Direction of Causation Models

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Caveats:

Hints, Allegations and things that must be said

The Direction of Causation is based upon the cross-twin cross-trait covariance

- In a twin model, the expected covariance matrix would be different if A causes B or if B causes A.
- If the cross-twin cross-trait covariance mimics the pattern of transmission of trait A, then A causes B

Power & Identification: To estimated causality you must have different modes of transmission

- ACE vs AE

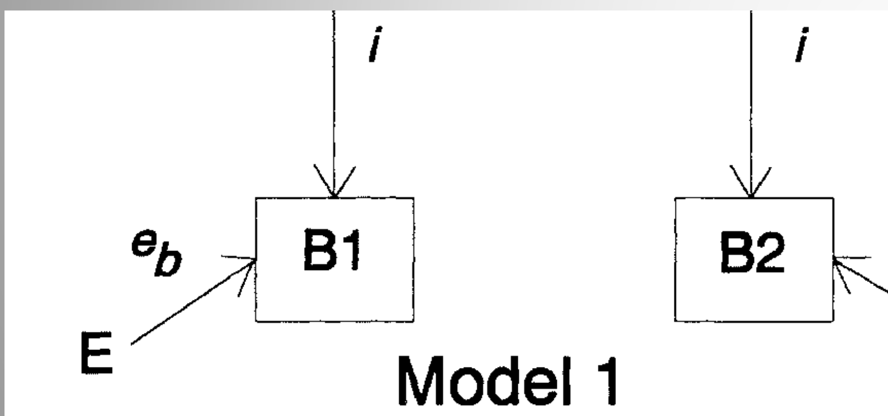
Measurement Error can drastically affect the estimation of the causal paths

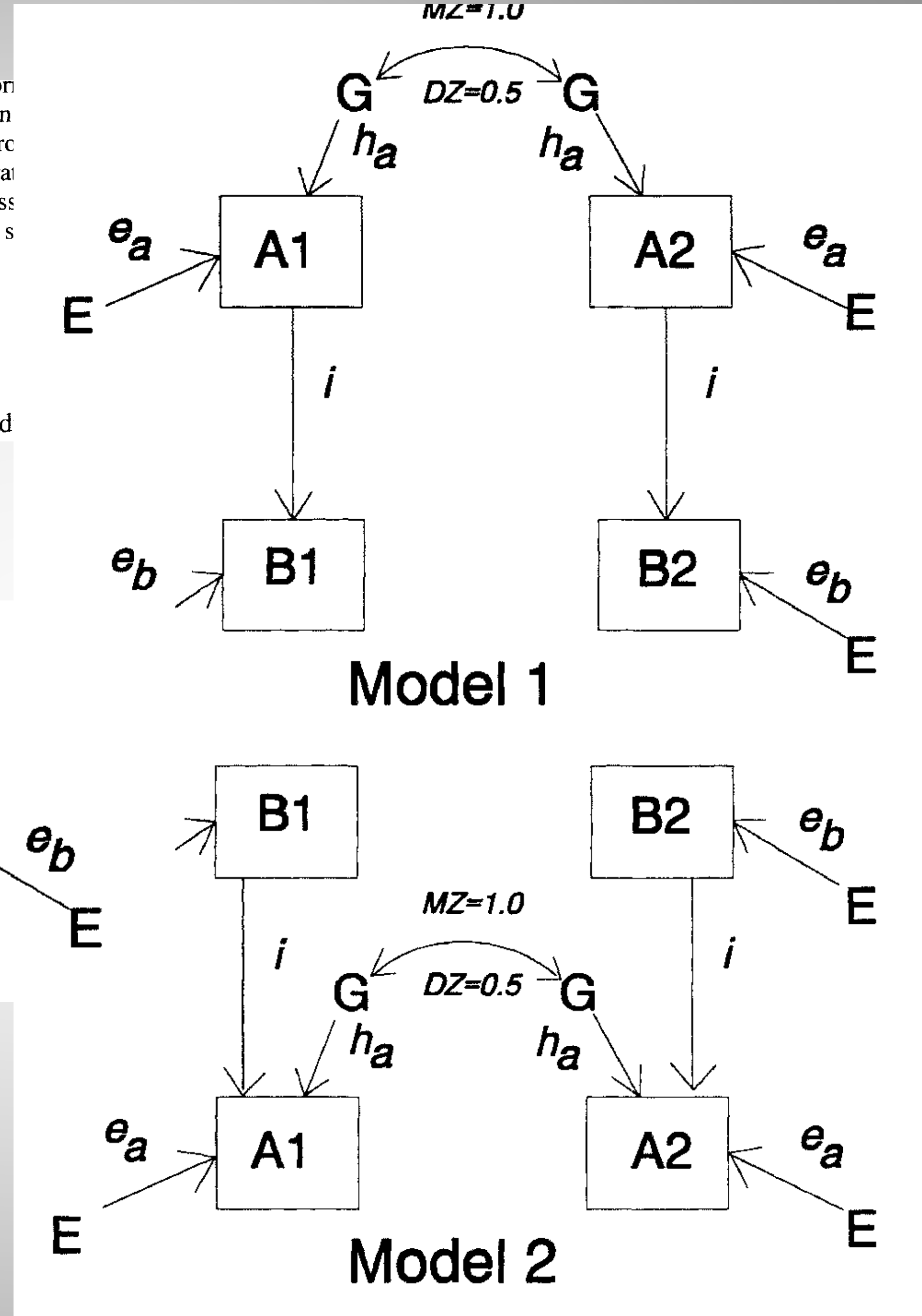
known. Failure to correctly specify a measurement model can lead to incor
of hypotheses. Difficulties can also occur when discriminating between
causative relationship and a correlation due to common genetic or enviro
determinants, but these occur in predictable situations. If these considera
taken into account in interpretation of results, the true nature of the ass
between traits can often be correctly identified, or at least included in a s
of best fitting models.

