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Personality traits and body weight: from accurate descriptions to tests of causation

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This dissertation is based on the following original publications and unpublished manuscript, which will be referred to in the text by their respective Roman numerals:

I. **Arumäe, K.**, Mõttus, R., & Vainik, U. (2023). Body mass predicts personality development across 18 years in middle to older adulthood. *Journal of Personality*, 1–15. https://doi.org/10.1111/jopy.12816

II. **Arumäe, K.**, Briley, D., Colodro-Conde, L., Mortensen, E. L., Jang, K., Ando, J., Kandler, C., Sørensen, T. I. A., Dagher, A., Mõttus, R., & Vainik, U. (2021). Two genetic analyses to elucidate causality between body mass index and personality. *International Journal of Obesity*, 1–8. https://doi.org/10.1038/s41366-021-00885-4

III. **Arumäe, K.**, Vainik, U., & Mõttus, R. A bottom-up approach to increase the predictability of BMI from personality traitsits. *Manuscript under review.*

IV. **Arumäe, K.**, Mõttus, R., & Vainik, U. (2022). Beyond BMI: Personality traits' associations with adiposity and metabolic rate. *Physiology & Behavior*, *246*, 113703. https://doi.org/10.1016/j.physbeh.2022.113703

The author of the current dissertation contributed to the publications as follows:

- in Studies I, III, and IV, conceptualized the studies and conducted the analyses;
- in Studies I–IV, wrote the manuscript as the first author.

INTRODUCTION

Several decades of research has tried to identify psychological correlates of body weight (e.g., M. A. Friedman & Brownell, 1995), much of it having been motivated by the objective to identify modifiable causes of excess weight. Not only is excess weight a risk factor for and likely cause of various physical diseases and mental disorders (Avila et al., 2015; Deng et al., 2016; Goodarzi, 2018; Larsson & Burgess, 2021), but it is also associated with increased health care expenditure, disability, lost productivity, and mortality, imposing a heavy economic burden on the affected individuals, their families, and societies at large (Tremmel et al., 2017). The scale of the problem is illustrated by the prevalence of overweight and obesity: about two billion people worldwide have been estimated to have excess weight, including over 600 million cases of obesity (Abarca-Gómez et al., 2017; The GBD 2015 Obesity Collaborators, 2017). The importance of finding ways to prevent and target excess weight is evident.

Researchers often believe that personality traits—people's enduring tendencies to feel, think, and behave in similar ways across different situations—are among the psychological characteristics affecting body weight. While personality traits, henceforth also simply referred to as traits, could exert their influences in many ways, they are most commonly thought to do so through trait-related weight-relevant behaviors—i.e., behaviors that affect either energy intake or expenditure. As traits are by definition highly stable, the behaviors characteristic of them tend to recur and thus their effects on weight should accumulate. For instance, a person with lower self-discipline may not be able to refrain from overeating and thus accumulate excess weight over time. By this line of reasoning, investigations of trait–body weight associations could indeed reveal some potential targets for weight-loss interventions.

Extensive research has shown correlations between body weight and various personality traits (reviewed in Gerlach et al., 2015 and Vainik et al., 2019). However, as I explain below, the descriptions of the associations may be incomplete and even biased due to issues relating to the measurement and conceptualization of the variables of interest. Furthermore, although traits are commonly assumed to influence body weight, causality between them is yet to be convincingly demonstrated. The four studies described in this dissertation addressed these issues. Before giving an overview of the studies, their main results and conclusions, I will outline how personality traits and body weight are defined and measured, summarize what is known of their development, and review previous research on their associations.

Personality traits

Conceptualization and measurement

People differ from each other in countless ways. According to the lexical hypothesis, which has been profoundly influential in the field of personality psychology, the most important ways in which people differ from one another become encoded as single terms in some or all human languages (Goldberg, 1990). Following this hypothesis, languages offer myriad ways to describe people's personality differences using trait-descriptive terms—terms that distinguish the behavior patterns of one human from those of another. In an early attempt to identify the personality-descriptive terms in the English language, Allport and Odbert (1936) found almost 18,000 such terms in a contemporary English dictionary, about 4,500 of which they classified as describing stable traits or those that seemed to "symbolize most clearly the "real" traits of personality" (p. 26). These terms often take the shape of adjectives like "sanguine", "tactful", or "crafty". Granted, languages do not correspond one-to-one in the terms they contain, but there is clearly a wealth of such terms—and also of traits they refer to.

The traits that such personality-descriptive terms represent can be thought of as forming a multivariate space or universe. Goldberg (1992) compared this multivariate trait space to the night sky: just as some stars are closer to each other, forming clusters, some traits are semantically similar and form clusters of traits. The clusters themselves can be thought of as broader traits reflecting the unions of their components—the narrower, finer-grained traits they are made up of. Yet, not all narrow traits fall neatly into a certain cluster. Some traits may be remote to any cluster or lie at an equal distance to more than one cluster. For instance, a trait reflecting talkativeness may be central to a broader trait called extraversion, but one reflecting exploration may fit equally well under extraversion and another broad trait called openness to experience. Which narrower traits belong to which broader ones is not always straightforward (Schwaba et al., 2020), but given how many traits there are, it is often advisable or even necessary to cluster the narrow traits.

Which traits exactly cluster together is a question that can be answered empirically. Although several competing solutions may be reasonable, near consensus concerning how to cluster traits had been reached by the 1990s (Goldberg, 1990). During the 20th century, several independent researchers set out to identify the fundamental dimensions of personality by factor-analyzing lists of trait-descriptive terms (see Digman, 1990 for a review). The results of the many independent studies converged and the conclusion appeared clear: the personality trait space can be broadly summarized with five clusters of traits (*dimensions* or *domains*) that are now known by the names extraversion, agreeableness, conscientiousness, openness to experience, and neuroticism (or its reverse, emotional stability). Together, these five domains are referred to as the Big Five (Goldberg, 1990) or, alternatively, the Five-Factor Model domains (McCrae & John, 1992). This five-factor structure replicates in many, although not all, languages in different parts

of the world (Heine & Buchtel, 2009; McCrae & Costa Jr., 1997; McCrae & Terracciano, 2005), attesting to its usefulness across many different societies and cultures. Although alternative factor structures have been proposed (notably the Big Six or HEXACO; Ashton & Lee, 2007) with their own advantages and disadvantages in comparison to the Big Five (Ashton & Lee, 2020), the latter is today the best-known and most used.

By far the most common approach to measuring human personality traits—the Big Five and others—is with self-report inventories. The self-report inventories may ask people to rate how accurately some adjectives (e.g., "sociable", "disorganized", "inquisitive") describe them, or to what extent they agree with more specific statements (e.g., "When visiting a new country, I like to learn some of the local language in advance" or "I prefer to spend Friday nights at home reading a book rather than going out to a loud party"). A host of self-report inventories that measure the Big Five have been developed and validated, ranging from long ones like the 300-item IPIP-NEO (Goldberg, 1999) and the 240-item NEO personality inventory (NEO-PI; Costa & McCrae, 1992a) to short and very short ones like the 44-item Big Five Inventory (BFI; John, 1991) and the 10-item BFI-10 (Rammstedt & John, 2007), to name a few. Because there is no known set of items that captures traits, either the Big Five or others, with perfect accuracy, each inventory uses different items and whether any inventory is objectively superior to all others is indeterminate. Therefore, traits as measured with specific inventories may be incomplete representations of traits as psychological constructs. To differentiate the two, I use uppercase initials when referring to traits as measured with inventories (e.g., Conscientiousness, Assertiveness) and lowercase initials when referring to the psychological constructs the inventories are meant to measure (e.g., conscientiousness, assertiveness).

The choice of personality measure depends on the specific research objectives. While longer inventories are more comprehensive and have superior validity (Hofmans et al., 2008; Kastner et al., 2012), shorter ones reduce assessment time and participant burden and are thus typically employed in large-scale studies. Another notable advantage of many longer inventories is that they enable measuring not only broad traits like the Big Five but also narrower traits within them. That is, personality traits are hierarchically organized: as each domain consists of narrower, partly-overlapping traits, they can be divided into clusters of subtraits that overlap (covary) even more strongly. For instance, each domain can be divided into two aspects (DeYoung et al., 2007) or a number of yet more specific facets. Although there is currently a lack of consensus on the number and content of these facets (Schwaba et al., 2020; Ziegler & Bäckström, 2016), two popular inventories—the IPIP-NEO and the NEO-PI—divide each Big Five domain into six facets. Finally, below facets in the hierarchy are nuances that constitute the narrowest level of personality measurement. Nuances are theoretically unidimensional, are the building blocks of all broader traits, and can in many cases be equated with the items of existing inventories (Condon et al., 2020; McCrae, 2015; Mõttus et al., 2019). So, for instance, the extraversion domain is made up of the aspects Enthusiasm and Assertiveness whereas examples of its NEO-PI

facets include Warmth and Activity, and its nuances may be represented by items that reflect liking flashy styles and feeling comfortable in crowds.

Each of the various trait levels is best suited for different objectives. While domains provide a common language for integrating research across many domains, lower levels of measurement enable finer-grained descriptions and often yield higher predictive accuracy than broader traits (Seeboth & Mõttus, 2018; Stewart et al., 2022). Although research most commonly relies on broader traits, the subunits they are made up of can be used in various ways to fulfill specific research objectives. For instance, when the objective is prediction, one could select items from a suitably large item pool that link with the outcome and apply factor analysis to identify traits that best predict it (e.g., Weiss and colleagues (2013) used this approach to pinpoint personality predictors of mortality). As another example, trait–outcome associations can be maximized by weighting traits (e.g., facets or items) by their correlation with the outcome and summing the products (e.g. Benning et al., 2005). By enhancing statistical power, this technique enables the discovery of otherwise weak associations. A similar approach is used in genetics: many genetic variants related to a phenotype are weighted by their associations with the measured phenotype to yield a predictive score called a *polygenic score* (PGS; Dudbridge, 2013). The aggregate of many personality facets or items could analogously be called a *polypersonality score* or *phenotypic personality score* (PPS).

