VU Research Portal

Reply to Turkheimer and Waldron and Wahlsten.

Vreeke, G.J.

published in

Human Development
2000

DOI (link to publisher)

[10.1159/000022657](https://doi.org/10.1159/000022657)

document version

Publisher's PDF, also known as Version of record

[Link to publication in VU Research Portal](#)

citation for published version (APA)

Vreeke, G. J. (2000). Reply to Turkheimer and Waldron and Wahlsten. *Human Development*, 43(1), 53-55.
<https://doi.org/10.1159/000022657>

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

E-mail address:

vuresearchportal.ub@vu.nl

Reply to Turkheimer and Waldron and Wahlsten

G.-J. Vreeke

Faculty of Social Sciences, University of Amsterdam, The Netherlands

Key Words

Analysis of variance · Behavior genetics · Cause and variation · Child rearing · Development · Dynamic systems · Interaction and additivity · Nature and nurture · Shared environment

To gain an overall picture of the role of the various sources that play a part in human development, it is surely in order to abstract from the intricate relations between genes, experience, and the environment. And surely there is no reason to suppose that one cannot learn something by using simplified models. Using an additivity model in a case where one knows that reality is interactive and complex (e.g., when the genesis of specific mental traits are at issue), one can still get a clue whether a particular variable makes a difference in a population and estimate the sense of its importance.

But, as I argued in my paper, it is quite another thing to make causal inferences, such as saying that genes and the unshared environment are largely responsible for the enduring features of our mental life, such as intelligence, personality, one's likes and dislikes, while the shared environment has hardly any causal effect on these traits. These conclusions, so I argued, are one station too far. It seems to me that making those inferences does require extra assumptions, and if these are not provided or are unavailable, one must settle for less grandiose conclusions.

Turkheimer and Waldron seem to agree, or don't they? They acknowledge that correlational statistical methods, as such, do not bear developmental conclusions. But they see this as a truth, which applies to all correlation-based studies. According to them, there is no difference between behavioral genetics use of the ANOVA method and its use in developmental psychology or any other field. So they object to my addressing

Editor's note: The action editor for this article was Claes von Hofsten.

KARGER

Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

© 2000 S. Karger AG, Basel
0018-716X/00/0431-0053\$17.50/0
Accessible online at:
www.karger.com/journals/hde

G.-J. Vreeke, Faculty of Social Sciences, Department of Education, University of Amsterdam, Wibautstraat 4
NL-1091 GM Amsterdam (The Netherlands)
Tel. +31 20 525 1242, Fax +31 20 525 1200
E-Mail Gertjan@educ.uva.nl

my criticism solely to the already much-plagued behavior geneticist. I think, however, that behavior genetics is a special case, and will try to spell out why I think so.

Turkheimer and Waldron appear to think that the relationship between correlational data and causal claims is fundamentally mysterious, at least in interesting and complex cases. They suggest that one should leave this matter as it is, which seems to imply that there is no answer anyway, adding that they and – indeed – I myself do not have an answer. I don't agree. The philosophical question of causality may be mysterious, but I do not think that the empirical issues related to correlation and causality are of this kind. On the contrary! Indeed, the section in my paper entitled 'Relating Variance and Development' is about that relationship. I shall restate the crux of my paper in this reply before turning to their remarks. I will conclude by briefly paying attention to Wahlsten's comments, which I take as a further confirmation of the concerns expressed in my paper.

My paper essentially addresses the role of the additivity assumption in behavior genetics. This assumption figures as a model which functions in connecting data with reality, or rather is taken as such. The additivity assumption says basically that genes and the environment can be seen as separately contributing to behavior. Both material and statistical claims support this assumption. These different types of claims are conceived as matching, that is, as mutually supportive, or at least as noncontradictory. Now, because those claims are held to match, the variables in behavior genetic research (genes, environment) can be seen as somehow representing material substances and thus be the proper element for entering a causal relationship. This would then allow the behavior geneticist to interpret data about variation in terms of causal effects (provided of course that limitations of method and design are reckoned with). What breeds trouble is the fact that the additivity model can hardly be seen as one that depicts the actual relationship between genes and the environment. In the study of humans, the notions of genes and of the environment can only function as abstract concepts that do not pick out content in a univocal way. And as animal studies indicate, the genes and the environment are – in a complex way – interdependent. These problems for the additivity model give rise to the question in what way the quantitative data of behavior genetics are to be interpreted in material (causal) terms.

Notice, however, that I did not conclude that these problems are 'nails in the coffin' of behavior genetics, as some critics would have it. Instead I argued that those problems can be seen as ones of interpretation. Moreover, I observed that most behavior geneticists do somehow accept the premises of this conclusion, even though they do not spend much time in seeking resolutions. Maybe they think, with Turkheimer and Waldron, that there are no answers to be found. Or perhaps they simply suspect interpretation. In the section of my paper 'Relating Variance and Development', I seek to find ways for dealing with the issue of interpretation. I envisage two types of directions to be taken. One actually shifts the interpretative issue (somewhat) to the background. According to this prospect, one should seek forms of statistical analysis that do match the material relation between genes and the environment, and which by that token would allow for developmental conclusions. The other, presumably more realistic, option is one of articulating an interpretive device that allows for translating variance in terms of development.

With this elucidation in mind, I can answer Turkheimer and Waldron's question of why I address my criticism of the causal use of ANOVA solely to behavior genetics research. I do not think the problem of relating variance and development applies ex-

clusively in the context of behavior genetics. But I do hold that behavior genetics is a special case. This is so because the additivity model is to a large extent *founded* upon correlation based statistics. In this sense ANOVA is not just another tool, it is part of the theoretical foundation of behavior genetics. This is generally not the case in developmental psychology or any other field, which usually utilizes separate causal theories as models to relate data and reality. However, note that in cases of absence of causal models to match a research design, causal inferences are usually seen as problematic, and rightly so! Non-experimental research may document that people who are fond of spinach live longer compared to people that never eat spinach, but does this allow for the conclusion that eating spinach *makes* one live longer? Who would not think that this is a hasty conclusion, much in need of empirical confirmation of a different kind? If on the other hand one starts from a (causal) model, things tend to be different. A researcher may predict that the emotional responsiveness of mothers toward their children leads to a secure attachment relation, for reasons that are clearly specified in a theory. In this case the model allows one to interpret correlations between responsiveness and secure attachment in causal terms. Of course, the model might be false, but it does offer further reasons, which are, or at least could be, of an empirical kind.

I turn now to the paper by Wahlsten. Wahlsten's paper is largely in support of my main thesis, that there is a gap between the assumptions that are basic to behavior genetics (the additivity model) and the reality that it is supposed to map (which is generally seen as interactive). Wahlsten offers important further reasons for holding on to this thesis. He points out in some detail how it is possible to partition variance in many situations 'even though the antecedent causes do not act separately during development' (Wahlsten, this issue). Wahlsten furthermore suggests in what way we might be able to map – with the aid of the ANOVA method – the interplay between genetic factors and the environment. His suggestion, however, does not offer much comfort for human behavior genetics, for Wahlsten holds that an experimental design is mandatory and adds a number of conditions related to sample size. Without the possibility to identify genes, however, there is no way to conform to these requirements. I can only add that in the meantime we should at least seek an interpretative device that helps close the gap between variance and development.

Copyright: S. Karger AG, Basel 2000. Reproduced with the permission of S. Karger AG, Basel. Further reproduction or distribution (electronic or otherwise) is prohibited without permission from the copyright holder.