

The Nature-Nurture Debate: New evidence and good news

Ragnhild Bang Nes

Abstract

How can behaviour genetics contribute to the future of public health and prevention? This paper reviews the new evidence from behaviour genetic studies on happiness and well-being and discusses its implications for public health care challenges. Most policy makers seem to be unaware of the important implications this research has for our understanding of the environment. It is concluded that we may benefit from greater use of genetically informative designs due to their potential for advancing our understanding of both genetic and environmental risk and protective mechanisms, which is necessary for making better, more fully informed policy decisions.

Behaviour genetics and happiness

Why are some people happy and others not? Why do people differ? This is the fundamental question of behaviour genetics which studies how genes and environments influence naturally occurring individual differences. By means of genetically informative data (adoption, twin, and family data), a mature mathematical framework, and sophisticated modelling techniques, observed differences between family members are apportioned into sources of variance due to genes, shared, and nonshared environments. Over the past decade, behaviour geneticists have entered the positive mental health field aiming to delineate the magnitudes and mechanisms by which these aetiological influences affect human happiness and well-being. This paper summarizes the recent behaviour genetic findings, and broadly

discusses the theoretical and practical implications of such research for public health and happiness.

The recent background

Recognition of the importance of genetics is one of the most profound and dramatic changes within the behavioural sciences during the past few decades (Plomin, DeFries, McClearn, & McGuffin, 2001). The behaviour genetic findings are impressively robust and indicate that virtually all psychological traits are at least moderately heritable. Quantitative genetic methodology and principles of population genetics were developed at the beginning of the 20th century. Since then there have been considerable changes in the prevailing views on the relative importance of genetic and environmental influences. A preoccupation with environmental influences on health and behaviour characterised the behavioural sciences until the 1960s (Rutter, Moffit, & Caspi, 2006). The behaviourist movement, which studied behaviour entirely independent of biology and evolutionary history, had a profound influence on most theories of learning and development from the 1920s to the 1960s. The idea of irreversible effects from early childhood experiences was highly influential (Rutter et al., 2006), and numerous studies reported profound effects from environmental risk factors such as adverse life experiences and parental behaviour on later development of mental health (e.g. Ainsworth, 1962). From the late 1960s, there was a considerable growth in behaviour genetics. Methodological strategies improved, and a large number of twin, adoption, and extended family studies clearly showed that mental health are caused by multiple genetic and environmental factors. By the early 1980s, environmental factors were considered less influential than previously assumed. This major shift was largely due to research documenting that:

1. Children have a strong impact on family functioning, not only the other way around (e.g. Bell, 1968).

2. Environmental experiences tend to make children growing up in the same family different rather than similar (Plomin & Daniels, 1987).
3. Correlations between environmental risk factors and mental health are often genetically mediated (Rutter et al., 2006).

These important findings underscore that i) separating environmental effects on the person from person effects on the environment (i.e. testing for the direction of causal influences) is necessary, ii) environmental influences often work in ways different from previously assumed, and iii) many claims in the research literature on environmentally mediated risks may be questionable due to considerable genetic influences on individual differences in exposure to life events.

Development and refinement of molecular genetic strategies in the 1990s led many researchers to believe in the possibility of identifying specific genes with causal effects on mental health and happiness. These expectations have not been met, perhaps largely because the traits relevant to biological psychology are characterised by a continuous distribution and influences from multiple genes and environmental factors that are often immeasurable, or entirely unknown. Due to small effects, genetic heterogeneity, and complex patterns of interplay with environmental factors, as well as psychometric difficulties, identification of susceptibility genes to psychological characteristics, in a robust and replicable manner, is highly complicated, if at all possible in the near future (Merikangas & Risch, 2003). Identification of single genes would also not elucidate the causal pathway of mental health and happiness. Genes operate in co-action with environmental factors as part of a multifactorial causation process, with effects usually being contingent on a multitude of environmental circumstances. Behaviour genetic designs do however offer novel tools and methods, including a mature mathematical framework and specialized computational tools, to address central issues in well-

being research with important implications for public efforts aiming to alleviate suffering and promote happiness and health.