In summary, a researcher is not limited to predefined trait structures but can use the responses to an inventory in whatever way achieves their particular objectives best.

Trait development and origins or trait differences

Where trait differences originate and how they develop throughout life provides the necessary context to make sense of their links with outcomes like body weight. Variation in behavior and reactivity patterns can already be observed in infants, even neonates, and these patterns have some stability across the first years of life (Planalp & Goldsmith, 2020; Rothbart & Ahadi, 1994). This supports the notion that personality traits have a genetic background—that people are not born as blank slates to be shaped exclusively by experiences. But neither do these early-life dispositions deterministically predict adult personalities. Despite having considerable stability, traits develop throughout life, even in old age (Bleidorn et al., 2022; Roberts & DelVecchio, 2000; Roberts & Mroczek, 2008; Specht et al., 2011), and even though traits follow typical trajectories—people tend to become more agreeable, conscientious, emotionally stable, and socially dominant with age (Roberts & Mroczek, 2008; Soto & John, 2012)—development is also subject to variation and anyone could deviate from the average trajectories considerably and in unpredictable ways (Beck & Jackson, 2020; Roberts & Mroczek, 2008; Schwaba & Bleidorn, 2018). So, for instance, one rather disorganized youth could become a quite organized adult while another stays at the same level of disorganization relative to his cohort.

The relative contributions of genetic and environmental factors to traits can be studied with behavior-genetic approaches. These studies make use of data from family members with different degrees of genetic relatedness to parse the variance in a phenotype (i.e., a measured trait) to genetic and environmental components. Most typically, three variance components are distinguished: additive genetic, common environmental, and unique environmental, respectively denoted by A, C, and E. While A constitutes a trait's heritable component—the sum of genetic effects on it—C includes factors shared by family members like socioeconomic status, diet, or the variety of books at home, and E refers to a person's unique experiences like differential parental treatment, accidents or friends not shared with family members. Shared environments make the members of a family more similar, unique environments contribute to their differences.

Traits' variance decomposition is most often done with data from monozygotic (MZ) and dizygotic (DZ) twin dyads and is enabled by the fact that MZ twin dyads are twice as similar in their segregating DNA ($r_A = 1$) as DZ dyads ($r_A = .50$), but MZ and DZ dyads are equally similar to each other in the two environmental variance components ($r_C = 1$, $r_E = 0$; Rijsdijk, 2002). Based on twin studies, the Big Five domains' heritabilities are about 40–50% on average (Bouchard, 1994; Vukasović & Bratko, 2015), although adjustment for measurement error (which falls under the E component) increases heritability to about two-thirds of the total phenotypic variability (Bouchard, 1994). The majority of the remaining variance is attributable to unique environmental factors and, to the surprise of many, common environmental factors have been shown to contribute little to nothing to traits' variability (Bouchard, 1994). To illustrate the latter, MZ twins reared apart are as similar personality-wise as MZ twins reared together (Bouchard et al., 1990), suggesting that the shared home environment does not make siblings any more similar.

But behavior-genetic studies say nothing about the specific factors—genes, biological processes, environmental features, or life events—that lead to personality differences. Concerning the former, several genome-wide association studies (GWASs) have attempted to identify the genetic variants (e.g., single nucleotide polymorphisms, SNPs) linked to personality traits (see Sanchez-Roige et al., 2018 for an overview), but found few replicable associations, each very weak. Even together, the identified variants account for minute proportions of phenotypic traits' variance. So, given that traits are heritable to a substantial degree, and that no SNPs with large effects apparently exist, the genetic architecture of personality traits is most likely highly polygenic—that is, they are influenced by numerous genetic variants with small effects (Chabris et al., 2013; Sanchez-Roige et al., 2018). This possibility is further evidenced by the fact that the number of genetic associations identified has increased along with sample sizes (Sanchez-Roige et al., 2018): as samples have grown, so has statistical power, allowing the detection of increasingly small effects. Perhaps this is not a surprising outcome. Given that genes code for proteins, not specific behavioral (or physical) characteristics, it may be unrealistic to expect specific genes to directly govern specific personality traits. Instead, genetic variants exert their influences on traits indirectly via complex

biological pathways (Abdellaoui & Verweij, 2021; Bouchard & Loehlin, 2001), making it commensurately harder to pinpoint the genetic mechanisms that underlie the traits.

An extensive literature has investigated the effects of more proximal biological factors and life experiences on traits. Although some studies have indeed linked traits to biological variables like neurotransmitter function, brain volume and connectivity (reviewed in DeYoung, 2010 and Yarkoni, 2015), there is currently no convincing evidence to support the links' generalizability. The same is true for life experiences: studies linking traits to events in the occupational (starting a job, retiring), family (marriage, childbirth), and other domains have overall yielded no robust results (Bleidorn et al., 2018). Several reasons could account for the failure to identify replicable influences. Life events' effects could be profound but transient and not reach stable traits (Schimmack et al., 2008). Further, although numerous nonshared environmental factors are believed to affect traits, their effects may be hard to detect due to being very small and/or idiosyncratic (Plomin & Daniels, 1987). Similarly, biological factors such as brain systems might not have one-to-one correspondences with traits (Matthews, 2018) but could instead interact with each other to influence traits (Yarkoni, 2015). This would align with the numerous genetic effects on traits, but make detecting any effects exponentially more difficult.

Body weight (or, more precisely, body fat)

Measuring body fat

Predicting consequential outcomes is often considered one of the most important objectives of personality research. Considering the number and severity of its health effects (Larsson & Burgess, 2021), body weight is an undeniably consequential outcome. Actually, to be precise, it is not body weight but rather (relative) body fat or adiposity that is behind the numerous health concerns. Body weight (or body mass) may give some indication of a person's excess weight but is not a credible indicator of body fat or its associated health risks when comparing people. That is, when a person with a sedentary lifestyle steps on a scale and realizes they have put on two kilograms, they may be reasonably confident that these two kilograms are attributable to increased fat mass, but using body weight (or mass) as a measure of body fat on a sample level would be misleading as it depends on factors like height and muscularity in addition to body fat. Because height and muscularity have different implications for health than body fat (R. R. Wolfe, 2006), body weight (or mass) would be a severely misleading health index. Thus, although this dissertation also uses the term "body weight" to refer to body fat, it is the latter that researchers generally intend to study.

There are several methods to measure body fat. Objective measurements using computed tomography, magnetic resonance imaging, and dual-energy X-ray absorptiometry are considered accurate in estimating fat mass as well as fat-free

mass which entails all other aspects of body composition including skeletal muscle and organs (Denton & Karpe, 2016; Seabolt et al., 2015). However, these methods are often unavailable to researchers as they require expensive equipment. More accessible means to estimate body fat *in vivo* include body composition analysis with bioelectrical impedance scales and measuring skin-fold thickness at various body sites with calipers (Seabolt et al., 2015). Despite being more accessible, even these measurements are rarely incorporated into large studies, potentially because the involved labor and time costs can be avoided with more convenient methods.

The most convenient approaches to obtain a measure of adiposity involve approximating it through anthropometric measurements like height, weight, waist and/or hip circumference. Body mass index (BMI), calculated by dividing weight in kilograms by height in meters squared, is the most widely used index of body fat. It is also the measure used to classify people into weight groups: for instance, someone with a BMI exceeding 30 kg/m^2 is considered as having obesity. Despite being practical and convenient, BMI and other adiposity indices are less accurate than more objective measurements and have varying levels of accuracy among different groups. For instance, BMI can overestimate adiposity in taller people and underestimate it in shorter people (Denton & Karpe, 2016). It also tends to overestimate fat mass in people with high muscle mass (Rothman, 2008) and may even correlate with lean mass more strongly than with fat mass, at least among men (Romero-Corral et al., 2008). The accuracy of BMI may additionally differ between age and ethnic groups (Prentice & Jebb, 2001). Moreover, using self-reports of height and weight instead of objective measurements by trained staff can also introduce bias as people may underreport their weight or overreport their height (Burke & Carman, 2017; Hill & Roberts, 1998; Jayawardene et al., 2014), although misreported measurements may be more of a problem in models with weight status as a categorical variable than for continuous BMI (Ng, 2019). And yet, while interpretations of studies using BMI should be mindful of these limitations, it still seems to represent adiposity with reasonable accuracy: BMI closely tracks population-level adiposity (Speakman et al., 2018) and several related health risks factors (blood pressure, cholesterol, triglycerides, glycemic and inflammatory traits; Bell et al., 2018).

Other common indices of adiposity include waist circumference and waist-to-hip ratio. Yet, a seemingly endless variety of alternative indices can be developed based on different combinations of height, weight, and other measurements taken at various body sites (Woolcott & Bergman, 2020). Although these alternatives have broadly similar limitations to BMI, they may be slightly more accurate overall (Woolcott & Bergman, 2020) or capture abdominal obesity and metabolic risk more closely (Janssen et al., 2004).

