NOBASCHOLAR

The Genetics of Well-being

By Espen Røysamb & Ragnhild Bang Nes, University of Oslo, Norwegian Institute of Public Health

Citation:
Røysamb, E., & Nes, R. B. (2018). The genetics of wellbeing. In E. Diener, S. Oishi, & L. Tay (Eds.), *Handbook of well-being*. Salt Lake City, UT: DEF Publishers. DOI:nobascholar.com

Abstract:

Research into the genetic and environmental origins of human wellbeing has made tremendous progress over the last two decades. Genetic effects on subjective wellbeing (SWB) are well established, and meta-analyses reveal average heritabilities in the 32-41% range. Behavior genetic studies also provide strong evidence for the causal role of environmental factors in SWB. In this chapter we review exciting developments within the intersecting fields of wellbeing and genetically informative research. We start by elaborating on the concept of heritability and the basics of twin studies. The beauty of the twin study lies in the potential to examine causal factors, without observing them, by the logic of a research design. Next, we review findings of genetic and environmental influences on SWB, and the role of such influences in the multivariate associations between different types of wellbeing, between wellbeing and ill-being, and between personality and wellbeing. We also outline recent developments within molecular genetics, and strategies to identify specific genes. A separate section addresses the issue of gene-environment interplay, and we discuss concepts such as vantage sensitivity and differential susceptibility. Throughout the chapter, we will seek to elaborate on what is known – and what is yet to be known.

Keywords: Genetic, Twin, Molecular genetics, Heritability, Wellbeing

---

To what extent, and in what ways, is wellbeing influenced by genes? What is the role and interplay of genetic and environmental factors in wellbeing and illbeing? Is the genetic effect on wellbeing due to personality-related genes? Which specific genes influence wellbeing? These are among the questions that researchers have addressed in recent years – and for which we do have some answers and are searching for more.

Recent meta-analyses have documented that the weighted average heritability of subjective wellbeing (SWB) is in the range of 32-41% (Bartels, 2015; Nes & Røysamb, 2015; Vukasovic, Bratko, & Butkovic, 2012). Heritability is, however, not a fixed statistic. There is considerable heterogeneity across population groups, measures and methods (Nes & Røysamb, 2015). There is also evidence of substantial heritability for personality traits, and the genetics of wellbeing appear to be partly about personality genes (Keyes, Kendler, Myers, & Martin, 2015; Weiss, Bates, & Luciano, 2008). Similarly, the genes that influence wellbeing appear to be partly the same as those protecting against depression (Haworth, Carter, Eley, & Plomin, 2017; Kendler, Myers, Maes, & Keyes, 2011; Nes, Czajkowski, Røysamb, Reichborn-Kjennerud, & Tambs, 2008; Nes et al., 2013).

Behavior genetic studies reporting heritability estimates also provide strong evidence for the substantial environmental effects involved. Causality of specific variables (e.g., life events) is generally hard to prove in correlational studies, but genetically informative studies reporting a heritability of 40% imply that 60% of the variance in wellbeing is caused by environmental factors, and random measurement error. Despite considerable progress in estimating genetic influences on wellbeing, several pieces of knowledge are still missing. Whereas behavior genetic studies, using twin, family and adoption designs, have been able to estimate total genetic effects (heritability), molecular genetic studies have so far had limited success in identifying the specific genetic variants involved (Okbay & al., 2016; Weiss et al., 2016). However, with collaborative efforts, extensive sample sizes and new technology for analyzing DNA, the field of molecular genetics is moving ahead at high speed.

In this chapter we aim to review some of the exciting recent findings on the role of genetic and environmental factors in wellbeing. We start by clarifying the concept of *heritability*, and move on to outline the logic involved in estimating heritabilities from twin and adoption studies. Next, we review findings pertaining to the role of genes and environment in wellbeing, and in the associations between wellbeing, illbeing and personality traits. Are the observed relationships between such phenotypes due to shared genes, shared environments or both? Next, we outline recent findings and prospects from molecular studies, and finally we address various types of interplay between genes and environment.

Heritability - What it is (Not) About.

What is the heritability of the number of fingers of human hands? Most of us have two hands and ten fingers, and we know that these features are genetic in origin. Without genes coding for hands and fingers in the developing fetus, we would not have any. Yet, the heritability of the number of fingers on human hands is probably quite low. How can that be? To understand the scientific concept of heritability (h^2) we need a simple formula:

$$Heritability = h^2 = \frac{V_g}{V_g + V_e} = \frac{V_g}{V_{tot}}$$

where V_g equals genetic variance, V_e is the environmental variance and V_{tot} represents the total variance (sum of all genetic and environmental effects). Thus, heritability is the proportion of the total variance that is due to genetic variance.

Now, returning to the number of fingers, in general there is not much variance in this figure. If everybody have ten fingers the variance is zero and it would not be meaningful to estimate the heritability (also, mathematically, division by zero is undefined). However, some people do not have ten fingers. This is usually due to accidents, which mostly are environmental in origin. The genetic variance in number of fingers is minimal. With the exception of rare mutations that generate other numbers of fingers, varying finger-numbers are due to environmental factors. Thus, the larger part of the total variance is environmental, rather than genetic, in origin – and therefore the heritability is low. This also implies that if the environmental variance for any trait is reduced in a population (e.g., less social inequalities or less accidents), the heritability increases, and with rising environmental variance (e.g., by introduction of an intervention), heritability will be reduced.

As heritability is about variance we need to keep in mind that it is a population statistic. It is not meaningful to translate a heritability estimate directly into an estimate of the genetic contribution to a single person's traits – regardless of whether the traits are wellbeing, personality, body height or number of fingers. For example, human body height has a heritability around 80% (Bergin et al., 2012; Silventoinen, Kaprio, Lahelma, & Koskenvuo, 2000; Yang et al., 2010). However, that does not mean that a person with height 170 cm has received 136 cm (170x80%) from her genes and 34 cm (170x20%) from the environment. Correspondingly, if the heritability of wellbeing is about 40% it does not mean that 40% of your wellbeing is genetic and 60% is environmental. In each one of us, the genetic and environmental factors have operated together in creating a unique blend that cannot be partitioned into percentages of effects.

In this sense, heritability is an estimate of explained variance in the same way that we can estimate the variance of wellbeing accounted for by personality traits or life events. When we obtain explained variance (R^2) in a multiple regression analysis, this parallels the estimate of heritability – the variance accounted for by genetic factors. It does not make sense to say that a certain life event explains for example 30% of a single individual's wellbeing. The same holds for genetic factors.

How then, may we envision the genetic effects on individuals without partitioning a person into genetic and environmental effects? One useful thought experiment includes imagining a person with 99 other identical twins, that is, in total 100 genetically identical persons. All differences between these 100 people would necessarily be due to environmental differences, and this group of people would have a certain mean, variance and total range of scores. Thus, the hypothetical wellbeing range among the 100 clones would represent the potential wellbeing range for each individual. In this sense, we all have a genetically based wellbeing set-point (in the sense of the average of our own clones), and a range to operate within (the range of our clones). Although this perspective involves a genetically based set-point, note that most of the 100 people will not be at this exact point: roughly half will be above and half below. This example may function as a basis for thinking about a person's wellbeing range as rather wide, with potential for both upward and downward movements. However, without the 99 other clones available, we

cannot know whether a particular person is actually below or above her own genetic midpoint.

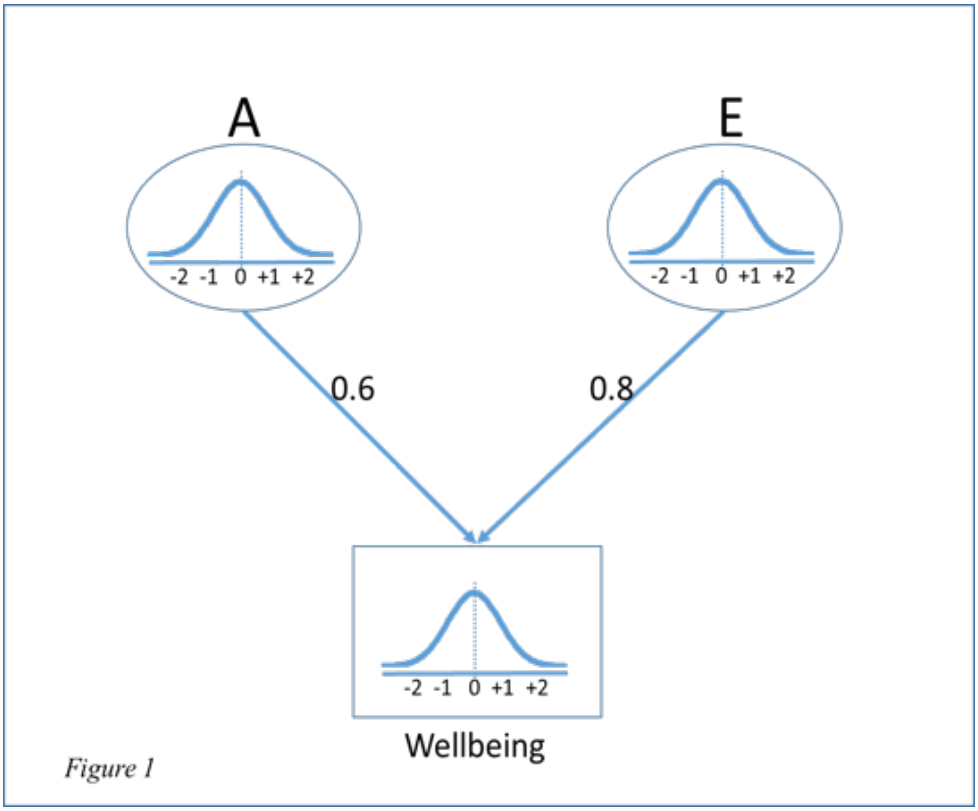

Figure 1

Figure 1 illustrates the effect of genetic and environmental factors on wellbeing. The latent factors (ovals) represent additive genetic effects (A) and environmental effects (E), and the observed (rectangular) variable represents wellbeing. In this illustration, all three variables are standardized (with *mean*=0 and *sd*=1). The effect of the genetic factor is set to 0.60, implying a heritability of 36% (0.6x0.6=.36), and the effect of the environmental factor is set to 0.80. Thus, in this scenario a person's wellbeing is given by the formula:

Wellbeing=0.60xA+0.80xE

A certain person X with a genetic disposition to happiness of -0.5 (i.e., 0.5 *sd* below the mean on the latent genetic variable A) but a favorable environment of +0.5 would end up with a wellbeing score of +.10 (i.e., 0.6x-0.5 + 0.8x0.5), whereas a person Y with a happy disposition of +1.0 but a total negative environmental load of -1.0 would have a wellbeing score of -0.20 (0.6x1.0 + 0.8x-1.0). Now, this illustration represents a basic model as it only includes an additive genetic and a non-shared environmental variable, and have disregarded potential correlations and interactions between them. We will return to the notions of gene-environment interplay later.