New evidence

Focus on well-being research in behaviour genetics, is a fairly recent development, reflecting the more general reorientation within the mental health field today towards a growing focus on positive indicators of mental health. So far only a few behaviour genetic studies have ventured into the positive mental health field, and most studies are based on simple, self-report measures of positive indicators such as *satisfaction with life* and *subjective well-being* (SWB). Although there are a limited number of studies, the findings are generally consistent and based on thousands of twins from several different countries, of which the majority is reared together, but some also reared apart. Results indicate that genetic influences are important for happiness and well-being, accounting for 35-50% of time-specific variance (heritability),^{*} and as much as 80% of long-term levels (Lykken & Tellegen, 1996; Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006). These results are consistent with a theory positing a general positivity factor, or readiness to perceive and interpret the world more or less positively, which is stable across time and strongly influenced by genes. Behaviour genetic studies have also recently indicated that the well-known association between well-being and stable personality traits such as extraversion (Lucas & Fujita, 2000) and neuroticism (Fujita, 1991) may be due to common genes (Weiss, Bates, & Luciano, 2008; Eid, Riemann, Angleitner, & Borkenau, 2003) possibly involved in susceptibility to experience positive and negative affect (Watson & Clark, 1997). Happiness and

^{*} These percentages represents amount of difference between people (i.e. in a particular environment at a particular time) attributable to genetic causes.

personality thus appear to be influenced, at least partly, by the very same set of genes.

The non-genetic influences on well-being and happiness largely reflect transient environmental effects which are individual-specific (Nes et al., 2006). This indicates that only minor, or negligible, influences are due to *shared environmental factors* which refer to environmental influences that make family members similar. The behaviour genetic findings thus convincingly show that familial resemblance essentially is due to shared genes, and not shared environments (Plomin & Daniels, 1987). These results fit well with findings on related variables, such as personality traits and liability to depression and distress (e.g. McGue, Bacon, & Lykken, 1993; Rijsdijk, Schnieder, Ormel, Sham, Goldberg, & Spector, 2003). The findings are similarly consistent with the vast research literature on well-being outside of the behavioural genetic domain which has documented surprisingly little influences from shared environmental factors (e.g. demographic factors) on subjective happiness despite most theoretical perspectives in psychology, ranging from psychoanalysis to social learning theory, assuming that major causes of individual variation are associated with shared aspects of the family.

Findings of mainly transient environmental effects on happiness and well-being are confirmed by different methods (Kahneman, Krueger, Schkade, & Schwartz, 2004). Along with findings from longitudinal research on related constructs (e.g. anxiety, depression, personality), the results collectively suggest that environmental influences are important, but do not exert long-lasting redirection or enduring changes unless exposure is continuous (e.g. Nes, Røysamb, Reichborn-Kjennerud, Harris, & Tambs, 2007; Rijsdijk et al., 2003; McGue et al., 1993; Diener, Sandvik, Pavot, & Fujita, 1992; Merikangas, Zhang, Avenevoli, Acharyya, Neuenschwander, & Angst, 2005).

Good news

Specific policy recommendations do not usually flow from behaviour genetics, and behaviour geneticists rarely make explicitly clear how their findings can be applied to prevent psychological problems or promote happiness and health. The behaviour genetic findings are commonly misunderstood, perhaps partly because the findings are poorly explained to policy makers and the general public. Results are simplified by researchers as well as the media, leading to inaccuracies, misapplications, and polarizations. Misunderstandings may also be due to the counterintuitive meaning behaviour geneticists assign to their concepts, such as the “shared environment”. To behaviour geneticists, the shared environment does not refer to family-wide influences that are objectively shared. It is a measure of environmental *effects* and used in a broad sense, encompassing prenatal influences, nutrition, and illness - not just family socialization processes. The behaviour genetic results most clearly communicated are usually the heritability estimates, which have few policy or practice implications. Workers in the social policy arena are thus often unaware that the most important discoveries made in behaviour genetics are concerned with how the environment, and not genes, affects human behaviour. Due for instance, to permitting control of genetic and social endowments that are unobserved in social science data sets, behaviour genetic research may be indispensable for testing hypotheses about environmental risk mediation. In the following I will outline some of the more specific contributions and potentials of behaviour genetics.