Weight trajectories and contributing factors

For most people, BMI rises throughout childhood and plateaus in adolescence or early adulthood, but for those who become obese, BMI tends to stabilize several years later—in the second half of their twenties (Buscot et al., 2018; Mattsson et al., 2019). In adulthood, BMI is generally rather stable (Herman et al., 2009). Very simply put, people gain weight when their energy balance is positive, that is, when they consume more energy than they expend. However, this simple statement does not do justice to the complexity of factors behind the various sorts of behaviors and physiological processes that contribute to energy intake and expenditure. Although body weight has a strong genetic component (Elks et al., 2012), environmental factors must also have an important role because population genetic changes cannot account for the threefold rise that was documented in global obesity rates in just a few decades—from 1975 to 2016 (Abarca-Gómez et al., 2017). Indeed, the steep increase seems to have been driven by changes in the global food system, specifically the increased accessibility of palatable and highly processed food leading to energy overconsumption (Swinburn et al., 2011). Yet, despite the obesogenic environment affecting everybody, the decisions that influence energy balance, even if automatic or subconscious (Swinburn et al., 2011), are ultimately up to the individual. Interindividual variability in body weight remains.

On average, twin studies have estimated BMI to be 75% heritable (Elks et al., 2012). The high heritability means that the risk of developing overweight or obesity is present already at birth, with weight at birth and in childhood being strong predictors of adulthood weight (Simmonds et al., 2016; Yu et al., 2011). Like personality traits, body weight is highly polygenic, but substantially more is known about its genetic contributors. Large-scale GWASs have identified over 1,100 genetic variants associated with BMI and other obesity traits (Buniello et al., 2019) that can also predict weight growth trajectories (Khera et al., 2019). Each of the identified variants has a small effect: the allele with the largest effect, found in the *FTO* gene, is associated with an extra 0.39 kg/m^2 (Speliotes et al., 2010). Despite body weight being polygenic, in extremely rare cases obesity is monogenic and without any environmental influence, being caused by a single mutation in genes related to the satiety hormone leptin (Dubern & Clement, 2012). Yet, the common polygenic and rare monogenic forms of obesity appear to share similar biological mechanisms as the genes associated with each tend to act in the central nervous system (Locke et al., 2015) and both have been linked to pathways associated with hedonic aspects of food intake (Loos & Yeo, 2022).

Environmental contributions to body weight are also multifaceted. Weight gain has been linked to factors as diverse as diet, sedentary lifestyle, sleep deprivation, socioeconomic status (SES), some medical conditions, certain medications, and adverse childhood experiences (Hemmingsson et al., 2014; Newton et al., 2017; S. M. Wright & Aronne, 2012). Another layer of complexity is added to the development of body weight by the fact that genetic and environmental forces can interact to determine who struggles with excessive weight and who stays effortlessly lean. For instance, adverse environments can amplify genetic risk: low SES

can magnify and high SES dampen the association between a polygenic score for BMI (PGS_{BMI}) and measured BMI (Liu & Guo, 2015). Similar interactions have been found with other environmental or lifestyle variables such as smoking, diet, physical activity, and obesogenic home environment (Goodarzi, 2018). Thus, it may be possible to alleviate genetic risk for obesity via lifestyle choices.

Links between personality traits and body weight

Cross-sectional correlations

Due to the great potential significance of linking body weight to traits, it is not surprising that numerous studies have explored their correlations. These studies have often used the NEO-PI, but also other inventories measuring the Big Five. Across the literature, each Big Five domain has been linked to BMI in some studies but not others (Armon et al., 2013; Bagnjuk et al., 2019; Brummett et al., 2006; Faith et al., 2001; Kim, 2016; Lahti et al., 2013; Mõttus et al., 2013; Shim et al., 2014; Sutin, Costa, et al., 2013; Sutin et al., 2011; Terracciano et al., 2009). Despite the individual studies' mixed results, reviews and meta-analyses converge on the conclusion that BMI has an inverse association with conscientiousness and appears overall unrelated to extraversion, openness, and agreeableness (Gerlach et al., 2015; Jokela et al., 2013; Vainik et al., 2019). Even the reviews, however, do not agree on whether BMI links with neuroticism or not. The health effects of this trait have been the subject of some controversy. In specific, although high neuroticism is otherwise detrimental to health, it has been hypothesized to become healthy in combination with high conscientiousness as worrying increases attentiveness to health and is thus preventive of adverse outcomes if appropriate action is taken (H. S. Friedman, 2000). Although it is unknown whether such an interaction could also apply to body weight, the possibility illustrates the complexity of trait–outcome associations and also highlights the necessity to look beyond domains: whereas worry may sometimes be health-protective, other features of neuroticism might not.

Indeed, studies on the finer-grained facet level have been revealing. A meta-analysis summarizing their results found links between BMI and 15 of the 30 NEO-PI facets (Vainik et al., 2019), among the strongest being links with Impulsiveness, Angry Hostility, Self-consciousness, Warmth, and Assertiveness (positive correlations), and Order, Self-discipline, and Activity (negative correlations). For one, these associations demonstrate that body weight links to a larger variety of traits than domain-level associations alone can show. But they also show that facets within a single domain can differ substantially in their links with body weight.

Within conscientiousness—the domain most consistently linked to BMI—links tend to be found with facets tapping into order, deliberation, and self-discipline, but not with facets reflecting competence or personal values (Sawhney et al., 2020; Sutin et al., 2018; Vainik et al., 2019). These differential associations are consistent with the idea that certain subtraits could be the "active ingredients" in

domains' associations with health (Chapman, 2013). But an outcome can also have meaningful relations with narrower traits without being related to the broader trait that subsumes them (e.g., Cooper et al., 1998). Regarding body weight, this is most clearly exemplified by extraversion. Namely, despite not being correlated with the domain, BMI has opposite-direction correlations with NEO-PI Extraversion's facets, linking positively with Warmth, Assertiveness, and Positive Emotions but negatively with Activity (Vainik et al., 2019). But a facet's subtraits can vary in their links with body weight. BMI's strongest facet-level correlation, the one with Impulsiveness (Sutin et al., 2011; Terracciano et al., 2009; Vainik et al., 2019), is driven purely by the two eating-related items it entails (Terracciano et al., 2009; Vainik et al., 2015), which the facet-level correlation does not convey. Failing to consider narrow traits could thus lead to misinterpretations.

Additional complexity in trait–body weight links has been revealed by studies showing moderating effects. Some studies have reported that the extraversion–BMI association is only observed among men (Brummett et al., 2006; Shim et al., 2014; Sutin et al., 2015). Likewise, a meta-analysis reported that the proportion of women in the sample moderated BMI's links with some of its facets (Vainik et al., 2019). Another possible moderator is cultural context: while the correlation with conscientiousness appears otherwise robust, studies in Japan, China, and South Korea have failed to find it (Shim et al., 2014; Sutin et al., 2015). Besides the possibility that the association varies between Western and Asian cultures, this result may be explained by the populations' different average body sizes (Vainik et al., 2019). In any case, it is apparent that the associations may not generalize across populations and may even vary between people within a population.

This diversity in the associations may be one reason that the reported links have been invariably weak. For instance, the meta-analytically estimated correlation between BMI and NEO-PI Conscientiousness was only –.04 and facet-level correlations did not tend to exceed $|r| = .06$ (all estimates adjusted for age, age^2, sex, and education; Vainik et al., 2019). These links are clearly weaker than effects tend to be in the psychological literature on average. One massive investigation found that across 25,000 social psychology studies, the average reported effect was $r = .21$ (Richard et al., 2003); another meta-analysis of over 700 individual-differences papers reported that $r = .11$, $r = .19$, and $r = .29$ corresponded to the 25th, 50th, and 75th percentiles of effect sizes, respectively (Gignac & Szodorai, 2016). Although associations between psychological and physical variables are bound to be weaker than the investigated associations among psychological constructs, the trait–BMI links do seem small in comparison. Still, they should not be dismissed as trivial. Modern views on effect sizes recognize that even very small effects like $r = .05$ can be consequential, particularly in cases where effects accumulate over time (Funder & Ozer, 2019). So, if traits do influence body weight, their effects may well accumulate as the trait-related, weight-relevant behaviors are repeated. Whereas the trait–BMI correlations may also have arisen spuriously—by way of common third variables such as genetics and early life experiences influencing personality traits and body weight mutually (Kern & Friedman, 2011)—causality is often assumed and important to investigate.

Causality from personality traits to body weight: empirical evidence and possible mechanisms

A number of longitudinal studies have tested traits' ability to predict body weight. While these studies have sometimes linked the Big Five domains to future BMI and future weight changes, their results have been no more consistent than those of cross-sectional studies, supporting different conclusions as to which traits in particular predict these outcomes (Armon et al., 2013; Bagnjuk et al., 2019; Brummett et al., 2006; Jokela et al., 2013; Magee & Heaven, 2011; Sutin et al., 2011; Terracciano et al., 2009). Even conscientiousness has not always predicted change in BMI (e.g. Armon et al., 2013; Bagnjuk et al., 2019; Magee & Heaven, 2011).

If personality traits do influence body weight, their influences could be exerted through various pathways. The previously noted and arguably most often discussed pathway involves health behaviors: personality traits could act on body weight via trait-related behaviors that contribute to diet (e.g., high self-discipline may help avoid unhealthy dietary choices) or physical activity (e.g., being high on the activity facet could facilitate calorie expenditure through the associated tendency to move more). But behaviors do not tend to account for trait–health outcome relationships in full (Mroczek et al., 2009; O'Súilleabháin et al., 2021). Various pathways involving physiological processes have also been suggested. One of them involves leptin, the hormone that inhibits hunger and signals the brain to stop eating (Sutin & Terracciano, 2017), as one study found more conscientious people to have higher levels of circulating leptin and, further, that the hormone mediated the conscientiousness–BMI link (Sutin, Zonderman, et al., 2013). As a possible explanation, it was proposed that people with lower conscientiousness are less sensitive to satiety which makes them more likely to overeat (Sutin & Terracciano, 2017). Another pathway could involve the stress response (Sutin & Terracciano, 2017): not only do neuroticism and related traits influence perceptions of and reactions to stressful events (Chapman et al., 2011), but they are also associated with elevated levels of the stress hormone cortisol (Nater et al., 2010), as is obesity (Björntorp & Rosmond, 2000).

