Common effect models: Positive or negative manifold?

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Abstract

Observing exclusively positive associations among a set of variables (i.e., a positive manifold) is a robust finding in many areas in psychology. These positive associations can be explained by positing an underlying common cause or, alternatively, through positive direct effects among the variables. Recently, the Kruis-Maris model has been proposed as a third alternative mechanism to explain a positive manifold. In the Kruis-Maris model, observed variables are causes of a latent variable, such that conditioning on the latent variable induces associations between the observed variables. In this paper, we analyze the difference between the Kruis-Maris model and the more familiar causal indicator model. Both are examples of common effect models, but they imply different relations between the response variables and the effect variable. Whereas conditioning on the common effect in the Kruis-Maris model implies a positive manifold among the response variables, we show that conditioning on the common effect in the causal indicator model implies a *negative* manifold. We demonstrate this result for two specific cases: (1) in which both the response variables and the effect variable are normally distributed variables, and (2) in which the effect variable is normally distributed but the response variables are dichotomous variables.

Keywords: common effect models, positive manifold, negative manifold

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When variables are all positively associated ([Holland & Rosenbaum, 1986](#)) these variables are said to feature a positive manifold. A positive manifold is a common observation in variables that are intended to measure the same thing. For example, the test scores on a set of items that are all intended to measure math ability typically all correlate positively with each other. Other examples of data that feature a positive manifold include personality data ([Musek, 2007](#)), test scores on cognitive tasks ([Jensen, 1998](#); [Spearman, 1904](#)) and psychopathology data ([Caspi et al., 2014](#); [Lahey et al., 2012](#)).

The positive manifold is often explained through the hypothesis that observed variables are causal effects of one or a few unobserved common causes. This is naturally compatible with factor modeling ([Spearman, 1904](#)). Another explanation that can be shown to be equivalent to the most dominant factor modelling techniques is that observed variables correlate positively because they interact with one another ([van der Maas et al., 2006](#)). This alternative explanation has resulted in network modelling techniques in which the observed variables are nodes in a network that are connected by an edge when they are conditionally associated ([Costantini et al., 2015](#); [Epskamp, Borsboom, & Fried, 2018](#); [van Borkulo et al., 2014](#)). [Kruis and Maris \(2016\)](#) recently introduced a third explanation: observed variables correlate positively with one another because one may (unknowingly) have selected only cases for which a common effect of the observed variables is present. We refer to this model as the Kruis-Maris model, which is a specific type of common effect model. The Kruis-Maris model as an alternative explanation for the positive manifold is remarkable because it is directly opposite in conceptualization to the common factor model in which the observed variables are functions of the latent factor instead of the other way around. Yet [Kruis and Maris \(2016\)](#) showed their model to be equivalent to the Item Response Theory (IRT) model, which can itself be described as a factor modeling technique ([Takane & De Leeuw, 1987](#)). In fact, the Kruis-Maris model, IRT model and Ising model (a network model for dichotomous variables) are all mutually equivalent. This equivalence

is interesting because the models reflect very different explanations for the observation of solely positive correlations (i.e., dependence on a common cause, conditioning on a common effect and positive direct effects; [Kruis & Maris, 2016](#)).

In the current paper we zoom in on two types of common effect models: we compare the Kruis-Maris model to the more familiar causal indicator model ([Bollen & Lennox, 1991](#); [Edwards & Bagozzi, 2000](#)). The causal indicator model is also a common effect model, but is not equivalent to the IRT model. The goal of the paper is to demonstrate that while the Kruis-Maris model, in which the effect is a quadratic function of the response variables, implies a *positive* manifold, the causal indicator model, in which the effect is a linear function of the response variables, implies a *negative* manifold among the response variables when conditioning on the common effect. To be precise, a causal indicator model with a single common effect implies a conditional covariance matrix given the effect that is a diagonal matrix *minus* a rank one matrix. This is the opposite structure of what is implied by a common cause model: a unidimensional factor model implies that the covariance structure is a diagonal matrix *plus* a rank one matrix.

We start by explaining how common cause models and network models provide alternative explanations for the positive manifold and explain the equivalence between the IRT model (a type of factor model) and Ising model (a type of network model). We then introduce the Kruis-Maris model as a third explanation for the positive manifold and explain how this model is equivalent to the Ising model and IRT model. Thereafter, we introduce the causal indicator model, which is a linear common effect model, and compare this model to the Kruis-Maris model, which is a quadratic common effect model. We prove that while the Kruis-Maris model, just like a common cause model, implies a positive manifold, the causal indicator model implies a negative manifold when conditioning on the common effect. We finish the paper by discussing some implications of this result.

1 Common cause vs direct effects as explanations for the positive manifold

One explanation for a positive manifold among a set of response variables is that the response variables are all effects of an underlying common cause. This explanation is consistent with reflective measurement in which a latent variable is identified by observing multiple indicators that are assumed to be effects of the latent variable, such that the shared variance among these indicators reflects their common cause (Bollen & Lennox, 1991; Edwards & Bagozzi, 2000). For example, if differences in math ability lead to differences in test scores on several math items, the shared dependence on math ability induces positive correlations among the test scores on these items. The test scores on math items can be used as *measures* of math ability because it is assumed that a higher math ability results in a higher probability of giving a correct response to each item. Since people with a better math ability have a higher probability correct for all math items, the test scores on these items will correlate positively over persons. This common cause explanation can be represented in a statistical model in which the observed variables are a function of a latent common factor.