The relativity of heritability

All of the biometric studies on happiness-related constructs have provided massive evidence against predetermination, reporting heritability estimates in the 30-50% range. Large-scale family studies have found even less genetic contributions. Genetic determinism is thus not warranted by the evidence. This is also no surprise. Pre-programmed well-being or despair would be

incompatible with the principle of evolution, which raw material is adaptation. The complexity of most behavioural systems necessarily implies that genes are not destiny - they contribute to probabilistic propensities. Far from representing the ultimate limits of what we may achieve, genes determine only the likelihood or probability of a particular outcome.

Heritability estimates are *relative* estimates, referring to genetic influences on individual differences, and indexes not only the direct genetic effects, but also the effects of gene-environment interplay. Heritability is estimated from correlations between family members, mostly identical and fraternal twins, and represents the amount of total variance in a given trait attributable to genetic factors - in a specific population at a specific point in time. Caution must therefore be taken in generalizing the heritability findings across populations. Heritability describes what is in a particular population at a particular time (Plomin et al., 2001), and refers to differences in populations, never to single individuals. This can best be illustrated with a concrete example. Heritability for body height is shown to be high, with heritability estimates up to about 0.90. This means that 90% of individual differences in body height in a specific population at a specific time are attributable to genetic differences. Despite body height being highly heritable, however, average height has increased over the last generations, leaving us on average taller than our grandparents. This is most certainly due to changes in nutrition; not changes in our gene pool. Behaviour genetics is not equipped to study causes of average height, or the causes of changes in average height, however. Behaviour genetics deal with the causes of individual differences. Environmental factors may thus have profound influence on average body height despite high heritability for body height. In many countries, including Norway, environmental factors inhibiting growth is not very prevalent. Due to the environment being fairly similar for most, environmental factors contribute little to individual height differences in such countries today. In societies in which some citizens have a poor diet and others not, environmental factors may have a substantial

impact on individual differences in height, and consequently the heritability estimate will be less.

The same holds for behaviour genetic research on happiness and well-being. High heritability does not necessarily limit the effectiveness of policy efforts aiming to raise average happiness. If environments change, so may the relative impact of genes and environment. Indeed, the more we improve our society, often the greater the genetic component in the remaining variation – thus, high heritability might reflect a social good, that is, of the successful elimination of much environmental inequality.

Genetic stability and environmental change

The vast research literature on happiness and well-being indicates that happiness is fairly stable across situations and the life span. This may partly reflect a dispositional tendency to experience life positively or negatively, and partly a cumulative effect of positive and negative life events (Brief, Butcher, George, & Link, 1993). Behaviour genetic research has shown genes to influence dispositions as well as exposure to life events. The few longitudinal behaviour genetic studies of happiness related constructs have reported two salient findings: i) genetic factors are important for generating stability, and ii) the environment is important for generating change. Whilst the genetic findings are commonly broadcasted, the environmental effects are often overlooked. Do strong and stable genetic effects indicate that opportunities for change are futile? Not at all! Well-being levels are not set in concrete. In fact, usually less than 50% of the time-specific variance is due to genes. At any given moment in time, environmental circumstances are as influential in determining our affective valence as genetically based dispositions. Long-term stability is also not accounting for more than half of the total variance, leaving change an important and constant factor in life. The limited stability of most environmental influences indicates that circumstantial boosts in well-being usually are short-lived, however, and that most people adapt to new circumstances. Thus,

we seem provided with an internal buffer which prevents drastic mood changes and defends against the negative potential inherent in the environment. How this genetic buffer works, is yet unknown. Genetic effects often operate on a level far removed from the DNA, and various indirect pathways (through individual choice of environments or exposure to life events), physiological, or biochemical processes may provide important links between happiness and genes.

The longitudinal results also highlight the importance of using both subjective and objective well-being indicators when monitoring populations and individuals. Measures of subjective happiness offer a break from conventional principles of utility, and happiness is irrevocably grounded in mental first-person experiences. However, global self report measures of well-being and happiness do not only reflect the objective quality of people's lives *per se*, but also the workings of our brains and our genes, which shape perceptions and responses to events. Global well-being measures may therefore be fallible estimates of the objective quality of people's lives, and "objective" (e.g. physical and mental health, environmental preservation and social wellness, individual and political freedom, etc) as well as subjective measures are necessary when surveying and monitoring populations. This also implies that policies improving meaningful, good, or pleasant experiences should be pursued also when there are no apparent changes in subjective happiness.