Yet another physiological pathway could involve chronic inflammation, the immune system's prolonged response to injury or infection linked to the development of a host of chronic diseases (Sanada et al., 2018). While inflammatory markers have been linked to personality traits, negative emotions, anxiety and mood disorders (Ong et al., 2018; Raison et al., 2006; Renna, 2021; Turiano et al., 2013; Wagner et al., 2019), systemic low-grade inflammation is also characteristic of excess weight as pro-inflammatory markers are typically elevated and anti-inflammatory markers decreased in people with obesity (Capuron et al., 2011; Forsythe et al., 2008). Consistent with this pathway, one 10-year longitudinal mediational study reported that two pro-inflammatory markers, interleukin-6 (IL-6) and C-reactive protein (CRP), mediated the relationship between conscientiousness and BMI (A. J. Wright et al., 2022). Of course, the various pathways

are not mutually exclusive. Although personality–biomarker–body weight pathways could additionally involve behaviors, such as those that help to reduce infections or stress, behavioral and biomarker pathways appear to be largely separate from each other (A. J. Wright et al., 2022).

However, taking the prior evidence together, no clear conclusions seem appropriate as to whether traits do affect body weight. Besides the longitudinal studies' results being inconsistent, they are no more than suggestive regarding causality. The studies' designs have enabled clarifying temporality—whether traits predict weight change—but they have not eliminated confounding and any observed association could have thus emerged due to some third factors, possibly exerting their effects on one variable more rapidly than on the other. While potential confounds like age, sex, and education are typically controlled for, the risk of confounding by others remains. Even the mediational studies may have reflected third variables' effects or reverse causality (Rohrer et al., 2022).

Reverse causation: body weight influencing personality traits

Influences from body weight to traits, which I call reverse causation in this dissertation, are far less commonly discussed than in the other direction (e.g., H. S. Friedman, 2019). Although arguably less relevant from a perspective of health advancement, influences in this direction would indicate a source of personality trait differences, informing personality theories. As I argue below after reviewing the relevant research, there is a good deal of reason to believe such effects exist.

A small number of studies have tested body weight as a predictor of traits. One longitudinal study reported that people whose body weight increased by 10% over 10 years increased in impulsivity and deliberation (Sutin et al., 2013). Another found that a 10% loss of or gain in body weight across eight years was associated with deviations from average trajectories of trait development: weight gain with decreases in extraversion, openness, and conscientiousness, and weight loss with maintenance of neuroticism and steeper declines in all other Big Five domains (Stephan et al., 2019). Yet another study reported that personality traits in older adulthood—measured at an average of 63 years—could be predicted from early-life weight: neuroticism had a quadratic association with birth weight while agreeableness and conscientiousness were predicted by slower weight gain in early life (Lahti et al., 2013). In contrast, another study found no evidence of BMI predicting change in any of the Big Five domains over a mean follow-up of five years across six large-scale cohort studies (Jokela et al., 2013). A systematic review did, however, find evidence for trait change after bariatric surgery—namely extraversion increasing and facets of neuroticism (anxiety, depression, self-consciousness, and impulsiveness) decreasing after surgical weight-loss intervention (Bordignon et al., 2017).

Regardless of the limited evidence, this direction of causality seems plausible in light of some possible mechanisms. As one option, inflammation could mediate the effects of body weight on traits instead of mediating traits' effects on weight.

Although no studies to my knowledge have directly explored it, current knowledge nevertheless seems to align with this chain of causality. Not only does weight loss in people with obesity result in the normalization of inflammatory marker levels (Bianchi, 2018; Forsythe et al., 2008), which could logically be expected because adipose tissue is a major source of inflammation (Forsythe et al., 2008), it also results in improvements in depression and anxiety (B. L. Wolfe & Terry, 2006). Given its emotional well-being outcomes, weight could feasibly also affect traits.

Another plausible pathway relies on social feedback. People with excess weight are perceived differently, often less favorably, than people within the normal weight range and are routinely stigmatized and discriminated against in various domains of life from education and employment to interpersonal relationships (Puhl & King, 2013). This, in turn, can lead to extreme emotional distress, anxiety, depression, and low self-esteem (Brewis et al., 2018; Puhl & King, 2013). Given the pervasiveness of weight-based discrimination and the severity of its consequences, these effects too could extend to stable traits. In fact, given the well-established effects of discrimination on the recipient's emotions, which could very plausibly affect thoughts and behaviors in turn, it would be hard to imagine body weight *not* affecting traits. And finally, a third pathway could involve the physical effects of body weight: given that very high body weight can limit physical functioning (Woo et al., 2007), it could put constraints on the actions a person is able to execute and thus objectively change their behavior, also affecting personality test responses.

In summary, although the results of longitudinal and bariatric-surgery studies seem to agree that impulsivity may respond to both weight gain and weight loss, evidence regarding other traits has been inconsistent. Yet, again, the prior evidence prohibits drawing conclusions regarding causality. Like the longitudinal studies, the bariatric-surgery trials did not eliminate confounding as they lacked randomization and did not control for normal personality development. Stronger tests of causality are needed.

Aims of the dissertation

To summarize, despite the wealth of studies, the evidence on trait–body weight links has been inconsistent and has not enabled drawing any conclusions on causality. The studies within this dissertation aimed to provide improved descriptions of the associations as well as some evidence on causality in them.

The first two studies addressed causality. Using longitudinal data, **Study I** combined evidence from two types of models to provide stronger tests of causality by minimizing confounding and to test the effects' direction. **Study II** applied Mendelian randomization analyses (using molecular genetic data) and behavior-genetic models using twin data.

The remaining two studies aimed to refine the descriptions of personality trait–body weight associations. **Study III** focused on how to optimally conceptualize traits for accurate descriptions: a large collection of items relating to BMI were factor-analytically aggregated to identify traits most strongly linked to BMI. In **Study IV**, formulas more accurate than BMI were used to estimate fat mass and fat-free mass to disentangle traits' relations to the two components of body composition.

TRIANGULATING CAUSALITY

The gold standard for testing causality in psychology is by way of experiment: people are randomized to intervention and control groups, the purported causal variable is manipulated, and changes in the supposed outcome variable are recorded. However, due to practical and ethical considerations, the links between personality traits and body weight are not approached in this way. In the following two studies, their links' compatibility with causality in either direction was assessed using longitudinal, molecular genetic (i.e., DNA), and twin data. The longitudinal analyses of **Study I** aimed to provide a stronger test of causality by improving control over confounding. **Study II** was the first to my knowledge that applied two types of genetic analyses, Mendelian randomization and Direction of Causation, to study trait–body weight associations.

Modeling longitudinal data to reduce confounding and test causal directionality

Among the various criteria of causality, temporality is one that is widely agreed on to be essential (Fedak et al., 2015). That is, the purported causal variable should precede the assumed outcome. However, even if this is shown to be true, longitudinal analyses do not provide convincing evidence for causality unless likely confounds can be ruled out. Of the many ways longitudinal data can be analyzed (e.g. Bainter & Howard, 2016), some are more successful at that than others and thus more causally informative.

One approach to reducing confounding involves testing *within-person correlations* or *correlated changes*. When the variables of interest have been measured at multiple time points, it is possible to assess whether within-person changes in them are correlated, which would suggest causality between them. A major strength of this approach is that it inherently controls for time-invariant (i.e., stable) confounds or individual differences, including unknown or unmeasured ones: that is, individual differences are accounted for just like they are in within-person experimental designs. This approach rules out a wide range of potential confounds including genes and early-life experiences. Yet, despite this major advantage, within-person correlations do have two important limitations.

First, although time-invariant confounds are controlled for, potential time-varying confounds are not. This limitation can be partially overcome by adding potential time-varying variables like age as covariates to the model. Of course, unknown or unmeasured third variables could still jeopardize these models' causal informativeness, but as long as such factors' influence is unknown, there is no way to determine whether or to what extent the results are confounded.

The second limitation of within-person correlations is that they do not test temporality and are thus agnostic to the direction of causation. To counteract this

limitation, within-person models can be supplemented with temporal (i.e., longitudinal or directional) models. For instance, longitudinal models can be applied bidirectionally to test whether either of the variables predicts change in the other. These temporal models do not provide strong evidence on causality by themselves because they can only partially tackle confounding (by including potential confounds in the models), but they complement within-person models (Daly et al., 2015): while the correlated-changes models indicate the presence or absence of likely causal links, the longitudinal models specify the likelier direction of causation.

In **Study I**, I used three waves of data from the Wisconsin Longitudinal Study (Herd et al., 2014) spanning 18 years (N = 12,235, 53% female, M_{age} = 53, and M_{BMI} = 27 at baseline) and applied both of the approaches in a multilevel modeling framework, taking into account the nested nature of the data—the three measurements being nested within the participants. Personality traits were assessed with a 29-item version of the Big Five Inventory (John, 1991); besides the five domains, I also assessed BMI's links with the individual items. For each included personality trait, I examined correlated changes with BMI as well as bidirectional relations with it, additionally accounting for age, age^2, sex, and education. The results indicated that no trait predicted change in BMI, but BMI did predict changes in, *and* had within-person correlations with, Agreeableness, Conscientiousness, and the items C4: Lazy, E1: Talkative, and E3: Full of energy ($|b^*|$ = .03 to .08 for all statistically significant associations).

