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Don't throw the bathwater in with the baby

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Abstract

The Presidential Address of the European Association of Developmental Psychology this year concerned how genes and environments interplay to shape loneliness and other developmental psychological relevant outcomes. This is a very welcome. However, when developmental psychology now is ready to integrate recent genetic and neuroscience knowledge and methods, I think it would be very wise to not uncritical go through the mistakes that have been done in other disciplines and instead learn from their hard lessons. I discuss some problems (genes versus environments, identifying gene for a phenotype, environments cause of developmental outcomes, and gene X environment interaction and epigenetics) and some suggestions for solutions which can be used to avoid throwing in the bathwater with the baby.

The Presidential Address of the European Association of Developmental Psychology this year concerned how genes and environments interplay to shape loneliness and other developmental psychological relevant outcomes. This is a very welcome. If we want to make progress in our understanding of the human nature we need to integrate both genetic and environmental research in our models and theories. When this is done, and the ideas and suggestions from the President are taken seriously by the members of the Association, it is likely that the quality of the research in developmental psychology would be greatly improved. However, when developmental psychology now is ready to integrate recent genetic and neuroscience knowledge and methods, I think it would be very wise to not uncritical go through the mistakes that have been done in other disciplines and instead learn from their hard lessons. Here are some problems that maybe can be avoided together with some ideas on what I believe is possible solutions.

Genes versus environments

The problem

In many disciplines, as well as in the public debate, there has in many cases been a conflict between opponents and proponents for genetic (or environmental) explanatory models, where the question often has been if it is genes **or** environments. Today (as most often previously) everyone “knows” that it is both genes **and** environments, and we have to understand their interplay. Nevertheless, even though this is kind of a truism, it is remarkable that some scholars just have this as a lip service, and that they anyway get stuck in their old paradigm.

Suggested solution

What is needed for true advances in the field of developmental psychology is (as is implicit in the Presidential Address) to design studies that really takes this truism into account. When

doing this, it might be good to remember the three “Laws of Behavior Genetics” (Turkheimer, 2000).

1. All human behavioral traits are heritable.
2. The effect of being raised in the same family is smaller than the effect of the genes.
3. A substantial portion of the variation in complex human behavioral traits is not accounted for by the effects of genes or families.

Identify a gene for a phenotype (gene hunting)

The problem

After identifying genes for Mendelian (monogenic) diseases (e.g., for cystic fibrosis or Huntington), many researchers used the last decades of the 20th century to try to find genes also for complex disorders in general and also more specifically within the field of psychiatry and psychology. Even though there are some success stories (e.g., one gene, Apo E, is today a well-known risk factor for Alzheimer’s disease) the failures are much more common. In the Presidential Address, association studies are suggested as one important approach. However, even though it might be fruitful, the problems should not be underestimated. It is very easy to find an association that is significant at the 0.05% level and it is in principle always possible to find a plausible biological mechanism for a found association. The literature is cluttered with this type of studies, however, rarely replicated and in principle never present in meta-analyses, at least not when publication bias is taken into account.

Suggested solution

The state of the art today is genome-wide association studies (GWAS). These studies are using microarrays to test for the role of up to millions of common genetic variants. The problem is when doing this, you also do millions of association tests; therefore, extremely large samples are needed to identify genome-wide significant effects. But it is working! Success stories started with macular degeneration, and after that new genes (or single nucleotide polymorphism; SNPs, “snips”) have been identified at an increasing pace. For example, about 50 SNPs for both Crohn’s disease and prostate cancer have to date been identified; a complete catalog is continuously updated at <http://www.genome.gov/gwastudies>. These advances became possible only when researchers started to establish worldwide consortia. There were initially problems within psychiatric disorders, but when large enough samples were pooled, genes for, for example, schizophrenia and bipolar disorders have been presented (Sullivan, 2012). However, an important lesson is that no gene seems to explain much of the variance and no gene seems to be neither necessary or (at least up to now) sufficient to cause disease. Instead, up to thousands of genes seem to be of importance even for such a “well defined” disorder as schizophrenia (Purcell et al., 2009). Another lesson is that the “usual suspects” (that is, the gene variants that have been claimed in association studies) are not replicated. Worldwide consortia have been established also for psychologically relevant phenotypes (e.g., cognitive abilities and personality), and should be possible also within developmental psychology. Interestingly, when a cognitive ability consortium studied the 12 specific genetic variants claimed to be important for general intelligence was evaluated, it was again shown that results from the earlier underpowered association studies were not replicated when using three independent datasets including 10,000 of well-characterized individuals (Chabris et al., In press).

Environments cause of developmental outcomes

The problem

For a long time psychological, behavioral and epidemiological studies have used different types of environmental indices (e.g., SES, stressful life events) to identify the causes for developmental outcomes. However, we all know that correlation is not equal to causation and reviews have noted how little we know about the true causes of mental health in children because research is stuck in the “risk factor” stage (Rutter, 2007). As alluded in Law 2 of Behavior Genetics above, “environmental” factors used in our studies (life events, divorce etc) are all influenced by genetic effects (Kendler & Baker, 2007), or in other words; we (partly) create our own context. Thus, it is still unclear whether putative early risk factors are truly causal or whether part—or most—of the associations with these risks are due to confounding from familial (both genetic and environmental) effects. For example, it has long been “well-known” that children to mothers who have smoked during pregnancy are at increased risk for criminality and drug use - and teratogenic effects of nicotine have been suggested - until it was recently shown that the entire effect of smoking during pregnancy on these outcomes was explained by common familial effects (D’Onofrio et al., in press; D’Onofrio et al., 2010).

The solution

As suggested by the Presidential Address, to make progress we need to integrate different disciplines. Here this means that when we know that familial effects are important for our “environmental” risk factors, we need to use study designs that can account for this. Randomized controlled trial is one possibility, but for much of the research in developmental psychology this would be ethically or practically impossible (e.g., we cannot randomize fetuses to smoke during pregnancy). The alternative approach is to use informative natural experiments (quasi-experimental study designs), including twin, adoption, and family studies, or random environmental events like earth quakes or tsunamis. Clearly, a field that is basing their theories on observational studies from only one child per family run a risk of creating more confusions than solutions.

The importance of gene X environment interaction and epigenetics

Finally a word on the last (and coming(?)) decade genetic research fashion: gene X environment interaction and epigenetics. Well-known are the results from Caspi and colleagues with reports of findings of gene by environment interaction for conduct disorder and depression (Caspi et al., 2002; Caspi et al., 2003). The research on “attachment” in rodents and their epigenetic and phenotypic consequences (Kaffman & Meaney, 2007) should also be of considerable interest for researchers within developmental psychology. However, it should also be noted that when meta-analyses have been done on gene-environment interaction results, and publication bias has been taken into account, the claimed associations have not been replicated (Risch et al., 2009) even though the jury still is out (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010). Regardless if the published interactions are true or not, gene X environment interaction studies will suffer from the same problems as association studies, only that the power issues are even worse (Eaves, 2006).

The future

I doubt my crystal bowl is much clearer than anyone else’s, but I am rather confident that gene X environment interactions and epigenetic effects will be found for developmental outcomes. On the other hand, I am equally confident that these types of effects will explain a rather limited amount of the variance. Twin and family studies have for over 100 years shown that genes have a substantial impact and the bulk of the variance will probably even

in the future be explained by the main effects of genes and the nonshared environment. Regardless, it is hard to conclude anything different than the Presidential Address; the integration of developmental psychology with genetics and brain science is necessary and will greatly improve the quality of research, but I think we should try to avoid throwing in the bathwater with the baby and instead learn from others mistakes.

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