The findings may also remind us that happiness and unhappiness are not ends, but means. Negative feeling states, although subjectively unpleasant, provides us with a necessary signal mechanism, a wake-up-call system to our consciousness to take notice of an adaptive problem in need to be solved, which subsequently motivates action to solve it (Hill & Buss, 2008).

The shared environment

The behaviour genetic finding of negligible influences from the shared environment on happiness and mental health is widely debated. Despite their shared upbringing, sibling similarity in well-being and mental health appears to be largely, or entirely, attributable to shared genes and not to shared environments. This claim is sometimes believed to indicate that family wide adversities, including hazards such as poverty, loss, and discord have little, or negligible, impact on risk and protection for mental health. This common misunderstanding may partly be caused by the counterintuitive meaning of the shared environmental construct. To behaviour geneticists, the “shared environment” signifies an inference derived from any evidence of sibling similarity which is not caused by genes. It reflects an effect from some “black box” environmental component which includes all non-genetic influences that produce family resemblance. If an objectively shared family wide factor, such as conflict or poverty, impinges differently on siblings in a family, the effect is classified as “non-shared”. The effect is only classified as shared if it in general – across families – tends to make siblings similar. Thus, lack of evidence for shared environmental influences should not be taken as indication that family factors are not important for our well-being or ill-being. Family features might quite possibly exert large effects, but tend to influence the different family members differently. What the behaviour genetic studies imply is essentially that environmental influences do not operate on a family-by-family basis (e.g. parenting style do not have general effects), but rather on an individual-by-individual basis (affect siblings differently), which clearly underscores the importance of studying more than one child per family when investigating environmental risk and protective factors.

Nevertheless, shared environmental contributions are sometimes found to be evident in designs assessing vulnerability factors more directly even when not apparent when treated as unmeasured inferred variance (Kendler, Neale, Prescott, Kessler, Heath, Corey,

& Eaves, 1996). And although many twin studies have stopped short at testing statistically for the existence of an environmental “black box” component (Caspi, Taylor, Moffitt, & Plomin, 2000), behaviour genetic methods do enable inclusion of measured environments in their models. For example, Caspi et al. (2000) explored whether neighbourhood deprivation could explain familiar aggregation of behaviour problems, and the extent to which this risk factor in fact was environmentally mediated. A major strength in this design was its potential to separate the “real” effect of deprived neighbourhood conditions which is difficult, if not unfeasible, in samples that are not genetically informative. The results clearly evidenced a significant effect from deprived neighbourhood conditions on children’s mental health above or beyond a genetic liability to behaviour problems.

Genetic effects on the environment & control for endowments

Behaviour genetic research has indicated that the vast literature on environmentally mediated risk is questionable. Many environmental measures widely used in psychology and public health work show genetic influences, because people create their own environments partly due to genetic reasons (the nature of nurture). Some supposedly environmental factors influencing happiness and health are in fact genetic (or at least partly so). This is due to environmental measures commonly being indirect measures of behaviour or personality. Such genetic effects on the environment do not disqualify intervention attempts, but certainly has implications for effective public health work. Correct measures of real effects from environmental risk and protective factors are critical when aiming to design effective interventions and promotion programs, or when monitoring the effects from such efforts. An important strength in behaviour genetic designs concerns its potential to control for confounding factors, like genetic endowments when investigating possible risks. Twins share the same womb, are born at the same time, live in the same family, and when identical, they are essentially genetic clones. Data from identical twins therefore allow us to control for

unobserved endowments such as preferences and capabilities due to genes, family background variables, and neighbourhood effects affecting well-being and happiness. Designs permitting control for such endowments have been shown to reach different conclusions on the effects from environmental factors. For example, Kohler and colleagues (2005) explored the effects from partnership and children on happiness using data from Danish identical twins. Several previous studies have shown children to produce decrements in marital satisfaction in most subpopulations in the US culture (Glenn & McGlanahan, 1982) with happiness levels rising to former levels first after the children leave their childhood home (Hakim, 2003).

