Twin Research for Everyone

From Biology to Health, Epigenetics, and Psychology

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CHAPTER

18

Happiness and well-being: The value and findings from genetic studies

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18.1 What is well-being?

Within the past decade, there has been increasing interest in well-being (WB) as a research topic across different disciplines, including the field of behavior genetics. Moreover, there is a growing global recognition of WB as an important public policy goal, as shown through population-based surveys initiated by governments with the aim of systematic consideration of WB to inform decisions.[1–3] In this chapter, we discuss the relevance of behavioral genetic (twin-)studies to increase our understanding of individual differences in WB.

The term "well-being" embodies a multitude of concepts with varying meanings depending on context and discipline. Here, we focus on the meaning of WB as employed in psychology and social sciences. It is important, though, to first briefly mention its philosophical origin. Two ancient philosophical traditions are relevant in this context: hedonism and eudaimonism.[4] The hedonist tradition dates back several centuries before Christ, to philosophers such as the Cyrenaics who believed that pleasure was the highest good, and central to happiness or WB.[5] Thus, in the ancient hedonist definition, WB or happiness is equated to the sum of one's pains and pleasures. Eudaimonism, on the other hand, has a definition that is quite different from the hedonist perspective. Influenced by Aristotle's virtue ethics, the eudaimonic view on happiness centers around living a virtuous life.[6] From this point of view, the greatest fulfillment in life will come with the realization of one's potential and finding meaning in life. These descriptions provide only a brief overview of the two philosophies, but they do illustrate the appreciable distinction that exists between their definitions of WB.

In the current psychological literature, a distinction is often made between "subjective" well-being (SWB) and "psychological" well-being (PWB). This distinction can be traced back to the ancient distinction between hedonic and eudaimonic WB,

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with SWB following from the hedonic tradition and PWB from the eudaimonic tradition. While different definitions exist, SWB is mostly characterized by high levels of positive affect, and low levels of negative affect, translating into a subjective evaluation of high satisfaction with life.[7] While life satisfaction reflects a more cognitive evaluation of WB that is not necessarily in line with hedonist ideas about happiness, the positive and negative affect dimensions of SWB are highly similar to the hedonist ideas about balancing pleasure and pains. Similar to how the eudaimonic definition was formulated as a response to the hedonic definition, the PWB definition was formulated as a response to the SWB definition. A critique of the SWB definition is that it does not capture important aspects of positive psychological functioning, such as self-fulfillment.[8] Therefore, PWB definitions of WB aim to include broader domains of positive functioning. For example, in Carol Ryff's definition of PWB, included domains are self-acceptance, positive relations with others, autonomy, environmental mastery, purpose in life, and personal growth.[8]

Theoretically, the distinction between SWB and PWB is clear. Empirically, however, the distinction is less clear-cut. While most research finds that WB is comprised of multiple related, yet conceptually distinct underlying dimensions,[9,10] discussion remains concerning the extent to which these underlying factors are correlated. Moreover, results indicate that there is a large overlap in the set of genes that influence SWB and PWB, with a higher genetic correlation than phenotypic correlation.[11,12] Additionally, many different measurement instruments are available to assess (different aspects of) WB.[13,14] This further complicates our interpretation of the interrelatedness of different WB constructs as these different measurement instruments might introduce additional variance.

Twin studies help us understand WB in multiple ways. First and foremost, by partitioning the variance of WB into genetic and environmental sources of variation, twin studies enable us to interpret the causes of individual differences in WB (Sections 18.2 and 18.3). Second, by examining the genetic and environmental sources of variation in phenotypes highly related to WB, we come one step closer to understanding the complexities of the WB construct (Section 18.4). The knowledge gained from existing twin studies of WB has fueled follow-up in-depth analyses in both genetic and environmental directions (Section 18.5), which again have led to the development of novel, more complex twin designs (Section 18.6). In what follows, we present these past, present, and future directions of research, demonstrating the transformational effect this research has had on our understanding of WB.

18.2 Earlier reviews on twin studies on well-being

In 2015, two comprehensive reviews on the causes of individual differences in WB were published.[15,16] Results of these twin-family studies into the genetic and environmental influences on WB revealed a range of heritability estimates, but when meta-analyses were used to estimate heritability across the studies the meta-analytic results converged on the heritability estimate. In the book chapter of Nes and

Roysamb, the weighted average heritability, across 13 independent studies including more than 30,000 twins (aged 12–88) from seven different countries, was estimated to be 40% (CI: 37%–42%).[16] Similarly, in the paper by Bartels, the weighted average heritability of WB, based on a sample size of 55,974 individuals, was 36% (34%–38%), while the weighted average heritability for satisfaction with life was 32% (29%–35%) (n = 47,750).[15]

These similar results, with an overlapping confidence interval, provide a more robust estimate of the genetic influence on WB. Both reviews and meta-analyses showed that both genetic and environmental influences are important for variation in WB. The meta-analyses indicate that genetic influences on WB are mainly additive and that the environmental influences appear to be nonshared.

18.3 New findings of twin studies on well-being

Since 2015, the twin design has been used in an additional 15 studies to investigate the heritability of WB using different measures of WB, and in combination with other variables, such as depression or social support, as described later. Fig. 18.1A and B summarizes the heritability estimates of all included twin studies in the earlier meta-analyses, and of the recent twin studies on WB. In addition, Table 18.1 summarizes the designs and findings of the recent twin studies of WB. The heritability estimates of the recent studies on WB vary somewhat (range: 0.27–0.67), but are mostly in line with the previous meta-analytic estimates. The effect of a shared environment is small but significant in a few studies in younger participants. In contrast to earlier studies, none of the recent studies reported evidence for nonadditive genetic effects.

