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REVIEW ARTICLE

Happiness in Behaviour Genetics: An Update on Heritability and Changeability

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Abstract In this paper we summarize recent behaviour genetic findings on happiness measured as life satisfaction (LS) and subjective wellbeing (SWB) and discuss important implications pertaining to stability and change, including the potential of individual and societal interventions. Broadly speaking, two main research strategies explore genetic and environmental influences on happiness, including *quantitative* and *molecular genetics.* Whereas molecular genetics seeks to trace the causal pathways from specific DNA variants, quantitative genetics estimates the magnitude of overall genetic and environmental influences without specifying actual DNA sequences and usually without specifying specific environmental circumstances. Molecular genetic studies have entered the happiness arena, but have shown mixed results. Most replicated findings are therefore based on quantitative genetics and derived from twin and family studies decomposing variation and co-variation into genetic, shared, and non-shared environmental sources. Recent meta-analyses of such studies report genetic influences (i.e., heritability) to account for 32–40 % of the variation in overall happiness (i.e., SWB, LS), and indicate that heritability varies across populations, subgroups, contexts and/or constructs. When exploring stable SWB levels, heritability is reported in the 70–80 % range, whereas momentary positive affect is often entirely situational. Happiness is thus heritable, stable, variable and changeable. What do these findings imply? Can happiness be raised as a platform in individuals and societies? We suggest that individual and societal interventions that target causal pathways and address both amplifying and compensatory processes (i.e., focus on developing strengths and mitigating risks)—thus providing for positive *gene-environment matchmaking*, are likely to be effective and longer lasting.

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1 Background

Emotions and motivation influence the way we perceive, evaluate, and respond psychologically, behaviourally, and physiologically, serving an important functional role by regulating how we interact and deal with our circumstances and surrounding context (Haybron 2014). By reflecting emotional and motivational responses, happiness constitutes an integral part of an ongoing functional process, a part of our biological make-up that serves fitness-enhancing functions by mobilizing appropriate responses to immediate circumstances and events. It is reported to facilitate availability of resources for coping with stress, for connecting with others, for innovation, and for approaching challenges (Fredrickson 2001)—and might act as a conspicuous fitness indicator by contributing to fitness-promoting activities such as access to mates and allies (Weiss et al. 2002). Happiness may thus be understood as a drive as well as an outcome, and consequently cause health and success as much as reflect these outcomes (Lyubomirsky et al. 2005). Genetic influences on happiness are therefore not very surprising.

A number of studies, using a range of genetically informative research designs, have shown that genetic influences are important for happiness and wellbeing, but with the genetic effects varying across different measures and environmental conditions. In this paper we aim to provide a brief introduction to the main methods and then to summarize findings for happiness measured as subjective wellbeing (SWB) or life satisfaction (LS) and perhaps more importantly, to discuss their implications—particularly those pertaining to stability, change, and intervention.

1.1 Genetically Informed Research

The foundations of quantitative and molecular genetics were developed already at the beginning of the twentieth century and much scientific efforts were vested on genetically informed research in medicine and biology. Within the behavioural and social sciences, research primarily focussed on environmental sources of human behaviour until the 1960s (Rutter et al. 2006) and influential perspectives like the behaviourist movement assumed that psychological characteristics could be understood entirely independent of biology, genetics, and evolutionary history. From the 1960s there was a considerable growth in genetic research and by the early 1980s, environmental influences were by many assumed to be less important than in previous decades. In the 1990s, development and refinement of molecular genetic strategies led many researchers to believe in the possibility for identification of specific genes with causal effects on most human characteristics including happiness. Yet, molecular genetics have yielded less substantive and replicable results than initially anticipated—and particularly so for psychological characteristics. This is perhaps mainly due to psychological characteristics being *multi-factorial* (i.e., influenced by multiple genetic and environmental processes operating individually and interactively) and *polygenetic* (i.e., many genes are involved) with risk and protective factors acting in a complex, probabilistic fashion rather than more deterministic. Most of the genetic variants involved are also likely to be *pleiotropic*, meaning that they are involved in multiple functions or characteristics. For example, some of the genetic polymorphisms that seem to

be related to happiness and wellbeing (e.g., 5-*HTPLLR*) are also involved in psychopathology (e.g., anxiety, depression). The specific genetic polymorphisms are therefore likely to code for "endophenotypic" neurobiological processes, such as neurotransmitter systems (e.g., serotonin, dopamine), that are involved in many different psychological outcomes. Such mechanisms might partly explain findings of partly overlapping sets of genes (i.e., 25–99 %) for different happiness indicators (Bartels and Boomsma 2009; Caprara et al. 2009; Keyes et al. 2010; Pluess 2015) as well as for happiness and personality (Hahn et al. 2013; Weiss et al. 2008), and happiness and psychopathology (Franz et al. 2012; Kendler et al. 2011; Nes et al. 2008, 2013; Okbay et al. 2016).

1.2 Quantitative Genetic Studies

Most human traits are influenced by both genetic and environmental factors (Polderman et al. 2015), but the finding of susceptibility genes to multi-factorial polygenetic psychological characteristics like happiness—in a robust and replicable manner, has proven complicated. Identification of causal genetic variants that explain much of the variation in happiness might not even be very likely in the future. It has turned out to be tremendously difficult even for characteristics with almost perfect reliability—like human body height (Turkheimer 2011, 2012; Wood et al. 2014). A recent large scale ($N = 298{,}420$) genome-wide association study (GWAS) of SWB identified only three credible genetic loci which explained a very low fraction of the variance (Okbay et al. 2016). The proteins that constitute the direct products of gene expression represent only an initial step in a long series of complex transactions involving innumerable factors leading to manifest, subjectively experienced happiness (Krueger and Johnson 2008). Being fundamentally subjective, this experience might also vary across different individuals.