In Item Response Theory (IRT) the observed variables are dichotomous variables and the probability correct for each item is a logistic function of the latent variable. In factor models the observed variables are continuous variables that are a linear function of the latent variable. Any unidimensional factor model or IRT model implies that the observed variables can always be recoded such that all correlations between the observed variables become positive. The explanation for a positive manifold implied by a unidimensional factor model or IRT model, is that the common factor can be interpreted as a common cause of the observed variables. The left panel in Figure 1 shows an example of a common factor model with four response variables (X_1 to X_4).

Another way to explain a positive manifold among a set of variables, is to assume mutual direct effects among these variables (van der Maas et al., 2006). For example, for a set of symptoms of major depression it may well be that the symptoms directly influence each other in such a way that having some problems (e.g., suffering from insomnia and worrying a lot), leads to more problems (e.g., concentration problems and

negative thoughts; [Cramer & Borsboom, 2015](#)). To the extent that the presence of symptoms leads to the presence (rather than absence) of more symptoms, symptoms will be positively associated. This explanation for the associations between observed variables can be expressed in a network model.

Pairwise Markov random fields are undirected networks in which the edges represent conditional dependencies and the absence of an edge represents conditional independence between two nodes given all other nodes in the network ([Lauritzen, 1996](#)). The Ising model ([Ising, 1925](#)) is an undirected network with dichotomous variables and the Gaussian Graphical Model (GGM; [Lauritzen, 1996](#)) is an undirected network model with Gaussian variables. Both the Ising model and GGM satisfy the pairwise Markov property, such that nodes are connected with an edge when the corresponding variables are conditionally dependent given all other variables in the network. The right panel in [Figure 1](#) shows an example of a Markov random field with four response variables (X_1 to X_4).

While providing substantively different explanations for the positive manifold, the Ising model and Multidimensional IRT model have been shown to be mathematically equivalent ([Epskamp, Maris, Waldorp, & Borsboom, 2018](#); [Kruis & Maris, 2016](#); [Marsman et al., 2018](#); [Marsman, Maris, Bechger, & Glas, 2015](#)). In the next section we elaborate on this equivalence.

2 Equivalence of the Ising model and IRT model

We here briefly introduce the Ising model and the unidimensional IRT model to give an intuition of why they both imply a positive manifold and how they are equivalent. Let $\mathbf{X}$ denote a vector of p dichotomous response variables of which the i th element takes on the value $X_i = 1$ or $X_i = -1$, and let $\mathbf{x}$ denote its realization. In the Ising model, the variables in $\mathbf{X}$ are a function of their direct neighbors. That is, a variable X_i is a function of all variables that are directly connected to this variable by an edge,

$$P(\mathbf{X} = \mathbf{x}) = \frac{\exp(\sum_{i=1}^p \mu_i x_i + \sum_{\langle ij \rangle} \omega_{ij} x_i x_j)}{\sum_{\mathbf{x}} \exp(\sum_{i=1}^p \mu_i x_i + \sum_{\langle ij \rangle} \omega_{ij} x_i x_j)}, \quad (1)$$

Figure 1. Common factor model (a) and Markov random field (b). Squares represent observed variables and the circles represent latent variables. Model A represents a common factor model. The latent variables ε_1 to ε_4 represent the errors, so that ε_i accounts for the unique variance of X_i that is not explained by η . Model B represents a Markov random field; edges represent conditional dependencies, and missing edges represent conditional independencies given the other two variables in the network. More specifically, when the response variables are dichotomous, Model A represents a unidimensional IRT model and Model B an Ising model. When the response variables are multivariate normal, Model A represents a unidimensional factor model and Model B represents a GGM.

where ω_{ij} denotes the edge weight connecting X_i and X_j , μ_i denotes the intercept of X_i and $\sum_{<ij>}$ takes the sum over all distinct pairs of variables X_i and X_j ($i > j$). $\sum_{\mathbf{x}}$ takes the sum over all 2^p realizations of $\mathbf{X}$ so that the denominator functions as a normalizing constant. The connectivity matrix $\mathbf{\Omega}$ is a symmetric $p \times p$ matrix consisting of all edge weights between the variables. The diagonal of $\mathbf{\Omega}$ is undefined. Any connectivity matrix with positive edge weights implies a positive manifold between the variables in $\mathbf{X}$. An example is the Curie Weiss model (Kac, 1968), which is an Ising model with a single (positive) parameter for all edge weights (i.e., all variables are connected by an edge and all edge weights are constrained to be equal),

$$P(\mathbf{X} = \mathbf{x}) = \frac{\exp(\sum_{i=1}^p \mu_i x_i + \sum_{<ij>} \omega x_i x_j)}{\sum_{\mathbf{x}} \exp(\sum_{i=1}^p \mu_i x_i + \sum_{<ij>} \omega x_i x_j)}. \quad (2)$$