How to Estimate Heritability

The elegance of twin studies lies in their potential to estimate genetic and environmental effects by the logic of a design, without necessarily measuring any specific genes or given environments (Plomin, DeFries, Knopik, & Neiderhiser, 2013; Røysamb & Tambs, 2016). In the classic twin design we collect data from *monozygotic* (MZ) and *dizygotic* (DZ) twins, and the observed similarity within pairs among MZ versus DZ twins represent a core element of information. MZ twins share 100% of their genes, whereas DZ twins share on average 50% of segregating genes, and we use this knowledge to test hypotheses about the influence of four major types of factors: *Additive genetic factors* (A) are correlated at unity among MZ twins and at 0.5 in DZ twin pairs. *Non-additive genetic influences* (D) also correlate 1.0 in MZ twins, and 0.25 (or below) in DZ twins. The non-additive effects include *dominance* (hence D), interaction at a certain locus whereby one allele (genetic variant) is dominant, and *epistasis*, which involves interaction between alleles at different chromosomal loci. Shared or *common environmental factors* (C) are per definition shared by the twins in a pair, for both MZ and DZ twins, and correlate at unity. C effects are often, but not

necessarily, expected to reside within the family. Note that C factors reflect environmental influences leading to similarity among twins. A certain parenting style or event in a family may or may not represent a C factor, depending on whether the effect on the twins is the same or different. Finally, *non-shared environmental influences* (E) include all factors that contribute to twin differences (including random error), and are therefore uncorrelated between twins in a pair (Plomin et al., 2013).

Broad-sense heritability (H^2) includes both additive (A) and non-additive (D) genetic effects, whereas narrow-sense heritability (h^2) includes additive effects only. A crude estimate of the heritability (disregarding D-effects for now) can be calculated based on the difference in observed twin-cotwin correlations across zyosity groups:

$$h^2 = 2(r_{MZ} - r_{DZ})$$

Thus, the heritability equals two times the difference between the MZ and DZ correlations. Correspondingly, the shared environmental variance (C) is given by the formula

$$c^2 = 2r_{DZ} - r_{MZ},$$

and non-shared environmental variance (E) is resolved by

$$e^2 = 1 - r_{MZ}.$$

These formulas can give important information about the magnitude of various effects, but more sophisticated methods are used to test specific models and obtain measures of uncertainty.

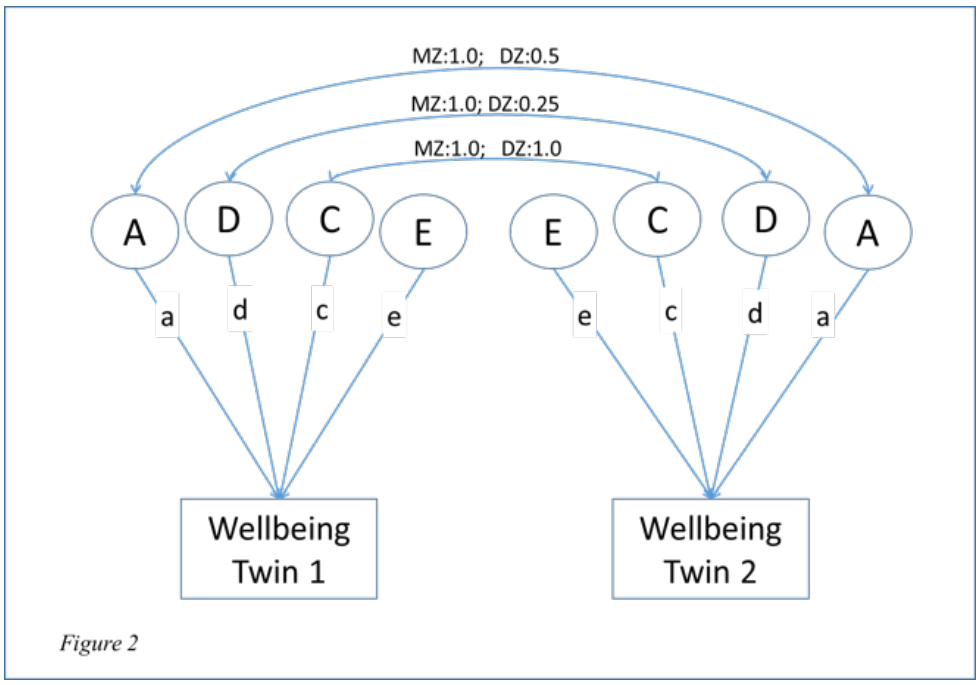

Figure 2

Figure 2 illustrates a basic biometric model with the four latent factors, A, D, C and E, and one observed wellbeing variable. Correlations between latent factors are shown for MZ and DZ twins. This is a univariate twin model, in the sense that only one observed variable is analyzed, but measured in two twins. The model can be tested as a structural equation model (SEM) in software such as Mplus, Mx or OpenMx (Boker et al., 2011; Muthen & Muthen, 2017; Neale, 1996). The twin-cotwin correlations for the latent factors, as shown in the figure, are fixed at the depicted values, for example in a two-group model with MZ and DZ twins, and the paths *a, d, c* and *e* are estimated. Typically, nested submodels (e.g., an AE model) are compared to full models (e.g., ACE) and the presence of the various latent factors is determined by the fit and the comparison of the different models. Note that the full ADCE model is underidentified in the classic twin design, which includes only MZ and DZ twins reared together. Therefore, the full model is typically an ACE, or ADE, model – depending on the structure of correlations.

The basic univariate model may be extended along several lines. First, sex-differences may be examined by testing for *quantitative* (different magnitude of effects for women and men) or *qualitative*

(partly different genes operating for women and men) effects (Neale, Røysamb, & Jacobson, 2006; Røysamb, Harris, Magnus, Vitterso, & Tambs, 2002). Testing of quantitative sex-differences can be performed in samples including only same-sex twins, whereas qualitative differences require opposite-sex DZ twins as well. Second, the univariate model may be extended into different multivariate models, including two or more phenotypes. The multivariate models comprise several subtypes, such as independent factor models, common factor models, phenotypic causality models, and Cholesky models (Neale et al., 2006; Røysamb & Tambs, 2016), which partly differ in their underlying assumptions. However, all may be informative regarding the shared and unique contributions of genes and environmental factors to a set of variables. For example, we know that wellbeing (e.g., life satisfaction) and illbeing (e.g., depression, anxiety) are negatively correlated in the population (Haworth et al., 2017; Nes et al., 2008; Nes et al., 2013). Twin studies may help resolve the question about whether this association is due to shared genetic factors or shared environmental factors (more about this below).

Genetically informative designs comprise twin (including MZ and DZ twins), family (including relatives of varying genetic relatedness, and step-relatives) and adoption (with or without twins involved) studies. The classic twin study represents the core logic and is the most widely used design. It has been criticized for limitations in distinguishing genetic and common environmental effects that contribute to similarity between twins, but a number of findings have been replicated in for example adoption studies, thus lending support to the general twin study findings (Plomin et al., 2013).

A recent meta-analysis of heritability estimates in *Nature Genetics* (Polderman et al., 2015) covered more than 2700 twin studies based on altogether 14,558,903 twin pairs, published during the last fifty years. The study included more than 17,000 phenotypes or traits and reported almost all of these to be heritable. Although genetic effects vary across traits, substantial heritabilities have been reported for phenotypes such as height and weight, metabolic functions, blood pressure, longevity, educational attainment, personality traits, drug use, mental disorders and wellbeing (Boomsma, Busjahn, & Peltonen, 2002; Plomin et al., 2013; Plomin, DeFries, Knopik, & Neiderhiser, 2016; Polderman et al., 2015). Thus, wellbeing is a human condition that falls into a widely-observed pattern of both substantial genetic and environmental origins.

Heritability Findings – What Do We Know?

A fair number of studies have reported on the genetic and environmental influences on SWB. The heritability estimates commonly range between 25 and 50% across the different studies (Bartels & Boomsma, 2009; Eid, Riemann, Angleitner, & Borkenau, 2003; Nes, Czajkowski, & Tambs, 2010; Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006; Røysamb et al., 2002; Røysamb, Tambs, Reichborn-Kjennerud, Neale, & Harris, 2003; Schnittker, 2008; Stubbe, Posthuma, Boomsma, & De Geus, 2005; Tellegen et al., 1988). The remaining variation tends to be accounted for by *nonshared*, rather than shared environmental influences. Table 1 gives a selection of estimates of genetic and environmental influences on SWB and closely related phenotypes.

Three *meta-analyses* have reported weighted average heritability estimates for wellbeing. First, Vukasovic et al. (2012) examined nine independent studies and reported an average heritability of 39%. Second, Bartels (2015) analyzed ten independent samples including a total of 55,974 individuals and obtained a general heritability estimate of 36% for SWB. She also constrained a set of analyses to life satisfaction, obtaining an estimate of 32%. Finally, Nes and Røysamb (2015) included 13 unique studies from seven countries, and used structural equation modelling to estimate average heritability. The best fitting model yielded a heritability of 40% in the total sample, and 41% in a more restricted sample (e.g., excluding outliers). These three meta-analyses varied in the inclusion criteria used, the studies included, and the statistical approaches adopted. Yet, they converge on finding average heritabilities in the range of 32 to 41%.

Notably, Nes and Røysamb (2015) also examined the variability of heritability estimates across studies, and found a significant and substantial deviation from homogeneity. Heritabilities varied beyond the level expected by random fluctuations, with roughly 70% of the variability due to true heterogeneity. Thus, the study confirmed the theoretical notion that heritability is not a fixed statistic, but can vary across populations and groups, and will depend on the range of environmental factors involved and the measures studied. This finding also accords with several studies reporting evidence of heritability-environment interaction (Johnson & Krueger, 2006; Nes, Røysamb, Harris, Czajkowski, & Tambs, 2010; van der Aa, Boomsma, Rebollo-Mesa, Hudziak, & Bartels, 2010).

Some of the biometric studies have shown the heritability of SWB to primarily reflect additive genetic effects (Nes et al., 2006; Røysamb et al., 2003; Schnittker, 2008). Other studies have indicated mainly non-additive genetic effects (Lykken & Tellegen, 1996; Stubbe et al., 2005). Perhaps the largest

biometric study of SWB to date, combining data from 6,600 twins and 54,450 nuclear family members, reported both additive and non-additive genetic influences. Broad-sense heritabilities were estimated to be 36 and 33 percent for males and females, respectively (Nes, Czajkowski, et al., 2010). This large-scale study also suggested additional effects such as assortative mating and a shared twin environment, the latter accounting for 8% of the total environmental variation.