Instead of providing insights into specific genomic regions underpinning variation in happiness and wellbeing, quantitative genetic studies typically use data from family members—such as twins, nuclear family members, or adoptees to partition the total variation algebraically into latent, unobserved genetic, shared (C), and non-shared (E) environmental contributions by means of path analysis and structural equation modelling (SEM) techniques. The estimated genetic contribution is commonly called *heritability* and represents the total effects from latent, unmeasured genetic sources likely to reflect influences from a large number of genes (i.e., polygenetic). The heritability estimate might reflect *additive genetic influences* (A) which comprise effects from individual genetic loci that combine additively and *non-additive genetic influences* (D) which reflect interaction between alleles (gene variants) at the same locus (dominance) or across loci (epistasis). *Broadsense* heritability (H^2) refers to effects from both additive and non-additive genetic influences, and *narrow-sense* heritability (h^2) refers only to additive ones. Expressed in percentage, heritability reflects the percentage of the total variation attributable to genetic factors. It is a group statistic with poor predictive value in any individual case referring to individual differences in populations arising from genetic differences.

Although the heritability estimate is the most frequently reported finding from quantitative genetics, it is important to note that the major strength of quantitative genetics is not the ability to estimate heritability as such. Quantitative genetic methods afford numerous possibilities, for example enabling multivariate analysis of the aetiology underlying co-occurrence, comorbidity, and heterogeneity, developmental continuity and change, sex differences, and correlations and interactions between putative genes and environmental factors. A major advantage of quantitative genetic designs concerns their

ability to control for genetic and social endowments that are unobserved in social science data sets. As people tend to create their own environments partly due to genetic influences, some factors assumed to be environmental might be partly genetic due for instance to constituting indirect measures of personality. Illustrating this important point, quantitative genetics have shown that some personal life events and difficulties such as financial problems are moderately (i.e., 39 %) heritable (e.g., Kendler et al. 1993). This is probably due to genetically influenced personality characteristics (e.g., sensation-seeking, being less cautious) rather than specific genes coding for indebtedness. The same mechanisms hold for exposure to positive experiences. For example, individuals with high scores on the personality trait of Openness tend to experience more controllable positive life events. This association appears to be primarily genetically mediated and unidirectional—from personality trait to life events (Kandler et al. 2012). Genetically informed data and quantitative genetic strategies may thus enable incrementally better causal inferences.

2 Findings

2.1 Heritability

Table 1 provides an overview of findings from the main quantitative genetic studies to date. Findings from such studies of overall happiness measures (e.g., SWB, LS) are fairly consistent, with genetic influences commonly found to account for approximately 20–50 % of the total variation. The estimates are based on thousands of twins from several different countries, the majority reared together, but some also reared apart (Bartels and Boomsma 2009; Bartels et al. 2010; Nes et al. 2006; Nes et al. 2010a, b; Røysamb et al. 2002; Røysamb et al. 2003; Schnittker 2008). Some studies have reported only additive genetic influences (i.e., narrow heritability) (Røysamb et al. 2002; Nes et al. 2005, 2006), whilst others have reported both additive and non-additive genetic effects (i.e., broadsense heritability) (Bartels and Boomsma 2009; Nes et al. 2010a, b). The few *specific* gene loci identified for SWB explain only a very small proportion of this overall estimate (Okbay et al. 2016). However, a recent study using whole-genomic information from a pooled sample of 11,500 unrelated genotyped individuals reported the lower bound of "common narrow heritability" of SWB to be 12–18 % after correction for measurement error (Rietveld et al. 2013). This suggests that genetic polymorphisms that are common in the population *together* account for nearly half of the overall heritability of SWB.[1]

When SWB is examined as a stable propensity or trait (e.g., stable SWB, or dispositional LS), heritability is usually higher and amounting to 70–90 % (Lykken and Tellegen 1996; Nes et al. 2006, 2013; McGue et al. 1993) with cross-time correlations between genetic factors estimated to range between 0.78 and 0.93 over a 6 year period (Nes et al. 2006; Paunio et al. 2009). The heritability of "dispositional happiness" (i.e., the stable component) is thus similar to the heritability estimates obtained for strongly heritable characteristics such as liability to schizophrenia and bipolar disorder (Lichtenstein et al. 2009; Sullivan et al. 2003), human body height (Silventoinen et al. 2003), and adult intelligence (Haworth et al. 2010).

By contrast, when happiness is estimated as a momentary state (e.g., current positive affect), heritability is often found to be low or entirely negligible (Menne-Lothmann et al. 2012; Riemann et al. 1998). In fact, using experience sampling methodology Menne-

[1] Genetic interaction effects (i.e., non-additive genetic variance) are difficult to identify in such studies.