Besides investigating the heritability of WB, many recent studies used the bivariate or multivariate approach to investigate the (genetic and environmental) covariance between WB and other variables. For example, Haworth and colleagues[17] reported moderate genetic correlations with depressive symptoms, Wang and colleagues[18] with social support, Wootton and colleagues[19] with positive and negative life events, and Luo et al.[20] reported a moderate genetic correlation with self-enhancement. Van t' Ent and colleagues[21] reported nonsignificant genetic correlations with subcortical brain volumes. In a small Polish twin sample, Milovanović et al.[22] and Sadiković et al.[23] investigated the covariance of life satisfaction with emotion regulation and personality traits. The genetic correlation with various forms of emotion regulation varied between 0.53–0.86. The genetic correlation between WB and personality traits varied from 0 (openness and agreeableness) to 0.61–0.71 (conscientiousness, extraversion, and neuroticism). The heritability of life satisfaction in relation to personality traits has also been investigated by Røysamb and colleagues in a larger sample.[24] The heritability of life satisfaction was estimated at 31% (22%–40%), of which 65% was explained by personality-related genetic influences (mainly neuroticism and extraversion). The remaining genetic variance was unique to life satisfaction.

Thege and colleagues[25] investigated genetic and environmental influences on happiness, life satisfaction, and general WB in a small Hungarian twin sample. The

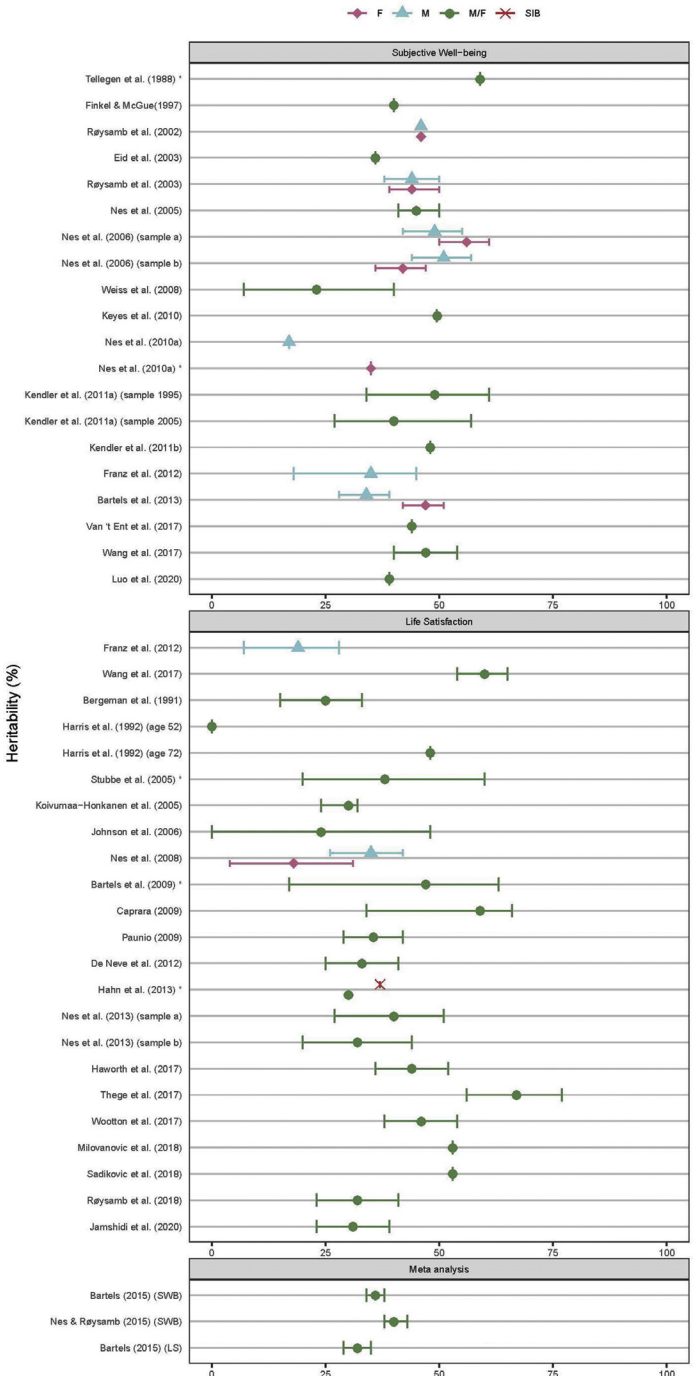

FIG. 18.1 (*Continued*)