Table 1.

Selected key twin studies of genetic and environmental influences on subjective wellbeing

First author	Year	Phenotype	Measure	N	Age	h^2	c^2	e^2
Tellegen	1988	WB	MPQWB	804		.40-.48	.13-.22	.38-.40
Røysamb	2003	SWB	SWB-index	6576	18-31	.44	-	.56
Stubbe	2005	LS	SWLS	5668	14-88	.38	-	.62
Nes	2008	LS	LS-item	8045	18-31	.17-.35	.00-.11	.66-.71
Schnittker	2008	Happiness	PA-scale	2330	25-74	.36	.06	.57
Weiss	2008	SWB	SWB-index	1946	25-74	.22	-	.78
Caprara	2009	LS	SWLS	856	23-24	.59	-	.41
Bartels	2009	SWB	SWLS, Cantril, SHS	5024	13-28	.36-.47	-	.53-.64
Keyes	2010	Emo.WB	PA-index, LS	1386	m=45	.50	-	.50
DeNeve	2012	LS	LS-item	1098		.33	-	.67
Franz	2012	LS, WB	LS-item, MPQWB	1226	51-55	.19-.35	.02	.63-.79
Haworth	2017	Happiness LS	SHS, BMSLSS	9463	16	.34-.44	.06-.11	.45-.60

BMSLSS=Brief Multidimensional Student Life Satisfaction Scale; SWLS=Satisfaction With Life Scale; SHS=Subjective Happiness Scale; MPQWB=Multidimensional Personality Questionnaire Well-being scale;

PA=Positive Affect; WB=Wellbeing; SWB=Subjective wellbeing; LS=Life satisfaction; h^2=heritability; c^2=common/shared environment (C); e^2=non-shared environment (E).

(Bartels & Boomsma, 2009; Caprara et al., 2009; De Neve, Christakis, Fowler, & Frey, 2012; Franz et al., 2012; Haworth et al., 2017; Keyes, Myers, & Kendler, 2010; Nes et al., 2008; Røysamb et al., 2003; Schnittker, 2008; Stubbe et al., 2005; Tellegen et al., 1988; Weiss et al., 2008)

The collective findings thus indicate that SWB is moderately heritable with differences in the magnitude of the heritability estimates likely to be related to the specific constructs explored, the psychometric qualities and the given research design, including the number and type of respondents (i.e., relative classes) – and the environmental context.

As noted above, the non-genetic influences on SWB primarily reflect *non-shared* environmental effects, and when not accounted for; measurement error (Bartels & Boomsma, 2009; Nes et al., 2006; Røysamb et al., 2003). *Shared* environmental influences appear to be minor, or entirely negligible. Thus, family resemblance for SWB appear to be mainly due to shared genes, and not to shared environments. It is important to note, however, that family factors might still impact on SWB. Rather than undermining the importance of family features, the biometric findings imply that environmental influences do not generally operate in a family-by-family fashion (e.g., parenting styles or socioeconomic conditions do not have general effects), but rather on an individual-by-individual basis (i.e., affect the different siblings in a family differently). Importantly, negligible influences from shared environments may primarily indicate that shared environmental influences are very small and smaller than the non-additive genetic effects.

How do the findings of heritability fit with findings of substantial *national differences* in wellbeing

(Diener, Helliwell, & Kahneman, 2010; Helliwell, Layard, & Sachs, 2017; Morrison, Tay, & Diener, 2011; Oishi & Schimmack, 2010; Tay & Diener, 2011)? It should be noted that heritability is a statistic referring to a variance component within a certain population, and as such has no say on differences across populations. It seems reasonable to assume that national differences in wellbeing are mainly environmental in origin (e.g., economy, governance, health care, education, culture, peace and safety). Thus, when including the cross-national differences in the equation, the total environmental variance will increase, and the 'global heritability' of wellbeing should consequently be reduced. Today there is also limited knowledge about systematic interaction effects between cultural and societal factors on the one hand and genetic factors on the other. Future studies should aim to disentangle how genetic expression is moderated by environmental factors also at the national and cultural level.

Multivariate Findings: Shared and Unique Factors

The same set of genes and environmental factors may underpin several correlated characteristics simultaneously. In fact, most genes involved in complex traits (e.g., SWB) tend to have pleiotropic effects - affecting a number of different characteristics, for example due to coding for "endophenotypic" neurobiological mechanisms (e.g., neurotransmitter systems) involved in different characteristics. Multivariate biometric designs permit us to decompose the correlation between variables (e.g., SWB and extraversion) into genetic and environmental sources. They also allow us to quantify the extent to which the genetic and environmental variation is specific to one characteristic only (e.g., SWB), and the extent to which it is shared by other characteristics (e.g., extraversion, depression). Such commonality or specificity is reflected in the genetic (r_g) and environmental (r_e) correlations between the given characteristics. The r_g and r_e may differ substantially from the genetic and environmental contributions to the covariance. For example, the genetic influence on two given traits may be minor (i.e., low genetic covariance). Yet, the genetic correlation could be very strong, indicating that the same genetic sources affect both traits (although weakly).

Partly overlapping genetic factors have been indicated for different wellbeing indicators. For example, by means of an extended twin-sibling design, Bartels and Boomsma (2009) reported correlations between four common SWB indicators (quality of life in general, quality of life at present, life satisfaction, subjective happiness) to largely reflect shared genes (i.e., the same genetic core). Partly overlapping sets of genes have also been reported for emotional, social, and psychological wellbeing (Keyes et al., 2010), and for self-esteem, SWB, and optimism (Caprara et al., 2009). In the latter study, the genetic correlations (r_g) were estimated to range from 0.80 (self-esteem and SWB) to 0.87 (SWB and optimism). The environmental influences were considerably more distinct, with environmental correlations (r_e) ranging from 0.18 to 0.32.

What about the association between SWB and *psychopathology*? Can SWB inform us about vulnerability to mental illness? The correlations between SWB and mental illness tend to be moderate and inverse, typically around -0.50 (Franz et al., 2012; Kendler, Myers, Maes, et al., 2011; Nes et al., 2008; Nes et al., 2013; Okbay et al., 2016). Moreover, a meta-analysis reported an average heritability of depression of 37% (Sullivan, Neale, & Kendler, 2000), thus paralleling that found for SWB. However, similar magnitudes of heritability for different characteristics do not per se inform us about the degree of shared genetic factors.

Biometric studies have indicated a partly different genetic and environmental etiology for positive and negative affect (Baker, Cesa, Gatz, & Mellins, 1992) as well as for optimism and pessimism (Plomin et al., 1992). A few studies have examined the genetic and environmental sources underpinning the negative associations between mental health problems and SWB and closely related constructs. Most of these studies are based on adult samples with mental health problems such as internalizing problems or disorders (i.e., anxiety and depression). For example, Vinberg, Bech, Kyvik, and Kessing (2007) investigated quality of life in first-degree relatives of patients with affective disorder, finding quality of life to be impaired in twins with an affected co-twin. Nes and colleagues examined the etiology underpinning the covariance between SWB (i.e., life satisfaction) and self-reported symptoms of anxiety and depression (Nes et al., 2008), and later on the association between dispositional SWB and life time major depression (Nes et al., 2013). In addition, Kendler, Myers, Maes, et al. (2011) examined the generality and specificity of etiological influences on the association between internalizing psychopathology (MDD, generalized anxiety disorder, panic attacks) and mental well-being (MWB), comprising emotional, psychological, and social well-being. All of these studies show a moderate inverse correlation between SWB and internalizing mental health problems, a substantial but far from complete overlap in genetic factors, and modest commonality of environmental influences (Kendler, Myers, Maes, et al., 2011; Nes et al., 2008). Thus, the genes that contribute to wellbeing are only partly the same as those protecting against depression. However,

environmental influences with a long-term impact on vulnerability to internalizing problems seem to influence SWB to a greater extent than environmental influences with mainly transient effects (Kendler, Myers, Maes, et al., 2011).

Although most studies to date are based on adults, a recent study of 4,700 pairs of 16-year-old twins reported similar findings (Haworth et al., 2017). This recent study has also indicated different patterns of overlap for life satisfaction and subjective happiness. More specifically, the phenotypic correlations between the constructs were highly similar. Yet, the authors observed stronger genetic links between life satisfaction and depression, than between subjective happiness and depression. About 45% of the genetic influences on life satisfaction and 70% of the genetic influences on subjective happiness were independent of the genetic factors underpinning internalizing symptoms. In line with previous studies, non-shared environmental influences were also in this study largely specific to each particular trait. Thus, certain environmental factors influence illbeing, whereas another, mostly independent set of environmental factors contributes to (low) wellbeing. The multidimensional nature of the etiological influences on wellbeing, and the overlap with mental health problems, has also been reported in a smaller study based on 613 pairs of middle-aged male twins (Franz et al., 2012).

Overall, the biometric studies thus indicate that SWB do not merely constitute the "other end" of a genetic liability to internalizing problems such as anxiety and depression. Some genetic factors for SWB convey protection against internalizing psychopathology, but high levels of SWB also reflect independent genetic sources associated with healthy, or salubrious psychological functioning. This specificity in etiological influences on SWB and internalizing problems suggests that different interventions might be needed for healing problems and promoting wellbeing, and that interventions may need to target partly different biological pathways.

The etiology underpinning associations between SWB and other mental health problems, is scarcely studied. Nevertheless, one study investigated common risk factors for (low) mental wellbeing and *externalizing* psychopathology (EP), the latter measured as a history of alcohol-related problems and smoking behavior last year (Kendler, Myers, & Keyes, 2011). The two latent constructs (i.e., EP and mental wellbeing) were modestly correlated (r = -0.28) in this study. The genetic and environmental risk factors for EP were negatively associated with wellbeing, and accounted for 7% and 21% of its genetic and environmental influences, respectively. Of note, when adding recent (last year) internalizing psychopathology (IP) to the model (i.e., controlling for genetic risk factors common to IP and EP), the genetic factors related only to EP (5% of the total genetic variation) were associated with higher wellbeing, while the unique environmental risk factors for EP were related to lower wellbeing. One tentative explanation of the positive genetic association between the unique EP factor and wellbeing could involve an underlying genetic disposition to for example extraversion that might contribute to both characteristics. Further, the authors suggested that shared genetic risk factors to EP and IP reflect genetic risk for EP associated with the negative consequences of substance use commonly decreasing wellbeing – such as anxiety and depression. The corresponding environmental influences were suggested to reflect psychosocial adversities that contribute to drug use and misuse, and in turn compromise wellbeing.