Key words: twins, causation, smoking, alcohol intake, lung function

INTRODUCTION

A well known shortcoming of cross-sectional and even longitud





or practically.

KEY WORDS: Twins; reciprocal causation; genetics.

INTRODUCTION

It is widely acknowledged that the existence of a correlation between two variables, measured at a single point in time, has no necessary implications about causation (Fisher, 1958). There are many ex-

the early environment has a direct causal influence on risk of psychopathology ($B \rightarrow A$) or because current psychopathology is biasing recall of early experiences ($A \rightarrow B$), or alternatively, both these processes may be operating simultaneously (recip-

Heath, Kessler, Neale, Hewitt, Eaves

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Table IV. Sample Sizes (*N* of Twin Pairs) Required for 80% Power of Rejecting False Unidirectional Hypothesis, for Selected Sets of Parameters Values of True Models[a]

	Trait *A*				Trait *B*			True model																
								$A \rightarrow B$ (Reject $B \rightarrow A$)								$B \rightarrow A$ (Reject $A \rightarrow B$)								
	e^2	h^2	d^2	c^2	e'^2	h'^2	c'^2	df	$i'=0.6$	$i'=0.5$	$i'=0.4$	$i'=0.3$	$i'=0.25$	$i'=0.2$	$i'=0.15$	df	$i=0.6$	$i=0.5$	$i=0.4$	$i=0.3$	$i=0.25$	$i=0.2$	$i=0.15$
I. No measurement error																								
1	0.25	0.5	0.25	0	0.25	0	0.75	1	93	114	157	248	337	503	861	2	111	141	195	313	432	652	1,127
	0.5	0.33	0.17	0	0.5	0	0.5	1	457	594	834	1,329	1,829	2,751	4,746	2	410	514	702	1,109	1,521	2,283	3,930
2	0.25	0.75	0	0	0.25	0	0.75	1	106	131	182	296	411	622	1,079	2	126	162	228	372	518	787	1,367

ORIGINAL ARTICLE

Testing Causality in the Association Between Regular Exercise and Symptoms of Anxiety and Depression

Marleen H. M. De Moor, MSc; Dorret I. Boomsma, PhD; Janine H. Stubbe; Gonneke Willemsen, PhD; Eco J. C. de Geus, PhD

Conclusion: Regular exercise is associated with reduced anxious and depressive symptoms in the population at large, but the association is not because of causal effects of exercise.

frontiers in GENETICS

ORIGINAL RESEARCH ARTICLE
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Regular exercise, subjective wellbeing, and internalizing problems in adolescence: causality or genetic pleiotropy?