Table 1 Overview of main studies

SWB	First author	Years	Study	N	Age	Respondents	Phenotype	Instrument	H^2/h^2	c^2	e^2
Momentary	Baker	1992	Independent	1020	16–98	Twins reared together, families	PA	ABS	–	.22–.56	
	Riemann	1998	Independent	600	18–70	Twins reared together	Positive mood	Mood scale	.00–.21	.07–.16	.71–.84
	Menne-Lothmann	2012	EFPTS	520	M = 27	Female twins reared together	Momentary PA	Four items	–	.00–.34	.66–1.0
Overall Time-Specific	Tellegen	1988	MTFS	804	–	Twins reared together/apart	WB	MPQWB	.40–.48	.13–.22	.38–.40
	Bergeman	1991	SATSA	848	50–87	Twins reared together/apart	LS	LSI-Z	.25	–	.75
	Harris	1992	SATSA	1448	26–87	Twins reared together/apart	LS	LSI-Z	.00–.50	.00–.17	0.55–.67
	Finkel	1997	MTFS	5556	>17	Twin Families	WB	MPQWB	0.40	–	0.60
	Røysamb	2002	NIPHTP	5140	18–31	Twins reared together	SWB	SWB index	.46–.54	–	.46–.54
	Røysamb	2003	NIPHTP	6576	18–31	Twins reared together	SWB	SWB index	.44	–	.56
	Eid[a]	2003	Independent	278	18–70	Twins reared together	PA	PA scale	.36	–	.64
	Takkinen	2004	Independent	434	64–76	Twins reared together	WB	CES-D WB	.30	.08	.61
	Stubbe	2005	NTR	5668	14–88	Twins and siblings	LS	SWLS	.38	–	.62
	Nes	2005	NIPHTP	8045	18–31	Twins reared together	SWB	SWB index	.45	–	.55
	Nes	2006	NIPHTP	8045	18–31	Twins reared together	SWB	SWB index	.42–.56	–	.44–.58
	Johnson	2007	MIDUS	1438	25–74	Twins reared together	LS	LS 3 items	.17–.45	.07–.15	.45–.76
	Nes	2008	NIPHTP	8045	18–31	Twins reared together	LS	LS	.17–.35	.00–.11	.66–.71
	Schnittker	2008	MIDUS	2330	25–74	Twins and siblings	Happiness (PA)	PA scale	.36	.06	.57
	Weiss	2008	MIDUS	1946	25–74	Twins reared together	SWB	SWB 3 items	.22	.00	.78
	Caprara.	2009	ITR	856	23–24	Twins reared together	SWB	SWLS	.59	–	.41
	Bartels	2009	NTR	5024	13–28	Twins and siblings	SWB, SH, QoL	SWLS, SHS, Cantril	.36–.47	–	.53–.64
	Paunio	2009	TFTC	19,151	18–95	Twins reared together	LS	LS 4 items	.29–.42	–	.58–.71

Table 1 continued

SWB	First author	Years	Study	N	Age	Respondents	Phenotype	Instrument	H^2/h^2	c^2	e^2
	Nes	2010	N/HUNT	60,000	18–80	Twins, siblings, parents	SWB	SWB index	.33–.36	.00–.12	.64–.67
	Bartels	2010	NTR	12,000	12–88	Twins and siblings	SH	SHS	.22–.41	–	.59–.78
	Keyes	2010	MIDUS	1340	M = 45	Twins reared together	EM	Six items, PA/LS	.48	–	.52
	Kendler	2011	MIDUS	1340	M = 45	Twins reared together	EM	Six items, PA/LS	.495	–	.505
	Van der Aa	2012	NTR	6773	13–20	Twins and siblings	QoL	Cantril	.33–.42	–	.58–.67
	Matteson[b]	2013	SIBS	615	M = 15	Adoptive/nonadptive families	WB	MPQWB	.42	–	.58
	DeNeve	2012	AddHealth	1098	–	Twins reared together	LS	LS	.33	–	.67
	Hahn[c]	2012	Independent	698	M = 40	Twins reared together	LS	LS	.30	.06	.64
	Franz	2012	VETSA	1226	51–55	Male twins reared together	PWB, LS, WB	PWB, MPQWB, LS	.19–.50	.01–.02	.49–.79
	Bartels	2013	NTR	10,610	12–20	Twins and siblings	SWB, SH, QoL	SWLS, SHS, Cantril	.34–.47	–	.53–.66
	Gatt	2014	TWIN-E	1669	18–61	Twins	SWB, LS	SWLS, COMPAS-W	.31–.43	–	.57–.69
Stable	McGue.	1993	MTFS	254	17–37	Twins reared together/apart	Dispositional WB	MPQWB	.95	–	.05
	Lykken	1996	MTFS	254	17–37	Twins reared together/apart	Dispositional WB	MPQWB	.80	–	.20
	Nes et al.	2006	NIPHTP	8045	18–31	Twins reared together	Dispositional SWB	SWB index	.80	–	.20
	Nes et al.	2013	NIPHTP	8045	18–36	Twins reared together	Dispositional LS	LS	.72	–	.28

SATSA The Swedish Adoption/Twin Study of Aging, *NIPHTP* Norwegian Institute of Public Health Twin Panel, *NTR* Netherland Twin registry, *MTFR* Minnesota Twin-Family Registry, *MIDUS* National Study of Midlife Development in the United States, *ITR* Italian Twin registry, *EFPTS* East Flanders Prospective Twin Survey, *VETSA* Vietnam Era Twin Registry Study, *TFTC* The Finnish Twin Cohort, *SIBS* Sibling Interaction and Behavior Study, *ABS* Affect Balance Scale (Bradburn 1969), *SWLS* Satisfaction With Life Scale (Diener et al. 1985), *MPQ* Multidimensional Personality Questionnaire (Tellegen 1982), *SHS* Subjective Happiness Scale (Lyubomirsky and Lepper 1999), *Cantril* Cantril's ladder (Cantril 1965), *SWB index* Subjective Well-Being index (Moum et al. 1990), *SWB Questionnaire* Subjective Well-Being Questionnaire (King and Landau 2003), *PA Scale* The Aggregated Positive Affect Scale (Riemann et al. 1998), *LS* Life Satisfaction single item, *PAS* Positive Affect Scale (MIDUS, Mrozek and Kolarz 1998), *QoL* quality of life, *PA* positive affect, *EM* emotional wellbeing; COMPAS-W scale (Gatt et al. 2014)

Momentary: current/recent/in-situation measures; Time-specific: general measure at single time-point; Stable: longitudinal data with two or more assessments. H^2/h^2 = Broad sense/narrow sense heritability; c^2 = common environment variance; e^2 = non-shared environment variance

[a] The estimates reflects influences from genetic and environmental influences on aggregated positive affect across five mood inducing experimental conditions

[b] Sample size refers to number of families. Each family consisted of at least one parent and two adolescent offspring

[c] This study includes multi-generation data from partly different samples. The estimates and demographic information listed in the table refer to twins only

Lothmann et al. (2012) reported momentary positive affect to be entirely attributable to environmental influences, indicating that positive affect may thrive in all in response to positive circumstances irrespective of genetic variation (i.e., environmental factors). Similar findings were also reported by Baker et al. (1992) who found positive affect to be primarily situational and entirely unrelated to genetic influences.

