Genes add risk to depression

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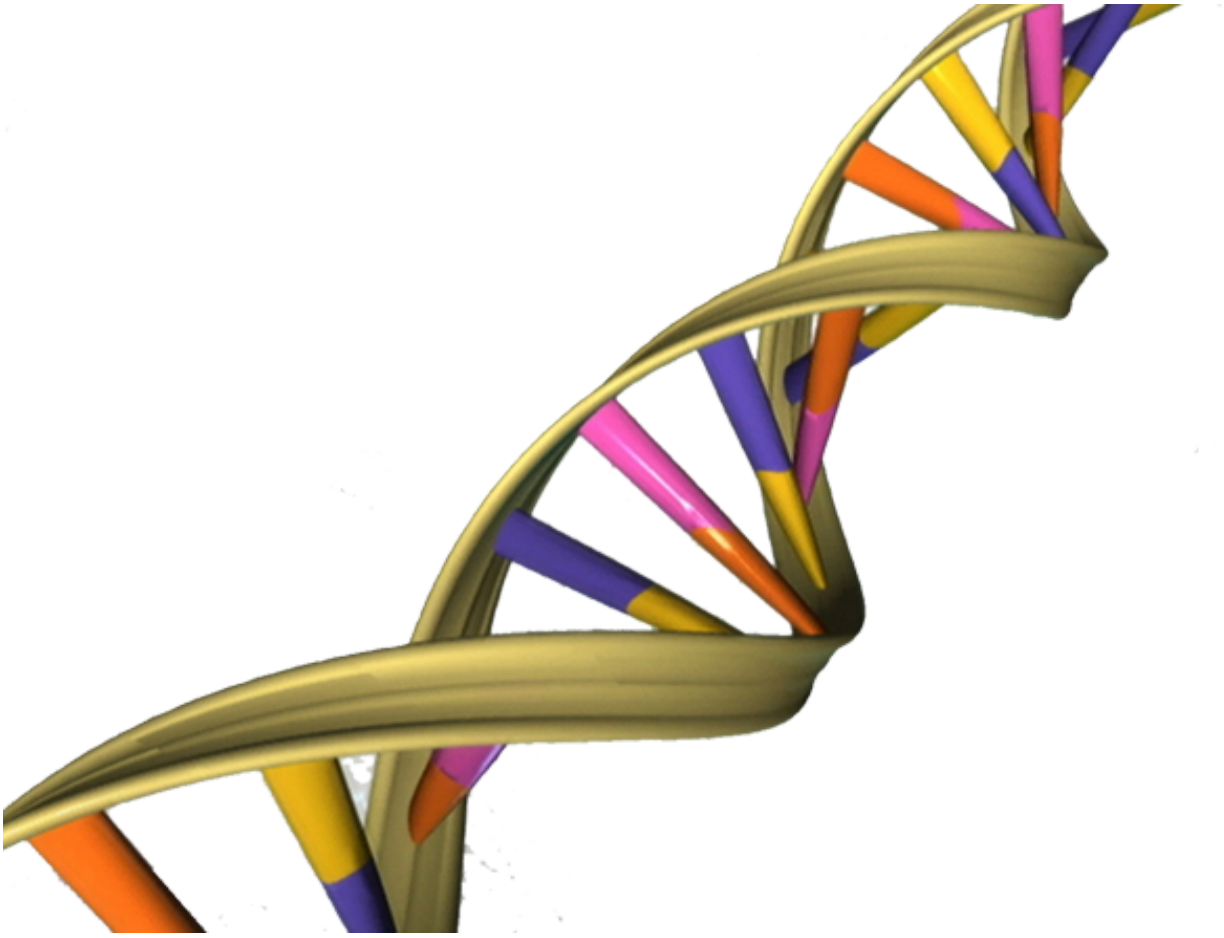

A depiction of the double helical structure of DNA. Its four coding units (A, T, C, G) are color-coded in pink, orange, purple and yellow. Credit: NHGRI

People born with a particular gene variant have a greater risk of developing depressions, a recent study from the Department of

Psychology at The University of Oslo shows.

