Direction of Causation Models

NATHAN A. GILLESPIE AND NICHOLAS G. MARTIN

Volume 1, pp. 496–499

in

Encyclopedia of Statistics in Behavioral Science

ISBN-13: 978-0-470-86080-9
ISBN-10: 0-470-86080-4

Editors

Brian S. Everitt & David C. Howell

Direction of Causation Models

In the behavioral sciences, experimental manipulation is often not an option when investigating direction of causation (DOC) and so alternative statistical approaches are needed. Longitudinal or two-wave data designs (*see* **Longitudinal Designs in Genetic Research**), while potentially informative, are not without their disadvantages. In addition to the cost and time required for data collection, these include stringent methodological requirements (see [3, 6]). When only cross-sectional data are available, a novel approach is to model direction of causation on the basis of pairs of relatives, such as twins measured on a single occasion (*see* **Twin Designs**) [1, 3, 6].

The pattern of cross-twin cross-trait correlations can, under certain conditions, falsify strong hypotheses about the direction of causation, provided several assumptions are satisfied (see [6]). One of these is that twin pair correlations are different between target variables, which is critical, because the power to detect DOC will be greatest when the target variables have very different modes of inheritance [3].

Figure 1 provides an illustrative example of DOC modeling based on cross-sectional data. Let us assume that variable A is best explained by shared (C) and nonshared (E) environmental effects, while variable B is best explained by additive genetic (A), dominant genetic (D), and nonshared (E) environment effects (*see* **ACE Model**). Under the "A causes B" hypothesis (a), the cross-twin cross-trait correlation (i.e., A_{t1} to B_{t2} or A_{t2} to B_{t1}) is $c_{AS}^2 i_B$ for MZ and DZ twin pairs alike. However, under the 'B causes A' hypothesis (b), the cross-twin cross-trait correlation would be $(a_{BS}^2 + d_{BS}^2) i_A$ for MZ and $(1/2 a_{BS}^2 + 1/4 d_{BS}^2) i_A$ for DZ twin pairs. It is apparent that if variables A and B have identical modes of inheritance, then the cross-twin cross-trait correlations will be equivalent for MZ and DZ twin