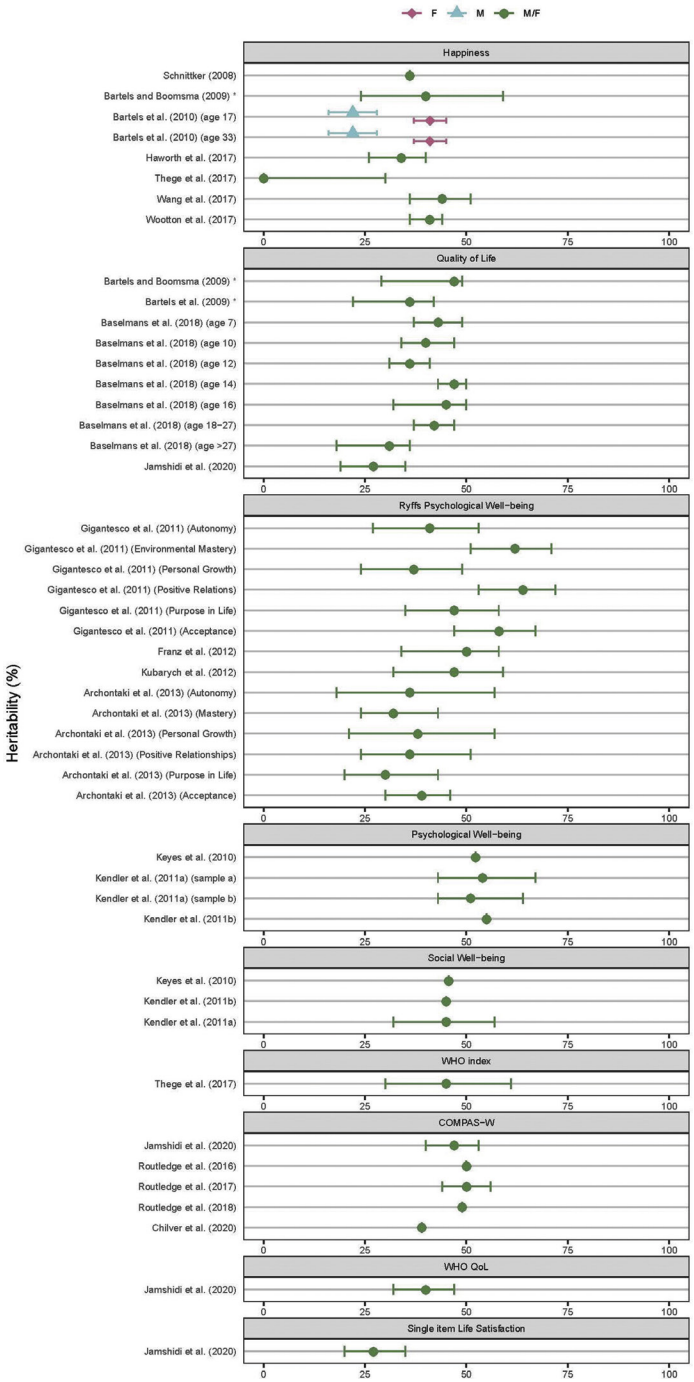

FIG. 18.1

(A) Heritability estimates for well-being. F = females, M = males, F/M = males and females, S = siblings. (B) Heritability estimates for well-being. F = females, M = males, F/M = males and females, S = siblings.

TABLE 18.1 Summary on twin studies for well-being since 2015.

References	Measure	Age	Sex	*N* twin pairs (MZ/DZ)	rMZ	rDZ	A (95 %CI)	C (95 %CI)	E (95 %CI)
Haworth et al.[17]	LS	16.32 (0.68)	M	1346 (693/653)	0.52	0.35	0.44 (0.36–0.52)	0.11 (0.05–0.18)	0.45 (0.42–0.48)
			F	1868 (989/879)	0.60	0.39			
			OS	1480		0.29			
	SHS		M	1347 (691/656)	0.43	0.19	0.34 (0.26–0.40)	0.06 (0.02–0.12)	0.60 (0.57–0.64)
			F	1867 (990/877)	0.41	0.30			
Thege et al. [25]	LS	43.27 (16.3)	M	37 (28/9)	0.67	0.46	0.67 (0.56–0.77)		0.33 (0.23–0.44)
			F	99 (72/27)					
	SHS				0.38	0.39	0.00 (0–0.30)	0.38 (0.10–0.61)	0.62 (0.45–0.82)
	WHO index				0.46	0.15	0.45 (0.30–0.61)		0.55 (0.39–0.70)
Van 't Ent et al.[21	Composite (SAT + HAP)	27.0 (4.1)	M/F	294 (171/123)	0.44	0.16	0.44		0.56
			Single	57 (32/25)					
			Siblings	87					
Wang et al.[18]	Positive Affect	17.85 (0.77)	M/F/OS	1133 (354/779)	0.49	0.19	0.47 (0.40–0.54)		0.53 (0.46–0.60)
	SHS				0.47	0.16	0.44 (0.36–0.51)		0.56 (0.49–0.64)

References	Measure	Age	Sex	*N* twin pairs (MZ/DZ)	rMZ	rDZ	A (95 %CI)	C (95 %CI)	E (95 %CI)
	LS				0.57	0.34	0.60 (0.54–0.65)		0.40 (0.35–0.46)
Wootton et al.[19]	LS	16.32 (0.68)	M	2200 (1142/1058)			0.46 (0.38–0.54)	0.10 (0.04–0.16)	0.44 (0.42–0.47)
			F	3058 (1640/1418)					
			OS	2370					
	SHS						0.41 (0.36–44)		0.59 (0.56–0.62)
Baselmans et al.[11]	Quality of Life		M/F/OS	42427 twins (16089/26338)					
		7			0.85	0.66	0.43 (0.37–0.49)	0.43 (0.37–0.49)	0.13 (0.12–0.16)
		10			0.79	0.62	0.40 (0.34–0.47)	0.41 (0.35–0.46)	0.20 (0.17–0.21)
		12			0.83	0.63	0.36 (0.31–0.41)	0.46 (0.41–0.50)	0.18 (0.17–0.20)
		14			0.46	0.25	0.47 (0.43–0.50)		0.53 (0.50–0.57)
		16			0.47	0.21	0.45 (0.32–0.50)		0.55 (0.51–0.59)
		18-27			0.42	0.16	0.42 (0.37–0.47)		0.58 (0.53–0.63)

(Continued)

TABLE 18.1 *Cont'd*

References	Measure	Age	Sex	*N* twin pairs (MZ/DZ)	rMZ	rDZ	A (95 %CI)	C (95 %CI)	E (95 %CI)
		>27			0.30	0.11	0.31 (0.18–0.36)		0.69 (0.64-0.75)
Reference	Measure	Age	Sex		rMZ	rDZ	A (95 %CI)	C (95 %CI)	E (95 %CI)
Milovanović et al.[22]/	LS	24.59 (7.11)	M	32 (23/9)	0.54	0.42	0.53		0.47
Sadiković et al.[23]			F	122 (98/24)					
			OS	28					
Røysamb et al.[24]	LS	57.11 (4.5)	M	537 (290/247)	0.35	0.05	0.32 (0.23–0.41)		0.68
			F	979 (456/523)	0.29	0.17			
Luo et al.[20]	Latent (LS + Affective WB)	18.29 (1.96)	M/F OS	492 (304/188) 116			0.39	0.12	0.49
Routledge et al.[27]	COMPAS-W	39.8 (12.7)	M	294 (202/92)			0.50		0.50
			F	449 (246/203)					
			OS	1483		0.16			
Routledge et al.[28]	COMPAS-W	39.77 (12.77)	M	598 twins (410/188)	0.51	0.22	0.50 (0.44–0.56)		0.50 (0.44–0.56)
			F	904 twins (496/408)					
Routledge et al.[29]	COMPAS-W	39.65 (12.73)	M	676 twins (472/204)			0.49		0.49
			F	979 twins (567/412)					

