On the Behavioral Consequences of Reverse Causality*

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Abstract

Reverse causality is a common attribution error that distorts the evaluation of private actions and public policies. This paper explores the implications of this error when a decision maker acts on it and therefore affects the very statistical regularities from which he draws faulty inferences. Applying the Bayesian-network approach of Spiegler (2016), I explore the equilibrium effects of a certain class of reverse-causality errors, in the context of an example with a quadratic-normal parameterization. I show that the decision context may protect the decision maker from his own reverse-causality error. That is, the cost of reverse-causality errors can be lower for everyday decision makers than for an outside observer who evaluates their choices.

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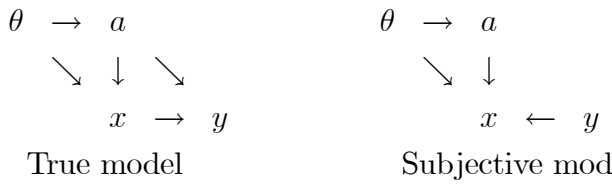
1 Introduction

Reverse causality is a ubiquitous error. Observing a correlation between two variables of interest, we often form an instinctive causal interpretation in one direction, even if true causation goes in the opposite direction. Reverse causality is often invoked as a warning to social scientists and medical researchers, to beware of naive causal interpretations of correlational data. For example, Harris (2011) famously criticized the developmental psychology literature for taking for granted that observed correlation between child personality and parental behavior reflected a causal link from the latter to the former. Harris argued that causality might go in the opposite direction: parental behavior could be a *response* to the child's innate temperament.

Clearly, when researchers and other outside observers interpret correlations under the sway of a reverse-causality error, their evaluation of private interventions or public policy may become distorted. But what happens when decision makers *act* on a reverse-causality error, such that their resulting behavior affects the very statistical regularities from which they draw inferences through their faulty prism?

For illustration, consider the developmental psychology example mentioned above and embed it in the following decision context. A counselor observes a child's exogenous characteristics and chooses a therapy. These two factors cause the child's behavior. Parental behavior is a response to the child's behavior and the counselor's therapy. However, the counselor operates under the perception that parental behavior is an independent variable that joins the list of factors that cause the child's behavior. This difference can be represented by two directed acyclic graphs (DAGs), which conventionally represent causal models (Pearl (2009)):



(Figure 1)

In this diagram, θ represents the child's characteristics, a represents the

counselor's action, x represents the child's behavior and y represents the parent's behavior. The counselor's subjective causal model departs from the true model by inverting the link between x and y and severing the link from a into y. That is, the counselor regards y as an exogenous variable that causes x, whereas in fact it is an endogenous variable and x is one of its direct cause.

The diagram is paradigmatic, in the sense that the error it represents could fit several real-life situations. In a monetary economics context, different specifications of the Phillips curve disagree over which of the two variables, inflation and employment, is a dependent variable and which is an explanatory variable. This can be viewed as a disagreement over the direction of causality (see Sargent (1999) and Cho and Kasa (2015) – I will discuss this example in detail in Sections 2 and 3). In a social policy context, θ represents initial socioeconomic or demographic conditions, a represents welfare policy, x represents poverty levels or income inequality, and y is a public-health indicator. According to this interpretation, public health is an objective consequence of income inequality or poverty, yet designers of social policy operate under the assumption that health is an exogenous, independent factor that affects the social outcome rather than being caused by it (for a survey of a literature that wrestles with the causal relation between income inequality and health, see Pickett and Wilkinson (2015)).

My objective in this paper is to take these familiar disagreements over the direction of causality and place them in a *decision* context, in which the objective data-generating process is affected by decision makers' behavior, which in turn is a response to statistical inferences that reflect their causal error. In this way, reverse causality ceases to be the exclusive problem of outside observers who make scientific claims, and becomes a payoff-relevant concern for decision makers with skin in the game. Is there a fundamental difference between the two scenarios? How does the decision context affect the magnitude of errors induced by reverse causality?