Most studies to date have investigated overall indicators (e.g., SWB, LS), however, and three recent meta-analyses based on partly overlapping, partly separate samples have independently reported the average heritability of SWB and LS to range between 32 and 41 % (Bartels 2015; Nes and Røysamb 2015; Vukasovic et al. 2012). This average estimate probably constitutes a lower bound as heritability is likely to be deflated by measurement error. Assuming an average reliability of 0.80 across the different happiness indicators included in these studies, and given an estimated heritability of 40 %, the findings translate into 50 % true heritability. Two of these meta-analyses (Vukasovic et al. 2012; Nes and Røysamb 2015) and several studies examining heritability-environment interaction (i.e., environmental moderation of heritability) have also shown that the heritability of overall SWB and LS varies across different indicators, strata, or subpopulations. Heritability of SWB has for example been shown to vary across gender (Nes et al. 2010a, b; Røysamb et al. 2002), socio-economy (Johnson and Krueger 2006), marital status (Nes et al. 2010b), and parental divorce (van der Aa et al. 2010). These findings underscore that there is no heritability of happiness as such—heritability depends on environmental factors and environmental variation.

The *heritability-environment interaction* described above constitutes one of several important types of gene-environment interplay that are commonly incorporated in the quantitative genetic variance components (A, D, C, E). Another type of interplay is *gene-environment interaction* (GxE) which refers to interaction between specific DNA sequences and specific measured environments. GxE might partly explain the "missing heritability problem"—the finding that specific genetic variants do not seem to account for much of the estimated heritability in psychological characteristics (i.e., the gap between the predictive and explanatory power of genes). A number of GxE studies, including several meta-analyses (Karg et al. 2011; Kim-Cohen et al. 2006; Risch 2009), indicate that individuals differ quite fundamentally in their response to the environment they are exposed to due to carrying specific genetic variants. Most of these studies examine associations between specific polymorphisms (e.g., *5-HTTLPR*) and risk of *negative* health outcomes (e.g., anxiety, depression, conduct disorder) given adverse life exposures (e.g., abuse, maltreatment, neglect). Few have examined more salubrious phenotypes and most GxE studies have been conducted within the diasthesis-stress perspective (Monroe and Simons 1991) which assumes that psychological outcomes (e.g., depression, low life satisfaction) result from an interaction between endogenous, pre-dispositional vulnerability factors (e.g., risk genes) and stress related to life experiences (e.g., maltreatment, financial difficulties). By contrast, the differential susceptibility framework (Belsky 1997; Belsky and Pluess 2009) predicts that some individuals—also for endogenous reasons—are disproportionally susceptible to both negative *and* positive environmental stimuli (i.e., being particularly malleable or plastic). Differential susceptibility thus pertains to situations in which given factors such as specific genetic polymorphisms that increase risk of adversity (e.g., depression, anxiety) given a harsh environment, contribute to a particularly positive development (e.g., optimism, wellbeing, self-esteem) given a positive, warm and supportive environment. More recently, the concept of *vantage sensitivity* (Manuck 2011; Pluess and Belsky 2013) has been used to refer to the "bright side" of differential susceptibility to indicate that some people for endogenous, genetic reasons benefit more than

others from positive life experiences. In support of the differential susceptibility perspective, some studies indicate that the short *5-HTTLPR* variant, which has been extensively studied in relation to anxiety and depression, not only confers heightened sensitivity to negative stimuli, but also to positive stimuli (Belsky and Pluess 2009; Belsky et al. 2009). This implies that carriers of the homozygous short alleles may benefit the most if exposed to a positive environment, including the lack of adversity. For example, one study examining differential susceptibility to parenting reported the association between supportive parenting and adolescents' positive affect to vary as a function of the youths' *5-HTTLPR* genotype (Hankin et al. 2011). Adolescents homozygous for the short variant of the *5-HTTLPR* were reported to exhibit lower levels of positive affect if experiencing unsupportive parenting, but higher levels of positive affect if experiencing positive and supportive parenting. The short variant of the *5-HTTLPR* may thus represent a *plasticity* allele rather than a vulnerability allele, coding for a general susceptibility to the environment in a "for better and worse" manner (Hankin et al. 2011).

Such GxE will commonly be incorporated, or concealed, in the standard variance components (i.e., A, C, E) in quantitative genetic designs. The same holds for a third type of interplay which is likely to be important to happiness and wellbeing, namely *gene-environment correlation* (rGE). Gene-environment correlation refers to genetic factors influencing exposure to a non-random sample of environments (i.e., the nature of nurture) and is known as *social selection* in the developmental literature and *reverse causation* or *confounding* in epidemiology. Commonly, rGE is classified as *passive, active*, and *evocative* (Scarr and Weinberg 1983). Passive rGE characterises situations in which individuals simply inherit both genes and environmental circumstances from their parents that reinforce each other—such as when children of emotionally stable, happy and optimistic parents inherit genes related to emotional stability as well as experience emotionally stable and supportive parenting (i.e., double advantage). Individuals are also active agents in selecting and shaping their surrounding environments (active rGE), and in turn, these environments reliably respond to their behaviour (evocative rGE), amplifying or strengthening genetically based dispositions. Children high in positive emotionality actively seek situations matching their partly genetic positivity disposition (active rGE) and are likely to elicit more supportive responses in parents and relevant others (evocative rGE). The processes of active and evocative rGE will be included in the heritability estimate due to reflecting heritable influences. Heritability thus reflects more than the direct genetic effects.