In the unidimensional IRT model, all variables in $\mathbf{X}$ are a function of a single latent variable η ,

$$P(\mathbf{X} = \mathbf{x} \mid \eta) = \prod_{i=1}^p \frac{\exp(x_i(a_i \eta + d_i))}{\sum_{x_i} \exp(a_i \eta + d_i)}, \quad (3)$$

where $\sum_{x_i}$ takes the sum over the two realizations of X_i . The item parameter d_i is an intercept and the item parameter a_i denotes the maximum slope of the item

characteristic function of X_i and is referred to as the discrimination parameter, where more discriminating items are characterized by a steeper slope. One can assume a distribution for the latent variable such that integrating over the latent variable in equation (3) to obtain the marginal probability distribution of $\mathbf{X}$ results in an Ising model with a connectivity matrix that is rank one (Marsman et al., 2015). In this case, all edge weights are a function of the p discrimination parameters. For any set of two variables for which the discrimination parameters have the same sign, the implied marginal association is then positive. This means that for the unidimensional IRT model (just like for its equivalent rank one Ising model) the response variables can always be recoded so that all marginal associations are positive (i.e., to obtain a positive manifold; Holland & Rosenbaum, 1986).

When all discrimination parameters in equation (3) are constrained to be equal, the so-called Rasch model (Rasch, 1960) is obtained. The Curie Weiss model is equivalent to a marginal Rasch model (Epskamp, Maris, et al., 2018; Kruis & Maris, 2016; Marsman et al., 2018). Both the Rasch model and Curie Weiss model use $p + 1$ parameters: p intercept parameters and one parameter for the slopes (Rasch model) or for the edge weights (Curie Weiss model).

The general equivalence between Ising models and multidimensional IRT models relies on two steps. Since (1) the diagonal of the connectivity matrix of an Ising model is undefined and thus can always be chosen so that a semi-positive definite matrix is obtained and (2) any semi-positive definite matrix can be expressed as an IRT model with as many latent variables as positive eigenvalues, there is an equivalent IRT model for any Ising model and vice versa. For a more extensive explanation of this equivalence we refer the reader to earlier papers (Epskamp, Maris, et al., 2018; Marsman et al., 2018, 2015).

3 The Kruis-Maris model

In a recent paper, Kruis and Maris (2016) propose a third model that is equivalent to the Ising model and IRT model. The Kruis-Maris model reflects another alternative

mechanism to explain the positive manifold, which is by conditioning on a common effect of the observed variables. [Kruis and Maris \(2016\)](#) show that if data are generated by an independence model (for which the state of one variable does not influence the state of another variable), but only cases are investigated for which a specific effect (a function of the observed outcomes) is present, a positive-manifold is obtained. That is, conditioning on the common effect induces positive associations that can be described by an Ising model with positive edge weights.

In this section, we provide an introduction to the Kruis-Maris model. Kruis and Maris present a formula for the joint distribution of $\mathbf{X}$, and a binary effect variable, E : $p(\mathbf{X} = \mathbf{x}, E = e)$. Here e denotes the realization of E , $e = 1$ indicating that the effect is present and $e = 0$ indicating that the effect is absent. The chain rule for probability gives us that the joint probability $p(\mathbf{X} = \mathbf{x}, E = 1)$ equals $p(\mathbf{X} = \mathbf{x}) \times p(E = 1 | \mathbf{X} = \mathbf{x})$. The Kruis-Maris model consists of these two probability distributions. We discuss each factor separately.

The first factor in the formula,

$$p(\mathbf{X} = \mathbf{x}) = \prod_{i=1}^p \frac{\exp(\mu_i x_i)}{\sum_{x_i} \exp(\mu_i x_i)}, \quad (4)$$

represents the marginal probability distribution of $\mathbf{X}$, in which μ_i denotes the intercept ranging from $-\infty$ to $+\infty$. This factor only includes intercepts and no interactions between the variables, so $\mathbf{X}$ is marginally independent. When μ_i is a large positive number then $x_i = 1$ results in a probability close to one and $x_i = -1$ results in a probability close to zero. When μ_i is a large negative number then $x_i = -1$ results in a probability close to one and $x_i = +1$ results in a probability close to zero. When μ_i equals zero, the probability for both possible values of x_i result in a probability of 0.5. The marginal probability distribution described above forms an empty Ising model in which all edge weights equal zero.

The second factor in the formula,

$$p(E = 1 | \mathbf{X} = \mathbf{x}) = \frac{\exp(\frac{1}{2}\omega[\sum_{i=1}^p x_i]^2)}{\exp(\frac{1}{2}\omega p^2)}, \quad (5)$$

implies that the probability of the effect being present depends on whether the variables in $\mathbf{X}$ are in the same state or in opposite states. The denominator functions as a normalizing constant so that when all variables are in the same state, the numerator equals the denominator and the probability for the effect being present equals one. Variables in the same state increase the probability of the effect being present while variables in opposite states decrease the probability of the effect being present. In the extreme, both response patterns in which all responses equal $+1$ as well as response patterns in which all responses equal -1 result in a probability of one for the effect being present, whereas a response pattern in which half of the responses equal -1 and half of the responses equal $+1$, results in a probability of one for the effect being absent. Conditioning on the effect being present implies that data patterns in which the response variables are in the same state have a high probability, thus inducing positive connections between the response variables. The conditional probability distribution of the response variables given that the effect is present is therefore similar to the probability distribution of the response variables implied by a Curie Weiss model in which the response variables also have thresholds and also tend to be in the same state.