To study these questions, I apply the modeling approach developed in Spiegler (2016,2020a), which borrows the formalism of Bayesian networks from the Statistics and AI literature (Cowell et al. (1999), Pearl (2009)) to analyze decision making under causal misperceptions. According to this

approach, a decision maker (DM henceforth) fits a DAG-represented causal model to an objective joint distribution over the variables in his model. The DM best-replies to the (possibly distorted) belief that arises from this act of fitting a misspecified causal model to objective data. Since the belief distortions may depend on the DM's strategy, it is appropriate to analyze subjectively optimal behavior as an equilibrium object. I apply this model to the specification of true and subjective causal models depicted in Figure 1.

I carry out the analysis in the context of an example with a quadratic-normal parameterization, such that the DM's payoffs are given by a quadratic loss function of a and x, and the joint distribution over the four variables is Gaussian. This parameterization helps in terms of analytic tractability and transparent interpretation of the results, which allows for crisp comparative statics. However, it is also appropriate because fitting a DAG-represented model to a Gaussian distribution is equivalent to OLS estimation of a recursive system of linear regressions (Koller and Friedman (2009, Ch. 7)). Causal interpretations of linear regressions permeate discussions of reverse causality in social-science settings. Assuming that the environment is Gaussian ensures that the linearity of the DM's model is not wrong per se – the misspecification lies entirely in the underlying causal structure, which is represented by the DAG.

The analysis of this simple model demonstrates the subtle equilibrium effects of decision making under reverse causality errors. One effect is that features of the model that would be irrelevant in a quadratic-normal model with rational expectations – specifically, the variances of the noise terms in the equations for x and y – play a key role when the DM exhibits the reverse-causality error given by Figure 1.

The results also illuminate the difference between committing a reverse-causality error as a "spectator" or as an "actor". A special case of the specification given by Figure 1 is that the action's objective effect on the outcome variables x and y is null. In this case, the DM can be viewed as a "spectator". This DM's reverse-causality error induces a wrong prediction of x and he suffers a welfare loss as a result. Compare this with a DM who

is an "actor", in the sense that his action has a non-null direct effect on y (but still no effect on x). I show that when this effect $fully$ offsets the direct effect of x on y (in the sense that $E(y \mid a, x) = x - a$), the DM's equilibrium strategy is as if he has rational expectations. This DM suffers no welfare loss as a result of his reverse-causality error. Thus, the decision context protects an "actor" from his error in a way that it does not for a "spectator". The lesson is that in some situations, reverse causality may be less of a problem for DMs than for the scientists who analyze their behavior.

2 The Model

A DM observes an exogenous state of Nature $\theta \sim N(0, \sigma_\theta^2)$ before taking a real-valued action a. The DM is an expected-utility maximizer with vNM utility function $u(a, x) = -(x - a)^2$, where x is an outcome variable that is determined by θ and a according to the equation

$$x = \theta - \gamma a + \varepsilon \tag{1}$$

Another outcome y is then determined by x and a according to the equation

$$y = x - \lambda a + \eta \tag{2}$$

The parameters γ, λ are constants that capture the direct effects of a on the outcome variables x and y. Assume $\gamma, \lambda \in [0, 1]$, such that these direct effects are of an "*offsetting*" nature. This restriction is motivated by the real-life situations alluded to in the Introduction. The terms $\varepsilon \sim N(0, \sigma_\varepsilon^2)$ and $\eta \sim N(0, \sigma_\eta^2)$ are independently distributed noise variables. The ratio of the variances of these noise terms, denoted

$$\tau = \frac{\sigma_\varepsilon^2}{\sigma_\eta^2} \tag{3}$$

will play an important role in the sequel.

A (potentially mixed) strategy for the DM is a function that assigns a

distribution over a to every realization of θ. Once we fix a strategy, we have a well-defined joint probability measure p over all four variables θ, a, x, y, which can be written as a factorization of marginal and conditional distributions as follows:[1]

$$p(\theta, a, x, y) = p(\theta)p(a \mid \theta)p(x \mid \theta, a)p(y \mid a, x) \tag{4}$$

The term $p(a \mid \theta)$ represents the DM's strategy. This factorization reflects the causal structure that underlies p and that can be described by the leftward DAG in Figure 1, which I denote G^*.[2]

I interpret p as a long-run distribution that results from many repetitions of the same decision problem. Our DM faces a one-shot decision problem, against the background of the long-run experience accumulated by many previous generations of DMs who faced the same one-shot problem (each time with a different, independent draw of the random variables $\theta, \varepsilon, \eta$).