I also created a composite score (called BMI's polypersonality score or PPS_{BMI}) of all 29 personality items by weighting each item by its correlation with BMI and subsequently summing the products. I did this with elastic net, a type of penalized regression commonly used to maximize predictor–outcome associations (Yarkoni & Westfall, 2017). Changes between BMI and PPS_{BMI} were correlated and directional analyses, once again, supported influences from BMI to personality traits but not vice versa.

Altogether, there was no evidence of personality traits influencing body weight. Yet, the bidirectional models showed variability in the associations, meaning that the trait–BMI links differed between people—which makes sense given that body weight as well as personality traits have many influences which may well affect everyone to a different degree. Thus, traits may still be relevant to body weight in some people or some circumstances despite this appearing not to be the case in general. For instance, conscientiousness or self-discipline may be protective against excess weight gain in households where enticing high-caloric snacks are consistently available but have no effect in homes with more strictly regulated food environments.

In the reverse direction, the results supported body weight having negative influences on agreeableness, conscientiousness, and sense of energy, and positive influences on laziness and talkativeness. Although the mechanisms can only be speculated on, it seems plausible that body weight affects agreeableness through social feedback. Given that how people are perceived and treated partly depends on their body weight—for instance, obesity is stigmatized and people with obesity

are routinely discriminated against (Puhl & King, 2013)—the persistent negative feedback could make the recipients behave less agreeably toward others in turn. Indeed, discrimination can affect emotions that could result in such behavior: in people with overweight and obesity, perceived weight discrimination has been shown to predict experiencing interpersonal stressors, feelings of anger and frustration, and having more, but also avoiding, arguments with others (Sutin et al., 2016).

The effects on conscientiousness, laziness, and energy could be attributed to the physical limitations associated with either excess adiposity (Woo et al., 2007) or its concomitant health conditions. Such physical limitations could constrain behavior and manifest as lower energy levels but also as trouble in managing day-to-day responsibilities at the usual capacity, thus leading to reporting lower conscientiousness and higher laziness. Importantly, this also suggests that people report being lazier as a result of gaining weight rather than the opposite, contesting the stereotype that laziness is a cause of weight gain (Brewis et al., 2018; Puhl & Heuer, 2009). Alternatively, body weight could affect traits through inflammation, which could serve to deplete the cognitive, emotional, and physical resources required to respond adaptively to challenging situations (Luchetti et al., 2014), leading to altered behavioral patterns.

The homogeneity of the sample (participants were graduates of Wisconsin high schools and their siblings in their middle to older adulthood) may call the generalizability of the results into question. Yet, similar associations observed in younger people born in Helsinki suggest that effects may generalize to younger ages and different populations: namely, slower growth of body weight from childhood to adulthood was associated with agreeableness and conscientiousness (Lahti et al., 2013). Because the sample was younger, these results also suggest that the associations (at least with agreeableness and conscientiousness) pertain to body weight specifically rather than deteriorating health which is common in older adulthood.

Two genetic analyses: testing causality with molecular genetic and twin data

The first of the two causally-informative approaches employed in **Study II**, Mendelian randomization, is a popular approach to elucidate causality in epidemiology (Haycock et al., 2016). This analysis uses the genetic variant(s) associated with a putative causal variable to test that putative causal variable's influence on an outcome. If the genetic variants do correlate with the outcome, the hypothesized causal association is supported. This is for two reasons: first, because a person's genetic makeup is determined before the measured phenotypes, eliminating the possibility of reverse causality, and second, because genes are inherited from parents randomly (each person randomly inherits a genotype, i.e., 0 to 2 copies of an allele), eliminating confounding. In the hierarchy of causal evidence,

Mendelian randomization has been placed below experimental but above observational studies (Davies et al., 2018).

To conduct a Mendelian randomization study, it is necessary to know at least one genetic variant associated with the putative causal variable. For BMI, several GWASs have been published and this information was readily available (Hemani et al., 2018; genotype–BMI associations were based on a sample of $N > 460{,}000$). In contrast, few genetic variants have been linked to personality traits, which precluded us from testing all Big Five domains' effects on BMI. Nevertheless, sufficient data were available for Neuroticism and its two facets, Worry and Depressive affect, as measured with the Eysenck Personality Questionnaire-Revised (Eysenck & Eysenck, 1968), based on a GWAS by Nagel et al (2018; Ns > 348,000). Thus, it was possible to calculate PGSs for BMI as well as the three personality traits by weighting the relevant SNPs by their correlations with the target phenotype and summing them (analogously with the calculation of PPS_{BMI} in **Study I**). While PGS_{BMI} comprised 957 SNPs, the personality traits' PGSs consisted of 59 to 109 SNPs. The four PGSs were calculated for 3,541 people in the Estonian Biobank (60% female, $M_{age} = 47$, $M_{BMI} = 26$) on whom we ran the Mendelian randomization analyses.

We also summarized 238 NEO-PI-3 items (i.e., the two eating-related items found in the inventory were excluded) with which personality traits had been measured in the Estonian Biobank data in a PPS_{BMI}. Weighting items by their correlations with BMI enhanced the associations, ensuring greater statistical power to detect an association. PPS_{BMI} was used as the personality phenotype that BMI's polygenic score was used to predict.

In summary, for Mendelian randomization, we used the $PGS_{Neuroticism}$, PGS_{Worry}, and $PGS_{Depressed\ affect}$ to predict BMI, and the PGS_{BMI} to predict PPS_{BMI}. Out of the personality variables, only PGS_{Worry} predicted BMI ($b^* = -.05$, $p < .001$), suggesting a protective effect of worry against excess weight and simultaneously demonstrating the necessity of testing the associations with narrower traits than domains. In the reverse direction, the PGS_{BMI} also predicted PPS_{BMI} ($b^* = .05$, $p < .001$). The vast majority of the items included in PPS_{BMI} had non-zero weights (specifically, 201 items), indicating that body weight may have had weak effects on many low-level traits.

The second approach, Direction of Causation (Heath et al., 1993), uses structural equation modeling to parse variance in the phenotypes of interest into the ACE components—as in classical twin studies, described in the introduction—and find the model of causation that best fits the data. Direction of Causation relies on the assumption that if a variable influences another, then its ACE components should be proportionally represented in the outcome variable. So, if a variable has contributions from the environmental factors shared by twins, then the C component should also be proportionally present in the purported outcome.

With Direction of Causation, we compared four alternative models: personality traits influencing BMI, BMI influencing personality traits, reciprocal causation, and no causation. We used data from twins from five countries (Australia, Canada, Denmark, Germany, and Japan), total $N = 5{,}424$ with a mean age of

30 years, a mean BMI of 23 kg/m^2, and 65% being female. Again, for statistical power considerations, we used PPS_{BMI} as the phenotype for personality.

As the results showed, the data supported causality from BMI to PPS_{BMI}. However, the reciprocal-influences model fit the data about as well (the difference between the two models' fits was borderline statistically significant at $p = .021$). Analyzing the bidirectional effects in the reciprocal model, we found that the effect of BMI on PPS_{BMI} was much larger ($b^* = .26$, $p < .001$) than the reverse effect ($b^* = –.04$, $p = .022$). Further, two directions having effects with different signs suggests a negative feedback loop operating between body weight and personality traits. This could be interpreted as a compensatory mechanism: for instance, increasing body weight could trigger personality trait changes, which in turn could act to reduce weight. Notably, however, the model of PPS_{BMI} influencing BMI fit the data by far the worst (ps < .001 compared to the reciprocal and correlated models), opposing the possibility of unidirectional effects from traits to body weight. As a follow-up, we conducted analyses with BMI subgroups and applied a structural equation modeling-based approach called LOSEM (Briley et al., 2015) to compare model fit at different BMI levels. Without going into detail on the nuances of these analyses, they suggested that the reciprocal model fit better at higher BMI levels, with the switch between the reverse and reciprocal model occurring at BMI ≈ 25, although even in the reciprocal models the effects of traits on body weight were weaker than reverse effects.

Together, the results of **Study II** indicated that effects predominantly flow from body weight to personality. Although the genetic analyses did not differentiate specific traits (aside from the neuroticism traits in Mendelian randomization) due to limited power, they nevertheless corroborated the direction of influences found in **Study I**. Because PPS_{BMI} is an aggregate of many traits, the results also suggest body weight has numerous effects.

Evaluating the evidence on causality

The plausibility of causality in an association can be evaluated by assessing if the available evidence matches the broadly accepted criteria of causality (outlined in Fedak et al., 2015). Arguably the most widely agreed-upon criterion is temporality: a cause must precede its consequences. Indeed, **Study I** indicated that body weight preceded conscientiousness, agreeableness, and several narrower traits, so for this direction of influence, the first criterion is met. According to a second criterion—consistency—similar associations between cause and effect should be obtained in studies with different populations, locations, and methods. As the two studies comprising three demographically distinct samples and three different approaches showed converging results, this second criterion also appears to be met. By a third criterion, the association should be strong enough. Although what exactly constitutes a strong enough effect is left unclear, modern views on effect sizes acknowledge that small effects (e.g., $r = .05$) can be consequential in real life, in the "not-very-long run" (Funder & Ozer, 2019). Especially given how

many variables likely contribute to personality traits, the effects observed in **Study I** (|*b**| = .03 to .08) seem sufficiently large to be considered meaningful. Fourth, the links should be plausible, meaning that they could be explained by feasible mechanisms consistent with the current state of knowledge. As discussed above, several mechanisms seem plausible in explaining the effects, including reduced energy levels, increased inflammation, and altered perceptions by others. So, the various criteria of causality seem to be at least tentatively met to be able to conclude that body weight can influence personality traits.