Although rGE has not been explicitly examined in most quantitative genetic studies on happiness-related constructs, Krueger et al. (2008) have shown that adolescents with a disposition to positive emotionality tend to elicit positive regard in their parents (evocative rGE). Likewise, Kandler et al. (2012) have found genetic influences to largely mediate associations between personality traits (i.e., Extraversion and Openness) and controllable positive life events. Caspi et al. (2005) have previously explored how individuals create, approach, or end up in circumstances that correlate with their stable disposition and shown that life experiences tend to reinforce the dispositions that initially pull the individual towards them (Caspi et al. 2005). Such niche building and co-responsive processes have rarely been explored for happiness-related characteristics within a quantitative genetic framework (Johnson and Krueger 2006; Krueger et al. 2008). However, Kandler et al. (2012) have shown that genetic factors play a major role in continuity and repetition of controllable positive events. Using non-twin data, Vaidya et al. (2002) have additionally shown that exposure to positive events might lead to an increase in Extraversion. Importantly, this latter study reported affective traits (e.g., positive affect) to be

consistently *less stable* than Big Five personality traits, and their stability was suggested to be *more influenced* by life events.

2.2 Environmental Influences

Environmental influences are clearly important to overall happiness and wellbeing, usually accounting for 50–70 % of the total variation—and even more when wellbeing is measured as a state more than a trait (Bartels 2015; Nes and Røysamb 2015; Menne-Lothmann et al. 2012). Quantitative genetic studies thus commonly show that *similarity* between family members—at least in adulthood, are attributable to shared genes—not to shared environments (i.e., C = 0). This finding accords well with findings from other strands of wellness research which have documented very modest influences from environmental influences that are typically shared such as demographic factors (Andrews and Withey 1976; Diener et al. 1999). The finding of negligible effects from shared environmental factors is not unique to quantitative genetic studies of happiness-related constructs. In fact, the finding is so common in quantitative genetics that Turkheimer (2000) coined it "the second law of behavioral genetics".[2] This "second law of behavioral genetics" has clearly posed a challenge to many practitioners and social scientists unfamiliar with quantitative genetics who commonly expect the effects of social forces (e.g., parenting style, sibship size, income, neighborhood factors) to be captured in the shared environmental component. It is therefore important to underscore that negligible influences from the shared environment do not imply that environmental factors (e.g., parenting, poverty, access to education) tend to be irrelevant to happiness and wellbeing. The finding indicates that environmental factors do not seem to operate on a family-by-family basis (e.g., parenting practices do not usually have general effects), but rather on an individual-by-individual basis (tend to affect the different siblings differently). The "second law of behavioral genetics" is perhaps less surprising when recalling that the shared environment (C) reflects environmental influences operating separately and independently from the genetic effects. Since happiness-related experiences probably mostly result from transactions between genetic and environmental factors, the effects will tend to appear in the variance components reflecting the genetic and unique environmental factors (Purcell 2002).

Interestingly, a few quantitative genetic studies—some of which are based on information from additional biological and social relationships (not only co-twins)—have evidenced shared environmental effects for happiness-related measures including SWB (Nes et al. 2010a, b), positive emotionality (Tellegen et al. 1988), positive affect (Baker et al. 1992), experimentally-induced positive moods (Riemann et al. 1998), and overall life satisfaction (Nes et al. 2008). For example, in a large study of SWB using data from nuclear families ($N = 54{,}540$) and twins ($N = 6620$), Nes et al. (2010a) examined several different types of shared environmental influences such as vertical cultural transmission (i.e., environmental influence from parents to off-spring), and different twin and sibling environments (e.g., environmental effects shared only by identical twins). No environmental transmission from parents to off-spring was indicated. However, a shared twin environment explained 8 % of the total environmental variation. So, twins were found to share more of the environment of importance to SWB than regular siblings and other first-degree relatives. As shown in Table 1, shared environmental influences also tend to affect momentary or short-term positive affect, with family resemblance often shown to be entirely due to context (i.e., shared environmental influences).

[2] The *first* law of behavioral genetics: all human behavioural traits are heritable.

Effects from shared environmental factors have also been indicated in designs assessing shared environments more directly. Examining shared environmental influences on familial aggregation of behaviour problems more directly, Caspi et al. (2000) reported a significant effect from deprived neighbourhood conditions on children's mental health above or beyond a genetic liability to behaviour problems. The risk of behaviour problems was thus partly *environmentally mediated*. To our knowledge, similar studies have not been undertaken for SWB.

2.3 Stability and Change

Numerous longitudinal studies have found SWB and LS levels to be fairly stable over time. Individuals who report being happy and satisfied at one time point thus often report being happy and satisfied also at later time points. Although cross-time correlations for SWB vary across samples and time-span, they rarely exceed 0.6, and rarely drop below 0.3 (Diener et al. 2013a). A given cross-time correlation of .50—which is a common finding in wellness research (Lucas and Donnellan 2007), implies that 50 % of the variance tends to reflect a stability factor with the remaining 50 % representing change or variation. SWB levels are therefore stable as well as variable. Findings from quantitative genetic studies indicate that the stability component is predominantly attributable to genes and suggest some kind of genetic "happiness baseline" from which our mood deviates in response to immediate circumstances and events. Such a mechanism seems practical and reasonable also from an evolutionary perspective. Human lives are commonly characterized by changes and unpredictable events that necessitate flexibility for optimal functioning. Those of our ancestors whose emotional experiences mapped more accurately to their situation clearly had a selective advantage (Nesse 2005). A baseline level of emotional functioning constitutes an important prerequisite for noticing and detecting important situational changes (e.g., threats, opportunities) required for such fitting responses to external demands. Lower fitness should therefore be associated with too rapid mood swings to the extremes as well as with entirely stable moods. In accordance with these expectations, recent experimental research indicates that too much positive (Gruber et al. 2013) as well as too much negative (Kashdan and Rottenberg 2010) emotion variability is often maladaptive, signaling psychological instability and psychopathology inconsistent with the concept of evolutionary fitness. Positive emotion thus seems to serve an adaptive function if it stays relatively stable over time.