The correct-specification benchmark

Suppose the DM correctly perceives the true model – i.e., he has "rational expectations". Then, he will choose a to minimize

$$E[(x - a)^2 \mid \theta, a]$$

where the expectation E is taken w.r.t the objective conditional distribution $p_{G^*}(x \mid \theta, a) \equiv p(x \mid \theta, a)$. This means that we can plug (1) into the objective function and use the fact that ε is independently distributed with $E(\varepsilon) = 0$. Hence, the rational-expectations prediction is that the DM will choose

$$a_{G^*}(\theta) = \frac{\theta}{1 + \gamma} \tag{5}$$

Note that y has no direct payoff relevance for the DM. Moreover, since y does not affect the transmission from θ and a to x, it is entirely irrelevant

[1] The factorization is invalid when some terms involve conditioning on zero probability events. Since this is not going to be a problem for us, I ignore the imprecision.

[2] Schumacher and Thysen (2020) study a principal-agent model in which the true causal model has the same form as G^*, although the nodes have different interpretations (in particular, the action variable corresponds to the initial node), and the agent's subjective causal model is quite different.

for the DM's decision, because y is a consequence of x and a. Therefore, the constant λ plays no role in the DM's rational action. Moreover, the variances of the noise terms ε, η are irrelevant for a_{G^*}.

Now relax the assumption that the DM correctly perceives the true model. Instead, he believes that the distribution over θ, a, x, y obeys a causal structure that is given by the rightward DAG in Figure 1, which I denote by G. Compared with G^*, the DAG G inverts the causal link between x and y, and also removes the link $a \to y$. This means that the DM regards y as an exogenous direct cause of x, instead of the consequence of x and a that it actually is.

The following are two examples of situations that fit this specification of the true and subjective DAGs G^* and G.

Example 2.1: Parenting
Here is a variant on the story presented in the Introduction. A parent observes characteristics of his child (possibly the child's home behavior in a previous period), captured by the state variable θ. He then chooses how toughly to behave toward the child, as captured by the variable a. The child's resulting home behavior is captured by some index x. The quality of the child's school interactions with teachers and peers, as measured by the index y, is a consequence of the child's and the parent's home behavior. However, the parent believes that school interactions are an independent driver of the child's home behavior, rather than a consequence of home interactions.

Example 2.2: Quantity setting
A large firm observes a demand indicator θ before setting its production quantity a. The price x is a function of demand and quantity. The variable y represents a competitive fringe that reacts to the price and the production quantity. However, the firm believes that the market agents that constitute the competitive fringe are not price takers, but rather independent decision makers that affect the price.

The quadratic loss function $u(x, a) = -(x-a)^2$ is not necessarily plausible in these examples. However, in both cases we can write down a sensible quadratic utility function (e.g. due to linear demand and constant marginal

costs in Example 2.2), and then redefine a, x, y via some linear transformation to obtain the quadratic loss specification, without any effect on our formal results (that is, the DM's action will be the same up to the above linear transformation). The reason is that under any quadratic utility function, the DM's optimal action is linear in $E_G(x \mid \theta, a)$, which is his subjective expectation of x conditional on θ and a.

Following Spiegler (2016), I assume that given the long-run distribution p, the DM forms a subjective belief, denoted p_G, by fitting his model G to p according to the Bayesian-network factorization formula. For an arbitrary DAG R over some collection of variables $x_1, ..., x_n$, the formula is

$$p_R(x_1, ..., x_n) = \prod_{i=1}^{n} p(x_i \mid x_{R(i)}) \tag{6}$$

where $x_M = (x_i)_{i \in M}$ and $R(i)$ represents the set of variables that are viewed as direct causes of x_i according to R. Formula (4) was a special case of (6) for $R = G^*$. Given our specification of the subjective DAG G, (6) reads as follows:

