

'Children suffer... that's what drives me'

Marinus van IJzendoorn met Jon Sutton at the European Congress of Psychology and talked about his research on differential susceptibility in child development

We're here in Stockholm, where I think the popular idea of dandelion children – those with 'resilient' genes who will do well in most conditions – actually originated. The idea of dandelions and orchids originates from Tom Boyce, he used the metaphor. He might be a little distressed because it's taken too literally. I agree – these may not be two classes, but a continuum of more or less openness to the environment.

Tell me how you came to it scientifically.

We were the first to do genetic research on the idea of differential susceptibility. The idea was already around on a theoretical level, for quite some time, and in fact was most active in the mid-1990s. The first studies were by Boyce and then Jay Belsky, but then it seemed to slow down a lot. We were entering into the field of genetics from the perspective of attachment theory, which is I think quite logical because attachment theory is based on evolutionary theory – it's the first evolutionary theory applied to human development besides Charles Darwin himself. John Bowlby was the first evolutionary psychologist you might say, it's now a very popular concept but he was the first to apply evolutionary thinking in a systematic way to human development.

So my colleague Marian Bakermans-Kranenburg and I went into this area of genetic research after having done twin studies, and we found – by accident, in a way – this interaction between DRD4, a dopamine-related gene, and sensitivity to environmental influences on children, developing differentially positively or negatively. That got us on the way in a series of studies on differential susceptibility.

Am I right in thinking that two psychologists at King's College London, Caspi and Moffit, had raw data and graphs in their 2002 and 2003 papers

pointing to this idea, but it didn't really get a foothold?

Well, their wonderful research prepared the way for gene by environment interaction research more broadly. But their study is really firmly grounded in the tradition of diathesis-stress and cumulative risk.

So in people who did *not* face severe or repeated stress, the risk alleles in question actually heightened *resistance* to stress and depression.

Carriers of risk alleles were more prone to develop, for example, anti-social behaviour or depression having grown up in a bad environment with lots of maltreatment experience. But the other side of the equation, the bright side, Caspi and Moffit didn't touch on. So absolutely groundbreaking studies, because for the first time in the human development area they opened up the way of thinking in terms of measured gene by observed environment interaction, but differential susceptibility is a two-sided phenomenon – the same risk alleles would also create more options to learn from a positive environment. That's quite unique to the idea of differential susceptibility and to the research that we did in Leiden.

And it was about that time that positive psychology was coming to the fore, so you were surfing the zeitgeist of looking on the bright side!

It might be that it's not by accident – about that time more people started to do research on the positive side of development, but again one-sided studies, of positive development in positive environments. But it is the power of the idea of differential susceptibility, that it covers both streams of research.

So tell me how you're linking it with

attachment here today.

It's quite a short talk, so I decided to talk about some of our recent research on attachment, especially how adult attachment representations are related to responses to infant crying and infant laughter, and how that might be influenced by oxytocin. We are doing a series of studies with oxytocin sniffs. We're interested in how people with a certain attachment style end up being harsh to their crying child, or remain calm and sensitive. Oxytocin might be one of the key issues in the chain from cognitive representations to behaviour.

The idea being that it's a kind of chemical spotlight, it makes social cues more salient?

Oxytocin is a hormone and neurotransmitter that is still not really determined in terms of its function. It's being considered the 'love hormone', or the 'cuddle chemical', but again that's a one-sided view. What we are finding is that it lowers the activity of fear centres, such as the amygdala, and elevates the activity of reward centres, such as the orbito-frontal cortex and the anterior cingulate cortex. But it is not effective in all people – we find that those with negative attachment experiences are less open to the effects of oxytocin. How that comes about, that's one of the big puzzles we are working on now.

A researcher once did the adult attachment interview on me, and said I was the most dismissively attached person they had met!

What would you predict for my reaction to oxytocin, and to my poor crying children?

Amazingly unscientific! The interview is not meant to conduct individual diagnoses, errors of measurement simply forbid it. Well, what I'm going to present is that without oxytocin, insecurely attached adults feel firstly more irritated by infant crying behaviour. Secondly we have a hand-grip measure, we teach the participants to exert full force and then we teach them to go for half-strength. They manage to do that. And then we have them listen to cry sounds. Insecurely attached individuals exert excessive force more often than the securely attached parents, when listening to this aversive crying.