In addition to exploring the “pure” effect from children and partnership on happiness, Kohler’s analyses enabled i) exploration of economic or rational choice fertility models that may be important in understanding continued partnership formation and childbearing in low fertility contexts, as well as ii) the set-point theory of happiness - as it pertains to partnership and childbearing. If happiness and well-being is partly dependent on genetic influences, personality, and family/childhood background, *and* these factors also affect selection into partnerships and the propensity to have children (which both literature and research suggests), results based on genetically uninformative data may be distorted. Kohler’s results show that partnership formation and childbearing do have persistent positive effects on happiness. His results thus contradict previous findings and raise questions about the primary dominance of genetic influences on long-term happiness as suggested by the set-point theory. His study also showed that men (at least Danish) gain greater happiness from partnership than women, and that a first-born child is an important, direct source of well-being in women, whereas additional children reduce their happiness levels. In contrast, a first child was found to contribute only indirectly in male respondents through increasing the probability of a current partnership, and additional children left their happiness unaffected. The results are discrepant with many

previous studies based on standard samples, and demonstrate the importance of controlling for endowments in research on risk and protective factors for happiness and health. They are also highly informative to social policy planners by showing that happiness gains are primarily associated with a first child and not additional ones. Such results corroborate demographic findings of low levels of childlessness in countries with fertility rates below 1.3, and indicate that women's (or couple's) motivations to have additional children might be more influenced by the costs and benefits of children, than their motivation to have at least one child. Desires for additional children may consequently be much more dependent on socioeconomic conditions and family policy. The results also suggest that changes in family policy aiming to increase fertility may not necessarily increase well-being in parents.

Conclusion

Understanding of how aetiological factors act at a population level may be an important step towards development and utilization of tools to improve health outcomes. The synthesis from The international Conference on Happiness and Public Policy (2007) points out that "a shift of development goals from economic prosperity to subjective well-being requires good measures of well-being and thorough studies of how public policies can impact these measures" (p. 40). Refined, accurate, and quantified knowledge on how environmental factors operate may be a critical step in this direction. Recommendations on policies have often assumed environmental risk mediation without testing alternative possibilities, such as genetic mediation. As environmental risks and protective factors appear to operate in rather different ways than previously assumed, failure to control for genetic influences on the environment, may lead to erroneous conclusions. By permitting control for genetic endowments, behaviour genetic methods may provide one of the best methodologies for studying environmental risk and protective mechanisms necessary for informed policy decisions. So far we are only in the beginning of

theorizing and testing models including such mechanisms in the field of happiness and well-being, and we need further knowledge on how the aetiological influences interplay to make appropriate societal actions.

Overwhelming focus on the reporting of genetic findings may divert interest and attention from the important work behaviour geneticists have done on the environment, which contrary to the many genetic findings do have important policy or practice implications. More behaviour geneticists should therefore try to make explicitly clear how their findings can be applied to prevent psychological problems and promote happiness and health. This might act to place the field more firmly within the environmental context, and stimulate further advanced research into the workings of gene-*and*-environment interplay which is necessary for making fully informed policy decisions.

References

- Ainsworth, M. D. (1962). The effects of maternal deprivation; a review of findings and controversy in the context of research strategy. *Deprivation of Maternal Care: A Reassessment of its Effects*. Geneva: World Health Organisation
- Bell, R. Q. (1968). A reinterpretation of the direction of effects in studies of socialization. *Psychological Review*, 75, 81-95.
- Brief, A. E., Butcher, A. H., George, J. M. & Link, K. E. (1993). Integrating bottom-up and top-down theories of subjective well-being: the case of health. *Journal of Personality and Social Psychology*, 64, 646-653.
- Caspi, A., Taylor, A., Moffitt, T. E., & Plomin, R. (2000). Neighbourhood deprivation affects children's mental health: environmental risks identified in a genetic design. *Psychological Science*, 11, 338-342.
- Eid, M., Riemann, R., Angleitner, A., & Borkenau, P. (2003). Sociability and positive emotionality: Genetic and environmental contributions to the covariation between different facets of extraversion. *Journal of Personality*, 71, 319-346.