$$p_G(\theta, a, x, y) = p(\theta)p(y)p(a \mid \theta)p(x \mid \theta, a, y)$$

The interpretation of this belief-formation model is as follows. The DM's DAG G is a misspecified subjective model. The DM perceives the statistical regularities in his environment through the prism of his incorrect subjective model. In other words, he fits his model to the long-run statistical data (generated by the true distribution p), producing the subjective distribution p_G. A related interpretation is that the objective distribution p is not transparently available to the DM. Instead, he needs to probe it via empirical queries. Our DM does not try to test his model; he only queries p to estimate it. He only performs the measurements that quantify the terms in the factorization formula (6), and does not take additional measurements that could refute his model. A third interpretation is based on Esponda and Pouzo (2016). We can regard G as a set of probability distributions – i.e. all distributions p' for which $p'_G \equiv p'$. The DM has a prior belief whose support coincides with this set. He goes through a process of Bayesian learning, by observing

a sequence of $i.i.d$ draws from p. The limit belief of this process is p_G (see Spiegler (2020a)).

The DM's subjective belief p_G induces the following conditional distribution over x, given the DM's information and action, by applying Bayes' rule to p_G:

$$p_G(x \mid \theta, a) = \frac{p_G(\theta, a, x)}{p_G(\theta, a)}$$

Since (θ, a) form an ancestral clique in G, $p_G(\theta, a) = p(\theta, a)$ (see Lemma 1 in Spiegler (2020b)). Since

$$p_G(\theta, a, x) = \int_y p_G(\theta, a, x, y) = \int_y p(y)p(\theta, a)p(x \mid \theta, a, y)$$

we obtain

$$p_G(x \mid \theta, a) = \int_y p(y)p(x \mid \theta, a, y) \tag{7}$$

The DM chooses a to maximize his expected utility w.r.t this conditional belief, which means minimizing

$$\int_x p_G(x \mid \theta, a)(x - a)^2 \tag{8}$$

Personal equilibrium

The behavioral model is potentially ill-defined because it is not obvious that $p_G(x \mid \theta, a)$ is invariant to the DM's strategy $(p(a \mid \theta))$. To resolve this ambiguity, Spiegler (2016) introduces a notion of "*personal equilibrium*", such that the DM's subjective optimization is defined as an equilibrium concept (this is a hallmark of models of decision making under misspecified models – see also Esponda and Pouzo (2016)). A strategy $(p(a \mid \theta))$ is a personal equilibrium if it always prescribes subjective optimal actions with respect to the belief $(p_G(x \mid \theta, a))$, which in turn is calculated when we take the DM's strategy as given.[3]

I focus on personal equilibria in *linear strategies*. A strategy $(p(a \mid \theta))$ is

[3]In general, this definition requires trembles in order to be fully precise. This subtlelty is irrelevant for our present purposes.

linear if there is some constant k such that $p(a \mid \theta)$ assigns probability one to $a = k\theta$ for every θ. When the DM plays a linear strategy, the objective joint distribution p over the four variables is Gaussian. It is well-known that in this case, p_G is Gaussian as well (see Koller and Friedman (2009, Ch. 7)). (My definition of linear strategies assumes a zero intercept. This restriction is without loss of generality, and made only to make calculations more transparent.)

Comment: The pure prediction case
When $\gamma = 0$, the DM's action has no causal effect on x. In other words, the DM faces a pure prediction problem: his subjectively optimal action is equal to his subjective expectation of x conditional on θ. Therefore, the link $a \to x$ can be omitted from G^*. This raises the following question: can we be equally cavalier about whether to include the link $a \to x$ in the subjective DAG G? Including the link means that the DM erroneously regards his action as a direct cause of x (but he is open to the possibility that when estimated, the measured effect will be null).

It turns out that the answer to our question is positive, in the following sense. When we remove the link $a \to x$ from G, we can no longer ignore the subtleties that called for treating subjective maximization as an equilibrium phenomenon. If we apply the concept of personal equilibrium, and impose in addition the requirement that the DM's equilibrium strategy is pure, then the model's prediction will be the same as when G includes the link $a \to x$. If the relation between a and θ were noisy – e.g. by introducing a random shock to the DM's behavior, or an explicit preference shock – this would cease to be true, and we would have to take a stand on whether G includes the link $a \to x$ – i.e. whether the DM understands that a has no causal effect on x.