Related to these findings, quantitative genetic studies indicate that SWB and stable personality traits (particularly the Big Five personality traits of neuroticism and extraversion) are closely genetically related (Hahn et al. 2013; Weiss et al. 2008). This may partly be due to personality and SWB reflecting common biological, activational, and neurochemical systems underpinning variation in motivation and emotional reactions—such as the *behavioural inhibition system* (BIS) that regulates withdrawal, caution, and anxiety in the presence of a threat and the *behavioural activation system* (BAS) that controls approach-related behaviour in the presence of reward (Steel et al. 2008). Reward anticipation commonly induces positive affect, and the BAS is thus closely associated with positive emotion. Receiving and savouring rewards are likewise linked with positive emotion, and neurochemical research indicates that at least two distinct biological reward systems are involved in positive emotional responses in the brain (Burgdorf and Panksepp 2006). One system (wanting, seeking system) is primarily involved in seeking rewards (appetitive, anticipatory behaviour), another system (liking system) is engaged in receiving reward and processing of sensory pleasures (e.g., hedonic tastes, pleasure, consummating).

These two systems seem to mediate reactions to constantly changing stimuli, but also to have relatively stable person-specific stimuli sensitivities manifested in personality traits.

Changes in happiness and wellbeing have been evidenced in a number of different research designs. Whereas positive affect tends to fluctuate in response to most environmental stimuli, SWB tends to change in response to formative events such as marriage and child birth (Dyrdal et al. 2011; Dyrdal and Lucas 2013; Luhmann et al. 2012), unemployment (Lucas et al. 2004), becoming disabled (Lucas 2007), or having a disabled child (Nes et al. 2014). Changes in SWB are also indicated in national comparison studies (Diener et al. 2013b; Veenhoven 2009), clinical psychology/psychotherapy research (Kessler et al. 2012), and intervention studies (Bolier et al. 2013; Sonja Lyubomirsky and Layous 2013; Seligman et al. 2005; Sheldon and Lyubomirsky 2007; Sin and Lyubomirsky 2009) including neuroplasticity studies (Tang et al. 2012; Hölzel et al. 2011; Davidson and McEwen 2012). The quantitative genetic studies that are published to date indicate that such changes commonly are due to environmental factors and may be relatively transient (e.g., Nes et al. 2006; 2013; Lykken and Tellegen 1996). Only about 20–25 % of the stability in SWB has been shown to reflect environmental sources (Lykken and Tellegen 1996; Nes et al. 2006, 2013), and environmental cross-time correlations have been estimated to 0.20–0.26 over a 6 year period (Nes et al. 2006). Environmental influences thus tend to phasically alter SWB levels, but not generally to accumulate much over time, and consequently to contribute only modestly to the observed stability. Similar findings are also indicated by different research methods outside quantitative genetics (Kahneman et al. 2004).

3 Implications for Intervention

What do these findings imply for individual and societal interventions aiming to increase happiness and wellbeing? A key point in understanding the quantitative genetic findings is that the estimated environmental variation (V_e) in standard quantitative genetic designs tends to include only naturally occurring environmental factors, and does not capture the effects of potential interventions unless a substantial proportion of the sample has been exposed. Adding a new intervention variance (V_i) to the existing V_e could theoretically reduce heritability and also increase wellbeing if a substantial proportion of the population was exposed. However, environmental interventions such as public health initiatives to create enriched or enhanced resources for all by eliminating environmental inequalities are perhaps more likely to reduce the total variation, increase heritability, and contribute to improved conditions for most. Investigating the impact on heritability for educational attainment after introduction of more liberal social and educational policies (i.e., more equal educational opportunities) in Norway after the Second World War, Heath et al. (1985) found that heritability for educational attainment increased in Norwegian males from 0.41 (born 1915–1939) to 0.67–0.74 (born 1940–1960). Better educational opportunities thus caused variation in educational attainment to depend more on innate abilities (i.e., higher heritability) as there were less environmental differences left to explain the total variation. Importantly, these findings did not imply that the intervention was ineffective in terms of improving educational opportunities in the population. High heritability might therefore partly reflect a welfare asset; the elimination of much environmental inequality. We are not aware of similar longitudinal studies examining SWB. However, Johnson and Krueger (2006) used a cross-sectional design to investigate "if money buys

happiness" and reported heritability to vary with socio-economy; to be higher among the more privileged and lower among the more disadvantaged. Thus, the heritability of SWB can differ, or change, in response to environmental conditions such as economy.

What about the change potential for individuals? As indicated, heritability refers to differences in populations and is not easily translated into individual effects. Developmental research on non-twin samples has shown that life span development may be characterised by individual differences in intra-individual change (Baltes and Nesselroade 1973), and inter-individual change trajectories have been reported for life satisfaction judgements (Mroczek and Spiro 2005). We do not yet know how hereditary factors may be involved in generating and sustaining such individual trajectories. More generally, however, we know that heritability varies across samples and subgroups, and that emotional flexibility and adaptation are important factors in human evolution. A notion of individual happiness-ranges is therefore likely and compatible with the quantitative findings reviewed.