- Fujita, F (1991). *An investigation of the relation between extroversion, neuroticism, positive affect, and negative affect*. Master's thesis. University of Illinois.
- Glenn, N. D., & McGlanahan, S. (1982). Children and marital happiness: Further specification of a relationship. *Journal of Marriage and the Family*, 44, 63-72.
- Hakim, C. (2003). *Work-Lifestyle Choices in the 21st Century: Preference Theory*. Oxford: Oxford University Press.
- Hill, S. E., & Buss, D. M. (2008). Evolution and subjective well-being. In M. Eid & R. J. Larsen (Eds.), *The Science of Subjective Well-Being*. (pp. 62 - 79). New York: The Guilford Press.
- Kahneman, D., Krueger, A. B., Schkade, D. A., Schwartz, N. & Stone, A. A. (2004). A survey method for characterizing daily life experience: The day reconstruction method. *Science*, 306, 1776-1780.
- Kendler, K. S., Neale, M. C., Prescott, C. A., Kessler, R. C., Heath, A. C., Corey, L. A., & Eaves, L. J. (1996). Childhood parental loss and alcoholism in women: a causal analysis using a twin-family design. *Psychological Medicine*, 26, 79-95.
- Kohler, H.-P., Behrman, J. R., and Skyttke, A. (2005). Partner + Children = Happiness? The Effect of Fertility and Partnerships on Subjective Well-Being. *Population and Development Review*, 31(3), 407-445.
- Lucas, R. E. & Fujita, F. (2000). Factors influencing the relation between extraversion and pleasant affect. *Journal of Personality and Social Psychology* 79, 1039-1056.
- Lykken, D. & Tellegen, A. (1996). Happiness is a stochastic phenomenon. *Psychological Science*, 7, 186-189.
- McGue, M., Bacon, S., & Lykken, D. T. (1993). Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental Psychology*, 29, 96-109.
- Merikangas, K. R. & Risch, N. (2003). Genomic Priorities and Public Health. *Science*, 302, 599-601.
- Merikangas, K. R., Zhang, H., Avenevoli, S., Acharyya, S., Neuenschwander, M. & Angst, J. (2005). Longitudinal Trajectories of Depression and Anxiety in a Prospective Community Study. *Archives of General Psychiatry*, 60, 993-1000.

- Nes, R. B., Czajkowski, N., Røysamb, E., Reichborn-Kjennerud, & Tambs, K. (2008). Well-being and ill-being: Shared environments, shared genes. *Journal of Positive Psychology*, 3 (4), 1-13.
- Nes, R. B., Røysamb, E., Reichborn-Kjennerud, T., Harris, J. R., & Tambs, K., (2007). Anxiety and depressive symptoms in young adults: Genetic and environmental influences on stability and change. *Twin Research and Human Genetics*, 10, 450-461.
- Nes, R. B., Røysamb, E., Tambs, K., Harris, J. R., & Reichborn-Kjennerud, T. (2006). Subjective well-being: Genetic and environmental contributions to stability and change. *Psychological Medicine*, 36, 1033-1042.
- Plomin R. & Daniels D. 1987. Why are children in the same family so different from one another? *Behavioural and Brain Sciences*, 10, 1-60.
- Plomin, R., DeFries, J.C., McClearn, G. E., & McGuffin, P. (2001) *Behavior Genetics*. New York: Worth Publishers, 4th edition.
- Rijsdijk, F. V., Schnieder, H., Ormel, J., Sham, P., Goldberg, D. P. & Spector, T. D. (2003). Genetic and environmental influences on psychological distress in the population: General Health Questionnaire analyses in UK twins. *Psychological Medicine*, 33, 793-801.
- Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry*, 47, 226-261.
- Rutter, M. (2006). *Genes and Behavior: Nature-Nurture Interplay Explained*. Malden, USA: Blackwell Publishing Ltd.
- Watson, D., & Clark, L. A. (1997). Measurement and mismeasurement of mood: Recurrent and emergent issues. *Journal of Personality Assessment*, 68, 267-296.
- Weiss, A., Bates, T. C., & Luciano, M. (2008). Happiness Is a Personal(ity) Thing: The Genetics of Personality and Well-Being in a representative Sample. *Psychological Science*, 19, 205-209.