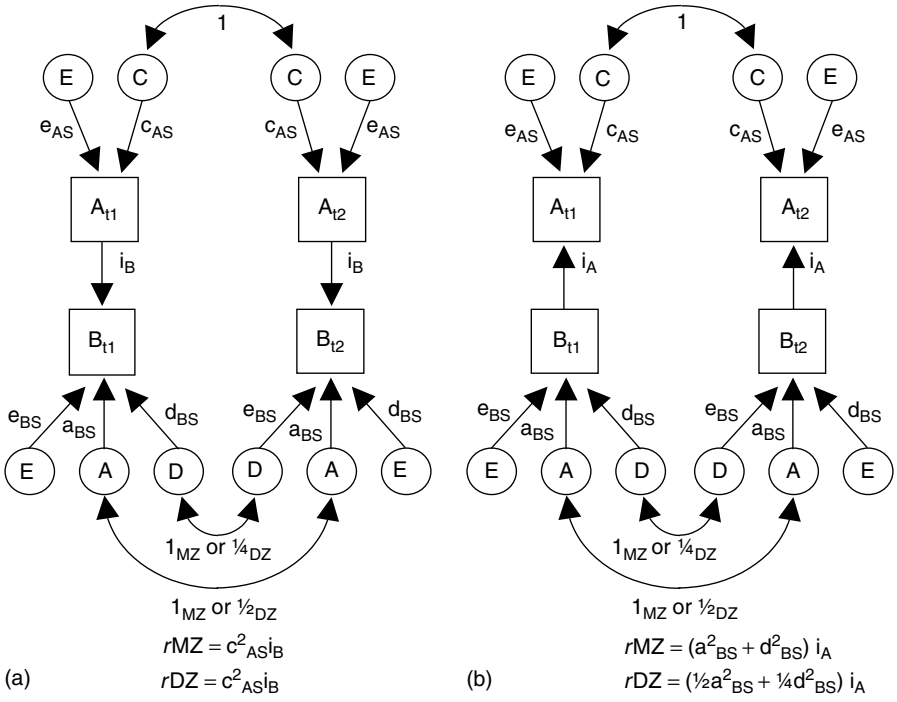

Figure 1 Unidirectional causation hypotheses between two variables A and B measured on a pair of twins. (a) Trait A causes Trait B and (b) Trait B causes Trait A. The figure also includes the expected cross-twin cross-trait correlations for MZ and DZ twins under each unidirectional hypothesis. Example based on simplified model of causes of twin pair resemblance in Neale and Cardon [5] and is also reproduced from Gillespie and colleagues [2]

pairs alike, regardless of the direction of causation, and the power to detect the direction of causation will vanish.

Neale and colleagues [7] have modeled direction of causation on the basis of the cross-sectional data between symptoms of depression and parenting, as measured by the dimensions of Care and Overprotection from the Parental Bonding Instrument [8]. They found that models that specified parental rearing as the cause of depression (parenting → depression) fitted the data significantly better than did a model that specified depression as causing parental rearing behavior (depression → parenting). Yet, when a term for **measurement error** (omission of which is known to produce biased estimates of the causal parameters [5]) was included, the fit of the 'parenting → depression' model improved, but no longer explained the data as parsimoniously as a common additive genetic effects model (*see* **Additive Genetic Variance**) alone (i.e., implying indirect causation).

Measurement error greatly reduces the statistical **power** for resolving alternative causal hypotheses [3]. One remedy is to model DOC using multiple

Table 1 Results of fitting direction of causation models to the psychological distress and parenting variables. Reproduced from Gillespie and colleagues [2]

	Goodness of fit					
Model	χ^2	*df*	$\Delta\chi^2$	Δdf	*p*	*AIC*
Full bivariate	141.65	105				−68.35
Reciprocal causation	142.12	106	0.47	1	0.49	−69.88
Distress[a] → Parenting[b]	152.28	107	10.63	2	**	−61.72
Parenting → Distress	143.13	107	1.48	2	0.48	−70.87
No correlation	350.60	108	208.95	3	***	134.60

Results based on 944 female MZ twin pairs and 595 DZ twin pairs aged 18 to 45.
[a]Distress as measured by three indicators: depression, anxiety, and somatic distress.
[b]Parenting as measured by three indicators: coldness, overprotection, and autonomy.
$*p < .05$, $**p < .01$, $***p < 001$.