To illustrate, consider a person who has not only one identical co-twin, but 99 'co-twins', all together constituting a group of 100 genetically identical individuals. Imagine also that these 100 people have an average genetic propensity for wellbeing (a potential set-point), thus having a zero score on a standardized latent genetic happiness factor. All differences between these one hundred 'twins' will necessarily be due to environmental differences. As 95 % of the 'twins' will be located within a range of ± 1.96 standard deviation, this can be seen as the *natural wellbeing range* for any individual, with a given genetic disposition. The happiness-potential of most individuals is thus potentially quite substantial.

To further exemplify the point of individual happiness ranges we simulated a situation of data showing a heritability of 40 %, thus reflecting the average heritability reported in a recent meta-analysis (Nes and Røysamb 2015). We created a large dataset ($N = 10{,}000$) and two standardized, normally distributed variables correlated at 0.40. This scenario corresponds to expected observations in a large real-life sample of MZ twins, given the typical finding of additive genetic and non-shared environmental effects accounting for the observed variance–covariance structure. Based on these data we calculated the absolute difference between the two variables to represent the intra-pair difference among MZ twins. The mean difference was 0.88 (*median* = 0.75) and 36.5 % of the sample had a difference larger than 1.00. In comparison, two standardized and uncorrelated random variables yield an average difference score of 1.12 (*median* = 0.95), representing the standardized expected difference between any *two random individuals*. These findings imply that in a population with heritability of 40 % (for wellbeing or any other phenotype), identical twins differ substantially, but are still more similar than random individuals. In the terminology of Cohens *d,* the mean difference between identical twins corresponds to a *large* effect size. Note that this simulation, and the reported results, is basically just an alternative way of representing the environmental variance of 60 %. Yet, we believe the findings are illustrative about the actual differences between genetically identical people, and thereby also the wellbeing range of most people. The simulation findings can be translated into scores on actual wellbeing instruments such as the Satisfaction With Life Scale (SWLS). The SWLS is one of the most widely used SWB scales worldwide, consists of five items and response options from 1 to 7, yielding a sum-score range of 5–35. In a review of the instrument, Pavot and Diener (1993) reported descriptive statistics for 36 different samples. The average mean value across samples was 21.9, and the average standard deviation was 6.6. Our simulation showed a mean MZ twin difference score of

0.88, which would translate into 5.8 (i.e., 0.88 × 6.6) points on the SWLS scale. Thus, MZ twins would on average differ almost six points on the well-established SWLS.

Turning now to matters of stability and change, the longitudinal twin studies of SWB have shown that approximately 80 % of the stable variance in dispositional happiness (i.e., stable variance) is attributable to genetic factors (Nes et al. 2006). Does this imply that environmental factors, such as happiness interventions, cannot have *lasting* effects? We believe the answer is no—for several reasons. First, as indicated above, the extant estimates are based on equations that do not include potential interventions—only naturally occurring activities and events. Second, even without specific interventions there are important within-pair differences over time. To illustrate, we conducted another simulation based on an MZ correlation of 0.80 consistent with the heritability of dispositional (stable) SWB. For two standardized random variables correlated at 0.80, the mean difference was 0.50 (*median* = 0.42). Despite high heritability, within-pair differences were still significant (i.e., moderate effect size) for the stable factor. As such, our data support the notion of environmental effects that are not only temporary, but possibly also long-term. It remains to be explored whether these factors include specific activities, life-choices, or life-events, as well as whether the stable influences from the environment represent long-term effects from past or persistent events, or more consistently occurring influences.

Overall, quantitative genetic findings thus indicate that happiness measured as overall SWB and LS is heritable, stable, variable and changeable. The findings support the notion of some kind of happiness baseline or "set-point". Yet, as human lives unfold, happiness naturally fluctuates—depending on external events and our own making of activities, relationships, and life-choices indicating that we are probably rarely at our "set-point" or baseline level. Indeed, should the "set-point" be operationalized as the mean value of lifetime SWB (Headey 2013), we will necessarily spend approximately half of the time above or below this mean level. Should the "set-point" be relatively fixed, in effect conceived as an intrinsic tendency (analogous to a disposition to body weight, cholesterol-level, or musical talent), there is no theoretical reason why we should not be able to increase the time spent above this fixed point, remain on the desired side of it, or indeed raise it as a platform. Happiness interventions are partly about nurturing the factors that contribute to moving up and remaining above a theoretical set-point, and growing evidence testify to their effectiveness (Bolier et al. 2013; Sin and Lyubomirsky 2009). For example, meditation techniques have been found to be associated with sustained wellbeing (Lutz et al. 2004) and neuronal growth in areas of the brain associated with positive emotion (Davidson and McEwen 2012). Even short-term interventions have indicated changes in both grey and white matter in the brain (e.g., Tang et al. 2012). Most complex human skills depend on long-term practice, however, and the majority of happiness interventions that are studied in the empirical literature do not involve the long-term persistent effort that is involved in acquisition of skills such as learning to play tennis or a musical instrument—or cultivating compassion, empathy and warm-heartedness as studied in long-term meditational practitioners. Most likely, changes in happiness dispositions or "set-points" are dependent, at least partly, on such persistent efforts. Interesting in this regard are findings suggesting that development of skills and growth of potentials often do not arise from enjoyment, but rather from commitment to the given activity (i.e., deliberate practice). In fact, the activities most relevant for persistent skill development are often rated as the least enjoyable, implicating that individuals need to be engaging in the activity and motivated to improve performance before they begin deliberate practice (Ericsson et al. 1993).