The fact that the effect variable here depends on whether variables are in the same state or in opposite states is the result of the square in the formula which results in a quadratic relation between the effect and the response variables. If the function in equation (5) would be a linear function, the probability of the effect would increase as more responses equalled $+1$, and decrease as more responses equalled -1 . [Kruis and Maris \(2016\)](#) show that the unidimensional Kruis-Maris model as presented above is equivalent to a Curie Weiss model, and the Ising model is equivalent to a Kruis-Maris model with multiple orthogonal effect variables.

4 Kruis-Maris models vs causal indicator models

In the structural equation modeling literature, psychometric models in which the latent variable is a common effect of the observed variables are not new ([Bollen & Bauldry, 2011](#); [Bollen & Lennox, 1991](#); [Edwards & Bagozzi, 2000](#); [MacCallum &](#)

[Browne, 1993](#)). In the models described in this literature, the common effect variable η is a linear function of the response variables:

$$\eta = \mathbf{x}^\top \boldsymbol{\gamma} + \zeta = \gamma_1 x_1 + \gamma_2 x_2 + \dots + \gamma_p x_p + \zeta. \quad (6)$$

In this equation ζ denotes the disturbance on the latent variable η , that is unexplained by the response variables $\mathbf{X}$. This model is typically referred to as a causal indicator model, since the response variables are indicators that cause the latent variable, although it is also known as the formative model, reflecting the idea that the latent variable is “formed” by the observed variables ([Edwards & Bagozzi, 2000](#)). An example with four observed variables is given in Figure 2. While the notion of common effect models is thus well established within psychology, it is interesting to notice that the models from this tradition are different from the Kruis-Maris model. We use this section to compare these two common effect model frameworks.

Although both the Kruis-Maris model and the causal indicator model include a latent variable that is a function of a set of observed variables, the relation between the effect variable and the response variables in the Kruis-Maris model described earlier is very different from the relation between the causal indicators and the latent common effect in the causal indicator model. Here we explain how these models differ and show that the models imply different conditional associations between the response variables. In the literature on causal indicator models both the latent effect and the observed causes are typically assumed to be Gaussian variables, although sometimes also categorical causal indicators are included ([Edwards & Bagozzi, 2000](#); [MacCallum & Browne, 1993](#)). The latent effect is a linear function of the causal indicators, which is different from the Kruis-Maris model in which the presence of the effect is primarily dependent on whether the causes are in the same state or in opposite states from each other.

Conditioning on the effect in the Kruis-Maris model induces positive associations between the response variables because the effect being present implies that the variables are likely to be in the same state. Conditioning on the common effect in a causal indicator model, however, induces negative associations. If two variables X_1 and

X_2 are marginally independent but both positively affect a third variable X_3 , conditioning on X_3 induces a negative dependency between X_1 and X_2 (Elwert & Winship, 2014; Pearl, 2000).

As an example of the linear causal indicator model, suppose that ‘complexity of the topic’ (X_1) as well as ‘perfectionism of the student’ (X_2) both positively predict the time it takes for a student to finish an assignment (X_3). Further, suppose that the perfectionism of the student is not related to what topic is assigned to this student so that X_1 and X_2 are marginally independent. The observation that a student handed in the assignment quickly induces a dependency between the complexity of the topic and the perfectionism of this student: observing that this student actually had a complex topic makes it less likely that this student is perfectionistic, and observing that the student is perfectionistic leads to the inference that the topic must have been rather simple, given the small amount of time that the student put in the assignment.

That conditioning on a common effect induces bias is well-known. This bias is relevant in several commonly occurring situations that involve conditioning on a common effect. For example, when one studies relations between cognitive tasks in a sample in which only people with an IQ score higher than some cut-off value are selected, or when one studies the relations between symptoms in a sample in which only people with a diagnosis are selected. In these cases one conditions on a common effect, and because in these examples the variables that are being considered all positively affect the common effect, this results in spurious negative relations between the observed variables that bias the existing correlations between these variables.

The above examples comply with the causal indicator model rather than with the Kruis-Maris model. This is the case because in these examples the effect variable is a linear function of the variables in $\mathbf{X}$, instead of being dependent on whether the variables are in similar or opposite states as is the case in the Kruis-Maris model. This difference between the common effect models becomes even more clear from taking a closer look at the implications of the Kruis-Maris model for one of these examples. Let the dichotomous causes X_1 to X_p represent the presence/absence of symptoms and let

the dichotomous effect variable represent whether someone is diagnosed. A linear common effect model would imply that the probability of a diagnosis increases as the number of symptoms increases.

The Kruis-Maris model, in contrast, implies that individuals for whom all symptoms are present have the same probability of being diagnosed as individuals for whom none of the symptoms are present, because for both these groups all symptoms are in the same state (all present or all absent). There may be causes and effects that follow the implications of the Kruis-Maris model, but for many examples in psychology a linear relation between the causes and effect seems more plausible. Because many of the examples of selection bias encountered in psychological research concern a linear relation between the causes and the effect (e.g., selecting people with a high sum score), we take the next section to formulate more formally what the effect is of conditioning on a common effect in the causal indicator model.

Figure 2. Causal indicator model. The latent common effect η is a linear function of the four observed variables $X_1 - X_4$. The double headed arrows that connect the observed variables represent the correlations between these variables.