Example 2.3: Inflation forecasting with a wrong Phillips curve
The following variation on Sargent's (1999) simplified version of the well-known monetary model due to Barro and Gordon (1983) fits the pure prediction case.[4] The variable θ represents a monetary quantity such as money

[4]This specification (with rational expectations) was also employed by Athey et al.

growth, which determines inflation x via (1). Independently, the private sector (corresponding to the DM in our model) observes θ and forms an inflation forecast a. The variable y represents employment, which is determined as a function of inflation and inflationary expectations via the Phillips curve (2). The parameter λ measures the extent to which anticipated inflation offsets the real effect of actual inflation; the case of $\lambda = 1$ captures the "new classical" assumption that only unanticipated inflation has real effects.

The private sector's inflation forecast is based on a misspecified Phillips curve, which regards y as an independent, explanatory variable and x as the dependent variable. Sargent (1999) and Cho and Kasa (2015) refer to this inversion of the causal inflation-employment relation in terms of an econometric identification strategy, and dub it a "Keynesian fit". The key difference between these papers and the present example is that they assume that the private sector has rational expectations; it is the monetary authority (which chooses θ) that operates under a misspecified model (Spiegler (2016) describes how to reformulate Sargent's example in the DAG language). In contrast, in the present example, it is the *private sector* that bases its inflation forecasts on a wrong Phillips curve.

3 Analysis

The following is the main result of this paper.

Proposition 1 *There is a unique personal equilibrium in linear strategies. The DM's strategy is*

$$a_G(\theta) = \frac{\theta}{1 + \gamma + \tau(1 - \lambda)} \tag{9}$$

Proof. The proof proceeds in two steps. First, I derive a general form of the DM's linear equilibrium strategy, having free parameters. I then calculate the value that these parameters must take.

(2005).

Step 1: For any equilibrium in linear strategies, there are constants c_0, c_1, c_2, c_3 such that the DM's equilibrium strategy takes the form:

$$a = E_G(x \mid \theta, a) = \frac{c_0 + c_1\theta + c_3 E(y)}{1 - c_2} \tag{10}$$

Proof: By assumption, the DM chooses a to minimize (8) at every θ. As we observed above, when the DM's equilibrium strategy is linear, p_G is Gaussian. Therefore, the conditional distribution $p_G(x \mid \theta, a, y)$ can be written as a regression equation

$$x = c_0 + c_1\theta + c_2 a + c_3 y + \psi \tag{11}$$

where c_0, c_1, c_2, c_3 are coefficients and $\psi \sim N(0, \sigma_\psi^2)$ is an independent noise term. Since G treats y and ψ as independently distributed error terms with $E(\psi) = 0$, (11) implies

$$E_G(x \mid \theta, a) = c_0 + c_1\theta + c_2 a + c_3 E(y) \tag{12}$$

It also follows from (11) that (8) can be rewritten as

$$\int_y p(y) E_\psi (c_0 + c_1\theta + c_2 a + c_3 y - a + \psi)^2 \tag{13}$$

where the expectation over ψ is taken w.r.t the independent distribution $N(0, \sigma_\psi^2)$. Note that the DM integrates over y as if it is independent of a because indeed, G posits that $y \perp a$. This assumption is wrong, according to the true model G^*.

Choosing a to minimize (13), and using the observation that $E(\psi) = 0$, we obtain the following first-order condition:

$$(c_2 - 1) \cdot [c_0 + c_1\theta + c_2 a + c_3 E(y) - a] = 0$$

This can be written as

$$(c_2 - 1) \cdot [E_G(x \mid \theta, a) - a] = 0$$

Plugging (12) and rearranging, we obtain (10). □

Step 2: Pinning down the values of c_0, c_1, c_2 and showing that $E(y) = 0$
Proof: Let us derive

$$E_G(x \mid \theta, a) = \int_y p(y) E(x \mid \theta, a, y) \tag{14}$$

As a first step, let us calculate $E(x \mid \theta, a, y)$. (Note that x is *not* independent of y conditional on θ, a; therefore, $E(x \mid \theta, a, y)$ need not coincide with $E(x \mid \theta, a)$.) Plugging (1) in (2), we obtain

$$x = \theta - \gamma a + \varepsilon = y + \lambda a - \eta \tag{15}$$

Therefore,

$$E(x \mid \theta, a, y) = E(\theta - \gamma a + \varepsilon \mid \theta, a, y) = \theta - \gamma a + E(\varepsilon \mid \theta, a, y) \tag{16}$$