So that's not necessarily expressing anger, it could be discomfort, that they find that more aversive.

Yes, and what in practice the response

"there is a gap between brain and behaviour, which is very intriguing"

would be. It could be an alarm signal that is more pronounced, it might also trigger harsh parenting. Crying behaviour is primordial attachment behaviour, it's one of the first behaviours that an infant can show to display discomfort, distress, stress... it's a proximity seeking behaviour, which John Bowlby wrote about quite extensively. But it's also a trigger for harsh parenting and child abuse, an epidemiological study showed that aversion to crying in the first half year is the stimulus for about 6 per cent of young mothers to slap the child, to smother the child, to really go into the direction of child abuse. You might imagine that it's a powerful trigger, because persistent crying is really a nuisance, that's for sure, for any person, but some people might have a lower threshold to react in a harsh way. What we see in the scanner is that listening to cry sounds compared to control sounds elevates the level of amygdala activation, because it's aversive, and oxytocin lowers that level of activation. What we hoped to find was that it's a mediating mechanism between attachment representation to those feelings of irritation and excessive force on the hand grip. We didn't find that, so there is a gap between brain and behaviour, which is very intriguing.

So it's not as simple as intervening on that pathway with a sniff of oxytocin when your baby starts crying.

Exactly. We can't really connect the three parts of the equation to each other... that's not unique to our lab, in the fMRI area the dominant paradigm is looking at the brain as a dependent variable, so what happens in the brain is the end product of a series of stimuli. For me this is totally unimportant, because what happens between your ears, no child or infant will ever see. What's important is how specific brain activity is expressed in behaviour. I'm interested in parenting, in child behaviour, I would like to know how brain activation affects parenting style and how it's made visible to the child, shaping the course of development. That's really a big puzzle still, and not even addressed in a lot of the neuroscientific studies on parenting.

A lot of your research is still very hands on with children and parents and behaviour, it's important not to take it that level of abstraction too far.

These are absolutely fantastic times to study parenting, with big advances in genetics, in brain research, in hormonal



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research. It all creates lots of opportunities to look at the mechanisms, the processes of how exactly parents are influencing children in their development, but we should refrain from using those types of devices for their own sake, because it's fancy, because it's creating these nice pictures. It's always a means to a goal and that goal is to understand better what happens between parents and children and to know more about how to intervene in families with an environment that is not so great for the child to grow up in.

In terms of creating positive environments, what do you think we can learn from the Scandinavians, given that we're here in Sweden? In the UK I think people look to here for an example of how it should be done; whereas in the UK some people say 'we go out to work and get paid badly so that we can pay other people badly

to look after our children badly'.

In Scandinavia the parents really seem to have the choice to be at home, both parents, to care for their infant in the first year of life. In the UK and other countries, maternity and particularly paternity leave is very brief, so people are obliged to have other forms of care.

From the perspective of attachment theory there's a misunderstanding around the concept of monotropy. There's lots of evidence that children are able to grow up in a network of attachment relationships. Sarah Hrdy has written about the survival value of alloparenting. It takes more than one person simply to collect all the food needed to have a child growing up to a reproductive age. You need a village to raise a child. So nothing against more caretakers in the environment of the child. The point is that in the first year of life, children are easily overwhelmed by all kinds of stimuli, they need more structure than older children. They are dependent on the moderation of stress by persons in the environment that they can rely upon. Attachment figures are in fact external stress moderators for infants in the first year of life.

Now if you put them into group care, that's really quite some stressful experience. Some children won't be bothered at all, depending on their temperament, but others will be. If parents don't have the choice of staying at home, I think that's bad, that's not enough options for parents of susceptible children who might need to be at home.

The 'orchids'?

Maybe, yes. So that's what I find distressing in the US, the UK, in Holland, that parents don't have the freedom to choose.

The second issue is of course quality of care. You can have bad care in both family and daycare environments with detrimental effects on the most susceptible children. In Scandinavian countries they seem to have strong regulations, and monitoring of them, to keep quality of care high. That's a lesson we should learn. Young children are worth this investment, according to economists like Heckman.