5 Causal indicator models imply a negative manifold when conditioning on the common effect

In the previous section we explained that conditioning on a common effect that linearly depends on two marginally independent variables induces a negative association

between the two causes (Pearl, 1988). We show here that the causal indicator model with a single normally distributed common effect variable that is a linear function of its causes, implies that all conditional covariances among the causes given the effect are negative. Just like Kruis and Maris (2016), we assume marginally independent causes. We conjecture that this result holds for all models in which the effect is a linear function of the predictor variables, but we here show it for two specific instances. We show that the conditional associations between the predictor variables given the effect variable are all negative for (1) the case in which both the predictor variables and the effect variable are normally distributed, and (2) the case in which the predictor variables are dichotomous variables and the effect variable is a normally distributed variable. We call the observation of solely negative associations a *negative manifold*.

Let the common effect variable η be a linear function of a vector $\mathbf{X}$ of p random variables X_i , as in equation (6):

$$\eta = \mathbf{x}^T \boldsymbol{\gamma} + \zeta,$$

where $\mathbf{x}$ is a vector of realizations of $\mathbf{X}$, $\boldsymbol{\gamma} \in \mathbb{R}^p$ is a vector of regression coefficients, and $\zeta \sim \mathcal{N}(0, \sigma^2)$. That is, the conditional distribution $p(\eta \mid \mathbf{x})$ is normal with mean $\mathbf{x}^T \boldsymbol{\gamma}$ and variance σ^2 .

Assuming that the predictor variables are independent of each other but all predict a single common effect, we conjecture that whenever the γ parameters are either all negative or are all positive, negative associations arise between the predictor variables $\mathbf{X}$ when conditioning on η . More specifically, the matrix of associations between the variables in $\mathbf{X}$ when conditioning on η is a diagonal matrix *minus* a rank one matrix in the case of normally distributed response variables or a zero matrix *minus* a rank one matrix with an undefined diagonal in the case of dichotomous response variables. Because the matrix that is subtracted is of rank one, the predictor variables can always be recoded such that a negative manifold arises, regardless of the sign of the γ parameters. We prove this for two particular cases, which we state as Theorems 1 and 2. In Theorem 1 we consider the case where the predictors X_i are independent normal random variables.

Theorem 1 *If the variable η is defined as in equation (6) using p independent normal predictor variables*

$$X_i \sim \mathcal{N}(\mu_i, \tau_i^2),$$

and the γ parameters all have the same sign, the conditional distribution $p(\mathbf{X} \mid \eta)$ implies a negative manifold.

The proof of Theorem 1 is given in Appendix A. Theorem 2 mirrors the results for Theorem 1 for the particular case of dichotomous predictor variables.

Theorem 2 *If the variable η is defined as in equation (6) using p independent dichotomous predictor variables that have the distribution as in equation (4), and the γ parameters all have the same sign, the conditional distribution $p(\mathbf{X} \mid \eta)$ implies a negative manifold.*

The proof of Theorem 2 is given in Appendix A, where it is shown that the conditional distribution $p(\mathbf{X} \mid \eta)$ is an Ising model with a fully negative connectivity matrix.

More precisely, the proof for Theorem 1 in Appendix A demonstrates that conditioning on the common effect in a causal indicator model implies a diagonal matrix *minus* a rank one matrix. The proof for Theorem 2 in Appendix A demonstrates that when the variables in $\mathbf{X}$ are dichotomous variables, conditioning on the common effect in a causal indicator model results in an Ising model with a connectivity matrix that is a zero matrix *minus* a rank one matrix. Note that the diagonal of this connectivity matrix is arbitrary as it drops out.

6 Positive versus negative manifold

In the previous section we presented a theorem stating that conditioning on a common effect in a causal indicator model induces conditional associations between the marginally independent observed variables that comply to a diagonal matrix (or zero matrix for dichotomous response variables) minus a rank one matrix. Because the matrix that is subtracted from the diagonal matrix is of rank one, this implies that the

Figure 3. Implied associations of a common cause model and causal indicator model. The associations between the response variables X_1 to X_4 (included in the matrix Σ) that result from the common cause model (model on the left) have the opposite sign of the conditional associations (included in the matrix $\Sigma|\eta$) that result from conditioning on the common effect in the causal indicator model (model on the right). Conditioning on a latent variable is visualized by drawing the circle inside a square.

observed variables can always be recoded so that this matrix is fully positive and hence subtracting this matrix from a diagonal matrix results in a matrix with solely negative off-diagonal elements. The associations implied by conditioning on the common effect thus feature a negative manifold. This is the opposite pattern of what is implied by a single common cause: the positive manifold.

A single common cause model implies that the covariance pattern among the observed variables is a diagonal matrix *plus* a rank one matrix. This implies that one can recode the items such that all factor loadings are positive and as such that all covariances among the observed variables are positive. In contrast, when the associations are the result of conditioning on a single common effect in a causal indicator model, one can always recode the items such that all γ parameters are positive and as such that all conditional covariances among the observed variables are negative.

The possible covariance structures that can be explained by a common cause, and those that can be explained by conditioning on a common effect are symmetric in the sense that for any covariance matrix that can be explained by a single common cause,

the off-diagonal elements can be multiplied by -1 to obtain a matrix that is consistent with a causal indicator model, and vice versa. Figure 3 depicts an example of this symmetry for four response variables.