Contrary to what might have been expected, however, there was no support for causality in the commonly assumed direction—from personality traits to body weight. Besides plausibility, the criteria of causality were not met. Yet, the common criteria of causality do not account for the fact that effects with complex traits tend to be probabilistic rather than deterministic: if an outcome is influenced by many factors (as body weight is), a change in a causal factor only increases the chance of a change in the outcome occurring, perhaps depending on some other causal factors. This would also mean that sample-level effects are expected to be weak. Another reason for weak sample-level effects would be that the effects are limited to certain subgroups. Based on the results, this seems to be the case. For one, the directional models of **Study I** indicated that the associations differ between people, leaving open the possibility that traits affect body weight in some people. Additionally, the twin models in **Study II** suggested that traits influence body weight in people with higher weight; personality traits may then exacerbate or prevent further weight gain in people with overweight or obesity. Considering the above, it would be premature to conclude that behavior change cannot have beneficial effects on body weight.

TOWARD MORE ACCURATE DESCRIPTIONS

As is known (and as the first two studies also showed), domains and even facets are not always sufficient to describe trait–outcome associations because narrow traits within them, such as those represented by single items, can differ in their associations with the outcome. **Study III** focused on tailoring traits to BMI in order to provide a description of the links that balances accuracy with parsimony. Another way to refine trait–adiposity links is to use measures more accurate than BMI to assess adiposity. **Study IV** aimed to clarify traits' relations to fat mass and fat-free mass by applying two other indexes besides BMI. Apart from helping make sense of trait–adiposity correlations, the following two studies' results can guide future explorations of causality: after all, attempts at explanation should start with accurate descriptions (Seeboth & Mõttus, 2018).

A bottom-up approach to identify traits linked with BMI

Besides the fact that a single Big Five domain's facets may well have different relations to BMI in both magnitude and direction, Big Five-based trait representations are limited in that they do not provide full coverage of the personality trait space—that is, there are numerous traits that do not fall into any of the five domains (Paunonen & Jackson, 2000) and are therefore likely not covered by inventories measuring the Big Five, even on the facet or item levels. Thus, Big Five-based trait–outcome descriptions cannot be considered comprehensive. **Study III** aimed to pinpoint the personality traits that body weight relates to, without relying on any pre-defined traits, to yield the most comprehensive possible, yet non-redundant, account of traits that relate to body weight.

Such an approach should begin with a dataset that covers the personality space as exhaustively as possible. Currently, the datasets that best match this requirement, and have fortunately been made freely available, are the datasets of the SAPA Project (Condon & Revelle, 2015). These datasets include 696 items selected to represent a broad set of personality constructs including but not limited to the Big Five, with data from people across the globe collected via online survey. As a feature of the SAPA data collection method, each respondent was only presented with a random subset of items, so the resulting data are massively missing completely at random—that is, the datasets had 87–88% missingness in personality items (but demographic data were collected from all). In the three SAPA datasets used in this study, the combined sample size was about 100,000, participants' mean age was 26–27 years and mean BMI 25 kg/m^2, 62–63% were female and 73–76% located in North America. I excluded two eating-related items ("Often eat too much" and "Love to eat") from the item pool as, having theoretically obvious links to body weight, they were of no interest as personality variables within the context of this study. From among the remaining 694 items, I selected for analysis those that correlated with BMI at a statistically significant

level ($p < .05$ after residualizing each item for age, age^2, sex, and continent) and aggregated them into traits reflecting BMI using exploratory factor analysis. For the sake of robustness, the item selection and factor analysis procedures were carried out in two separate datasets, so that only the items and factors that replicated across the datasets were retained.

The procedure revealed 14 factors which I named Self-control, Activity, Disorganization, Anger, Conventionality, Liveliness, Talkativeness, Obedience, Worry, Preference for the Familiar, Adventure-seeking, Altruism, Impulsivity, and Mood Swings (listed in the order of decreasing correlations with BMI), each consisting of at least three items. Some of the traits represented by the factors have been frequently correlated with BMI—for instance, activity, (dis)organization, and anger (Vainik et al., 2019)—corroborating the well-known associations but also lending credence to the approach itself. Others, such as obedience, altruism, and mood swings, have rarely or never been linked to body weight in the literature, perhaps because the traits are not included in common inventories (which appears to be the case for obedience and mood swings) or, if they are, potentially because they are measured with different items than in the current study (this may be the case for altruism).

Further, as a test of the empirically-derived factors' usefulness, I compared BMI's correlations with the resulting factors to its correlations with Big Five domains and facets as measured with the IPIP-NEO in the same dataset. The absolute values of the empirically-derived factors' correlations with BMI ranged from .03 to .14 ($Mdn_{|r|} = .08$), being considerably stronger than the Big Five domains' (.00 to .04, $Mdn_{|r|} = .01$) or facets' (.00 to .09, $Mdn_{|r|} = .02$) correlations, indicating that the factors can provide a more accurate representation of BMI's personality trait correlates.

Next, I evaluated BMI's associations with each of the three different trait types (empirically-constructed factors, domains, or facets) collectively. In elastic net models trained and tested on different subsets of the data, the 14 factors together predicted BMI three times more accurately ($r = .15$) than either the domains or facets did ($r = .05$ for both). For comparison, all 694 items together predicted BMI at $r = .24$; thus, the factors thus collectively captured most of the total predictive accuracy of the item pool (63%). Notably, these predictive accuracies were likely greatly dampened by the massive missingness in the datasets. For instance, a study comparing elastic net models' performance at different levels of imposed data missingness found prediction of BMI to be 269% higher at complete data compared to 90% missingness (Elleman et al., 2020). But regardless of prediction accuracy in the absolute sense, the current results still suggest that the factors are a good compromise between using standard traits like domains or facets and using a large number of items in capturing BMI-relevant personality variance.

All in all, the results of **Study III** corroborated but also extended previous knowledge on which traits body weight is related to. The main limitation of this study, given its aim of providing a detailed description of trait–body weight associations, was that the item pool, despite including nearly 700 items and having

more content breadth than any typical inventory, was likely still incomprehensive in its coverage of the personality trait space, meaning that traits relevant to body weight may have been missed. Nevertheless, the identified factors could guide further research by highlighting what traits (composed of which exact items) to use in tests of causality and enable increased statistical power to detect such associations owing to their stronger correlations with BMI.

Delineating traits' associations with fat mass and fat-free mass

The links can be further refined by improving measurement accuracy of adiposity. Because BMI conflates fat mass with lean mass, its correlations with personality traits could be systematically biased. Other, more accurate indices should yield more accurate correlations. **Study IV** explored personality traits' correlations with one such index and attempted to shed light on how BMI's correlations with traits are biased by distinguishing the contributions of fat mass and fat-free mass to these correlations.

For these aims, I used previously validated formulas to calculate relative fat mass (RFM) and basal metabolic rate (BMR). The former, RFM, is an estimate of whole-body body fat percentage that tracks fat mass more accurately than BMI does (Woolcott & Bergman, 2020) and can be calculated through height, waist circumference, and sex. The latter, BMR, is a measure of an organism's required energy to power crucial life-sustaining functions in the strictest sense, but can also be thought of as a proxy for lean mass given that lean mass accounts for the vast majority of variance in BMR (Dulloo et al., 2010; Weinsier et al., 1992). BMR can be estimated through height, weight, age, and sex (Mifflin et al., 1990). I combined the two indices to determine which traits relate to fat mass and lean mass, and to gauge which of BMI's correlations are driven by either component of body composition. If a trait that correlates with BMI has a stronger link with RFM than with BMR, its correlation is likely driven by fat mass; in the other case, it is likely driven by lean mass.

I applied these formulas in subsamples of Estonian Biobank (*N*s for the various analyses ranging from 2,547 to 3,535, M_{age} = 47, M_{BMI} = 26, 57% female in the full sample) where personality traits had been measured using the NEO-PI-3. Because RFM and BMR were estimated from partially overlapping variables, I first tested their correlations with each other and with BMI to assess whether the formulas were able to appropriately distinguish fat mass and fat-free mass. I found that to be the case: RFM and BMR were correlated at .23 in women and .16 in men, whereas the expected correlation between fat and lean mass is around .26 (Dulloo et al., 2010); thus, the two formulas had not produced overly similar measures. For further analyses, I residualized RFM, BMR, and BMI each for age, age^2, and sex; to distinguish the unique variance of fat and lean mass, I additionally residualized RFM for BMR and vice versa in the next set of analyses.

The results showed that RFM's correlations with traits were largely similar to BMI's as evidenced by their personality profiles' correlations. Specifically, RFM's and BMI's correlations with the 30 facets (Figure 1) were themselves correlated at $r = .89$. This estimate corresponds to previous studies where BMI's personality profile has correlated with the profiles of waist and hip circumference, waist-to-hip ratio, and skinfold thickness at $r = .85$ to .99 (Sutin et al., 2011; Terracciano et al., 2009). Although there thus appear to be no dramatic differences between different adiposity traits' personality profiles, suggesting that different indicators tend to correlate with the same traits, there is still some variability which may indicate that some indices provide more accurate correlations than others.

Additionally, the personality profiles' correlations do not reveal how strongly the different adiposity indices correlate with personality traits, but trait-level associations clarified that RFM tended to have stronger links than BMI with the traits. Not only did RFM correlate more strongly than BMI with various traits (assessed with Williams' test), but it also correlated with several traits that had no correlations with BMI—and most of these associations remained significant after additionally adjusting for BMR. Notably, RFM correlated negatively with Openness and its facets O4: Openness to Actions and O6: Openness to Values, which is generally not the case for BMI (Jokela et al., 2013) but is consistent with the oft-reported link between openness and healthier eating habits (see Lunn et al., 2014 for a review). This supports the conclusion that personality traits may be more relevant to adiposity than studies had previously shown.