References	Measure	Age	Sex	*N* twin pairs (MZ/DZ)	rMZ	rDZ	A (95 %CI)	C (95 %CI)	E (95 %CI)
Chilver et al.[30]	COMPAS-W	40.0 (13.0)	M/F	422 (292/130)	0.43	0.08	0.39		0.61
Jamshidi et al.[31]	COMPAS-W LS WHO (psych QoL) Single item LS Single item QoL	39.64 (12.73)	M/F Single	1660 (968/334) 158			0.47 (0.40–0.53) 0.31 (0.23–0.39) 0.40 (0.32–0.47) 0.27 (0.20–0.35) 0.27 (0.19–0.35)		0.53 (0.47–0.60) 0.69 (0.61–0.77) 0.60 (0.53–0.68) 0.73 (0.65–0.80) 0.73 (0.65–0.81)
Well-being intervention	Composite (SHS + LS)	16.55(0.51)	M	158 (67/91)	0.55	0.32	Before: 0.48 (0.20–0.64)	0.07 (0.0–0.30)	0.44 (0.36–0.55)
Haworth et al.[32]			F	217 (100/117)	0.50	0.25	During: 0.45 (0.19–0.60)	0.06 (0.0–0.26)	0.49 (0.39–0.60)
					0.53	0.20	Follow-up: 0.48 (0.26–0.60)	0.03 (0.0–0.20)	0.49 (0.40–0.60)

SHS, subjective happiness scale; LS, life satisfaction; F, Females; M, Males; OS, opposite sex pairs.

results indicate a heritability of life satisfaction and general WB of 67% and 45% with no shared environmental effects. Happiness had a negligible heritability (0%), whereas 38% of the variance was explained by the shared environment. Due to the small sample, these results should be interpreted with caution.

A recent study in a Dutch twin sample[26] investigated the contribution of genetic and environmental factors on WB and depression across the lifespan. Genetic factors explained a substantial part of the phenotypic variance in WB during childhood, adolescence, and adulthood (range 31%–47%). In the younger samples, shared environmental influences explained a large part of the variation, but these disappeared with age. Regarding the association between WB and depression, the contribution of genetic factors increased from childhood to adolescence, meaning that environmental factors are important in explaining the relationship between WB and depressive symptoms in childhood, while in adolescence genetic factors play a larger role.

Whereas most recent studies used the most popular WB measures (e.g., the Satisfaction with Life Scale, Subjective Happiness Scale, or Cantril ladder), Routledge and colleagues[27–29] designed the COMPAS-W scale. The COMPAS-W scale is a composite index of subjective (hedonic) and psychological (eudaimonic) WB. The heritability of WB measured using this scale was estimated at 50%. Additionally, about half of the genetic influences on WB were shared with symptoms of depression and anxiety. Furthermore, Chilver et al. reported a small genetic association between WB and brain activation, as reflected by electroencephalography (EEG) power.[30] Recently, Jamshidi and colleagues compared the heritability estimates of the COMPWAS-W scale, Satisfaction with Life scale, and single-item measures of life satisfaction and quality of life.[31] Heritability estimates ranged from 23% to 47%, with the heritability of single-item questions being lower than multiple-item scales.

Lastly, Haworth and colleagues[32] investigated the effect of a WB intervention on the genetic and environmental variance components. The intervention lasted 10-weeks and consisted of online kindness and gratitude tasks. WB improved during the intervention and was significantly higher at follow-up. The contribution of genetic influences to the phenotypic variance remained consistent before, during, and after the intervention (respectively, 48%, 45%, and 48%). The contribution of nonshared environmental influences also remained constant, but new nonshared environmental influences emerged over time in response to the intervention. Thus, genetic influences stayed largely the same, whereas new environmental influences explained the changes in WB in response to the intervention.

To summarize, although the studies in the previous meta-analyses and the 15 newer studies use different types of contexts, WB measures, and sample sizes, the results seem to converge on a heritability estimate of about 40 to 50%.

18.4 Related phenotypes

As described in the previous sections, WB is not a unitary construct. Besides the multidimensionality of the construct itself in terms of its definition, there are also many phenotypes that are closely related to WB. We can identify different classes

of these related phenotypes: unfavorable outcomes that are negatively related to WB (e.g., depressive symptoms[26]), related but clearly distinct traits such as personality characteristics,[12] and highly related phenotypes that are sometimes difficult to conceptually separate from WB. In this section, we focus on the insights that twin studies have brought us for this last class of phenotypes. Specifically, we focus on optimism, meaning in life, self-esteem, and resilience.

18.4.1 Optimism

Optimism can be defined as the general expectation of positive versus negative outcomes in different domains of life and is often measured using the Life Orientation Test (LOT) or LOT-revised (LOT-R).[33] In the context of WB, optimism is related to lower negative emotions and higher, positive affect, and life satisfaction.[34–36] A large meta-analysis estimated phenotypic correlations of around 0.50 between optimism and the different aspects of WB.[37]

Fig. 18.2A provides an overview of the heritability estimates reported for optimism from the existing literature. All studies in this figure used the 6-item LOT-R to measure optimism,[19,38–43] with the exception of Plomin et al.[44] and Yuh et al.,[45] in which the 4-item LOT was used, and Mavioğlu et al.[38] and Whitfield et al.[46] whom used the 3-item LOT-R. Plomin et al.[44] were the first to study the causes of individual differences in optimism. Using a twin/adoption design, a heritability of 23% was reported for LOT-measured optimism, with the remaining 77% of the variance being accounted for by nonshared environmental factors. As depicted in Fig. 18.2A, the heritability estimates from later studies do not differ substantially across different studies, even though there is much variability in the confidence intervals.

18.4.2 Meaning in life

The meaning in life construct, like WB, knows many different operationalizations. One popular view on meaning in life is that it is a tripartite structure, consisting of three distinct subdomains: comprehension (one's life making sense), purpose (a sense of direction in life), and mattering (a sense of life having inherent value).[47,48] The relationship between WB and meaning in life is complex. While meaning in life can be viewed as an important *part of* eudaimonic/psychological WB,[8] it might also be interpreted as a route to or consequence of WB.[49,50] A correlation of around 0.50 has been reported between meaning in life and WB, i.e. life satisfaction or psychological WB.[51–53]

There have only been a few twin studies so far focusing on meaning in life (see Fig. 18.2A). As is evident from Fig. 18.2A, most studies find heritability estimates that are medium in effect, ranging from 33% to 52%.[18,19,54] Yet, Thege and colleagues report a heritability of 0% in their analysis of meaning life. However, given that the sample in this study was smaller than the previously described studies, these findings should be interpreted with caution.[41]

