicating that, ultimately, their influence does not determine the child's future health behaviour. Scientifically, parents could not have done health behaviour teaching significantly better. Their adult's child's life is really influenced now by genetics and child's own chosen environment.

4.5 Future directions

In the field of behavioural genetics there is dearth of research in long prospective parent-child association studies which extend into later adulthood and more such research is needed for the different health behaviours. One rare good example of such a study examined the association between parents' their children's physical activity from the age of 9 to the age of 49 (Kaseva et al., 2017). Intergenerational studies on heritability show weak proof for causality because they

are almost without exception cross-sectional, have very short follow up or are inadequately based in theory (Pearl, 2009). A few studies were prospective association studies but the follow-up mainly ended when children were in their early 20s. For smoking there wasn't a single prospective estimation for the correlation between parent's and children's smoking behaviours, and for dietary behaviours there was only one. Whilst there were multitude of twin studies, there is a lack of evidence in the elderly population of the effect of heredity to their lifestyle. A more specific gap is in the heritability of alcohol consumption as a health behaviour. Studies were mainly focused on alcohol use disorders. Ultimately, there appears to be a complete deficiency in the topic of heritability of health behaviours which as a whole hasn't been studied in any way either by twin- or family studies.

4.6 Conclusion

Health behaviours exhibit heritability, manifesting partial influence in childhood but more notably in adults. Smoking behaviour and alcohol consumption were more strongly inherited than dietary patterns and physical activity. In later stages of life, substance use behaviours demonstrated a minor shared environment influence, whilst it had no effect on dietary patterns and physical activity. These results underline the importance of the effect of individual's unique environment in adult life and provide solid ground for compassionate attitude towards patient's with even poor health behaviours.

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The Supplementary

Supplementary table S1. Details of the family and cross-sectional studies reporting heritability of eating behaviours

Author + year	Country	Age (ethnicity)	Sample size	Behaviour outcome	Main result - Correlation	95% CI, p-value, S.E.	Stydy type	Additional effect of shared environment	95% CI, p-value	Notes
Mirmiran et al., 2022	Iran	8-77 years	6,949	Daughter's energy intake living with parents to mom	0,19	p = 0,04	Three-generational follow up study			
Mirmiran et al., 2022	Iran	8-77 years	6,949	Daughter's energy intake living independantly to mom	0,12	p = 0,69	Three-generational follow up study			
Mirmiran et al., 2022	Iran	8-77 years	6,949	Son's energy intake living with parents to mom	0,11	p < 0,01	Three-generational follow up study			
Mirmiran et al., 2022	Iran	8-77 years	6,949	Son's energy intake living independantly to mom	0,01	p < 0,01	Three-generational follow up study			
Hosseini-Esfahani et al., 2022	Iran	8-77 years	6,949	Daugter's healthy eating living with parents (HEI score)	0,25	p = 0,07	Three-generational follow up study			
Hosseini-Esfahani et al., 2022	Iran	8-77 years	6,949	Daugter's healthy eating living independently (HEI score)	0,07	p < 0,01	Three-generational follow up study			
Hosseini-Esfahani et al., 2022	Iran	8-77 years	6,949	Son's healthy eating living with parents (HEI score)	0,34	p <0,01	Three-generational follow up study			
Hosseini-Esfahani et al., 2022	Iran	8-77 years	6,949	Son's healthy eating living independently (HEI score)	0,03	p <0,01	Three-generational follow up study			
Lahmann et al., 2017	USA	offspring university aged	218	Food neophobia	0,6	95% CI, 0,46-0,71	Cross-sectional			
Lahmann et al., 2017	USA	offspring university aged	218	Picky eating	0,75	95%CI; 0,51-0,75	Cross-sectional			
Lahman et al., 2017	Austrlia	offspring age 18-23	4,034	Food intake (by food groups)	0,12-0,29	p < 0,001	Cross-sectional			
Van den Berg et al., 2013	The Netherlands	19-92 years	1,690	Healthy dietary intake	0,32	p<0,01	Family structure			
Van den Berg et al., 2013	The Netherlands	19-92 years	1,690	Unhealthy dieatry intake	0,27	p<0,01	Family structure			
Wang et al., 2011	Mostly USA	1-30 years	Not shown	Energy intake	0,17	95%CI; 0,18-0,24	Cross-sectional			
Wang et al., 2011	Mostly USA	1-30 years	Not shown	Fat intake	0,19	95%CI; 0,13-0,28	Cross-sectional			

Supplementary table S2. Details of the twin studies reporting heritability of eating behaviours in children