Personality and Wellbeing

What is the relationship between personality traits and wellbeing? To what extent do people's *extraversion* and *neuroticism* influence their happiness and life satisfaction? And what is the role of genetic and environmental factors in these associations?

Personality psychology focuses on a number of different tendencies and traits. Within the trait perspective, the five factor model (FFM) of personality has obtained a dominant role. The *Big Five* factors comprise five broad personality traits, including neuroticism, extraversion, openness to experience, agreeableness and conscientiousness. A number of studies have examined relations between these personality traits and wellbeing, and already in 1998, a meta-analysis concluded that in particular neuroticism is a strong (negative) predictor of life satisfaction and happiness (DeNeve & Cooper, 1998). In addition, extraversion and partly conscientiousness show substantial relations to wellbeing whereas the findings involving agreeableness, and in particular openness, are mixed and mainly indicate weak or negligible associations (DeNeve & Cooper, 1998; Lucas & Diener, 2008; Vitterso, 2001).

The importance of personality for wellbeing contrasts findings of the limited effect of demographic and background variables. In general, age and sex differences in wellbeing are usually minor or totally absent, and variables such as educational level, income or geographical location appear to explain considerably less variance in wellbeing than personality traits do (Diener, Suh, Lucas, & Smith, 1999; Lucas & Diener, 2009). Also, whereas the impact of negative and positive life events (e.g., divorce, accidents, winning a lottery) are mostly short-lasting, the associations between personality and wellbeing

tend to be enduring.

Personality traits are influenced by genetic factors, and studies have shown substantial heritabilities (Boomsma et al., 2002; Jang, Livesley, & Vernon, 1996; Vassend, Røysamb, Nielsen, & Czajkowski, 2017). Given the genetic influence on both personality traits and wellbeing, and the phenotypic associations between them, it seems pertinent to address the question of what role genes and environment play in generating relations between the two sets of characteristics. Is the heritability of wellbeing due to personality related genetic factors? Are there unique genetic influences on wellbeing, independent of personality? Are there environmental factors that influence both personality traits and wellbeing?

A few recent studies have addressed these questions. Weiss et al. (2008) found that both genetic and environmental factors contributed to the relations between wellbeing and three of the Big Five traits, namely neuroticism, extraversion and conscientiousness. However, the authors found no unique genetic effects on wellbeing. That is, the entire heritability of wellbeing was accounted for by genetic factors in personality. Correspondingly, Hahn, Johnson, and Spinath (2013) reported shared genetic factors for personality and wellbeing. Associations were found for extraversion and neuroticism, but not conscientiousness, and the personality-related genetic factors explained the entire heritability of wellbeing. Finally, Keyes, Kendler, Myers, and Martin (2015) studied a global latent factor of flourishing, finding substantial genetic overlap with personality. However, contrasting the two previous studies, the authors also found a unique genetic influence on wellbeing, unrelated to personality traits.

Some studies have also moved beyond the general level of the five broad factors and examined associations between *personality facets* and wellbeing. For example, given that extraversion is related to wellbeing, researchers have investigated which particular facets (e.g., warmth, sociability, novelty seeking, assertiveness, positive emotions), are the most important drivers of this association. One study found the facet of positive emotions in extraversion, and the facet of depression in neuroticism to be consistent predictors of life satisfaction (Schimmack, Oishi, Furr, & Funder, 2004). More recently, Quevedo and Abella (2011) reported depression, but not positive emotions, to be important, and also found a unique association for the facet of achievement striving in conscientiousness. Next, Albuquerque, de Lima, Matos, and Figueiredo (2012) replicated the finding of depression and positive emotions as central, but also found an effect from the facet of vulnerability in neuroticism.

Whereas these studies examined associations between personality facets and wellbeing at the phenotypic level, ongoing efforts try to disentangle the role of genes and environment in the facet-wellbeing relations. A recent study identified four facets that were particularly important for wellbeing, namely anxiety and depression in neuroticism and positive emotions and activity in extraversion (Røysamb, Nes, Czajkovski, & Vassend, 2017; Røysamb, Nes, & Vassend, 2014). These four facets accounted for the larger part of the genetic variance in life satisfaction, but there was also a unique genetic factor in wellbeing. Environmental factors also contributed to the associations between personality facets and wellbeing, and were the major source of unique variance in wellbeing.

The studies on personality facets and wellbeing are somewhat divergent on which specific factors that contribute uniquely to wellbeing, and this divergence is probably partly due to different measures being used, different levels of facets, and different populations being sampled. Still, the studies seem to replicate and converge on pointing to the depression facet of neuroticism and positive emotions in extraversion as important. It is noteworthy that out of 30 facets in the NEO-PI model, these two *basic emotional tendencies* seem more important for wellbeing than facets such as sociability (gregariousness), competence, warmth, excitement seeking, self-discipline and trust.

Stability and Change

Our levels of wellbeing fluctuate, but tend to be relatively stable over time, with substantial variance shared with personality traits. The stable variance in wellbeing typically amounts to approximately 50% of the total variation, and the heritability of this stable component is usually high – in the 70% to 90% range (Lykken & Tellegen, 1996; McGue, Bacon, & Lykken, 1993; Nes et al., 2013; Nes et al., 2006). As such, the heritability of stable, or dispositional wellbeing resembles that of strongly heritable characteristics like human body height (Silventoinen et al., 2003) and adult intelligence (Haworth et al., 2009). Biometric studies have also shown the genetic influences on wellbeing to be fairly stable, reporting cross-time correlations between the genetic factors (r_g) in the 0.8 to 0.9 range. By contrast, corresponding correlations for environmental factors (r_e) are reported in the 0.2 to 0.3 range (Nes et al., 2006; Paunio et al., 2009).

How do findings of (genetic) stability fit with evidence of changeability? National comparison studies have indicated quite substantial population changes over time (Diener, Tay, & Oishi, 2013; Helliwell et al., 2017; Veenhoven, 2009). Wellbeing levels also change in response to formative events

(Dyrdal, Røysamb, Nes, & Vitterso, 2011; Luhmann, Hofmann, Eid, & Lucas, 2012; Nes et al., 2014), psychotherapy and interventions (Bolier et al., 2013; Lyubomirsky & Layous, 2013; Seligman, Steen, Park, & Peterson, 2005). Longitudinal twin studies show that environmental factors account for the major share of change variance, in the 80-90% range (McGue et al., 1993; Nes et al., 2006). Interestingly, as the remainging change is due to genetic factors, these findings suggest that partly different genes affect wellbeing at different ages and life stages.

Interventions have proved effective despite substantial heritability and stability. A recent twin study by Haworth et al. (2016) showed significant improvements in wellbeing along with reductions in internalizing problems over a 10-week intervention involving kindness and gratitude tasks. The authors reported increased wellbeing despite high heritability (48%) and the same genetic influences were operating throughout the intervention. The overall magnitude of environmental influences also remained stable, but new non-shared environmental influences emerged over time in response to the intervention. In summary, wellbeing is both heritable and changeable (Røysamb, Nes, & Vitterso, 2014), and whereas genetics play a major role in the stability of wellbeing, environmental factors play an equally important role in generating change.

Molecular Genetic Findings

Development and refinement of molecular genetics in the 1990s led many to assume that specific genes with causal effect on happiness, health and illness would be easily identified. These expectations have not been met, perhaps primarily due to such traits being *multi-factorial* and *polygenetic* (i.e., many genes with differing effects are involved in genetic variation) with risk and protective factors acting in a complex, probabilistic fashion. Additionally, complex characteristics like wellbeing are likely to be *pleiotropic* (i.e., the genes are involved in multiple functions such as biochemical processes throughout the brain). Due to very modest effects, genetic heterogeneity, and intricate patterns of interplay with environmental influences - as well as psychometric challenges; the relationship between the genotype (i.e., genetic constitution) and the phenotype (i.e. observed characteristic) is far from understood. Nevertheless, molecular genetics have made immense progress over the past few years, and wellbeing is now clearly placed on the molecular genetic research agenda and examined by means of a number of different strategies and techniques.

Molecular genetics examine the structure and function of specific genes, aiming to trace the causal pathways from the DNA to a given phenotype (e.g., SWB) using a multitude of research strategies. Traditionally, molecular genetics employed linkage analyses and candidate gene association studies, and SWB has been studied using these methods. For example, by means of a linkage design, Bartels et al. (2010) reported a signal at the end of the long arm of chromosome 19 and a second suggestive linkage peak at the short arm of chromosome 1 for SWB measured with the Subjective Happiness Scale. However, the sample was fairly small and the linkage peaks were not clearly significant.

Several candidate gene studies have focused on the *5-HTTLPR* (associated with decreased availability of the serotonin transporter protein) and suggested a link between the neurobehavioral effects of the *5-HTTLPR* and enhanced sensitivity to motivationally relevant stimuli. Recent reports also suggest that the short *5-HTTLPR* variant is likely to confer heightened sensitivity to both negative and positive stimuli (Beevers et al., 2011; Belsky et al., 2015; Belsky & Pluess, 2009b; Fox, Zougkou, Ridgewell, & Garner, 2011). The “short” variant might therefore constitute a plasticity factor rather than a risk factor, coding for a general sensitivity to the environment.

Genome-wide association studies (GWAS) constitute a widely-used approach. GWAS are not hypothesis driven and commonly sequence all or most of the genome, usually focusing on correlations between particular traits (e.g., SWB) and common gene variations (i.e., *polymorphis*ms), or structural variations in the DNA such as *copy number variations* (CNVs). GWAS usually test for a million genetic markers and permit scanning the whole genetic sequence with simplicity and low cost, and may thus include a large number of participants. In a recent GWAS of SWB (n = 298,420), depressive symptoms (n = 161,460), and neuroticism (n = 170,911), Okbay et al. (2016) identified three variants associated with SWB, two associated with depressive symptoms, and 11 associated with neuroticism. These findings support the view that GWAS may successfully identify genetic associations with large sample sizes. However, mixing of different measures make the discovered associations somewhat difficult to interpret. The heritabilities estimated in this study were based on specific genetic variants, or *single-nucleotide polymorphisms* (SNPs) - and were also quite low (0.04 for SWB, 0.05 for depressive symptoms, and 0.09 for neuroticism). Overall, the effect sizes reported in this GWA study imply that to account for even a moderate share of the heritability estimated in twin studies, hundreds - or perhaps more likely thousands of genetic variants will be required.

High genetic correlations (r_g > .75) between SWB, positive affect, neuroticism, and depressive symptoms suggest a common, or partially shared etiology. By means of a multivariate genome-wide meta-analysis (n= 958,149) of these four traits - collectively referred to as "the well-being spectrum", the authors reported 63 significant independent signals, of which 29 were not previously identified (Baselmans et al., 2017).