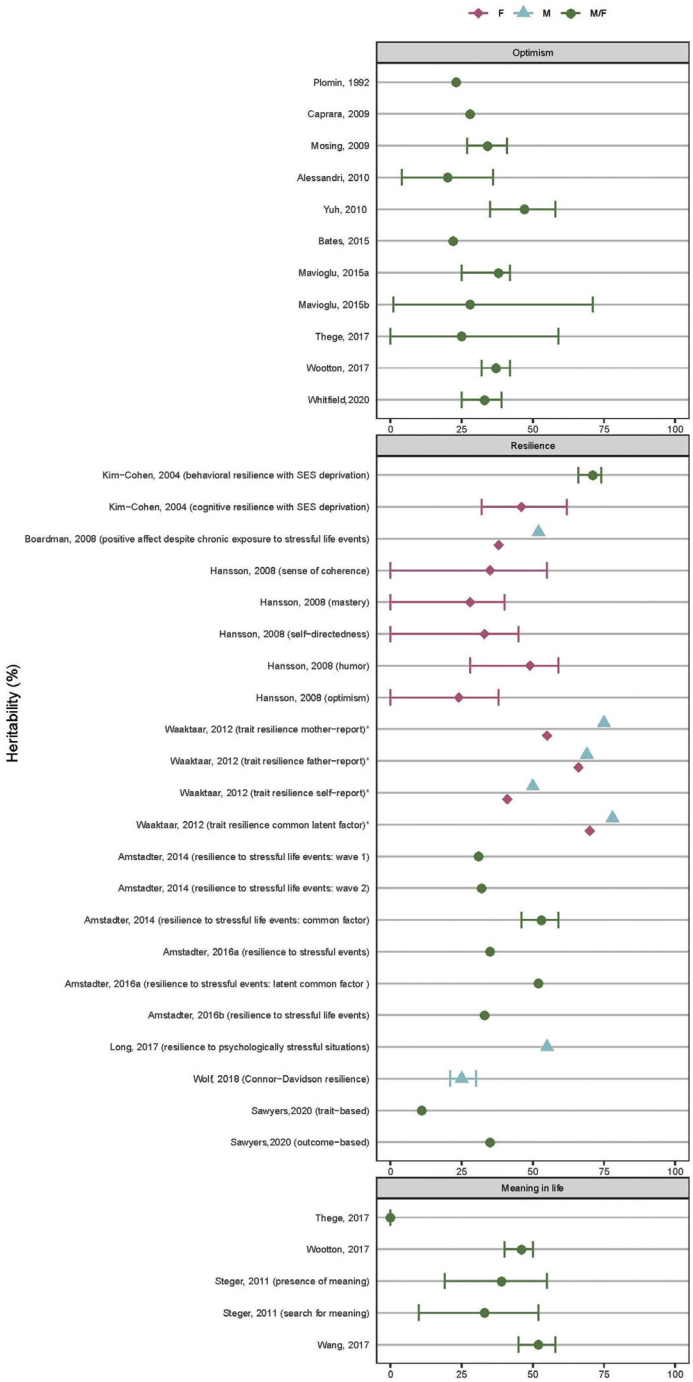

FIG. 18.2 *(Continued)*

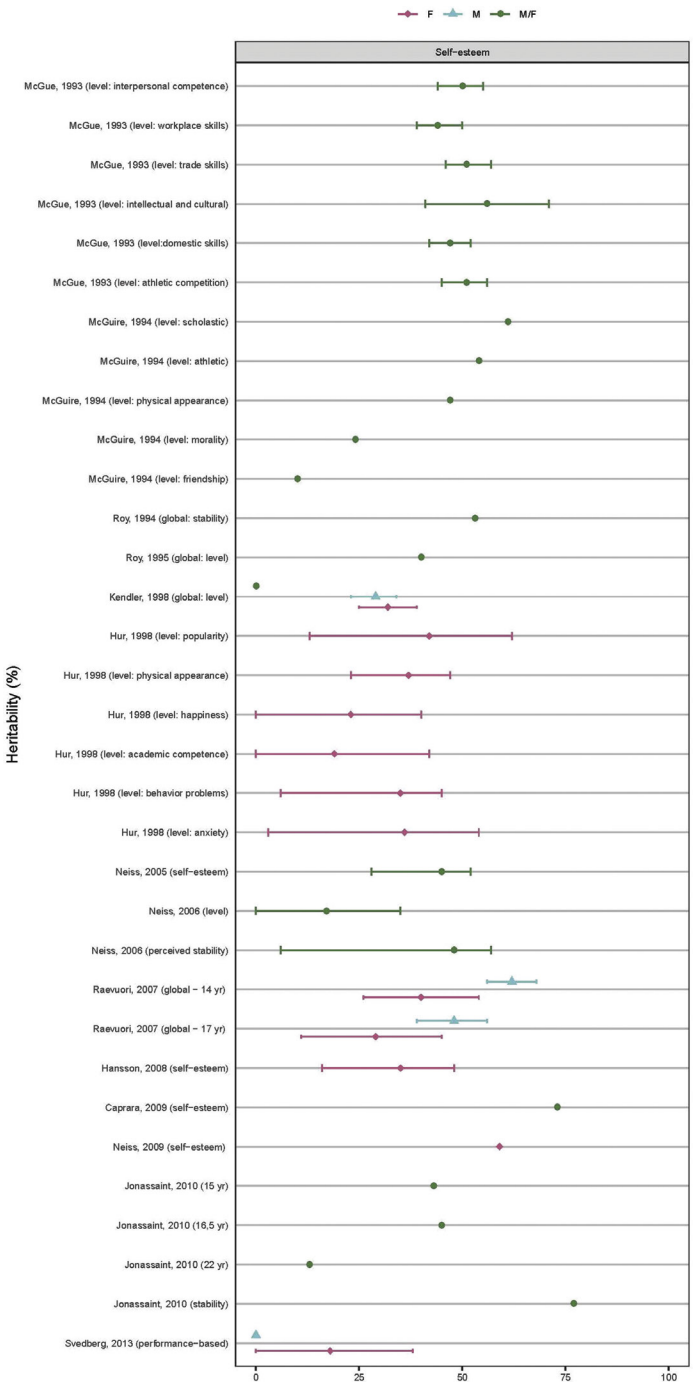

FIG. 18.2

(A) Heritability estimates reported for well-being related phenotypes. F = females, M = males, M/F = males and females. (B) Heritability estimates reported for well-being related phenotypes. F = females, M = males, M/F = males and females.