Author + year	Country	Child age (ethnicity)	Sample size	Behaviour outcome	Main result - Additive genetic effect	95%CI, p-value	Stydy type	Additional effect of shared environment	95%CI, p-value2	Notes
Warkentin et al., 2022	Portugal	10-year old	172	Food responsiveness (urge to eat)	0,69	95%CI: 0,41-0,93	Twin			
Warkentin et al., 2022	Portugal	10-year old	172	Slowness in eating	0,69	95%CI: 0,19-0,94	Twin			
Warkentin et al., 2022	Portugal	10-year old	172	Food fussiness	0,7	95%CI: 0,08-0,95	Twin			
Kan et al., 2022	UK	5-year olds	2,402	External eating	0,50	95%CI: 0,43-0,58	Twin	0,42	95%CI: 0,34-0,49	
Kan et al., 2022	UK	5-year olds	2,402	Food responsiveness (urge to eat)	0,60	95%CI:0,51-0,69	Twin	0,29	95%CI: 0,20-0,38	
Herle et al., 2017	UK	5-year olds	2,054	Emotional over eating	0,07	95%CI: 0,06-0,09	Twin	0,9	95%CI: 0,89-0,92	
Herle et al., 2017	UK	5-year olds	2,054	Emotional under eating	0,07	95%CI: 0,06-0,09	Twin	0,91	95%CI: 0,90-0,92	
Fildes et al., 2016	UK and The Netherlands	3-year old (British)	2,686	Food fussiness	0,78	95%CI: 0,73-0,82	Twin	0,05	95%CI: 0,02-0,09	
Faith et al., 2013	USA	4-7 year old (Diverse american	132	Food neophobia (Child Food Neophobia Scale, CFNS)	0,72	p = 0.28	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Does not eat enough (2,5-years old)	0,89	95%CI: 0,75-0,96	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Does not eat enough (9-years old)	0,56	95%CI: 0,28-0,78	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats too much (2,5-years old)	0,87	95%CI: 0,70-0,95	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats too much (9-years old)	0,55*	95%CI: 0,25-0,77	Twin			*variation explained by nonadditive genetic variance
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats too fast (2,5-years old)	0,71	95%CI: 0,49-0,87	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats too fast (9-years old)	0,44*	95%CI: 0,18-0,66	Twin			*variation explained by nonadditive genetic variance
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Fussy about food (2,5-years old)	no value		Twin	0,70	95%CI; 0,51-0,84	
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Fussy about food (9-years old)	0,85	95%CI; 0,59-0,96	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats between meals (2,5-years old)	0,24	95%CI; 0,02-0,51	Twin	0,71	95%CI;0,46-0,89	
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats between meals (9-years old)	0,81	95%CI; 0,66-0,91	Twin			
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats different meal (2,5-years old)	no value		Twin	1,0	95%CI; 0,99-1.00	
Dubois et al., 2013	Canada	2,5 and 9-years old	692	Eats different meal (9-year old)	no value		Twin	0,70	95%CI; 0,54-0,81	
Llewellyn et al., 2008	UK	10-12 years	254	Eating rate	0,62	95%CI: 0,45-0,74	Twin			

Supplementary table S3. Details of the twin studies reporting heritability of eating behaviours in adults