Three mutually exclusive groups of correlation matrices can be distinguished: those for which the pattern of positive versus negative correlations is consistent with a common cause model, those for which it is consistent with a causal indicator model, and those which are inconsistent with both models. An example of a pattern of positive and negative correlations that is inconsistent with both the common cause model and the causal indicator model is a covariance matrix for $p > 2$ variables with only one negative covariance. For such a covariance structure it is impossible to recode the variables such that either a positive or negative manifold is obtained.

7 Discussion

We compared the Kruis-Maris model (Kruis & Maris, 2016) to the more familiar causal indicator model (Edwards & Bagozzi, 2000). The models imply different relations between the effect variable and the response variables. In the Kruis-Maris model the relationship between the effect variable and response variables is a quadratic relationship; the effect variable has a higher probability of being present when more response variables are in the same state. In the causal indicator model the effect variable is a linear function of the response variables. The Kruis-Maris model and causal indicator model also differ in what they imply for the conditional associations among the response variables. Whereas the Kruis-Maris model implies positive associations between variables because the effect variable has a higher probability of being present when the response variables are in the same state, the causal indicator model implies negative associations between variables that positively affect the common effect.

In the last part of the paper we proved that a causal indicator model with a single latent common effect implies solely negative associations, which we dubbed *a negative manifold*, between the response variables conditioned on the common effect. This result reveals a symmetry in the association patterns that can result from a common cause

and from conditioning on a common effect; when the response variables are effects of a single common cause the response variables can always be recoded such that a positive manifold is obtained. In contrast, when the associations between response variables are induced by conditioning on the common effect in a causal indicator model, the variables can always be recoded such that all associations are negative.

The result of a negative manifold was derived under the assumption of marginally independent response variables. This assumption is highly unlikely for psychological variables and thus it is a far more plausible scenario that the response variables are marginally associated (for example due to direct effects) and that conditioning on a common effect induces bias to these associations rather than turning the covariance matrix into a fully negative matrix. The phenomenon that conditioning on a common effect may induce spurious associations and as such bias the covariance structure is referred to in the literature as collider bias or endogenous selection bias ([Elwert & Winship, 2014](#); [Greenland, 2003](#); [Pearce & Richiardi, 2014](#)). The proof of Theorem 1 shows that the conditional distribution of $\mathbf{X}$ given η is a diagonal matrix minus a rank one matrix, in which the diagonal matrix represents the marginal covariance matrix of the variables in $\mathbf{X}$. Thus, when the response variables are not marginally independent, this matrix will no longer be a diagonal matrix, and thus the rank one matrix is subtracted from whatever marginal covariance structure exists between the response variables. For example, if the response variables feature strong positive marginal correlations, then conditioning on a common effect will shrink these correlations since a rank one matrix is subtracted from the fully positive matrix. The proof in Theorem 2 for dichotomous variables can be extended in a similar way. Future research may reveal whether the specific covariance structure that is induced by conditioning on a common effect as demonstrated in this paper (i.e., subtracting a rank one matrix from the marginal associations), can help identify selection bias and correct for it.

To conclude with an answer to the question raised in the title of this paper: a common effect model implies a positive manifold when the relation between the response variables and the common effect is quadratic, but implies a negative manifold

when the relation between the common effect and response variables is linear.

Appendix A

Proof of Theorem 1

The marginal expectation of η is equal to $\mathbb{E}(\eta) = \sum_{i=1}^p \mathbb{E}(X_i)\gamma_i = \boldsymbol{\mu}^\top \boldsymbol{\gamma}$, and its marginal variance is equal to

$$\text{Var}(\eta) = \mathbb{E}(\text{Var}(\eta \mid \mathbf{X})) + \text{Var}(\mathbb{E}(\eta \mid \mathbf{X})) = \sigma^2 + \text{Var}(\mathbf{X}^\top \boldsymbol{\gamma}) = \sigma^2 + \sum_{i=1}^p \tau_i^2 \gamma_i^2.$$

The covariance between η and X_j is equal to

$$\text{Cov}(\eta, X_j) = \text{Cov}\left(\sum_{i=1}^p X_i \gamma_i + \zeta, X_j\right) = \sum_{i=1}^p \gamma_i \text{Cov}(X_i, X_j) = \gamma_j \text{Cov}(X_j, X_j) = \gamma_j \tau_j^2.$$

Thus, the joint distribution of $\mathbf{X}$ and η is given by

$$\begin{pmatrix} X_1 \\ \vdots \\ X_p \\ \eta \end{pmatrix} \sim \mathcal{N} \left(\begin{pmatrix} \mu_1 \\ \vdots \\ \mu_p \\ \sum_{i=1}^p \mu_i \gamma_i \end{pmatrix}, \begin{pmatrix} \tau_1^2 & \dots & 0 & \gamma_1 \tau_1^2 \\ \vdots & \ddots & \vdots & \vdots \\ 0 & \dots & \tau_p^2 & \gamma_p \tau_p^2 \\ \gamma_1 \tau_1^2 & \dots & \gamma_p \tau_p^2 & \sigma^2 + \sum_{i=1}^p \tau_i^2 \gamma_i^2 \end{pmatrix} \right).$$