Meike Bartels[1,2]*, Marleen H. M. de Moor[1,2], Niels van der Aa[1,2], Dorret I. Boomsma[1,2] and Eco J. C. de Geus[1,2]

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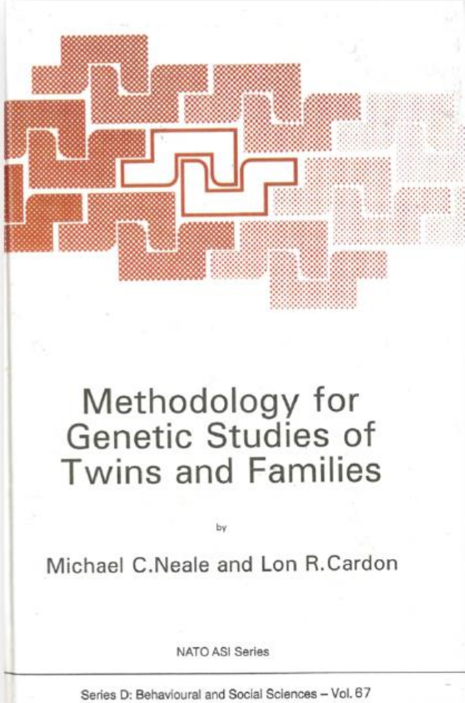
sectionally and longitudinally. We conclude that exercise behavior is associated with fewer internalizing problems and higher levels of SWB. The association largely reflects the effects of common genetic factors on these traits.

A Twin-Sibling Study on the Relationship Between Exercise Attitudes and Exercise Behavior

Charlotte Huppertz · Meike Bartels · Iris E. Jansen · Dorret I. Boomsma · Gonneke Willemsen · Marleen H. M. de Moor · Eco J. C. de Geus

present as well. Furthermore, after taking genetic pleiotropy into account, our data were compatible with a causal association between exercise attitudes and exercise behavior. Replication in longitudinal studies is now needed to more firmly establish this causality and its direction.



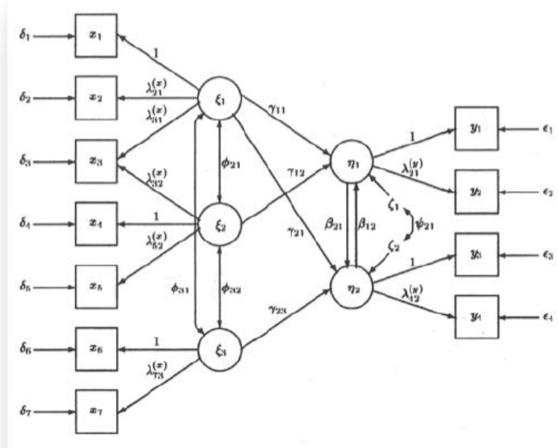


Figure 6.1: Path diagram for hypothetical model.

6.4.4 Submodel 3B: only y- and η-variables

When only NY and NE are specified, the program assumes the model

$$\mathbf{y} = \mathbf{\Lambda}_y \boldsymbol{\eta} + \boldsymbol{\epsilon} \tag{6.11}$$

$$\boldsymbol{\eta} = \mathbf{B}\boldsymbol{\eta} + \boldsymbol{\zeta} \tag{6.12}$$

or equivalently

$$\mathbf{y} = \mathbf{\Lambda}_y (\mathbf{I} - \mathbf{B})^{-1} \boldsymbol{\zeta} + \boldsymbol{\epsilon} \,, \tag{6.13}$$

with implied covariance matrix

$$\mathbf{\Sigma} = \mathbf{\Lambda}_y (\mathbf{I} - \mathbf{B})^{-1} \mathbf{\Psi} (\mathbf{I} - \mathbf{B}')^{-1} \mathbf{\Lambda}_y' + \mathbf{\Theta}_\epsilon \,.$$

```
## Covariance between the items due to the latent factors
FacCovMZ    <-    mxAlgebra( expression= FacLoadtw %&% (cause %&% symVmz), name="FacCovMZ" )
FacCovDZ    <-    mxAlgebra( expression= FacLoadtw %&% (cause %&% symVdz), name="FacCovDZ" )
```