Author + year	Country	Age (ethnicity)	Sample size	Behaviour outcome	Main result - Additive genetic effetct or correlation	95%CI, p-value	Stydy type	Additional effect of shared environment	95%CI, p-value2	Notes
Leeming et al., 2022	UK	18-89	1,858	Length of eating window	0,33	95%CI; 0,24-0,41	Twin			
Herle et al., 2020	Spain	43-69	698	Cognitive restraint eating	0,09	95%CI; 0,0-0,34	Twin	0,26	95%CI; 0,04-0,40	
Herle et al., 2020	Spain	43-69	698	Uncontrolled eating	0,22	95%CI; 0,0-0,53	Twin	0,22	95%CI; 0,0-0,45	
Herle et al., 2020	Spain	43-69	698	Emotional eating	0,26	95%CI; 0,0-0,47	Twin	0,1	95%CI; 0,0-0,35	
Masip et al, 2020	Finland	31-37	3,977	Emotional and external eating	0,36	95%CI; 0,29-0,42	Twin			
Masip et al, 2020	Finland	31-37	3,977	Snacking	0,39	95%CI; 0,32-0,45	Twin			
Masip et al, 2020	Finland	31-37	3,977	Infrequent and unhealthy eating	0,48	95%CI; 0,42-0,54	Twin			
Masip et al, 2020	Finland	31-37	3,977	Avoidant eating	0,36	95%CI; 0,29-0,42	Twin			
Lopez-minguez et al.,2019	Spain	46-69	106	Breakfast timing	0,56	95%CI; 0,28-0,74	Twin			
Lopez-minguez et al.,2019	Spain	46-69	106	Lunch timing	0,38	95%CI; 0,07-0,62	Twin			
Lopez-minguez et al.,2019	Spain	46-69	106	Dinner timing	no value		Twin	0,39	95%CI; 0,14-0,59	
Lopez-minguez et al.,2019	Spain	46-69	106	Midpoint of intake	0,64	95%CI; 0,40-0,79	Twin			
Song, et al., 2013	Korea	31-57	1,361	Restrained eating (female)	0,41	p<0,05	Prospective twin + family member			
Song, et al., 2013	Korea	31-57	1,361	Restrained eating (male)	0,21	p<0,05	Prospective twin + family member			
Song, et al., 2013	Korea	31-57	1,361	External eating (female)	0,28	p<0,05	Prospective twin + family member			
Song, et al., 2013	Korea	31-57	1,361	External eating (male)	0,23	p<0,05	Prospective twin + family member			
Song, et al., 2013	Korea	31-57	1,361	Emotional eating (female)	0,3	p<0,05	Prospective twin + family member			
Song, et al., 2013	Korea	31-57	1,361	Emotional eating male	0,21	p<0,05	Prospective twin + family member			
Sung et al., 2010	Korea	20-65	2,144	Restrained eating	0,31	p<0,05	Twin			
Sung et al., 2010	Korea	20-65	2,144	Emotional eating	0,25	p<0,05	Twin			
Sung et al., 2010	Korea	20-65	2,144	External eating	0,25	p<0,05	Twin			
Keskitalo et al., 2008	UK and Finland	17-82	1,326	Cognitive restraint eating (female)	0,45-0,76	range within 95%CI	Twin			
Keskitalo et al., 2008	UK and Finland	17-82	1,326	Cognitive restraint eating (male)	0,0-0,56	range within 95%CI	Twin			
Keskitalo et al., 2008	UK and Finland	17-82	1,326	Emotional eating (female)	0,07-0,54	range within 95%CI	Twin			
Keskitalo et al., 2008	UK and Finland	17-82	1,326	Emotional eating (male)	0,0-0,47	range within 95%CI	Twin			
Keskitalo et al., 2008	UK and Finland	17-82	1,326	Uncontrolled eating (female)	0,53-0,80	range within 95%CI	Twin			
Keskitalo et al., 2008	UK and Finland	17-82	1,326	Uncontrolled eating (male)	0,17-0,70	range within 95%CI	Twin			
Tholin et al., 2005	Sweden	23-29	1,564	Restrained eating (male)	0,59	95%CI; 0,52-0,66	Twin			
Tholin et al., 2005	Sweden	23-29	1,564	Emotional eating (male)	0,6	95%CI; 0,52-0,67	Twin			
Tholin et al., 2005	Sweden	23-29	1,564	Uncontrolled eating (male)	0,45	95%CI; 0,36-0,53	Twin			

Supplementary table S4. Details of the twin studies reporting heritability of food intake in adults

Author + year	Country	Age (ethnicity)	Sample size	Behaviour outcome	Main result - Additive genetic effect	95%CI, p-value, ± = standard error)	Stydy type	Additional effect of shared environment	95%CI, p-value2	Notes
Matison et al., 2023	Australia	65-90	374	Vegetable intake (female)	0,55	95%CI; 0,43-0,65	Twin			
Matison et al., 2023	Australia	65-90	374	Vegetable intake (male)	0,23	95%CI; 0,02-0,45	Twin			
Matison et al., 2023	Australia	65-90	374	Fruit intake (female)	0,28	95%CI; 0,13-0,41	Twin			
Matison et al., 2023	Australia	65-90	374	Fruit intake (male)	0,04	95%CI; 0,00-0,22	Twin			
Leeming et al., 2022	UK	18-89	1,858	Breakfast consumption	0,11	95%CI; 0,02-0,21	Twin			
Leeming et al., 2022	UK	18-89	1,858	Eating window	0,33	95%CI; 0,24-0,41	Twin			
Treur et al., 2017	The Netherlands	Mean age 33,5 (SD = 15,3)	10,368	Coffee consumption	0,53	95%CI; 0,48-0,58	Twin			
Treur et al., 2017	The Netherlands	Mean age 33,5 (SD = 15,3)	8,060	Caffeine consumption	0,49	95%CI; 0,47-0,58	Twin			
Treur et al., 2016	The Netherlands	Mean age 33,5 (SD = 15,3)	8,586	Sugar consumption	0,48	95%CI; 0,38-0,57	Twin			
Scheibehenne et al., 2014	USA	Average 59 (SD = 14 years)	5,543	Dieatry variety (female)	0,27	Figure	Twin	0,14	Figure	
Scheibehenne et al., 2014	USA	Average 59 (SD = 14 years)	5,543	Dieatry variety (male)	0,30	Figure	Twin	0,15	Figure	
Hasselbalch et al., 2010	Denmark and Finland	20-27 (Finnish)	2,009	Bread intake (female)	0,4	95%CI; 0,33-0,49	Twin			
Hasselbalch et al., 2010	Denmark and Finland	20-27 (Finnish)	2,009	Bread intake (male)	0,37	95%CI; 0,28-0,45	Twin			
Hasselbalch et al., 2010	Denmark and Finland	18-67 year olds (Dansih)	575	Bread intake (female)	0,23	95%CI; 0,07-0,38	Twin			
Hasselbalch et al., 2010	Denmark and Finland	18-67 year olds (Dansih)	575	Bread intake (male)	0,26	95%CI; 0,10-0,40	Twin			
Teucher et al., 2007	UK	18-80	3,262	Fruit and vegetable eating pattern (female)	0,43	95%CI; 0,28-0,58	Twin	0,11	95%CI; 0,0-0,23	
Teucher et al., 2007	UK	18-80	3,262	Four other eating patterns (female)	0,41-0,48	95%CI; 0,34-0,54	Twin			

