

*Everybody is different when it comes to assessing their subjective well-being. It is likely that the differences in people's genetic makeup contribute to long-lasting differences in their subjective well-being. Find out from Philipp Koellinger, Lars Bertram, and Gert G. Wagner, who are experts in genetic studies, about the extent to which we are responsible for our own happiness.*



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## **We are largely responsible for our own happiness**



### **Scientists identify genes related to well-being, depression, and neuroticism**

Well-being is of great concern to all of us. It is obvious that the environment, especially individuals' economic situation, has quite an impact on well-being. However, we intuitively know that people are different in terms of their subjective perception of their well-being. We know people who are happy with their lives despite their difficult circumstances. And we also know people who are always less satisfied than others regardless of their circumstances.

Experts in the field have formulated one hypothesis suggesting that the differences in people's genetic makeup contribute to long-lasting differences in their subjective well-being. In an attempt to test the existence of genes related to well-being, an interdisciplinary consortium of 178 researchers

from around the world studied anonymised genetic data from almost 300,000 people. They discovered new genes associated with well-being in a study [published early this year in \*Nature Genetics\*](#). The findings show diverse genetic links between life satisfaction, happiness, depression and neuroticism.

Using an approach called [genome-wide association testing](#) the team found three DNA sequence variants (known as [single nucleotide polymorphisms or SNPs](#)) to be linked with happiness and satisfaction with life. The team also identified more than a dozen other SNPs associated with neuroticism and depression. Even though these new findings can be considered as unambiguous, as they are statistically significant, they only explain a small proportion of the differences in terms of well-being between individuals in the population.

## **Power of genetic variants**

Individual genes have only tiny effects on well-being. However, this does not contradict the often substantial heritability of happiness and related personality traits. Indeed, several members of the same family often display striking similarities. Remarks like “she’s just like her grandmother” are common in families. Yet, this resemblance within families is not merely due to one gene. Rather, it can be attributed to combinations of genes that jointly contribute to traits like happiness. With the exception of twins who are ~100% genetically identical—referred to as monozygotic—the combination of genetic patterns passed on from parents to children are unique to the each individual. Yet, different combinations of genes can lead to similar effects in people.

## **Nature and Nurture**

Our subjective well-being is, ultimately, influenced by both our genes and our environment. To explain this logic, it is useful to take an example. Let's assume that genetic variants influence how extroverted an individual is. If we consider that being more extroverted helps a person make more friends—and, in turn, become happier and more satisfied with their lives—changes to this intermediate environmental channel could have drastic effects on well-being. Indeed, the genetic association might not be found in environments where the number of friends a person has is less strongly related to extroversion; for instance, in a close-knit community where people tend to know each other personally.

Conversely, social circumstances can increase life satisfaction. This may happen even when the gene pool of the population does not change. For example, if people react negatively to social inequality, it may be possible to increase the average well-being through redistribution of resources like income.

## **Limited remedy**

Regardless of the level of heritability of well-being, the environment could still have a large impact on people's well-being. To illustrate this point, let's consider the famous example given by economist Arthur Goldberger. If genes were responsible for all the variation in eyesight allowing people to see without any external help, anybody suffering from eyesight problems could overcome their difficulties by wearing glasses. Indeed, the non-genetic intervention of glasses often counteracts

100% of the genetic effects on eyesight.

In medical genetics, genetic testing can predict the occurrence of some diseases, such as Huntington's disease or certain forms of Alzheimer's disease, with great certainty. By contrast, genetic research into traits involving multiple genes, such as subjective well-being, cannot be expected to provide an accurate prediction. This is due to the tiny effects exerted by the individual DNA variants and their potentially complex interaction with the environment.

## Future developments

Genetic studies can, however, help to identify biological mechanisms that are relevant for mental health. Recently published [findings in schizophrenia research](#) provide a pertinent example. Using genome-wide association analyses similar to those applied in studies of subjective well-being, researchers identified over 100 genetic variants contributing to the risk of schizophrenia, each with a small effect. Some of the identified genes appear to affect the regulation of the immune system.

Follow-up research has since confirmed that specific biological mechanisms involved in the immune response do indeed contribute to the risk of schizophrenia. These mechanisms were previously unknown and may point the way toward new approaches to the diagnosis and treatment of this psychiatric disorder.

Genetic studies could help reveal some of the genetics variants associated with health and well-being. This growing knowledge should empower each of us to get a bit closer towards goals such as happiness.

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Bertram and Wagner are co-principal investigators of the [Berlin Ageing Study II](#) (BASE-II). Koellinger is one of the principle investigators and co-founders of the Social Science Genetic Association Consortium (SSGAC) and one of the main authors of the study cited which also used BASE-II data.

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