The covariance matrix $\boldsymbol{\Sigma}$ can be partitioned in four submatrices,

$$\boldsymbol{\Sigma} = \left(\begin{array}{c|c} \boldsymbol{\Sigma}_{11} & \boldsymbol{\Sigma}_{12} \\ \hline \boldsymbol{\Sigma}_{21} & \boldsymbol{\Sigma}_{22} \end{array} \right) = \left(\begin{array}{ccc|c} \tau_1^2 & \dots & 0 & \gamma_1 \tau_1^2 \\ \vdots & \ddots & \vdots & \vdots \\ 0 & \dots & \tau_p^2 & \gamma_p \tau_p^2 \\ \hline \gamma_1 \tau_1^2 & \dots & \gamma_p \tau_p^2 & \sigma^2 + \sum_{i=1}^p \tau_i^2 \gamma_i^2 \end{array} \right).$$

The conditional distribution of $\mathbf{X}$ given η is also multivariate normal. The conditional expected value of $\mathbf{X}$ given η (Anderson, 1958) is given by

$$E(\mathbf{X} \mid \eta) = \boldsymbol{\mu} + \boldsymbol{\Sigma}_{12} \boldsymbol{\Sigma}_{22}^{-1} (\eta - \mathbb{E}(\eta)),$$

which is equal to

$$\begin{pmatrix} \mu_1 \\ \vdots \\ \mu_p \end{pmatrix} + \begin{pmatrix} \gamma_1 \tau_1^2 \\ \vdots \\ \gamma_p \tau_p^2 \end{pmatrix} \times \frac{\eta - \sum_{i=1}^p \mu_i \gamma_i}{\sigma^2 + \sum_{i=1}^p \tau_i^2 \gamma_i^2}.$$

The conditional covariance matrix of $\mathbf{X}$ given η is

$$\Sigma(\mathbf{X} \mid \eta) = \Sigma_{11} - \Sigma_{12}\Sigma_{22}^{-1}\Sigma_{21},$$

which is equal to

$$\begin{pmatrix} \tau_1^2 & \dots & 0 \\ \vdots & \ddots & \vdots \\ 0 & \dots & \tau_p^2 \end{pmatrix} - \frac{1}{\sigma^2 + \sum_{i=1}^p \tau_i^2 \gamma_i^2} \times \begin{pmatrix} \tau_1^2 \gamma_1 \\ \vdots \\ \tau_p^2 \gamma_p \end{pmatrix} \begin{pmatrix} \tau_1^2 \gamma_1 \\ \vdots \\ \tau_p^2 \gamma_p \end{pmatrix}^T.$$

All off-diagonal elements of this conditional covariance matrix are negative whenever the γ parameters have the same sign. This means that the variables in $\mathbf{X}$ can always be recoded such that a negative manifold is obtained for the conditional associations between the variables in $\mathbf{X}$ given η .

Proof of Theorem 2

The conditional distribution $p(\eta \mid x_1, \dots, x_p)$ is the normal distribution,

$$p(\eta \mid \mathbf{x}) = \frac{1}{\sqrt{2\pi}\sigma} \exp\left(-\frac{1}{2\sigma^2}(\eta - \mathbf{x}^T \boldsymbol{\gamma})^2\right),$$

and the marginal distribution $p(X_1, \dots, X_p)$ is given in equation (4), as

$$p(\mathbf{x}) = \prod_{i=1}^p p(x_i) = \prod_{i=1}^p \frac{\exp(x_i \mu_i)}{\sum_{x_i} \exp(\mu_i)},$$

where $\mu_i \in \mathbb{R}$ is an intercept. Together, these two distributions specify the joint distribution

$$p(\eta, \mathbf{x}) = p(\eta \mid \mathbf{x}) p(\mathbf{x}) = \frac{1}{\sqrt{2\pi}\sigma} \exp\left(-\frac{1}{2\sigma^2}(\eta - \mathbf{x}^T \boldsymbol{\gamma})^2\right) \prod_{i=1}^p \frac{\exp(x_i \mu_i)}{\sum_{x_i} \exp(\mu_i)}.$$

We derive the conditional distribution $p(\mathbf{x} \mid \eta)$ from this joint distribution,