Supplementary table S5. Details of the twin and family studies reporting heritability of smoking behaviour

Reference and year	Country	Age of study population	Sample size	Behavior outcome	Main result - Additive genetic effect or risk ratio	95%CI, p-value	Study type	Additional effect of shared environment	95%CI, p-value2	Notes
Dongmeng er al., 2022	China	24-51	23,250	Cigarette smoking - yes/no (only male)	0,23	95%CI; 0,16-0,29	Twin	0,54	95%CI; 0,48-0,60	
Mezquita et al, 2018	Spain	47-73	2,285	Lifetime smoking (never, quitted, smoking) (female)	0,49	95%CI; 0,17-0,87	Twin	0,12	95%CI; 0,06-0,21	
Mezquita et al, 2018	Spain	47-73	2,285	Lifetime smoking (never, quitted, smoking) (male)	0,87	95%CI; 0,73-0,95	Twin			
Mezquita et al., 2018	Spain	47-74	2,285	Number of years smoked (female)	0,88	95%CI; 0,77-0,94	Twin			
Mezquita et al., 2018	Spain	47-75	2,285	Number of years smoked (male)	0,79	95%CI; 0,64-0,89	Twin			
Maes et al., 2018	USA and Australia	18≤	50,318	Smoking initiation (female)	0,55	not shown	Extenden twin study (Kinship design)	0,11	not shown	Also information from parents, siblings, spouses and children
Maes et al., 2018	USA and Australia	18≤	50,318	Smoking initiation (male)	0,53	not shown	Extenden twin study (Kinship design)	0,18	not shown	Also information from parents, siblings, spouses and children
Treur, 2017	The Netherlands	Age mean 32,5	10,386	Current smoking	0,76	95%CI; 0,70-0,79	Twin			
Bao et al., 2016	China	25≤ (only male)	12,916	Smoking behaviour (male)	0,26	95%CI; 0,19-0,34	Twin			
Bao et al., 2016	China	25≤ (only male)	12,916	Onset age of smoking (male)	0,05	95%CI; 0,0-0,14	Twin			
Bao et al., 2016	China	25≤ (only male)	12,916	Smoking cessation (male)	0,31	95%CI; 0,0-0,74	Twin			
Do et al., 2015	USA	12-32 years	2,804	Smoking initiation (14-15 old)	0,54	95%CI; 0,22-0,84	Twin	0,29	95%CI; 0,05-0,56	
Do et al., 2015	USA	12-32 years	2,804	Smoking initiation (16-17 old)	0,85	95%CI; 0,77-0,90	Twin			
Do et al., 2015	USA	12-32 years	2,804	Smoking initiation (22-32 old)	0,79	95%CI; 0,66-0,88	Twin			
Do et al., 2015	USA	12-32 years	2,804	Current quantity smoked (14-15 old)	No value		Twin	0,85	95%CI; 0,68-0,94	
Do et al., 2015	USA	12-32 years	2,804	Current quantity smoked (16-17 old)	No value		Twin	0,89	95%CI: 0,78-0,95	
Do et al., 2015	USA	12-32 years	2,804	Current quantity smoked (22-32 old)	0,55	95%CI; 0,28-0,76	Twin			
Seglem et al,. 2015	Norway	12-18 years	1,394 families	Lifetime smoking	0,37	95%CI; 0,35-0,40	Twin + information from parents	0,56	95%CI; 0,53-0,64	
Öncel et al., 2014	Turkey	15-45	618	Smoking initation	no value		Twin	0,89	not spesified	
Öncel et al., 2014	Turkey	15-45	618	Nicotine dependence (FTND)	0,80	not spesified	Twin			FTND = Fagerstrom test of Nicotine Dependence), In males heriability
Zhang et al., 2012	China	27-49 (only male)	900	Cigarette smoking (male)	0,683	95%CI; 0,57-0,78	Twin			
Vink and Boomsma, 2011	The Netherlands	18-25	5,008	Smoking behaviour (female)	0,21	95%CI; 0,0-0,49	Twin	0,57	95%CI; 0,32-0,77	
Vink and Boomsma, 2011	The Netherlands	18-25	5,008	Smoking behaviour (male)	0,66	95%CI; 0,29-0,86	Twin	0,14	95%CI; 0,0-0,46	Sex difference was not significant
Lessov-Schlaggar et al., 2006	China	24 and over (only male)	1,010	Current smoking (male)	0,751	95%CI; 0,57-0,88	Twin			