18.4.3 Self-esteem

The scientific study of self-esteem is one that has produced an abundance of literature. Often measured using the Rosenberg self-esteem scale,[55] self-esteem can be defined as one's affective or evaluative appraisal of the self, or the extent to which a person (dis)likes him- or herself.[56] Two components are central in the assessment of self-esteem: the level (i.e., the general appraisal of yourself), and the stability over time of this appraisal.[57] Moreover, we can interpret an individual's general self-esteem, but within a person self-esteem can also vary across different domains (e.g., intellectual, cultural). The correlation with WB is strong, as estimates of 0.50 and higher were reported.[58,59]

In a literature review performed in 2002, the results from behavioral genetic studies on self-esteem thus far were summarized.[57] For the review, results were split up for the level and stability of self-esteem, and within these categories, for general and domain-specific self-esteem. For the level of self-esteem, the results from different studies did not always converge. Nevertheless, overall it seems that about 30%–40% of individual differences in self-esteem level can be explained by genetic factors and that the remaining variation is accounted for by unique (but not shared) environmental factors. For domain-specific self-esteem levels similar results were reported, with heritability estimates around 50%, and a small or no role for shared environmental influences, both in childhood and adulthood. Yet, depending on which domain was studied, there is quite some variation in the estimates. For example, McGuire et al.[60] examined the level of self-esteem in the following five domains: scholastic competence, athletic competence, physical appearance, morality, and friendship. While the heritability of self-esteem in the scholastic domain was estimated at 61%, the heritability for self-esteem in friendship was substantially lower, 10% (also depicted in Fig. 18.2B). While there was less literature available for self-esteem stability, the heritability of stability in self-esteem seems to be similar to or even higher than the heritability at one time-point, with heritability estimates a little over 50%. This was true for both global self-esteem and domain-specific self-esteem.

Since this meta-analysis, many other twin studies on self-esteem have been published (see Fig. 18.2B). As can be seen in Fig. 18.2B, the heritability estimates varied considerably. However, this is likely due to the different definitions used for self-esteem, and the different age groups examined. For example, Jonassaint reported that in early adulthood, self-esteem is almost completely determined by the unique environment, with no role for genetic factors.[61] Raevuori and colleagues looked at genetic and environmental factors affecting self-esteem in boys and girls from age 14 to 17.[62] Their results show that the heritability of self-esteem is higher in boys than in girls in this age group.

18.4.4 Resilience

Psychological resilience can be defined as an individual's ability to recover after the experience of stress or trauma, returning to an optimal mental state, or as the

psychological outcome after adverse events.[63] Resilience and WB have been associated with many studies with a phenotypic correlation of around 0.50, and especially strong links between resilience and the cognitive and affective components of WB have been reported.[64] Resilience has been studied in different twin studies (Fig. 18.2A), but again with varying definitions of the construct. For example, Kim-Cohen and colleagues investigated individual differences in behavioral and cognitive resilience of children after economic deprivation (defined as lower antisocial behavior and higher IQ than predicted), and reported heritability estimates of 71% and 46%, respectively.[65] Hansson and colleagues performed analyses on specific resilience concepts (sense of coherence, mastery, self-directedness, self-worth, humor, and optimism), and estimated a moderate heritability of around 33%. Analyses of scales aimed at measuring psychological resilience reveal an even greater range of variation in heritability estimates. For example, using the Connor-Davidson Resilience Scale,[66] Wolf and colleagues estimated the explained variance of additive genetic effects and shared environment in military male twins at 25% and 15%, respectively.[67] However, in adolescents, the heritability of a latent resilience factor was estimated at 78% and 70% in boys and girls, using the Ego-Resilience scale.[68,69]

Alternatively, studies may use an outcome-based measure of resilience instead of questionnaires aimed at measuring resilience directly (trait-based).[70] For example, in a first study, resilience was defined as the residual of positive affect after controlling for stressors.[71] The heritability estimates were higher in men (52%) than in women (38%). In addition, Amstadter and colleagues defined resilience as the residual of internalizing symptoms after controlling for the number of stressful life events.[72] At two-time points, the heritability was stable, around 31%, with no sex differences. Sawyers et al. (2020) compared the etiology of trait-based and outcome-based resilience.[73] Only 15% of the heritability of outcome-based resilience was shared with trait-based resilience. In summary, variation in the definition of resilience (and in the sample) leads to a lot of variation in heritability estimates, demonstrating the need for a universal or commonly agreed-upon definition for resilience.

18.4.5 Multivariate models of positive psychological traits

Multivariate twin designs can answer the question how much of the phenotypic correlation between traits is accounted for by genetic and environmental factors. In addition, the overlap in genetic and environmental factors underlying multiple traits can be assessed. In other words, these designs help us understand why traits are related or tend to co-occur.

For example, a study by Caprara and colleagues[40] assessed the associations between self-esteem, optimism, and WB (in terms of life satisfaction). The analyses indicated a large overlap in genetic causes, with genetic correlations between 0.80 and 0.87. Likewise, Wootton and colleagues[19] investigated whether positive life events were genetically associated with SWB and related positive psychological traits including subjective happiness, life satisfaction, optimism, hopefulness, and gratitude measured at the age of 16. The WB traits were positively genetically correlated with

positive life events, and negatively with negative life events. However, these genetic correlations were moderate, ranging approximately from −0.5 to 0.5.

While the above studies are just two examples of studies applying multivariate models to WB and related traits, these types of investigations are becoming more frequent, and are fueling follow-up genetic molecular studies. In the next section, it will be shown how studies like these help with the design of so-called "multivariate genome-wide association meta-analysis,"[74] where the genetic overlap between related traits is used to increase power for genetic analyses.

To summarize, positive psychological traits, such as optimism, meaning in life, self-esteem, and resilience are related to WB with phenotypic correlations of around 0.50. Although the estimated heritability is tied to the specific construct, definition, sample, context, and methods used, around one-third of the variance in the related phenotypes can be explained by genetic factors, similar to what has been reported for WB. In addition, multivariate twin models show strong genetic correlations between WB and other positive psychological traits. These findings help us to further understand the complex nature of WB.