Genetic influences for SWB have also been examined using Genome-wide Complex Trait Analysis (GCTA), which allows for estimation of the variance accounted for by all the common SNPs on the genome. Using this method, Rietveld et al. (2013) reported that up to 18% of the variance in SWB can be explained by cumulative additive effects of genetic polymorphisms that are frequent in the population. Other contemporary methods include the use of polygenic scores, resembling a sum or factor-score for multiple genetic loci and their associated weights. Polygenic scores may allow us to test genetic overlap between different dimensions of wellbeing, to detect and control for genetic confounding in the outcomes, and to explore gene-environment interaction (Plomin, 2013; Weiss et al., 2016).

Yet another molecular genetic strategy using RNA transcripts as outcomes (i.e., "expression profiling", "transcriptome profiling") has been used to examine SWB. Genes which are transcribed from genomic DNA (i.e., transcriptome) constitute an important determinant of cellular function. Using such procedures, Fredrickson et al. (2013) have reported divergent transcriptional response to adversity (CTRA; pro-inflammatory, anitviral and antibody-related genes) for hedonic and eudaimonic wellbeing despite the two constructs being highly correlated, suggesting that the two types of wellbeing engage distinct gene regulatory programs despite similar effects on overall wellbeing. These findings have been subject to heated debate however (Brown, MacDonald, Samanta, Friedman, & Coyne, 2014), but have been partly replicated (Fredrickson et al., 2015).

In summary, different genetically informative designs have provided somewhat divergent estimates of the genetic influence on wellbeing. First, twin studies (and adoption/family studies) typically yield heritability estimates in the 25-50% range. Second, genome-wide complex trait analysis, examining genome similarities in unrelated individuals, report heritabilities based on common polymorphisms up to 18% (Rietveld et al., 2013). Third, genome wide association studies, so far have only identified specific genetic variants that explain 4% of the variance in wellbeing (Okbay et al., 2016). The apparent discrepancy between quantitative genetic studies and molecular genetic studies is often referred to as *missing heritability* (Manolio et al., 2009; Plomin, 2013; Yang, Zeng, Goddard, Wray, & Visscher, 2017). Why do molecular genetic studies not find the same heritabilities as twin studies? Notably, only quantitative genetic studies (twin/adoption/family studies) are able to capture the entire genetic effects, including rare genetic variants, epistasis and dominance effects – and there is a general consensus that the estimates from such studies are mostly valid (Manolio et al., 2009; Polderman et al., 2015; Visscher, Yang, & Goddard, 2010). Possible explanations for the lower estimates found in molecular genetic studies include the limited ability to examine non-additive effects, rare mutations, epigenetics, and a high number of tiny effects. Note that the genome wide significance level is typically set at $p<0.00000005$, instead of the ordinary p-levels of 0.05, 0.01 and 0.001. This is due to the excessive multiple testing involved, and implies that huge sample sizes, in the range of hundreds of thousands of participants, are required to identify small effects (Manolio et al., 2009; Okbay et al., 2016). Note also that missing heritability has a certain parallel in 'missing environment'. Although we know from twin studies that environmental factors in total account for roughly 60% of the variance in wellbeing, and numerous studies have identified correlates of wellbeing, we have limited evidence about which specific factors that are truly causally related to wellbeing and thereby add up to this percentage.

The recent interest in the molecular genetic underpinnings of wellbeing testifies to the importance of these fields. However, there are still few published studies, most findings have not, yet, been replicated, and the scientific understanding of the molecular genetic mechanisms in SWB is rather limited, thus awaiting new discoveries. Based on the combined findings from molecular and quantitative genetics it seems fair to conclude that the total genetic effect on wellbeing is substantial. Yet there is no major happiness gene that explains most of the heritability, but rather a high number of genetic variants, each with very tiny effects, which appear to operate and interact in complex ways.

A Structural Model of Wellbeing and Illbeing

As outlined above there are substantial negative correlations between wellbeing and illbeing, and genetic factors contribute to these associations (Franz et al., 2012; Nes et al., 2008). Yet, wellbeing and illbeing are not necessarily polar opposites and a nuanced picture of relationships is emerging. Illbeing comprises various conditions such as depression, anxiety, drug abuse, eating disorders and psychoses – and the relations to wellbeing vary across disorders (Keyes, 2007; Nes et al., 2008; Ryff et al., 2006; Røysamb

et al., 2011).

Moreover, as the general field of wellbeing research has moved forward, a number of concepts and terms have been launched. In addition to examining SWB, researchers have proposed terms such as psychological wellbeing (Ryff, 1989; Ryff & Singer, 1996), mental wellbeing (Keyes et al., 2010), social wellbeing (Keyes, 1998; Shapiro & Keyes, 2008), and a general distinction between hedonic and eudaimonic wellbeing (Disabato, Goodman, Kashdan, Short, & Jarden, 2016; Goodman, Disabato, Kashdan, & Barry, 2017; Ryan & Deci, 2001; Waterman, 2008). Here, we mainly refer the interested reader to the relevant literature for these different concepts and types of wellbeing. Yet we see it fitting to integrate some of the current knowledge into a general structural model, in order to illustrate some of the mechanisms of environmental and genetic factors.

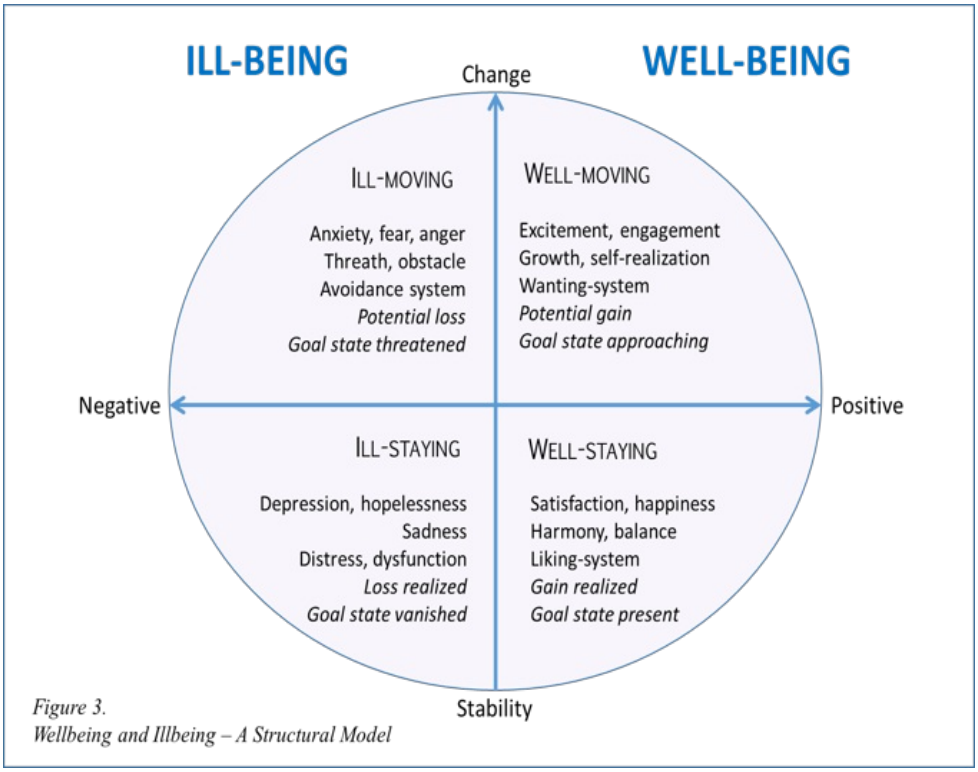

Figure 3.
Wellbeing and Illbeing – A Structural Model

Figure 3 shows a *structural model of wellbeing and illbeing*, also termed the Well/Ill-Staying/Moving (WISM) model (Røysamb & Nes, 2016). This model is partly based on the circumplex model of affect (Posner, Russell, & Peterson, 2005; Russell, 1980) and recent findings of relationships between mental disorders and different types of wellbeing (Kendler, Myers, & Keyes, 2011; Nes et al., 2013; Ryff et al., 2006).

The model involves two basic dimensions: One *positive-negative* dimension and one of *stability-change*. Environments can be (or perceived to be) positive or negative to us, and may represent continuity and stability, or development and change – with all shades of grey in between. Central to the model is the notion that humans have various ideals, needs or *goal states* (Emmons & Diener, 1986; Lyubomirsky, Sheldon, & Schkade, 2005; Oishi & Diener, 2001; Ryan, Huta, & Deci, 2008). We may experience the presence of an obtained goal state (e.g., a university grade, a good relationship, or a home), in which we are satisfied, happy and in harmony. This condition is termed *well-staying*. Sometimes we are not yet in this state, but nevertheless moving towards it (e.g., climbing a mountain, applying for a job, dating a wonderful person); we are in a process involving excitement, growth and fulfilling of potentials – thus we are *well-moving*. In other situations we realize there are threats to our goal states (e.g., a potentially failed exam, a dream-date meets someone else, or serious illness emerges); we experience fear, anxiety or anger, and are basically *ill-moving*. Note that both well-moving and ill-moving are partly future-oriented. Finally, when a goal state is lost or has vanished (e.g., failed exam, lost job, relationship break-up), we might experience sadness, depression and hopelessness – a situation of *ill-staying*.

In this model, wellbeing comprises both well-staying and well-moving. Illbeing correspondingly involves ill-staying and ill-moving. As we face life's challenges and opportunities, we perceive and

respond to environmental factors (e.g., events, people, circumstances) that are seen as good or bad, and as involving continuation of the current situation, or change. We all have a repertoire of evolutionary based tendencies (e.g., fear, sadness, joy) with which we respond (Belsky & Pluess, 2009a; Hill & Buss, 2008; Izard, 2009). However, there are also individual differences in the threshold for activation and in the strength of response, and these differences seem to be partly genetic. Thus, finding a heritability of 40% for SWB tells us about the genetic contribution to individual differences in seeking, creating and responding to environmentally based life challenges and opportunities. As life happens, and is constructed, mastered and celebrated, we all move around in this space of illbeing and wellbeing with the help of our genetic make-up. A *good life* may involve movement mainly between the well-staying and well-moving areas. However, some inevitable trips to the illbeing side might result in important turning-points, contrasts and sources of meaning, and thereby a potential return into the wellbeing sphere.