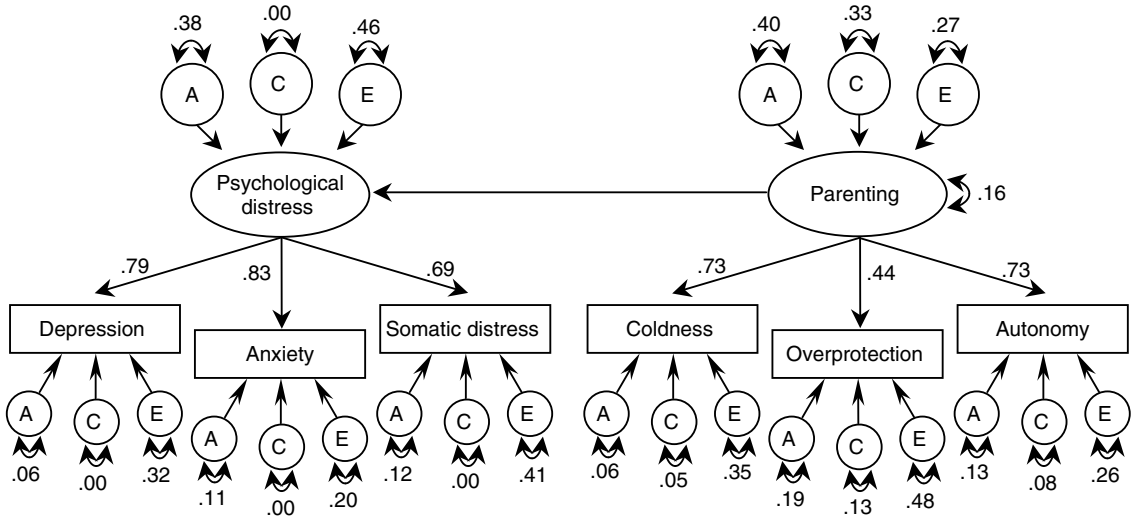

Figure 2 The best-fitting unidirection of causation model for the psychological distress and PBI parenting dimensions with standardized variance components (double-headed arrows) and standardized path coefficients. Circles represent sources of latent additive genetic (A), shared (C), and nonshared (E) environmental variance. Ellipses represent common pathways psychological distress and parenting. Reproduced from Gillespie and colleagues [2]

indicators [3–5]. This method assumes that measurement error occurs, not at the latent variable level but at the level of the indicator variables, and is uncorrelated across the indicator variables [5]. Gillespie and colleagues [2] have used this approach to model the direction of causation between multiple indicators of parenting and psychological distress. Model-fitting results are shown in Table 1.

The 'parenting → distress' model, as illustrated in Figure 2, provided the most parsimonious fit to the data. Unfortunately, there was insufficient statistical power to reject a full bivariate model. Therefore, it is possible that the parenting and psychological distress measures were correlated because of shared genetic or environmental effects (bivariate model), or simply arose via a reciprocal interaction between parental recollections and psychological distress. Despite this limitation, the chief advantage of this model-fitting approach is that it provides a clear means of rejecting the 'distress → parenting' and 'no causation' models, because these models deteriorated significantly from the full bivariate model. The correlations between the parenting scores and distress measures could not be explained by the hypothesis that memories of parenting were altered by symptoms of psychological distress.

Despite enthusiasm in the twin and behavior genetic communities, DOC modeling has received little attention in the psychological literature, which is a shame because it can prove exceedingly useful in illuminating the relationship between psychological constructs. However, as stressed by Duffy and Martin [1], these methods are not infallible or invariably informative, and generally require judgment on the part of the user as to their interpretation.

References

[1] Duffy, D.L. & Martin, N.G. (1994). Inferring the direction of causation in cross-sectional twin data: theoretical and empirical considerations [see comments], *Genetic Epidemiology* **11**, 483–502.

[2] Gillespie, N.G., Zhu, G., Neale, M.C., Heath, A.C. & Martin, N.G. (2003). Direction of causation modeling between measures of distress and parental bonding, *Behavior Genetics* **33**, 383–396.

[3] Heath, A.C., Kessler, R.C., Neale, M.C., Hewitt, J.K., Eaves, L.J. & Kendler, K.S. (1993). Testing hypotheses about direction of causation using cross-sectional family data, *Behavior Genetics* **23**, 29–50.

[4] McArdle, J.J. (1994). Appropriate questions about causal inference from "Direction of Causation" analyses [comment], *Genetic Epidemiology* **11**, 477–482.

[5] Neale, M.C. & Cardon, L.R. (1992). Methodology for genetic studies of twins and families, *NATO ASI Series*, Kluwer Academic Publishers, Dordrecht.

[6] Neale, M.C., Duffy, D.L. & Martin, N.G. (1994a). Direction of causation: reply to commentaries, *Genetic Epidemiology* **11**, 463–472.

[7] Neale, M.C., Walters, E., Heath, A.C., Kessler, R.C., Perusse, D., Eaves, L.J. & Kendler, K.S. (1994b). Depression and parental bonding: cause, consequence, or genetic covariance? *Genetic Epidemiology* **11**, 503–522.

[8] Parker, G., Tupling, H. & Brown, L.B. (1979). A parental bonding instrument, *British Journal of Medical Psychology* **52**, 1–10.

NATHAN A. GILLESPIE AND NICHOLAS G. MARTIN