Epigenetic mechanisms are also relevant to individual change and have been suggested as a unifying principle in the aetiology of multi-factorial and complex traits. Epigenetic

mechanisms reflect long-lasting changes in gene expression that are not associated with changes in the DNA (nucleotide) sequence (Tsankova et al. 2007). Such functionally relevant changes may be induced by environmental factors (e.g., maternal behaviour, physical exercise), may even be reversible (Heard et al. 2010), and passed over generations (York et al. 2005). Epigenetic mechanisms illustrate the complex bidirectional relationship between genetic and environmental influences in development. We are aware of only one epigenome-wide association study exploring epigenetic mechanisms associated with wellbeing (i.e., LS). In this study, two autosomal sites reached genome-wide significance after bonferroni correction (Baselmans et al. 2015). Epigenetic reseach has thus entered the wellbeing field.

Since happiness and wellbeing reflect continuous interplay between genes and environments, it should be possible to alter genetically based "set-points" by altering activities and circumstances. For example, a person with a highly creative disposition (e.g., writing, musical talent) might alter her "set-point" by creating a life conducive to expressing her potential. In this sense, we might have multiple potential "set-points" depending on the life-situations and activities we create or are exposed to. This way of conceptualizing "set-points" fits well with findings suggesting that life-changes like losing one's job may alter individual happiness "set-points" (Lucas 2007; Lucas et al. 2004). The notion of multiple potential "set-points" fits similarly well with findings of national differences and national changes in wellbeing following environmental improvements.

The notion of *positive gene-environment interplay* or *gene-environment matchmaking* (Røysamb et al. 2014) invites us to use these findings actively by creating happiness-enhancing interventions, policies, activities, and environments permitting flourishing of individual genetic potentials that simultaneously buffer against vulnerability and risk. The processes involved are implicitly present in the recent focus on personalized medicine (Gordon 2007) and treatment-matching (Gastfriend and McLellan 1997)—and incorporated in a number of happiness enhancing strategies. One example being to "use signature strengths in a new way" (Seligman et al. 2005) which involve identifying individual top character strengths and exploring new ways to use these particular strengths. Given that character strengths are partly heritable (Steger et al. 2007), such interventions take into account that identifying personal characteristics like genetic potentials might predict differential effectiveness and yield more effective results. At a more general level, *person-activity fit* has been proposed as the key moderator of the effects from positivity enhancing activities (Lyubomirsky and Layous 2013; Sheldon and Lyubomirsky 2007). Optimal matchmaking may include both amplifying and compensatory processes, with amplifying referring to active creation of positive gene-environment correlations in which activities provide opportunities to express particular (partly genetic) talents and potentials. Compensatory processes might include identification and acceptance of weaknesses, barriers, and vulnerabilities (e.g., anxieties, addictions, lack of impulse-control) that limit behavioral flexibility or impede optimal development—followed by activities that build competencies or create circumstances to balance or mitigate risks, or channel them into more functional pathways. Optimal matchmaking thus focusses on both the internal and external factors important for affecting change, and both amplifying and compensatory processes (i.e., interactionism). Research outside behaviour genetics has also shown intrinsically motivating strengths to be particularly useful in promoting wellbeing and indicate that a balanced approach that simultaneously target the most and the least developed strengths (i.e., amplifying and compensatory interventions) might be more effective (Young et al. 2015). Our key point is that gene-environment interplay occurs naturally, that some variants of such interplay are highly beneficial for wellbeing, and that happiness intervention studies could benefit from explicitly addressing genetic and environmental matchmaking.

Gene-environment matchmaking pertains to happiness-raising or strength-building related to genetically influenced, individual talents and vulnerabilities. Interventions targeting individual and population wellbeing might also be effective regardless of genetic variation-albeit with effects differing across individuals. Environmental improvements such as universal access to education, health services, secure employment, safety, green spaces, and low corruption are highly likely to increase mean values of wellbeing and happiness in populations. Nations or communities with high wellbeing scores may be seen as doing well at creating environments that allow for optimal "set-points", or afford *default contentment* which is suggested as a natural setting for most individuals in the absence of adverse conditions (Grinde 2016). Interventions that permit environmental improvement and individual effort (e.g., skills learning) to mutually fashion each other might be very particularly valuable. Training contributes to development of skills across both genetic variation and the age span. As skills tend to beget skills, and positive adaptation at one developmental stage tends to provide a foundation for successful encounters also at subsequent stages (Heckman 2006), childhood might be the optimal time to build personal resources and counteract vulnerability. In line with findings from quantitative and molecular genetics, including differential susceptibility and vantage sensitivity, this suggests that primary intervention efforts such as promotion of wellbeing capacities and skills (e.g., emotion differentiation, self-regulatory abilities, conscientiousness) in children and parents might constitute a vital strategy for sustained individual and population wellbeing, particularly if drawing upon information on genetic and environmental barriers and strengths.

Genetically informative designs are likely to assist us in developing more effective interventions regardless of whether they are individual therapies or universal interventions (e.g., education and social policy changes). However, knowledge about how to overcome genetic risk, and about the importance of genetic factors in explaining individual differences in response to treatment and intervention (i.e., who will benefit the most from what environmental advantages), is still limited. Genetically sensitive studies have shown that momentary positive affect and reward experiences may thrive in all irrespective of genetic variation (Menne-Lothmann et al. 2012). Yet, happiness understood as immediate positive feeling states, tends to be of a transient kind unless manifested in personality traits and we know relatively little about how, and for whom, such experiences may accumulate and contribute to sustained wellbeing. As indicated by genetically sensitive studies, people are active agents in selecting and shaping their surrounding environments (i.e., gene environment correlation) due to stable, partly genetic propensities and differ in their sensitivity to the environment and the responses they elicit. To date we have very limited understanding of how to break down adverse gene-environment interplay and frame favourable interplay—in individuals and different segments of the population. Our understanding of the long-term consequences of such interventions—of what is favourable for whom in the long run, is also very limited. We believe, however, that interventions that target causal pathways and focus on developing strengths as well as mitigating risks, at the individual and societal level—providing for positive gene-environment matchmaking—are likely to be useful and their impact longer lasting.

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