Lessov-Schlaggar et al., 2006	China	24 and over (only male)	1,010	Heavy smoking (male)	0,66	95%CI; 0,0-0,88	Twin	0,09	95%CI; 0,0-0,71	
Broms et al., 2006	Finland	24-88	19,760	Amount smoked (female)	no value		Twin	0,47	95CI; 0,36-0,58	
Broms et al., 2006	Finland	24-88	19,760	Amount smoked (male)	0,54	95%CI; 0,45-0,62	Twin			
Broms et al., 2006	Finland	24-88	19,760	Age at initiation (female)	0,35	95%CI; 0,28-0,43	Twin	0,51	95%CI; 0,44-0,57	
Broms et al., 2006	Finland	24-88	19,760	Age at initiation (male)	0,59	95%CI; 0,49-0,69	Twin	0,19	95%CI; 0,10-0,27	
Broms et al., 2006	Finland	24-88	19,760	Smoking cessation (female)	0,50	95%CI; 0,39-0,60	Twin			
Broms et al., 2006	Finland	24-88	19,760	Smoking cessation (male)	0,58	95%CI; 0,50-0,65	Twin			
Hamilton et al., 2006	USA	18-44	64,718	Smoking initation (female)	0,32	95%CI; 0,24-0,39	Twin	0,48	95%CI; 0,41-0,54	
Hamilton et al., 2006	USA	18-44	64,718	Smoking initation (male)	0,71	95%CI; 0,67-0,75	Twin	0,12	95CI%; 0,09-0,16	
Hamilton et al., 2006	USA	18-44	64,718	Smoking persistence	0,55	95%CI; 0,44-0,66	Twin	0,09	95%CI; 0,0-0,17	
Vink et al., 2005	The Netherlands	Mean age 30,5	3,144	Smoking initiation	0,44	not shown	Twin	0,51	not shown	
Vink et al., 2005	The Netherlands	Mean age 30,5	3,144	Nicotine dependence	0,75	not shown	Twin			
Madden et al., 2004	Australia, Sweden and Finland	18-45	43,766	Regular smoking (female)	0,46	95%CI; 0,40-0,53	Twin	0,35	95%CI; 0,19-0,53	Shared environment at age 18-25, 26-35 and 36-45 was 0,45; 0,35 and 0,26
Madden et al., 2004	Australia, Sweden and Finland	18-45	43,766	Regular smoking (Asutralian men)	0,57	95%CI; 0,49-0,64	Twin	0,15	95%CI; 0,0-0,36	Shared environment at age 18-25, 26-35 and 36-45 was 0,26; 0,09 and 0,11
Madden et al., 2004	Australia, Sweden and Finland	18-45	43,766	Regular smoking (Scandinavian men)	0,57	95%CI; 0,49-0,65	Twin	0,27	95%CI; 0,12-0,40	Shared environment at age 18-25, 26-35 and 36-45 was 0,33; 0,29 and 0,19
Li et al., 2003	USA, Finland, Australia and	18≤	57,742	Smoking initiation (female)	0,55	95%CI; 0,47-0,64	Twin meta-analysis	0,24	95%CI; 0,12-0,35	
Li et al., 2003	USA, Finland, Australia and	18≤	57,742	Smoking initiation (male)	0,37	95%CI; 0,29-0,45	Twin meta-analysis	0,49	95%CI; 0,42-0,57	
Li et al., 2003	USA, Finland, Australia and	18≤	57,742	Tobacco use (female)	0,46	95%CI; 0,22-0,69	Twin meta-analysis	0,28	95%CI; 0,12-0,45	
Li et al., 2003	USA, Finland, Australia and	18≤	57,742	Tobacco use (male)	0,59	95%CI; 0,54-0,63	Twin meta-analysis	0,08	95%CI; 0,0-0,16	
Vink et al., 2003	The Netherlands	12-40 years	6,501-8,155	Risk of smoking if one parent smokes (12-15 year old)	1,38-2,46	95%CI; 0,85-3,90	Cross-sectional study			
Vink et al., 2003	The Netherlands	12-40 years	6,501-8,155	Risk of smoking if both parents smoke (12-15 year old)	2,16-3,06	95%CI; 1,19-5,64	Cross-sectional study			
Vink et al., 2003	The Netherlands	12-40 years	6,501-8,155	Risk of smoking if one parent smokes (21-40 year old)	1,18-1,41	95%CI; 0,19-1,74	Cross-sectional study			
Vink et al., 2003	The Netherlands	12-40 years	6,501-8,155	Risk of smoking if both parents smoke (21-40 year old)	1,51-1,62	95%CI; 1,05-2,16	Cross-sectional study			