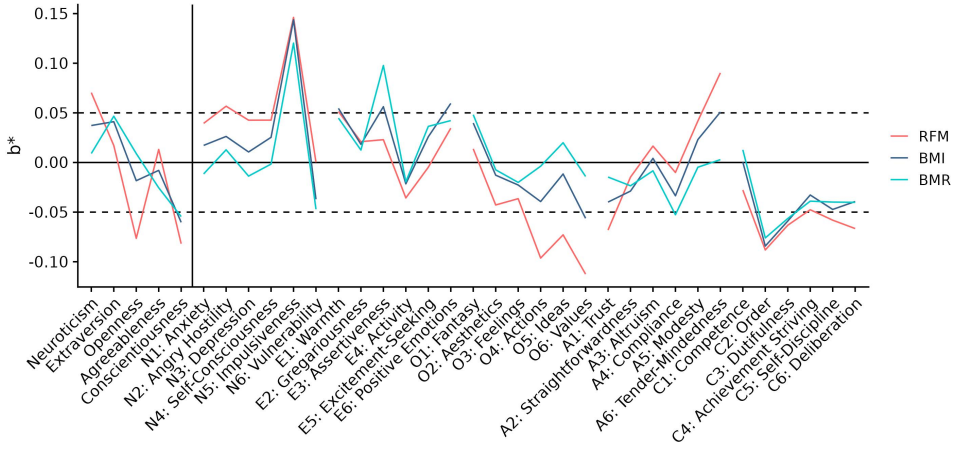

Figure 1. Personality trait profiles of RFM, BMI, and BMR. Dashed lines indicate thresholds for statistical significance ($\alpha = .05$; false discovery rate correction applied). Adapted from **Study IV**.

BMR's profile was as similar to BMI's as RFM's was ($r = .89$), suggesting that trait–BMI correlations reflect fat and lean mass' correlations equally strongly. Whereas BMR also correlated with various traits, only one link remained significant after residualizing for RFM—namely, the link with E3: Assertiveness.

Various studies have previously reported a correlation between assertiveness and BMI (Sutin et al., 2011; Terracciano et al., 2009; Vainik et al., 2019), creating an apparent association between assertiveness and higher adiposity. The current data, in contrast, suggest this association is driven by lean mass and more assertive people tend to have larger lean mass (e.g., muscle mass) instead.

Aside from clarifying traits' associations with lean mass, the correlation between BMR and assertiveness is interesting from a behavioral ecology perspective as it appears to support the existence of the pace-of-life syndrome. The pace-of-life syndrome hypothesis posits a covariation between behavioral and metabolic traits: bold, aggressive, and active individuals should have a faster life-history strategy (i.e., grow faster, have offspring earlier, and have shorter lives) and expend more energy than shy, unaggressive, and passive individuals (Careau et al., 2011; Réale et al., 2010). Support for this hypothesis has come from many studies showing metabolic rate to covary with traits like aggressiveness, activity, exploration, and dominance in, for instance, dogs, mice, and various species of fish (Careau & Garland, 2012). Whereas these links have rarely been tested in humans, with the two studies published on them having likely been underpowered to detect them (Bergeron et al., 2021; Terracciano et al., 2013), the current results suggest that such links may indeed extend to humans, given the conceptual similarity between dominance and assertiveness.

The major limitation of **Study IV** was the indirect estimation of adiposity and lean mass. Given their higher accuracy, body composition measures obtained with more direct methods would yield more reliable estimates of their correlations with traits. Yet, that the two phenotypes' correlation did not exceed their expected similarity suggests that they were differentiated adequately.

SUMMARY AND CONCLUSIONS

Across numerous previous studies, the main motivation for investigating personality traits' associations with body weight has been to uncover some potentially modifiable causes for excess weight. A far less frequently addressed possibility is that body weight could instead, or additionally, affect personality traits. Whereas correlations, both cross-sectional and longitudinal, have been shown previously between BMI and numerous traits, evidence on causality in these associations has been scarce. **Studies I** and **II** of this dissertation tested causality with three analyses and several diverse samples. Both studies indicated that causality predominantly flows from body weight to personality traits. While the genetic analyses of **Study II** detected effects from BMI to the weighted sum of many personality items (PPS_{BMI}), suggesting that body weight may influence numerous narrow traits, the longitudinal analyses of **Study I** examined associations with specific traits, identifying influences on agreeableness, conscientiousness, and several finer-grained traits.

The possibility that body weight influences personality traits is of particular interest from the perspective of personality theories. Despite extensive research, specific influences on personality traits have thus far remained elusive (Bleidorn et al., 2020; Jackson et al., 2017) and they have even been thought to be empirically unidentifiable (Turkheimer et al., 2014). In contrast, the two causally-informative studies suggest body weight to be one potential source of personality differences. Current knowledge is consistent with these effects operating through various pathways including social feedback, physical limitations, or physiological processes like inflammation.

However, there was no convincing evidence for generalizable influences from traits to body weight. This calls into question the widespread assumption that conscientiousness or any other trait commonly associated with body weight helps prevent or exacerbates excess weight accumulation, at least to a notable degree. Importantly, however, the lack of sample-level associations does not contradict effects in this direction altogether. It could be that the effects pertain to certain groups of people, perhaps being moderated by factors such as the home environment. After all, causality between complex traits tends to be probabilistic and a trait may or may not influence body weight in any given individual, possibly dependent on the numerous other factors contributing to it. Moreover, the results specifically describe the effects of naturalistic trait development and may not generalize to deliberate trait change, which could still lead to weight change that is both statistically detectable and practically meaningful.

Besides exploring causality, another objective of the studies was to provide more accurate descriptions of trait–body weight associations. Based on prior research, body weight was known to have small correlations with conscientiousness and various narrower traits, such as activity, order, self-discipline, anger, assertiveness, and impulsiveness. The two descriptively-focused studies of this dissertation replicated several, although not all, of these links, while also revealing some novel ones. More specifically, with the exploratory factor analysis of BMI-

related items, **Study III** replicated the associations with activity, organization, and anger, but also identified several new associations with traits such as altruism, obedience, conventionality, preference for the familiar, and mood swings. The novel associations attest that some links are overlooked when relying exclusively on pre-existing trait taxonomies as some weight-relevant traits may not be covered by them.

Additionally, **Study IV**, which used an improved adiposity estimate—RFM—revealed openness and some of its facets to be among the strongest personality trait correlates of body fat, along with conscientiousness and a few of its facets. Although both of these domains are associated with healthier dietary habits (Lunn et al., 2014) and the evidence is thus in principle consistent with the traits preventing excess weight accumulation via eating habits, based on the causally-informative **Study I** it appears more plausible that the link with openness may be spurious and that the direction of effects could be reversed for conscientiousness. However, these possibilities should be considered with caution as the results were based on different samples. Further, the results contradicted a link between assertiveness and body fat, suggesting that the trait's link with BMI is driven by lean mass instead.

Finally, both descriptive studies indicated that traits are more closely linked with body weight than would be concluded based on previous evidence. In **Study III**, BMI was more strongly correlated with most factor-analytically derived traits than with either the IPIP-NEO domains or facets. In **Study IV**, RFM generally had more pronounced correlations than BMI with NEO-PI domains and facets. But even the two studies may have underestimated the links. For one, in studies where the anthropometric measurements are self-reported such as **Study III**, associations between traits and body weight could be suppressed by systematic relations between traits and the tendency to underreport body weight (Roehling et al., 2008). And, as is the case in correlational studies generally, correlations are additionally attenuated by measurement error in the variables they involve (Padilla & Veprinsky, 2012), which was likely somewhat reduced but certainly not eliminated by the use of RFM instead of BMI in **Study IV**. All of this suggests that the reported correlations may underestimate the real-life relevance of body weight to personality traits and vice versa.

In summary, while the four studies have provided new evidence on the typical ways traits relate to body weight, the associations should be interpreted with their potential complexity in mind—for instance, the links may vary between people or be non-linear, a possibility beyond the scope of the four studies. This complexity also serves as a reminder that the sample-level associations do not apply to every individual. Just like a correlation, especially a small one, says nothing about any given individual—for instance, one's weight status is not sufficient to make accurate judgments about their personality traits—a sample-level causal association says nothing about effects in a specific person. Nonetheless, beyond helping make sense of body weight–personality trait links, the new evidence can guide further tests of causality. The descriptive studies' results in particular could inform new causal hypotheses.

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SUMMARY IN ESTONIAN

Isiksusejooned ja kehakaal: täpsetest kirjeldustest põhjuslike seoste testimiseni

Arvukad uuringud on näidanud, et kõrgema kehakaaluga inimesed erinevad normaalkaalulistest mitmete isiksusejoonte poolest. Näiteks käivad kõrgema kehakaaluga kaasas keskmiselt veidi suurem impulsiivsus, aktiivsus ja soojus ning veidi väiksem meelekindlus (Vainik et al., 2019). Sageli on nende uuringute eesmärgiks olnud tuvastada ülekaalu põhjustavaid tegureid – ennekõike muudetavaid, sekkumistele alluvaid tegureid. Uurijate seas levinud arvamuse kohaselt võiksid isiksusejooned mõjutada kehakaalu eelkõige läbi kalorite tarbimist või kulutamist mõjutavate käitumiste: kuna isiksusejooned on ajas üsnagi stabiilsed ning nendega seotud käitumised kipuvad korduma, peaks relevantsete käitumiste efekt energiabilansile aja jooksul ka kaalunumbris kajastuma. Näiteks võib meelekindlal, suure enesedistsipliiniga inimesel olla lihtsam kaloritega liialdamist ning seega ka ülekaalu vältida. Siiani puudub aga veenev tõendusmaterjal, mis võimaldaks väita, et mõni isiksusejoon tõepoolest kaalutõusu põhjustaks või ära hoiaks – kirjeldatud korrelatsioonid võivad olla ka mittepõhjuslikud ehk kujunenud kolmandate muutujate ühistel mõjudel. Ometi on küsimusel selge praktiline olulisus, sest liigset kehakaalu seostatakse mitmete haiguste tekkega (Larsson & Burgess, 2021).