18.5 Specific molecular genetic and environmental influences

The introduction of this chapter already briefly mentioned that twin studies on WB fueled more in-depth analyses of genetic and environmental effects. To help frame the importance of findings from twin and family studies, it is useful to view them in conjunction with findings that probe the role genetic and environmental factors using other methods. Behavioral genetic studies have revealed that a substantial part (∼40%) of the variation in WB can be attributed to genetic influences and an obvious next step is to try to identify genomic regions associated with WB.

The first reliable molecular evidence for the genetic complexity of WB came from a method called GCTA (genome-wide complex trait analysis), where the proportion of phenotypic variance explained by all genome-wide SNPs (single nucleotide polymorphisms—DNA sequence variation of a single nucleotide) is estimated by comparing the phenotypic and genetic similarity across a group of unrelated individuals.[75] In a pooled sample of ∼11.500 unrelated genotyped Swedish and Dutch participants, WB was measured using the positive affect subscale of the Center for Epidemiology Studies Depression Scale (CES-D). Based on this approach, it was estimated that 12%–18% of the variance in WB was accounted for the additive effects of the SNPs measured on genotyping platforms.[76]

Next, the development of genome-wide association studies (GWASs), allowed for the first identification of specific genetic variants associated with WB. In a GWAS, millions of genetic variants are measured and regressed on a phenotype in a large group of individuals. In this way, the association between each genetic variant and an outcome of interest is tested with a strong correction for multiple testing, so that the chance of finding false positives is greatly reduced. The first successful GWAS for

WB ($N = 298{,}420$) was performed in 2016. This study led to the identification of 3 genetic variants associated with WB (defined as life satisfaction and positive affect).[77] The SNPs had estimated effects in the range of 0.015–0.018 s.d. per allele (each $R^2 \approx 0.01\%$). The high genetic correlations ($r_g > 0.75$) between life satisfaction, positive affect, neuroticism, and depressive symptoms suggest a common liability and this common liability was leveraged to increase the power to identify associated genetic variants. To this end, the latest GWAS for WB combined these three traits and coined them "the WB spectrum." In this study, 304 independent significant variant-phenotype associations were identified for the WB spectrum, with 148 and 191 associations specific for life satisfaction and positive affect, respectively. Biological annotation revealed evidence for enrichment of genes differentially expressed in the subiculum (part of the hippocampus) and enrichment for GABAergic interneurons. However, even with this progress, the identified variants account for only a small percentage of the variation, meaning that we still have a long road ahead. The first and only epigenome-wide association study approach, to identify differentially methylated sites associated with individual differences in WB, reports two sites (cg10845147, $P = 1.51 * 10–8$ and cg01940273, $P = 2.34 * 10–8$) that reached genome-wide significance following Bonferroni correction. Four more sites (cg03329539, $P = 2.76* 10\text{-}7$; cg09716613, $P = 3.23 * 10–7$; cg04387347, $P = 3.95 * 10–7$; and cg02290168, $P = 5.23 * 10–7$) were considered to be genome-wide significant when applying the widely used criterion of an FDR q value < 0.05. Gene ontology (GO) analysis highlighted enrichment of several central nervous system categories among higher-ranking methylation sites. However, replication of these results is warranted in larger samples.

Twin studies already taught us that about 40% of individual differences in WB can be explained by genetic factors. These follow-up analyses taught us about the genetic complexity of WB, with likely thousands of variants contributing to the trait. These studies also revealed that each genetic variant only contributes a tiny amount to the variation in WB, so that we cannot speak of a single "happiness gene" or a few "happiness genes" that assert substantial influence on WB.

While there is substantial genetic influence on variation in WB, the remaining majority of variance is caused by environmental influences. Again, while twin-and family-studies tell us something about the relative influence of the environment, they do not clarify which environmental influences are important. We can draw a few conclusions from the existing literature on the association between WB and environmental factors. On the socioenvironmental side, it seems that factors associated with social connectedness, such as the quality of social contacts[78] and social support[79] are important for WB. However, on the more contextual/physical environment side, there is not a lot of consensus on which environmental factors are important. Not only do studies produce contradicting results, there seems to be a lack of meta-analytic oversight. This lack of meta-analyses can mostly be explained by the fact that studies used varying designs, making it difficult to compare outcomes. There are some overview studies for specific environmental factors from the WB literature in general, but these studies also fail to present conclusive evidence. For example, Lovell and colleagues examined the association between exposure to biodiverse environments and WB and conclude that there is some evidence for a

small positive effect, but that much of the evidence is inconclusive.[80] Similarly, Vanaken, and Danckaerts[81] and Houlden and colleagues[82] examined the literature related to the relation between green space exposure and WB in children and adults, respectively. They both conclude there is limited evidence for a positive effect. Unfortunately, even though there is much literature examining the associations between different environmental variables and WB, it seems we are far from having a complete picture of these environmental influences.

For future research in this area, it is important that we continue with large-scale investigations into these environmental factors. For example, more homogeneity can be achieved by employing a design that is similar to that used in GWA studies, but includes WB and multiple environmental factors instead of multiple genetic variants. By performing such "environment-wide association studies," we can study the effect of environmental variables in different populations and geographical levels in a consistent manner. Ni and colleagues already applied such a design for WB, where they assessed the association between 194 psychosocial and behavioral factors and physical, mental, and social WB in a large Hong Kong sample.[83] They reported that only depressive symptoms, life satisfaction, and happiness were simultaneously associated with these three domains of WB. To develop a full picture of the WB *exposome* (i.e., the collective of exposures people experience, and how these exposures influence WB), it is important we continue this progress by studying other types of environmental factors in an environment-wide context, such as the physical and social environment. Moreover, as we have seen in this chapter, there is a considerable genetic influence on WB. Environmental factors are also partly under genetic control,[84] meaning that exposure to certain environments might be driven by genetic factors. Therefore, to fully understand the association between WB and environmental factors, this gene-environment interplay also needs to be considered. As mentioned earlier, there is a lot of inconsistent results from studies examining the environment in relation to WB. Part of this inconsistency might be explained by the fact that most studies do not use genetically sensitive designs. Twin research can help us elucidate the extent to which covariation between WB and environmental factors is genetic in nature, for instance using bivariate designs that partition covariance into genetic and environmental sources.