Supplementary table S6. Details of the family studies reporting heritability of drinking behaviours

Reference, year	Country	Age of study population	Sample size	Behaviour outcome	Main result - Correlation or odds ratio	95%CI, p-value or S.E.	Study type	Additional effect of shared environment	95%CI, p-value	More information
Sipilä et al., 2023	Finland	21-28 and 31-37	2,969 and 2,269	Father's problem drinking to child's problem drinking	0.12-0.18	p<0.001	Prospective parent-child resemblance			Problem drinking: Malmö-modified Michigan Alcoholism Screening test
Sipilä et al., 2023	Finland	21-28 and 31-37	2,969 and 2,269	Mother's problem drinking to child's problem drinking	0.09-0.14	p<0.01	Prospective parent-child resemblance			
Sipilä et al., 2023	Finland	21-28 and 31-37	2,969 and 2,269	Parent's problem drinking to child's problem drinking at age 21-28	0.09-0.18	p<0.01	Prospective parent-child resemblance			
Sipilä et al., 2023	Finland	21-28 and 31-37	2,969 and 2,269	Parent's problem drinking to child's problem drinking at age 31-39	0.11-0.19	p<0.001	Prospective parent-child resemblance			
Sipilä et al., 2023	Finland	21-28 and 31-38	2,969 and 2,270	Parent's heavy drinking to child's heavy drinking	0.12-0.19	p<0.001	Prospective parent-child resemblance			
Parra et al., 2020	Finand	28	6,963	Parents alcohol consumption (child age 16) on child's alcohol use disorder at 28 (r)	n-0,09-0,05	not significant	Longitudinal prospective study			n = negative
Kosty et al. 2020	USA	14-30	739	Parental alcohol use disorder to child's alchol use disorder (r)	0,02-0,25	not significant	Longitudinal epidemiological study of			
Saunders et al., 2017	USA	14-29	7,270	Alcohol consumption resemblance (r) at age 14	0,12	In papers figures	Longitudinal paren-offspring resemblance			
Saunders et al., 2017	USA	14-29	7,270	Alcohol consumption resemblance (r) at age 17 an 24	0,25	In papers figures	Longitudinal paren-offspring resemblance			
Saunders et al., 2017	USA	14-29	7,270	Alcohol consumption resemblance (r) at age 29	0,19	In paper's figures	Longitudinal paren-offspring resemblance			
Saunders et al., 2017	USA	14-29	7,270	Alcohol dependence symptomps resemblance (r) at age 17	0,18	In paper's figures	Longitudinal paren-offspring resemblance			
Saunders et al., 2017	USA	14-29	7,270	Alcohol dependence symptomps resemblance (r) at age 20 and 29	0,11	In paper's figures	Longitudinal paren-offspring resemblance			
Alati et al., 2014	Australia	13-17	715	Childrens drinking if fahter drinks (OR)	1,4	95%CI; 1,0-1,9	Prospective parent-child resemblance			
Alati et al., 2014	Australia	13-18	715	Childrens drinking if mother drinks (OR)	2,77	95%CI; 1,9-4,1	Prospective parent-child resemblance			
Pedersen & von Soest, 2013	Norway	28	2558	Parental alcohol consumption to child's alcohol consumption at 28 (r)	0,09-0,12	p<0.001	Longitudinal prospective study			
Pedersen & von Soest, 2013	Norway	28	2558	Parental binge drinking to child's drinking at 28 (r)	0,10-0,17	p<0.001	Longitudinal prospective study			
Englund et al., 2008	USA	26-old (only male)	178	Bieng a heavy drinker at 26-old (to mothers drinking frequency at 16) - OR	1,75	(95%CI; 1.11-2.70)	Longitudinal prospective study			
Pitkänen et al., 2008	Finland	42	347	Parental drinking when children lived at home to child's problem drinking (r)	0.31	p<0.001	Follow-up study from 8 to 42			
Merline et al., 2008	USA	22-35	21,137	Heavy parental drinking to child's alcohol abuse and depedence symptoms at age 35 (r)	0.12-0.16	p<0.001	Follow-up study			
Merline et al., 2008	USA	22-35	21,137	Parental drinking in childhood to child's alcohol use last 30 days age 22-35 (r)	0.05-0.10	p<0.001	Follow-up study			
Pagan et al., 2006	USA	22-35	21,137	Parental drinking in childhood to child's heavy drinking at age 22-35 (r)	0.05-0.11	p<0.001	Follow-up study			