$$\begin{aligned}
p(\mathbf{x} \mid \eta) &= \frac{p(\eta, \mathbf{x})}{p(\eta)} \\
&= \frac{p(\eta \mid \mathbf{x}) p(\mathbf{x})}{\sum_{\mathbf{x}} p(\eta \mid \mathbf{x}) p(\mathbf{x})} \\
&= \frac{\frac{1}{\sqrt{2\pi}\sigma} \exp\left(-\frac{1}{2\sigma^2}(\eta - \mathbf{x}^\top \boldsymbol{\gamma})^2\right) \prod_{i=1}^p \frac{\exp(x_i \mu_i)}{\sum_{x_i} \exp(\mu_i)}}{\sum_{\mathbf{x}} \frac{1}{\sqrt{2\pi}\sigma} \exp\left(-\frac{1}{2\sigma^2}(\eta - \mathbf{x}^\top \boldsymbol{\gamma})^2\right) \prod_{i=1}^p \frac{\exp(x_i \mu_i)}{\sum_{x_i} \exp(\mu_i)}} \\
&= \frac{\exp\left(-\frac{1}{2\sigma^2}(\eta - \mathbf{x}^\top \boldsymbol{\gamma})^2 + \mathbf{x}^\top \boldsymbol{\mu}\right)}{\sum_{\mathbf{x}} \exp\left(-\frac{1}{2\sigma^2}(\eta - \mathbf{x}^\top \boldsymbol{\gamma})^2 + \mathbf{x}^\top \boldsymbol{\mu}\right)} \\
&= \frac{\exp\left(-\frac{1}{2\sigma^2} \left[\eta^2 - 2\eta \mathbf{x}^\top \boldsymbol{\gamma} + \mathbf{x}^\top \boldsymbol{\gamma} \boldsymbol{\gamma}^\top \mathbf{x}\right] + \mathbf{x}^\top \boldsymbol{\mu}\right)}{\sum_{\mathbf{x}} \exp\left(-\frac{1}{2\sigma^2} \left[\eta^2 - 2\eta \mathbf{x}^\top \boldsymbol{\gamma} + \mathbf{x}^\top \boldsymbol{\gamma} \boldsymbol{\gamma}^\top \mathbf{x}\right] + \mathbf{x}^\top \boldsymbol{\mu}\right)} \\
&= \frac{\exp\left(\frac{1}{\sigma^2} \eta \mathbf{x}^\top \boldsymbol{\gamma} - \frac{1}{2\sigma^2} \mathbf{x}^\top \boldsymbol{\gamma} \boldsymbol{\gamma}^\top \mathbf{x} + \mathbf{x}^\top \boldsymbol{\mu}\right)}{\sum_{\mathbf{x}} \exp\left(\frac{1}{\sigma^2} \eta \mathbf{x}^\top \boldsymbol{\gamma} - \frac{1}{2\sigma^2} \mathbf{x}^\top \boldsymbol{\gamma} \boldsymbol{\gamma}^\top \mathbf{x} + \mathbf{x}^\top \boldsymbol{\mu}\right)} \\
&= \frac{\exp\left(\mathbf{x}^\top \left(\frac{1}{\sigma^2} \eta \boldsymbol{\gamma} + \boldsymbol{\mu}\right) + \mathbf{x}^\top \left(-\frac{1}{2\sigma^2} \boldsymbol{\gamma} \boldsymbol{\gamma}^\top\right) \mathbf{x}\right)}{\sum_{\mathbf{x}} \exp\left(\mathbf{x}^\top \left(\frac{1}{\sigma^2} \eta \boldsymbol{\gamma} + \boldsymbol{\mu}\right) + \mathbf{x}^\top \left(-\frac{1}{2\sigma^2} \boldsymbol{\gamma} \boldsymbol{\gamma}^\top\right) \mathbf{x}\right)},
\end{aligned}$$

which is an Ising model with intercepts $\boldsymbol{\mu} + \frac{1}{\sigma^2} \eta \boldsymbol{\gamma}$ and a fully negative connectivity matrix $-\frac{1}{\sigma^2} \boldsymbol{\gamma} \boldsymbol{\gamma}^\top$.

To see that this is in fact an Ising model note that the model can be rewritten as follows. Let $\boldsymbol{\mu}^* = \boldsymbol{\mu} + \frac{1}{\sigma^2} \eta \boldsymbol{\gamma}$ denote the vector of conditional intercepts so that $\mu_i^* = \mu_i + \frac{1}{\sigma^2} \eta \gamma_i$ denotes the intercept for X_i conditional on η . Let $\boldsymbol{\Omega} = -\frac{1}{\sigma^2} \boldsymbol{\gamma} \boldsymbol{\gamma}^\top$ denote the conditional connectivity matrix, so that $\omega_{ij} = -\frac{1}{\sigma^2} \gamma_i \gamma_j$, represents the edge weight connecting X_i and X_j conditional on η . We can rewrite the above model as follows:

$$\begin{aligned}
p(\mathbf{x} \mid \eta) &= \frac{\exp\left(\mathbf{x}^\top \left(\frac{1}{\sigma^2} \eta \boldsymbol{\gamma} + \boldsymbol{\mu}\right) + \mathbf{x}^\top \left(-\frac{1}{2\sigma^2} \boldsymbol{\gamma} \boldsymbol{\gamma}^\top\right) \mathbf{x}\right)}{\sum_{\mathbf{x}} \exp\left(\mathbf{x}^\top \left(\frac{1}{\sigma^2} \eta \boldsymbol{\gamma} + \boldsymbol{\mu}\right) + \mathbf{x}^\top \left(-\frac{1}{2\sigma^2} \boldsymbol{\gamma} \boldsymbol{\gamma}^\top\right) \mathbf{x}\right)} \\
&= \frac{\exp\left(\mathbf{x}^\top \boldsymbol{\mu}^* + \mathbf{x}^\top \boldsymbol{\Omega} \mathbf{x}\right)}{\sum_{\mathbf{x}} \exp\left(\mathbf{x}^\top \boldsymbol{\mu}^* + \mathbf{x}^\top \boldsymbol{\Omega} \mathbf{x}\right)} \\
&= \frac{\exp\left(\sum_{i=1}^p x_i \mu_i^* + \sum_{\langle ij \rangle} x_i x_j \omega_{ij}\right)}{\sum_{\mathbf{x}} \exp\left(\sum_{i=1}^p x_i \mu_i^* + \sum_{\langle ij \rangle} x_i x_j \omega_{ij}\right)}.
\end{aligned}$$

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Additional information

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