To conclude, while there are still hurdles to be overcome and many unanswered questions, considerable progress has been made over the past years in identifying genetic and environmental factors that influence WB. The above paragraphs already outlined some of the steps that have been/need to be taken to advance our understanding of WB. However, what was not mentioned yet is the way in which (extended) twin designs can help us further our understanding of WB. In Section 18.6, we elaborate on some interesting future directions for this type of research.

18.6 Future directions

In this final section, we present some interesting extensions of the classical twin design in terms of designs and outcome measures. More specifically, we discuss

the use of ecological momentary assessment, causality in terms of MZ difference models, and nuclear twin family designs (NTFDs).

18.6.1 Well-being fluctuations

Almost all existing twin studies examining the heritability of WB assess WB with questionnaires about general WB, happiness, or life satisfaction. However, like many other complex human traits, feelings of WB (e.g., mood) fluctuate over time and across different contexts.[85–87] The heritability of momentary WB (e.g., how happy do you feel in this moment?) has been assessed twice and resulted in low or even negligible estimates.[88,89] In a small twin sample, Riemann and colleagues measured moods across mood-inducing situations and estimated the heritability around 8%–16%.[88] More recently, Menne-Lothman et al. investigated momentary positive affect in female twins using the experience sampling method and reported a heritability of 0%.[89] The variance in momentary WB was completely explained by the environment, that is, the mood-inducing situation.

The heritability of fluctuations in WB has not been investigated, even though individual differences in WB and mood fluctuations have been reported. Some people show relatively stable levels of WB over the day and/or week, while others fluctuate a lot.[90–92] One way to capture the fluctuations and dynamic nature of WB is by applying an ecological momentary assessment (EMA) design. EMA involves the repeated assessment of the momentary experiences and moods of participants in real-time and in their natural environment.[93] Due to technological advances EMA studies can be conducted more easily with smartphones, a device ubiquitously present in our society. Future twin studies should make use of such designs to explore the contribution of genetic and environmental effects to the stability and fluctuations of momentary WB.

18.6.2 MZ difference/causality

Twin studies can also be used to investigate causal relationships between variables, as this design controls for genetic and shared environmental confounding. The co-twin control model makes use of discordant MZ (and DZ) twin pairs to determine whether an observed association is consistent with a causal effect of an exposure on an outcome.[94] For example, if MZ twins differ on an exposure variable, and also differ on the outcome (e.g., WB), we can conclude that the association between the variables is not due to confounding genetic or shared environmental factors affecting both variables as MZ twins share 100% of their genes.

In the field of WB, this causality analysis has only been applied to investigate the causal relationships between WB and exercise behavior[95] and mortality.[96,97] Stubbe and colleagues[95] reported that, even though exercisers were on average more satisfied with their lives and happier than nonexercisers, no evidence for a causal effect of exercise on WB was present using the co-twin method. Sadler and colleagues[96] and Saunders and colleagues[97] did find a causal association between higher WB and

lower mortality. Twin differences in WB predicted differential mortality within discordant pairs. Although the discordant twin method or (MZ) twin difference design is powerful to explore likely causal pathways between (environmental) factors and traits or outcomes, in the field of WB, the application of these methods is scarce. Future studies can use this powerful design to explore various causal influences on WB using twin samples.

18.6.3 Nuclear twin family design

In a classical twin study, the observed covariance between MZ and DZ twin pairs is used to make inferences about the relative influence of genes and environment. While the design has led to many important insights for WB (as was summarized in this chapter), it does have some limitations. In these models, we can only estimate as many parameters as there are pieces of information available. Since the unique environment parameter (e) can be estimated in all models (given that e is 1 minus the MZ correlation), and we observe the MZ and DZ covariance, we have three pieces of information available to us when using the classical twin design. This means we can only estimate three parameters: either additive genetic effects (a), shared environmental effects (c), and unique environmental effects (e), or (a), (e) and dominant genetic effects (d).

To increase the number of parameters that can be estimated, a simple solution is to include more family members between which we can estimate covariation. In the nuclear twin family design (NTFD), data on parents are included in addition to the twin data, meaning that we now also include the covariance between the parents, and the covariance between parents and children.[98] This allows for the simultaneous estimation of C and D, and also for the estimation of other interesting parameters: the potential effect of assortative mating, and potential vertical transmission. Assortative mating occurs when two spouses are more similar to each other than would be expected under a random mating pattern. While this can have many causes, the result is that these spouses are genetically more similar than two random individuals. Vertical transmission, in the context of the NTFD, describes the influence of the familial environment from nongenetic effects passed from parents to offspring. In the case of WB, this would mean that parental WB influences offspring WB through its effect on the familial environment. Importantly, the parental phenotype is influenced by the parental genotype. This vertical transmission from parental phenotype on offspring phenotype is thus not completely independent from genetic influences.

Thus, by including data on parents, the NTFD allows for the estimation of more parameters, and also provides more accurate estimates of the model parameters. Naturally, the design can be extended to include more family members (e.g., nontwin siblings) resulting in better-powered designs.[99] Importantly, this design is not new: it has been applied to many traits and has been improved by different people over the years. Yet, for WB, it seems that such an extended twin design has only been applied once. In a study in Norwegian twins and parents, Nes, Czajkowski, and Tambs[100] applied the NFTD to estimate nonrandom mating, cultural transmission, and shared environmental effects specific for regular siblings and twins. Their analyses revealed

the presence of nonrandom mating (spousal correlation of 0.26) and significant influence of the shared twin environment. The effect of vertical (cultural) transmission was estimated to be negligible. As this was the only extended NFTD study to date, it is not yet clear whether these results are consistent across different cultures/measures of WB. An interesting future direction would thus be to replicate these findings in different studies and with different measures.

18.7 Conclusion

The aim of this chapter was to summarize existing behavioral genetic research on WB, to show how this has directed WB research, and to provide a glance into directions for future research. While the last meta-analyses on twin studies for WB were published only five years ago, since then the field has developed rapidly: the first (300) genetic variants for WB were identified, the field is increasingly doubting existing definitions of WB and acknowledging the interrelatedness of different WB-related phenotypes, and are thinking about how to improve models for estimating sources of variation in WB. While there was first a focus on quantity, where the goal was to obtain the largest sample size possible at the cost of simple phenotyping, we are now transitioning to a focus on quality, with promising improvements in the measurement (e.g., EMA) and analyses (i.e., genetically informative designs) of WB ahead.

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