Gene-Environment Interplay

It is increasingly acknowledged that genetic and environmental factors are neither independent nor static. Rather, the genetic and environmental factors transact and interplay throughout development. Several types of gene-environment interplay are likely to be important for wellbeing. *Heritability-environment interaction* constitutes one type of such interplay and refers to environmental moderation of genetic effects. This type of interaction has been indicated in several biometric studies of SWB. The heritability of SWB has for example been shown to vary across gender (Nes, Czajkowski, et al., 2010; Røysamb et al., 2002), socio-economy (Johnson & Krueger, 2006), marital status (Nes, Røysamb, et al., 2010), and parental divorce (van der Aa et al., 2010). In one study, the genetic and environmental contributions to SWB were indicated to vary across marital status in both men and women, with lower heritability observed among the married (Nes, Røysamb, et al., 2010). Marriage or marital-like relationships are thus associated with greater impact of environmental influences on SWB.

Another type of interplay is *gene-environment interaction* (GxE), which refers to interaction between specific DNA variants and specific measured environments. A number of GxE studies and meta-analyses (Karg, Burmeister, Shedden, & Sen, 2011; Kim-Cohen et al., 2006; Risch et al., 2009) have found that individuals often differ quite markedly in how they respond to their environment depending on whether they carry specific genes. Most such studies have investigated associations between specific polymorphisms (e.g., *5-HTTLPR*) and vulnerability to *negative* health outcomes (e.g., depression, conduct disorder) given *negative* life circumstances (e.g., abuse, maltreatment) (Caspi et al., 2003; Moffitt, Caspi, & Rutter, 2006). Recently more salubrious phenotypes have also been explored. The concept of *vantage* sensitivity (Pluess & Belsky, 2011) indicate that some people for genetic reasons may benefit more than others from positive life experiences. The related construct of *differential susceptibility* (Belsky, 1997; Belsky & Pluess, 2009a) refers to some individuals being disproportionally susceptible to both negative *and* positive environmental influences – thus they are particularly malleable or plastic. So, differential susceptibility pertains to situations in which given factors - like specific genetic polymorphisms, may increase risk of adversity (e.g., depression, anxiety) given a harsh environment, but may contribute to particular benefits (e.g., optimism, wellbeing, self-esteem) given a positive and supportive environment.

The concept of *gene-environment match-making* (Røysamb, Nes, & Vitterso, 2014) builds on gene-environment interplay and suggests a pathway to increased wellbeing through pursuing and creation of environmental conditions that allow for flourishing of genetic potentials. People have various (genetically influenced) potentials, and these may be nourished and allowed to develop when matched with an appropriate environment. For example, a person with a genetic talent for creativity or musicality would benefit from creating and seeking environments that are conducive to these potentials. Such match-making occurs naturally, for example when we choose to go into sports or academia or get new friends. However, acknowledging the phenomenon also provides a basis for developing interventions and exercises that build on the importance of such match-making. The notion of gene-environment match-making is a new concept, awaiting further exploration. But it corresponds to the well-established *diathesis-stress* model of mental disorders (Belsky & Pluess, 2009a; Monroe & Simons, 1991), which postulates that disorders occur as a result of a genetically influenced vulnerability in combination with stressful life events. The diathesis-stress model can be seen as focusing on gene-environment *mismatch*, and as such the match-making perspective represents an antidote to this model.

Gene-environment interplay will commonly be concealed in the standard variance components (i.e., A, C, E). The same goes for another common type of gene environment interplay, namely *gene-environment correlation* (rGE), known as *social selection* in the developmental sciences and *reverse causation* or *confounding* in epidemiology and refers to the fact that genetic factors tend to imply exposure to a non-random selection of environments (i.e., the nature of nurture). rGE is usually classified as *passive,*

active, and *evocative* (Scarr & Weinberg, 1983). Individuals tend to inherit their parents' genes and environment and these are likely to reinforce each other (passive rGE): children of happy and optimistic parents inherit "positivity" genes *and* experience positive and supportive parenting (i.e., double advantage). Individuals also actively select and shape their environments (active rGE), and in turn, these environments reliably respond to their behavior (evocative rGE), amplifying or strengthening genetic traits and dispositions. Happy, sociable, and optimistic children actively seek situations matching their partly genetic positivity disposition (active rGE) - and tend to evoke more positive and supportive responses in others (evocative rGE). Such active and evocative rGE will be incorporated in the heritability estimate. That is, the effect is genetic in origin but the mechanism in play can be environmental. Consequently, heritability reflects more than direct genetic effects.

Most biometric studies on wellbeing have not examined such niche building and co-responsive processes explicitly (Johnson & Krueger, 2006; Krueger & Johnson, 2008). However, Krueger and colleagues have shown that youths with high levels of positive emotionality tend to elicit positive regard in their parents (Krueger, South, Johnson, & Iacono, 2008). Additionally, Kandler, Bleidorn, Riemann, Angleitner, and Spinath (2012) have reported genetic factors to mediate associations between personality traits (i.e., Extraversion and Openness) and controllable positive life events - and shown that genetic factors play a major role in continuity and repetition of controllable positive events. The nature of nurture, and the nurture of nature suggest fascinating ways in which genes and environments interact and correlate, and we are only beginning to understand the processes involved.

Conclusion

Subjective wellbeing is influenced by genetic factors. There is solid evidence of substantial heritability for different wellbeing components. Correspondingly, there is evidence for equally strong, or stronger, causal effects of environmental factors. Nevertheless, despite these replicated findings (Bartels, 2015; Nes & Røysamb, 2015), heritability is not a fixed statistic. Rather, as a representation of explained variance, heritability is found to vary significantly across studies – probably due to differing constructs and measures, differing demographics, and differing environmental and cultural conditions.

Genetic factors seem to play an important role for associations between different aspects of wellbeing, including life satisfaction and positive affect, as well as eudaimonic components. Likewise, associations between wellbeing, illbeing and personality are also substantially influenced by genetic factors. The genes that protect against depression are partly overlapping with those that contribute to wellbeing.

Despite exciting progress in studies of genetic and environmental effects on wellbeing, this is also a field where new questions arise continuously. Future research is needed to identify specific genes, and specific environments, involved in components of wellbeing and illbeing – and in their associations. We need a more profound understanding of the mechanisms at play and the way the underlying factors interact. As such, the intersection of genetics and wellbeing promises to be a tremendously exciting research area in the years to come.

References

Albuquerque, I., de Lima, M. P., Matos, M., & Figueiredo, C. (2012). Personality and subjective well-being: What Hides behind global analyses? *Social Indicators Research, 105*(3), 447-460.

Baker, L. A., Cesa, I. L., Gatz, M., & Mellins, C. (1992). Genetic and environmental influences on positive and negative affect: Support for a two-factor theory. *Psychology and Aging, 7*(1), 158-163.

Bartels, M. (2015). Genetics of wellbeing and its components satisfaction with life, happiness, and quality of life: A review and meta-analysis of heritability studies. *Behavior Genetics, 45*(2), 137-156.

Bartels, M., & Boomsma, D. I. (2009). Born to be happy? The etiology of subjective well-being. *Behavior Genetics, 39*(6), 605-615.

Bartels, M., Saviouk, V., de Moor, M. H., Willemsen, G., van Beijsterveldt, T. C., Hottenga, J.-J., . . . Boomsma, D. I. (2010). Heritability and genome-wide linkage scan of subjective happiness. *Twin Research and Human Genetics, 13*(2), 135-142.

Baselmans, B. M. L., Jansen, R., Ip, H. F., van Dongen, J., Abdellaoui, A., van de Weijer, M. P., . . . Bartels, M. (2017). Multivariate genome-wide and integrated transcriptome and epigenome-wide analyses of the well-being spectrum. *bioRxiv*.

Beevers, C. G., Marti, C. N., Lee, H. J., Stote, D. L., Ferrell, R. E., Hariri, A. R., & Telch, M. J. (2011). Associations between Serotonin Transporter Gene Promoter Region (5-HTTLPR) Polymorphism and gaze

bias for emotional information. *Journal of Abnormal Psychology, 120*(1), 187-197.

Belsky, J. (1997). Variation in susceptibility to environmental influence: An evolutionary argument. *Psychological Inquiry, 8*(3), 182-186.

Belsky, J., Newman, D. A., Widaman, K. F., Rodkin, P., Pluess, M., Fraley, R. C., . . . Roisman, G. I. (2015). Differential susceptibility to effects of maternal sensitivity? A study of candidate plasticity genes. *Development and Psychopathology, 27*(3), 725-746.

Belsky, J., & Pluess, M. (2009a). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*(6), 885-908.

Belsky, J., & Pluess, M. (2009b). The nature (and nurture?) of plasticity in early human development. *Perspectives on Psychological Science, 4*(4), 345-351.

Bergin, J. E., Neale, M. C., Eaves, L. J., Martin, N. G., Heath, A. C., & Maes, H. H. (2012). Genetic and environmental transmission of Body Mass Index fluctuation. *Behavior Genetics, 42*(6), 867-874.

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., . . . Fox, J. (2011). OpenMx: An open source extended structural equation modeling framework. *Psychometrika, 76*(2), 306-317.

Bolier, L., Haverman, M., Westerhof, G. J., Riper, H., Smit, F., & Bohlmeijer, E. (2013). Positive psychology interventions: a meta-analysis of randomized controlled studies. *Bmc Public Health, 13* .

Boomsma, D., Busjahn, A., & Peltonen, L. (2002). Classical twin studies and beyond. *Nature Reviews Genetics, 3*(11), 872-882.

Brown, N. J. L., MacDonald, D. A., Samanta, M. P., Friedman, H. L., & Coyne, J. C. (2014). A critical reanalysis of the relationship between genomics and well-being. *Proceedings of the National Academy of Sciences of the United States of America, 111*(35), 12705-12709.

Caprara, G. V., Fagnani, C., Alessandri, G., Steca, P., Gigantesco, A., Sforza, L. L. C., & Stazi, M. A. (2009). Human optimal functioning: The genetics of positive orientation towards self, life, and the future. *Behavior Genetics, 39*(3), 277-284.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., . . . Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301*(5631), 386-389.

De Neve, J.-E., Christakis, N. A., Fowler, J. H., & Frey, B. S. (2012). Genes, economics, and happiness. *Journal of Neuroscience, Psychology, and Economics, 5* (4), 193-211.

DeNeve, K. M., & Cooper, H. (1998). The happy personality: A meta-analysis of 137 personality traits and subjective well-being. *Psychological Bulletin, 124*(2), 197-229.

Diener, E., Helliwell, J. F., & Kahneman, D. (2010). *International differences in well-being*. New York, NY: Oxford University Press; US.

Diener, E., Suh, E. M., Lucas, R. E., & Smith, H. L. (1999). Subjective well-being: Three decades of progress. *Psychological Bulletin, 125*(2), 276-302.