Slowly, our society has begun to realise that depression is a serious public health problem. An estimate shows that mental illness costs NOK 60-70 million a year only in Norway, and depression accounts for a big share of this sum. But worse than the expense is the human suffering caused by depression. And yet, depression has been relatively under-researched, and we are not at all good at treating depressions: Only about 60 per cent of patients get better with treatment, regardless of which treatment they get.

Wrong basis for treatment

Part of the problem, according to post doctor Rune Jonassen, is that treatment is based not on what would most benefit the patient, but on therapists' preferences or on assumptions based on cost-benefit analyses.

"National guidelines for treatment encourage therapists to use the treatment which statistically seems to benefit the largest number of patients. But no form of therapy is inherently better than others. Neuroscientific research indicates instead that different people will benefit from different treatments," Jonassen says.

Then how can you tell what sort of treatment this particular patient should receive? Jonassen is doing research on brain function and the relationship between emotions and mental illness, in addition to working as a clinical psychologist. He has surveyed ten years' worth of research on a particular gene and its function in the brain. The results have been published in the journal Progress in Neurobiology.

He concludes that treatment needs to be much more based on knowledge about the ways in which the brain processes emotion under various circumstances. These are genetically coded functions, and the genetic

variation between people is substantial.

We react differently to change

"The more information we get about what a few crucial genes do, the better we understand the various paths that may lead to risk of, or robustness against, mental illness. This in turn will help us understand the mechanisms involved in development and treatment of depression," he says.

The gene Jonassen and his colleagues have focused their research on, regulates the neurotransmitter serotonin in the brain. The gene is closely connected with activity in certain brain structures which help us notice emotionally relevant things in our vicinty.

"It seems that adaptation is a key word when it comes to this gene's involvement in the development of depression. People who carry a particular variant of the gene, are more receptive to both negative and positive stimuli in their surroundings. In other words, we are not talking about a "gene for depression", but rather a gene which is involved in the brain's susceptibility to change. This means that people with the gene variant have a higher risk than others for developing a depression when faced with stressful life circumstances," Jonassen explains.

"Our complex brain has given humans a very sophisticated system for regulating emotions. Some people are more emotionally engaged than others in the first place; they have very quick emotional reactions. But different people also cope differently with the emotional reactions they have. Both these things are coded in our genes."

No need for gene mapping

All this means that our genes influence several possible pathways that may lead to depression, and these different pathways require different treatments. The big challenge is the translation of our knowledge about genes and basic brain functions into practical tools which may be of use in treatment.

The good news is that psychologists already have a large array of treatments at their disposal. To match these treatments with basic biology takes interdisciplinarity, but otherwise it need not be very complicated. Experimental psychology already offers simple tests which may reveal the various ways in which an individual can be at risk for depression.

According to Jonassen, mapping all the patient's genes in order to determine his or her treatment, is neither necessary nor useful.

"After all, far from every carrier of a specific gene develops depression. They're just a little more vulnerable than others under certain circumstances. If we pick one treatment, for instance cognitive therapy, and use it on all depressive patients, we know with certainty that a large percentage will receive no benefit. They will need a different approach, based on the mechanisms which triggered the depression in their individual cases."

Europe calls for research

The European Commission recently announced a roadmap for mental health research in Europe. Until now, mental illnesses have received far less attention from researchers than somatic illnesses. Given our growing understanding of how much suffering and disability is caused by mental illness, everyone agrees that research into these issues must take a higher priority.

Jonassen hopes to see more research which aims to translate neuroscientific knowledge into clinical treatment.

"The depression is the end result, but the way there will vary from one person to the next. There is a vast trove of information from, for example, neuroimaging, wich might be of use for therapists. The difficulty lies in bridging the gap between this knowledge and clinical treatment," he says.

More information: "Serotonin transporter polymorphisms (5-HTTLPR) in emotion processing: Implications from current neurobiology," *Progress in Neurobiology*, Volume 117, June 2014, Pages 41-53, ISSN 0301-0082, dx.doi.org/10.1016/j.pneurobio.2014.02.003

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