You've researched a huge range of topics, from the aftermath of genocide through sleeping children to adoption. What's the common thread, what values drive you in that work?

The most important perspective is the influence of the environment, parenting and the family context on child

development. You know of course the book by Judith Harris, that parents are not important at all because it's all genes which drives development? There's now a very popular book in Holland by Dick Swaab called *We Are Our Brains*. So in the past 20 years we witness a very strong main current that defines child development as a kind of autonomic process driven by genes and brain structures, with only marginal influence for the environment – prenatally, maybe, but whether after birth the work is really done, that's what I doubt.

So you're driven to counter that at a personal level as well as a scientific level?

Neglect of the environment is a big misunderstanding and, in the end children suffer... That's what drives me. If you follow it through, with the brain as ultimate cause of any developmental process, you can't even criticise that children grow up in an orphanage environment. We did studies in orphanages in different parts of the world, and what we see is that for every single month they stay in that environment there is a bigger lag in their development of weight, height and head circumference compared to their peers growing up in families. Cognitive development goes into the range of mental retardation. If they are adopted, you see a tremendous catch-up in cognitive development, IQ recovers to a normal level, a difference of 15–20 IQ points. This would be hard to explain on the basis of genes and brains being the causal drivers of development.

That recovery, that resilience, brings us back to the positive and negative effects of the environment... it's not too late to intervene.

Differential susceptibility theory makes clear that some children are quite robust, it doesn't matter too much what environment they're raised in as long as it meets minimum standards. That's quite hopeful because there's quite a few 'just

good enough' environments around. But there are also a lot of children who are very open to environmental pressures, these orchid children, who would really flourish in a better environment. That potential is going to be wasted if we feel it's only genes and brains that create development. It's a waste of talent, a waste of potential, if we are seduced by a deterministic view of child development.

Genes are important, but it's the interplay with the environment, and too often that's lip service – on the part of those who study the genes, and on the

taking you next?

There's distressingly little experimental research done on gene by environment interactions. We were the first to do a gene by environment experiment – changing the environment and seeing how that interacts with genes. It's so much more powerful statistically. We have to work on better assessments of the environment, better assessments of genotype (for example genetic pathways), but also better designs to be able to really test and examine differential susceptibility and gene by environment interplay in general. So what we would like to do are large-scale experimental studies in which we have a closer look at the mechanism itself. We plan to use fMRI as a pre- and post-test assessment, to see if differences in brain activation mediate the effects of our intervention on the behaviour of parents and children. It is trying to get a more detailed and mechanistic view of how interventions work more effectively in certain subgroups of participants who are more open to the environment.

I think we're going to continue our work with the oxytocin sniffs, because it's intriguing how it is moderated by childhood experiences. It's still shown in only three or four experimental studies. This is shaky in terms of the assessment of childhood experiences, so the first step will be to see how it is moderated by adult attachment representations, but it would be great if we could also include it in longitudinal studies where we may observe negative childhood experiences moderating the effects of oxytocin.

Is there a lot of funding in that area?

We just received a seven million euro grant from the national science foundation to conduct experimental studies on differential susceptibility. But the pharmaceutical industry is not really interested in our oxytocin research. I just read Ben Goldacre's *Bad Pharma* and felt lucky that industry does not see any profit in oxytocin. Maybe this is the reason why published results of oxytocin studies are diverging and sometimes disappointing. Our recent meta-analysis in *Translational Psychiatry* shows the problems of clinical applications of oxytocin. I love to do independent research because it is difficult enough without a big company looking over your shoulder and having an interest in the outcome.



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part of those who study the environment. You need a concept like differential susceptibility to study, in detail, the interactions between the two facets.

Have you had your own behavioural genes assayed, or would you not think that's important because it all depends on the interplay anyway?

These concepts, genes and environment, they all work on the level of samples, they don't work on the level of the individual. It's a misunderstanding if you feel one might predict the individual course of life on the basis of candidate genes, one gene in more than 20,000, without any insight into the environment... but even if you had exact information about the environment past and present, I still think on the individual level prediction would be quite disappointing.

Looking to your own future, can you predict where this research path is

reading

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