Supplementary table S7. Details of the twin studies and family structure estimate reporting heritability of drinking behaviours

Reference, year	Country	Age of study population	Sample size	Behaviour outcome	Main result - Additional genetic effect or correlation	95% CI, p-value or S.E.	Study type	Additional effect of shared environment	95% CI, p-value	More information
Clarke et al., 2021	UK	18-99	19,377	Alcohol consumption (units per week)	0,18	S.E.= 0,02-0,06	SNP-based + kindship model	0,45	S.E. = 0,03	
Clarke et al., 2021	UK	18-99	19,377	Risk for problem drinking (CAGE score)	0,19	S.E.= 0,03	SNP-based + kinship model			
Zheng et al., 2019	Canada	13-17	877	Alcohol use (early onset and low drinking group)	0,27-0,38	95%CI; 0,01-0,70	Twin	0,22	95%CI; 0,0-0,53	
Zheng et al., 2019	Canada	13-17	877	Alcohol use (never drinking group)	0,38	95%CI; 0,24-0,51	Twin			
Mbarek et al., 2015	The Netherlands	43,3 age mean	15,388*	Alcohol dependance (AUDIT)	0,60	95%CI; 0,55-0,69	Twin			AUDIT = Alcohol Use Disorders Indetification test. *sample size spesified to the outcome
Verhulst et al., 2015	Scandinavia, USA, Australia and UK	mainly over 18	103,530	Alcohol use disorders (AUD)	0,49	CI95%; 0.43-0.53	Meta-analysis of twin and adoption studies	0,10	95%CI; 0.03–0.16	
Zhang et al., 2012	China	27-49 (only male)	900	Drinking alcohol (male)	0,79	95%CI;0,69-0,86	Twin			
van Beek et al., 2012	The Netherlands	15-32	8,398	Abuse and dependece (CAGE score) age 15-20	0,18	not shown	Twin	0,45	not shown	
van Beek et al., 2012	The Netherlands	15-32	8,398	Abuse and dependece (CAGE score) age 21-29	0,50	not shown	Twin	0	not shown	
van Beek et al., 2012	The Netherlands	15-32	8,398	Abuse and dependece (CAGE score) age 30-32	0,28	not shown	Twin	0,12	not shown	
Sartor et al., 2009	Australia	24-36	5,382	First alcohol use	0,36	95%CI; 0,21-52	Twin	0,15	95%CI; 0,02-0,27	
Sartor et al., 2009	Australia	24-36	5,382	Alcohol dependence	0,53	95%CI; 0,45-0,61	Twin			
Hansell et al., 2008	Australia	19-90	12,045	Alcohol consumption age 23-39	0,31	95%CI; 0,23-36	Twin			
Hansell et al., 2008	Australia	19-90	12,045	Alcohol consumption age 28-90	0,47	95%CI; 0,43-0,51	Twin			
Hansell et al., 2008	Australia	19-90	12,045	Alcohol dependence age 23-39	0,46	95%CI;0,37-49	Twin			Dependence was revised DSM-III-R and DSM-IV criteria
Hansell et al., 2008	Australia	19-90	12,045	Alcohol dependence age 28-90	0,46	95%CI; 0,42-0,51	Twin			
Lessov-Schlaggar et al., 2006	China	24≤	1,010	Current drinking (only male)	0,6	95%CI; 0,0-0,88	Twin	0,15	95%CI; 0,0-0,72	
Lessov-Schlaggar et al., 2006	China	24≤	1,010	Amount of alcohol consumed (only male)	0,424	95%CI; 0,0-0,92	Twin	0,39	95%CI; 0,0-0,83	
Hopfer et al., 2003	Finland, The Netherlands,	Adolescent	13,948	Alchohol use at 15-16	0,34	not shown	Review of twin and adoption studies	0,58	not shown	
Hopfer et al., 2003	Finland, The Netherlands,	Adolescent	13,948	Alchohol use at 17 and older	0,43	not shown	Review of twin and adoption studies	0,47	not shown	
Rose et al., 2001	Finland	14	3,283	Drinking initiation (female)	0,18	95%CI; 0,10-0,29	Twin	0,76	95%CI; 0,68-0,83	
Rose et al., 2001	Finland	14	3,283	Drinking initiation (male)	no value		Twin	0,76	95%CI; 0,68-0,83	