Teisalt võib põhjuslikkus kulgeda vastupidises suunas: kehakaalust isiksusejoonteni. Kuigi seda põhjuslikkuse suunda kajastatakse kirjanduses võrdlemisi harva, paistab kehakaalu mõju isiksusejoontele usutav, arvestades kehakaalu teadaolevaid tagajärgi. Näiteks on rasvumus seotud diskrimineerimisega pea igas eluvaldkonnas alates haridusest ja tööst kuni inimsuheteni, mis põhjustab tugevaid negatiivseid emotsioone (Brewis et al., 2018; Puhl & King, 2013). Võib uskuda, et järjepideva ebavõrdse kohtlemise mõju laieneb ka kogeja mõtetele ja käitumistele ning üldistub seega isiksusejoontele. Teiseks kaasnevad ülekaaluga füsioloogilised muutused, sealhulgas vere põletikumarkerite sisalduse kõrgenemine (Forsythe et al., 2008), ning kaalulangusega ärevuse ja depressiooni sümptomite paranemine (B. L. Wolfe & Terry, 2006). Kui need seosed peaksid väljendama kaalu põletikumarkerite poolt vahendatud mõjusid, võiks kaal analoogselt ka isiksusejoontele mõjuda.

Käesoleva doktoritöö neljast uuringust kahes testiti põhjuslikkust isiksuseomaduste ja kehakaalu vahel. **Uurimus I** kasutas Wisconsini longituuduuringu kolme mõõtmislaine andmeid, mis sisaldasid nii kehamassiindeksit (KMI) kui Suure Viisiku isiksusedimensioone. Põhjuslikkuse testimiseks kasutati kaht tüüpi mitmetasandilisi mudeleid: ajas koosmuutumist testivad mudelid ning ajalist järgnevust testivad mudelid. Ühegi isiksusejoone mõju kehakaalule ei tuvastatud. See-eest olid tulemused kooskõlas kehakaalu mõjudega meelekindluse ja sotsiaalsuse dimensioonidele ning kolmele kitsamale isiksusejoonele (energilisusele, jutukusele ja laiskusele). Tulemused võivad olla seletatavad kehakaalu

sotsiaalsete ja tervisemõjudega. Näiteks võib negatiivne sotsiaalne tagasiside (diskrimineerimine) vähendada kõrgema kehakaaluga inimeste sotsiaalsust (koostöövalmidust ja teistega arvestavust). Teiseks võivad kehakaalu mõjusid vahendada põletikumarkerid. Viimaks võib kasvav kehakaal anda märku tervise allakäigust kõrgemas eas, millega kaasnev langus energiatasemes ja raskused igapäevastes toimingutes võivad viia väiksema meelekindluse ja suurema laiskuse hinnanguteni. Muuhulgas viitavad tulemused sellele, et kaalutõus ei tulene laiskusest, vaid põhjustab kõrgemaid laiskuse hinnanguid.

Uurimuses II viidi läbi kaks geneetilist analüüsi. Neist esimene rakendas Eesti geenivaramu andmetel Mendeli randomiseerimist. Selle analüüsimeetod põhineb asjaolul, et geenikoodid kujunevad juhuslikult, enne ühegi fenotüübi välja arenemist. Nii viitab seos ühe muutujaga seotud geenivariantide ja teise muutuja vahel põhjusliku seose olemasolule ja suunale (Haycock et al., 2016). Näiteks leiab muutuja A mõju muutujale B kinnitust, kui A-ga seotud geenivariandid ennustavad B-d. Analüüsiti ärevuse, depressiivsuse ja neurootilisuse mõjusid KMI-le ning KMI mõju isiksusele. Tulemustest ilmnes, et ärevuse polügeenne skoor (teisisõnu geneetiline eelsoodumus ärevuseks) ennustas madalamat KMI-d. Depressiivsuse ja neurootilisuse polügeensetel skooridel seos KMI-ga puudus. Vastupidise suunaga mõjude tuvastamiseks kombineeriti paljude isiksuseväidete vastused kokku üheks tunnuseks – fenotüüpseks isiksuseskooriks – mis tagas seoste tuvastamiseks vajaliku statistilise jõu. Ilmnes, et ka KMI polügeenne skoor ennustas fenotüüpset isiksuseskoori. Seda seost võib tõlgendada kui kehakaalu mõjule mitmetele kitsastele isiksusejoontele.

Teises analüüsis kasutati viiest riigist pärit mono- ja disügootsete kaksikutepaaride andmeid. Käitumisgeneetiline analüüs näitas taas kehakaalu mõju isiksusejoontele (nn fenotüüpsele isiksuseskoorile). Kaalugruppe eristav kordusanalüüs näitas, et kõrgema kaaluga (KMI > 25) inimeste seas olid mõjud kahesuunalised, kuid kehakaalu mõju isiksusele oli siiski märkimisväärselt tugevam. Kuigi kehakaalu mõju isiksusele tundub domineerivat, võivad isiksusejooned kehakaalu mõjutada seega just kõrgema kaalu juures.

Ülejäänud kahe uuringu eesmärgiks oli läbilõikeliste seoste varasemast täpsem kirjeldamine. Varasemad uuringud on seoste kirjeldamisel tuginenud peamiselt Suure Viisiku isiksusedimensioonidele ning nende alaskaaladele. Seda võib pidada puuduseks, sest ükski isiksuseomaduste taksonoomia, ka Suur Viisik, ei ole kõikehõlmav. Niisiis on võimalik, et kehakaalu seosed on paremini kirjeldatavad kas isiksusejoonte (dimensioonide või ka alaskaalade) kitsamate komponentidega või hoopis joontega, mida levinud küsimustikes ei leidu. **Uurimuses III** analüüsiti võimalikult kõikehõlmavat isiksuseväidete kogumit (ligi 700 väidet) sisaldavaid SAPA projekti andmestikke. Avastava faktoranalüüsiga koondati KMI-ga korreleeruvad väited faktoritesse. Protseduuri tulemusena leiti 14 faktorit, millest osad esindasid juba teadaolevalt kehakaaluga seostuvaid isiksusejooni (aktiivsus, korralikkus, viha) ning teised väljendasid varasemates töödes kirjeldamata seoseid (altruism, konventsionaalsus, kuulekus, meeleolukõikumised, tuttava eelistamine). Ootuspäraselt korreleerus KMI leitud faktoritega tugevamini kui Suure Viisiku dimensioonide või nende alaskaaladega.

Uurimuses IV kasutati seoste kirjeldamiseks KMI-st täpsemat keha rasvaprotsendi indeksit. Kuigi KMI on lihtsasti arvutatav ja teadustöös üldlevinud rasvaprotsendi näitaja, kipub KMI rasvamassi osades inimgruppides üle- ja teistes alahindama, mis võib omakorda rasvaprotsendi ja teiste muutujate seoste hindamisel kaasa tuua süsteemseid kaldeid. Näiteks kipub KMI suure lihasmassiga inimeste rasvamassi üle hindama ja seega on võimalik, et KMI seosed isiksusejoontega on põhjustatud rasvamassi asemel rasvavabast massist (lihasmassist). Uurimuses arvutati kahe ja poole tuhande geenidoonori KMI ning lisaks sellele veel kaks kehakompositsiooni näitajat: suhteline rasvamass (KMI-st täpsem rasvaprotsendi näitaja, arvutatav pikkuse, soo ja vööümbermõõdu kaudu) ning baasmetabolismi kiirus (lihasmassi suuruse kaudne näitaja, arvutatav pikkuse, kaalu, vanuse ja soo kaudu). Tulemused näitasid, et mitmed isiksusejooned korreleerusid tugevamalt suhtelise rasvamassi kui KMI-ga. Suhteline rasvamass korreleerus tugevaimalt meelekindluse ja avatusega ning mõlema dimensiooni alaskaaladega. Baasmetabolism seevastu seostus vaid ühe alaskaalaga: kehtestavusega. Kuigi seda isiksusejoont on varasemalt seostatud KMI-ga, näitavad siinsed tulemused, et korrelatsiooni taga on kehtestavuse seos rasvavaba, mitte rasvamassiga, nagu varem arvatud.

Kokkuvõttes toetasid doktoritöös kirjeldatud tulemused kehakaalu mõju isiksusejoontele. Arvukad eelnevad uurimused on püüdnud tuvastada isiksuseerinevusi põhjustavaid tegureid, kuid siiani edutult. On ka arvatud, et selliseid tegureid ei olegi võimalik empiiriliselt tuvastada. Siinsed tulemused aga osutavad, et kehakaal võib olla üks isiksusejooni mõjutavatest teguritest. Vastupidiselt levinud arvamusele ei leidnud aga kinnitust isiksusejoonte mõju kehakaalule. Samas ei ole tõendusmaterjal piisav selliste mõjude välistamiseks: näiteks on võimalik, et kehakaal muutub isiksusejoonte tahtliku muutmise tulemusel, mida selle doktoritöö raames ei käsitletud. Kahes kirjeldavas uurimuses leitud seosed pakuvad sisendit uute põhjuslike hüpoteeside püstitamiseks ja nende edasiseks uurimiseks.

PUBLICATIONS

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