Diener, E., Tay, L., & Oishi, S. (2013). Rising income and the subjective well-being of nations. *Journal of Personality and Social Psychology, 104*(2).

Disabato, D. J., Goodman, F. R., Kashdan, T. B., Short, J. L., & Jarden, A. (2016). Different types of well-being? A cross-cultural examination of hedonic and eudaimonic well-being. *Psychological Assessment, 28*(5), 471-482.

Dyrdal, G. M., Røysamb, E., Nes, R. B., & Vitterso, J. (2011). Can a happy relationship predict a happy life? A Population-based study of maternal well-being during the life transition of pregnancy, infancy, and toddlerhood. *Journal of Happiness Studies, 12*(6), 947-962.

Eid, M., Riemann, R., Angleitner, A., & Borkenau, P. (2003). Sociability and positive emotionality: Genetic and environmental contributions to the covariation between different facets of extraversion. *Journal of Personality, 71*(3), 319-346.

Emmons, R. A., & Diener, E. (1986). A goal-affect analysis of everyday situational choices. *Journal of Research in Personality, 20*(3), 309-326.

Fox, E., Zougkou, K., Ridgewell, A., & Garner, K. (2011). The serotonin transporter gene alters sensitivity to attention bias modification: Evidence for a plasticity gene. *Biological Psychiatry, 70*(11), 1049-1054.

Franz, C. E., Panizzon, M. S., Eaves, L. J., Thompson, W., Lyons, M. J., Jacobson, K. C., . . . Kremen, W. S. (2012). Genetic and environmental multidimensionality of well- and ill-being in middle aged twin men.

Behavior Genetics, 42(4), 579-591.

Fredrickson, B. L., Grewen, K. M., Algoe, S. B., Firestine, A. M., Arevalo, J. M. G., Ma, J., & Cole, S. W. (2015). Psychological well-being and the human conserved transcriptional response to adversity. *PLoS One, 10*(3).

Fredrickson, B. L., Grewen, K. M., Coffey, K. A., Algoe, S. B., Firestine, A. M., Arevalo, J. M. G., . . . Cole, S. W. (2013). A functional genomic perspective on human well-being. *Proceedings of the National Academy of Sciences of the United States of America, 110*(33), 13684-13689.

Goodman, F. R., Disabato, D. J., Kashdan, T. B., & Barry, S. (2017). Measuring well-being: A comparison of subjective well-being and PERMA. *The Journal of Positive Psychology*. doi: DOI: 10.1080/17439760.2017.1388434

Hahn, E., Johnson, W., & Spinath, F. M. (2013). Beyond the heritability of life satisfaction-The roles of personality and twin-specific influences. *Journal of Research in Personality, 47* (6), 757-767.

Haworth, C. M., Carter, K., Eley, T. C., & Plomin, R. (2017). Understanding the genetic and environmental specificity and overlap between well-being and internalizing symptoms in adolescence. *Developmental Science, 20*(2).

Haworth, C. M., Nelson, S. K., Layous, K., Carter, K., Jacobs Bao, K., Lyubomirsky, S., & Plomin, R. (2016). Stability and change in genetic and environmental influences on well-being in response to an intervention. *PLoS ONE [Electronic Resource], 11* (5), e0155538.

Haworth, C. M., Wright, M. J., Martin, N. W., Martin, N. G., Boomsma, D. I., Bartels, M., . . . Plomin, R. (2009). A twin study of the genetics of high cognitive ability selected from 11,000 twin pairs in six studies from four countries. *Behavior Genetics, 39*(4), 359-370.

Helliwell, J. F., Layard, R., & Sachs, J. (2017). *World Happiness Report 2017*. Retrieved from: http://worldhappiness.report

Hill, S. E., & Buss, D. M. (2008). Evolution and subjective well-being. In M. Eid & R. J. Larsen (Eds.), *The science of subjective well-being* (pp. 62-79). New York, NY: Guilford Press.

Izard, C. E. (2009). Emotion theory and research: Highlights, unanswered questions, and emerging issues. *Annual Review of Psychology, 60* , 1-25.

Jang, K. L., Livesley, W. J., & Vernon, P. A. (1996). Heritability of the big five personality dimensions and their facets: A twin study. *Journal of Personality, 64*(3), 577-591.

Johnson, W., & Krueger, R. F. (2006). How money buys happiness: Genetic and environmental processes linking finances and life satisfaction. *Journal of Personality and Social Psychology, 90* (4), 680-691.

Kandler, C., Bleidorn, W., Riemann, R., Angleitner, A., & Spinath, F. M. (2012). Life events as environmental states and genetic traits and the role of personality: A longitudinal twin study. *Behavior Genetics, 42*(1), 57-72.

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The Serotonin Transporter Promoter Variant (5-HTTLPR), stress, and depression meta-analysis revisited evidence of genetic moderation. *Archives of General Psychiatry, 68*(5), 444-454.

Kendler, K. S., Myers, J. M., & Keyes, C. L. M. (2011). The relationship between the genetic and environmental influences on common externalizing psychopathology and mental wellbeing. *Twin Research and Human Genetics, 14*(6), 516-523.

Kendler, K. S., Myers, J. M., Maes, H. H., & Keyes, C. L. M. (2011). The relationship between the genetic and environmental influences on common internalizing psychiatric disorders and mental well-being. *Behavior Genetics, 41*(5), 641-650.

Keyes, C. L. M. (1998). Social well-being. *Social Psychology Quarterly, 61*(2), 121-140.

Keyes, C. L. M. (2007). Promoting and protecting mental health as flourishing - A complementary strategy for improving national mental health. *American Psychologist, 62*(2), 95-108.

Keyes, C. L. M., Kendler, K. S., Myers, J. M., & Martin, C. C. (2015). The genetic overlap and distinctiveness of flourishing and the big five personality traits. *Journal of Happiness Studies, 16*(3), 655-668.

Keyes, C. L. M., Myers, J. M., & Kendler, K. S. (2010). The structure of the genetic and environmental influences on mental well-being. *American Journal of Public Health, 100* (12), 2379-2384.

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W., & Moffitt, T. E. (2006). MAOA, maltreatment, and gene-environment interaction predicting children's mental health: New

evidence and a meta-analysis. *Molecular Psychiatry, 11*(10), 903-913.

Krueger, R. F., & Johnson, W. (2008). Behavioral genetics and personality: A new look at the integration of nature and nurture. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research, 3rd edition* (pp. 287-310). New York: Guilford Press.

Krueger, R. F., South, S., Johnson, W., & Iacono, W. (2008). The heritability of personality is not always 50%: Gene-environment interactions and correlations between personality and parenting. *Journal of Personality, 76*(6), 1485–1521.

Lucas, R. E., & Diener, E. (2008). Personality and subjective well-being. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research (3rd ed.)* (pp. 795-814). New York, NY: Guilford Press; US.

Lucas, R. E., & Diener, E. (2009). Personality and subjective well-being. In E. Diener (Ed.), *The science of well-being: The collected works of Ed Diener* (pp. 75-102). New York, NY: Springer Science + Business Media.

Luhmann, M., Hofmann, W., Eid, M., & Lucas, R. E. (2012). Subjective well-being and adaptation to life events: A meta-analysis. *Journal of Personality and Social Psychology, 102* (3), 592-615.

Lykken, D., & Tellegen, A. (1996). Happiness is a stochastic phenomenon. *Psychological Science, 7*(3), 186-189.

Lyubomirsky, S., & Layous, K. (2013). How do simple positive activities increase well-being? *Current Directions in Psychological Science, 22*(1), 57-62.

Lyubomirsky, S., Sheldon, K. M., & Schkade, D. (2005). Pursuing happiness: The architecture of sustainable change. *Review of General Psychology, 9* (2), 111-131.

Manolio, T. A., Collins, F. S., Cox, N. J., Goldstein, D. B., Hindorff, L. A., Hunter, D. J., . . . Visscher, P. M. (2009). Finding the missing heritability of complex diseases. *Nature, 461*(7265), 747-753.

McGue, M., Bacon, S., & Lykken, D. T. (1993). Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental Psychology, 29*(1), 96-109.

Moffitt, T. E., Caspi, A., & Rutter, M. (2006). Measured gene-environment interactions in psychopathology concepts, research strategies, and implications for research, intervention, and public understanding of genetics. *Perspectives on Psychological Science, 1*(1), 5-27.

Monroe, S. M., & Simons, A. D. (1991). Diathesis stress theories in the context of life stress research - implications for the depressive-disorders. *Psychological Bulletin, 110*(3), 406-425.

Morrison, M., Tay, L., & Diener, E. (2011). Subjective well-being and national satisfaction: findings from a worldwide survey. *Psychological Science, 22*(2), 166-171.

Muthen, L. K., & Muthen, B. O. (2017). *Mplus user's guide*. Retrieved from Los Angeles, CA: https://www.statmodel.com/download/usersguide/MplusUserGuideVer_8.pdf

Neale, M. C. (1996). Genetic analyses with Mx. *Behavior Genetics, 26*(6), 593-593.

Neale, M. C., Røysamb, E., & Jacobson, K. (2006). Multivariate genetic analysis of sex limitation and G x E interaction. *Twin Research and Human Genetics, 9* (4), 481-489.

Nes, R. B., Czajkowski, N. O., Røysamb, E., Orstavik, R. E., Tambs, K., & Reichborn-Kjennerud, T. (2013). Major depression and life satisfaction: A population-based twin study. *Journal of Affective Disorders, 144*(1-2), 51-58.

Nes, R. B., Czajkowski, N., Røysamb, E., Reichborn-Kjennerud, T., & Tambs, K. (2008). Well-being and ill-being: Shared environments, shared genes? *The Journal of Positive Psychology, 3*(4), 253-265.

Nes, R. B., Czajkowski, N., & Tambs, K. (2010). Family matters: Happiness in nuclear families and twins. *Behavior Genetics, 40*(5), 577-590.

Nes, R. B., & Røysamb, E. (2015). The heritability of subjective well-being: Review and meta-analysis. In M. Pluess (Ed.), *Genetics of Psychological Well-Being* (pp. 75-96). Oxford: Oxford University Press.

Nes, R. B., Røysamb, E., Harris, J. R., Czajkowski, N., & Tambs, K. (2010). Mates and marriage matter: Genetic and environmental influences on subjective well-being across marital status. *Twin Research and Human Genetics, 13*(4), 312-321.

Nes, R. B., Røysamb, E., Hauge, L. J., Kornstad, T., Landolt, M. A., Irgens, L. M., . . . Vollrath, M. E. (2014). Adaptation to the birth of a child with a congenital anomaly: A prospective longitudinal study of maternal well-being and psychological distress. *Developmental Psychology, 50*(6), 1827-1839.