Supplementary table S8. Details of the family and twin studies reporting heritability of physichal activity

Reference, year	Country	Age of study population	Sample size	Behaviour outcome	Main result - Additional genetic effect or correation	95%CI, p-value or S.E.	Study type	Additional effect of shared environment	95%CI, p-value	More information
De Geus, 2023	The Netherlands and other	All aged	5,098	Device-based total physichal activity and moderate to vigorous activity	0,48	95%CI; 0,30-0,66	Family study estimate (meta-analysis)			
De Geus, 2023	The Netherlands and other	All aged	137,695*	Self-reported total physichal activity and moderate to vigorous activity	0,21	95%CI; 0,14-0,28	Family study estimate (meta-analysis)			*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	29,147*	Self-reported leisure time physichal activite and voluntary exercise behaviour	0,29	95%CI; 0,22-0,36	Family study estimate (meta-analysis)			*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	42,879*	Different type of physichal excercise (chidren under 12)	0,19-0,36	95%CI; 0,10-0,43	Twin (meta-analysis)	0,51-0,62	95%CI; 0,42-0,67	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	39,710*	Different type of physichal excercise (adolescent)	0,42-0,47	95%CI; 0,35-0,55	Twin (meta-analysis)	0,23-0,28	95%CI; 0,13-0,38	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	148,830*	Different type of physichal excercise (adults)	0,37-0,54	95%CI; 0,30-0,59	Twin (meta-analysis)	0,02-0,03	not significant	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	148,830*	Device-based total physichal activity (adults)	0,54	95%CI; 0,48-0,59	Twin (meta-analysis)	0,02	not significant	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	148,830*	Self-reported total physichal activity (adults)	0,37	95%CI; 0,30-0,44	Twin (meta-analysis)	0,03	not significant	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	20,911*	Voluntary physichal activity (girls under 12)	0,24	95%CI; 0,18-0,30	Twin (meta-analysis)	0,62	95%CI; 0,57-0,67	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	20,602*	Voluntary physichal activity (boys under 12)	0,36	95%CI; 0,28-0,43	Twin (meta-analysis)	0,51	95%CI; 0,42-0,60	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	19,051*	Voluntary physichal activity (female adolescent)	0,42	95%CI; 0,35-0,50	Twin (meta-analysis)	0,28	95%CI; 0,19-0,38	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	20,659*	Voluntary physichal activity (male adolescent)	0,47	95%CI; 0,39-0,55	Twin (meta-analysis)	0,23	95%CI; 0,13-0,33	*sample size spesified to the outcome
De Geus, 2023	The Netherlands and other	All aged	237,588	Physichal activity (girls under 12)	0,26	Not shown	Family- and twin study estimate (meta-analysis)			
De Geus, 2023	The Netherlands and other	All aged	237,588	Physichal activity (boys under 12)	0,35	Not shown	Family- and twin study estimate (meta-analysis)			
De Geus, 2023	The Netherlands and other	All aged	237,588	Physchal activity (adolescent)	0,42	Not shown	Family- and twin study estimate (meta-analysis)			
De Geus, 2023	The Netherlands and other	All aged	237,588	Physichal activity (adults)	0,45	Not shown	Family- and twin study estimate (meta-analysis)			
Yang et al., 2022	Finland	9-49 years	3,596	Parental leisure time physical activity to child's (adult female)	0,19	p=0,002	Longitudinal parent-child accociation, cohort			
Yang et al., 2022	Finland	9-49 years	3,596	Parental leisure time physical activity to child's (adult male)	0,22	p<0,001	Longitudinal parent-child accociation, cohort			
Kaseva et al., 2017	Finland	9-49 years	3,596	Parental physichal activity to child's physichal avctivity at age 9-24 (r)	0,10-0,20	p<0,01	Prospective study (30-years), parent-child			Except females at the age 24 corraletd 0,07 p= 0,005
Kaseva et al., 2017	Finland	9-49 years	3,596	Parental physichal activity to child's physichal avctivity at age 27-49 (r)	0,05-0,13	p<0,01	Prospective study (30-years), parent-child			Only significant correlations mentioned
Kaseva et al., 2017	Finland	9-49 years	3,596	Father's physichal activity to son's at 46 years old	0,21	p<0,001	Prospective study (30-years), parent-child			
Kaseva et al., 2017	Finland	9-49 years	3,596	Father's physichal activity to son's at 24-49 years old	0,08-0,21	p<0,01	Prospective study (30-years), parent-child			Only significant correlations mentioned
Laine, 2007	Finland	63-76	434 (only women)	Physichal activity	0,28	Not shown	Twin	0,13	Not shown	A study done for master's thesis