Nes, R. B., Røysamb, E., Tambs, K., Harris, J. R., & Reichborn-Kjennerud, T. (2006). Subjective well-being: Genetic and environmental contributions to stability and change. *Psychological Medicine, 36*(7), 1033-1042.

Oishi, S., & Diener, E. (2001). Goals, culture, and subjective well-being. *Personality and Social Psychology Bulletin, 27*(12), 1674-1682.

Oishi, S., & Schimmack, U. (2010). Culture and well-being: A new inquiry into the psychological wealth of nations. *Perspectives on Psychological Science, 5*(4), 463-471.

Okbay, A., Baselmans, B. M., De Neve, J. E., Turley, P., Nivard, M. G., Fontana, M. A., ... & Gratten, J. (2016). Genetic variants associated with subjective well-being, depressive symptoms, and neuroticism identified through genome-wide analyses. *Nature Genetics, 48*(6), 624-633.

Okbay, A., Baselmans, B. M. L., De Neve, J. E., Turley, P., Nivard, M. G., Fontana, M. A., . . . Study, L. C. (2016). Genetic variants associated with subjective well-being, depressive symptoms, and neuroticism identified through genome-wide analyses *Nature Genetics, 48*(12), 624–633.

Paunio, T., Korhonen, T., Hublin, C., Partinen, M., Kivimaki, M., Koskenvuo, M., & Kaprio, J. (2009). Longitudinal study on poor sleep and life dissatisfaction in a nationwide cohort of twins. *American Journal of Epidemiology, 169*(2), 206-213.

Plomin, R. (2013). Commentary: Missing heritability, polygenic scores, and gene-environment correlation. *Journal of Child Psychology and Psychiatry, 54* (10), 1147-1149.

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral genetics* (Sixth edition). New York: Worth Publishers.

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2016). Top 10 replicated findings from behavioral genetics. *Perspectives on Psychological Science, 11*(1), 3-23.

Plomin, R., Scheier, M. F., Bergeman, C., Pedersen, N. L., Nesselroade, J., & McClearn, G. (1992). Optimism, pessimism and mental health: A twin/adoption analysis. *Personality and Individual Differences, 13*(8), 921-930.

Pluess, M., & Belsky, J. (2011). Prenatal programming of postnatal plasticity? *Development and Psychopathology, 23*(1), 29-38.

Polderman, T. J. C., Benyamin, B., de Leeuw, C. A., Sullivan, P. F., van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics, 47*(7), 702-709.

Posner, J., Russell, J. A., & Peterson, B. S. (2005). The circumplex model of affect: An integrative approach to affective neuroscience, cognitive development, and psychopathology. *Development and Psychopathology, 17*(3), 715-734.

Quevedo, R. J. M., & Abella, M. C. (2011). Well-being and personality: Facet-level analyses. *Personality and Individual Differences, 50*(2), 206-211.

Rietveld, C. A., Cesarini, D., Benjamin, D. J., Koellinger, P. D., De Neve, J.-E., Tiemeier, H., . . . Bartels, M. (2013). Molecular genetics and subjective well-being. *PNAS Proceedings of the National Academy of Sciences of the United States of America, 110*(24), 9692-9697.

Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J., . . . Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *JAMA, 301*(23), 2462-2471.

Røysamb, E., Harris, J. R., Magnus, P., Vitterso, J., & Tambs, K. (2002). Subjective well-being. Sex-specific effects of genetic and environmental factors. *Personality and Individual Differences, 32* (2), 211-223.

Røysamb, E., Kendler, K. S., Tambs, K., Orstavik, R. E., Neale, M. C., Aggen, S. H., . . . Reichborn-Kjennerud, T. (2011). The joint structure of DSM-IV Axis I and Axis II fisorders. *Journal of Abnormal Psychology, 120*(1), 198-209.

Røysamb, E., & Nes, R. (2016). Genes, environment and core features of eudaimonic wellbeing. In J. Vitterso (Ed.), *Handbook of eudaimonic well-being* (pp. 233-252). Springer.

Røysamb, E., Nes, R., Czajkovski, N., & Vassend, O. (2017). Genes, personality and wellbeing. A twin study of traits, facets and life satisfaction. *Submitted for publication*.

Røysamb, E., Nes, R., & Vassend, O. (2014). *Genes, personality and life satisfaction*. Paper presented at the 15th International Congress on Twin Studies, Budapest.

Røysamb, E., Nes, R. B., & Vitterso, J. (2014). Well-being: Heritable and changeable. In K. Sheldon & R. E. Lucas (Eds.), *Stability of Happiness* (pp. 9-31). Elsevier.

Røysamb, E., & Tambs, K. (2016). The beauty, logic and limitations of twin studies. *Norsk Epidemiologi, 26*(1-2), 35-46.

Røysamb, E., Tambs, K., Reichborn-Kjennerud, T., Neale, M. C., & Harris, J. R. (2003). Happiness and health: Environmental and genetic contributions to the relationship between subjective well-being, perceived health, and somatic illness. *Journal of Personality and Social Psychology, 85* (6), 1136-1146.

Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39* (6), 1161-1178.

Ryan, R. M., & Deci, E. L. (2001). On happiness and human potentials: A review of research on hedonic and eudaimonic well-being. *Annual Review of Psychology, 52* , 141-166.

Ryan, R. M., Huta, V., & Deci, E. L. (2008). Living well: A self-determination theory perspective on eudaimonia. *Journal of Happiness Studies, 9*(1), 139-170.

Ryff, C. D. (1989). Happiness Is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology, 57* (6), 1069-1081.

Ryff, C. D., Love, G. D., Urry, H. L., Muller, D., Rosenkranz, M. A., Friedman, E. M., . . . Singer, B. (2006). Psychological well-being and ill-being: Do they have distinct or mirrored biological correlates? *Psychotherapy and Psychosomatics, 75*(2), 85-95.

Ryff, C. D., & Singer, B. (1996). Psychological well-being: Meaning, measurement, and implications for psychotherapy research. *Psychotherapy and Psychosomatics, 65*(1), 14-23.

Scarr, S., & Weinberg, R. A. (1983). The Minnesota adoption studies - genetic-differences and malleability. *Child Development, 54*(2), 260-267.

Schimmack, U., Oishi, S., Furr, R. M., & Funder, D. C. (2004). Personality and life satisfaction: A facet-level analysis. *Personality and Social Psychology Bulletin, 30* (8), 1062-1075.

Schnittker, J. (2008). Happiness and success: Genes, families, and the psychological effects of socioeconomic position and social support. *American Journal of Sociology, 114* (Suppl 1), S233-S259.

Seligman, M. E. P., Steen, T. A., Park, N., & Peterson, C. (2005). Positive psychology progress: Empirical validation of interventions. *American Psychologist, 60*(5), 410-421.

Shapiro, A., & Keyes, C. L. M. (2008). Marital status and social well-being: Are the married always better off? *Social Indicators Research, 88*(2), 329-346.

Silventoinen, K., Kaprio, J., Lahelma, E., & Koskenvuo, M. (2000). Relative effect of genetic and environmental factors on body height: differences across birth cohorts among Finnish men and women. *Am J Public Health, 90* (4), 627-630.

Silventoinen, K., Sammalisto, S., Perola, M., Boomsma, D. I., Cornes, B. K., Davis, C., . . . Kaprio, J. (2003). Heritability of adult body height: A comparative study of twin cohorts in eight countries. *Twin Research, 6*(5), 399-408.

Stubbe, J., Posthuma, D., Boomsma, D., & De Geus, E. (2005). Heritability of life satisfaction in adults: A twin-family study. *Psychological Medicine, 35*(11), 1581-1588.

Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: Review and meta-analysis. *American Journal of Psychiatry, 157* (10), 1552-1562.

Tay, L., & Diener, E. (2011). Needs and subjective well-being around the world. *Journal of Personality and Social Psychology, 101*(2), 354-365.

Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K. J., Rich, S., & Segal, N. L. (1988). Personality similarity in twins reared apart and together. *Journal of Personality and Social Psychology, 54* (6), 1031-1039.

van der Aa, N., Boomsma, D. I., Rebollo-Mesa, I., Hudziak, J. J., & Bartels, M. (2010). Moderation of genetic factors by parental divorce in adolescents' evaluations of family functioning and subjective wellbeing. *Twin Research and Human Genetics, 13* (2), 143-162.

Vassend, O., Røysamb, E., Nielsen, C. S., & Czajkowski, N. O. (2017). Musculoskeletal complaints, anxiety-depression symptoms, and neuroticism: A study of middle-aged twins. *Health Psychology, 36*(8), 729-739.

Veenhoven, R. (2009). Well-being in nations and well-being of nations: Is there a conflict between

individual and society? *Social Indicators Research, 91*(1), 5-21.

Vinberg, M., Bech, P., Kyvik, K. O., & Kessing, L. V. (2007). Quality of life in unaffected twins discordant for affective disorder. *Journal of Affective Disorders, 99*(1-3), 133-138.

Visscher, P. M., Yang, J., & Goddard, M. E. (2010). A commentary on 'common SNPs explain a large proportion of the heritability for human height' by Yang et al. (2010). *Twin Research and Human Genetics, 13*(6), 517-524.

Vitterso, J. (2001). Personality traits and subjective well-being: Emotional stability, not extraversion, is probably the important predictor. *Personality and Individual Differences, 31*(6), 903-914.

Vukasovic, T., Bratko, D., & Butkovic, A. (2012). Genetic contribution to the individual differences in subjective well-being: A meta-analysis. *Drustvena Istrazivanja, 21*(1), 1-17.

Waterman, A. S. (2008). Reconsidering happiness: A eudaimonist's perspective. *The Journal of Positive Psychology, 3*(4), 234-252.

Weiss, A., Baselmans, B. M. L., Hofer, E., Yang, J. Y., Okbay, A., Lind, P. A., . . . Luciano, M. (2016). Personality polygenes, positive affect, and life satisfaction. *Twin Research and Human Genetics, 19*(5), 407-417.

Weiss, A., Bates, T. C., & Luciano, M. (2008). Happiness is a personal(ity) thing: The genetics of personality and well-being in a representative sample. *Psychological Science, 19*(3), 205-210.

Yang, J., Benyamin, B., McEvoy, B. P., Gordon, S., Henders, A. K., Nyholt, D. R., . . . Visscher, P. M. (2010). Common SNPs explain a large proportion of the heritability for human height. *Nature Genetics, 42*(7), 565-569.

Yang, J., Zeng, J., Goddard, M. E., Wray, N. R., & Visscher, P. M. (2017). Concepts, estimation and interpretation of SNP-based heritability. *Nature Genetics, 49*(9), 1304-1310.