It follows that in order to get an explicit expression for $E(x \mid \theta, a, y)$, we need to derive $E(\varepsilon \mid \theta, a, y)$. Toward this end, rearrange (15) to obtain

$$\varepsilon + \eta = y + (\lambda + \gamma)a - \theta$$

Therefore,

$$E(\varepsilon \mid \theta, a, y) = E(\varepsilon \mid \varepsilon + \eta = y + (\lambda + \gamma)a - \theta)$$

Since $\varepsilon \sim N(0, \sigma_\varepsilon^2)$ and $\eta \sim N(0, \sigma_\eta^2)$ are independent variables, we can use the standard signal extraction formula to obtain

$$E(\varepsilon \mid \theta, a, y) = \beta \cdot (y + (\lambda + \gamma)a - \theta) \tag{17}$$

where

$$\beta = \frac{\sigma_\varepsilon^2}{\sigma_\varepsilon^2 + \sigma_\eta^2} = \frac{\tau}{1 + \tau} \tag{18}$$

Plugging (17) in (16), we obtain

$$E(x \mid \theta, a, y) = (1-\beta)\theta + (\beta\lambda + \beta\gamma - \gamma)a + \beta y$$

Plugging this expression in (14), we obtain

$$E_G(x \mid \theta, a) = (1-\beta)\theta + (\beta\lambda + \beta\gamma - \gamma)a + \beta E(y)$$

Plugging this expression in (10), we obtain

$$a_G(\theta) = \frac{1-\beta}{1-\beta\lambda+\gamma(1-\beta)}\theta + \frac{\beta}{1-\beta\lambda+\gamma(1-\beta)}E(y)$$

Plugging this in (1) in (2) and repeatedly taking expectations, we obtain $E(y) = 0$ such that $a_G(\theta)$ is given by (9).

We have thus pinned down $c_0 = 0$, $c_1 = 1-\beta$ and $c_2 = \beta\lambda - \gamma(1-\beta)$ in (10). The value of c_3 is irrelevant because $E(y) = 0$. Since $\lambda, \gamma \in [0,1]$ and $\beta \in (0,1)$, $c_2 \neq 1$ such that (10) is well-defined and unique. ■

The DM's equilibrium strategy $a_G(\theta)$ has several noteworthy features, in comparison with the correct-specification benchmark $a_{G^*}(\theta)$.

Sensitivity to the state of Nature θ

The coefficient of θ in the expression for $a_G(\theta)$ is lower than $1/(1+\gamma)$, which is the correct-specification benchmark value. This means that the DM's reverse-causality error results in diminished responsiveness to the state of Nature. The reason is that the DM erroneously believes that θ is not the only independent factor affecting x (apart from a). The extent of this rigidity effect depends on τ, as explained below.

The relevance of τ

In the correct-specification benchmark, the variances of the error terms in the relations (1)-(2) are irrelevant for the DM's behavior. This is a consequence of the quadratic-normal specification of the model. In contrast, the DM's reverse-causality error lends these variances a crucial role. When $\tau = \sigma_\varepsilon^2/\sigma_\eta^2$ is small – corresponding to the case that the equation for x is precise relative to the equation for y – $a_G(\theta)$ is closer to the correct-specification benchmark

$a_{G^*}(\theta)$. In contrast, when τ is large, the departure of $a_G(\theta)$ from $a_{G^*}(\theta)$ is big. In particular, in the $\tau \to \infty$ limit, $a_G(\theta)$ is entirely unresponsive to θ.

The intuition for this effect is as follows. When forming the belief $E_G(x \mid \theta, a)$, the DM integrates over y as if it is an exogenous, independent variable (this is the DM's basic causal error) and estimates the conditional expectation $E(x \mid \theta, a, y)$ for each value of y. This conditional expectation represents a signal extraction problem, and therefore takes into account the relative precision of the equations for x and y. When τ is small, y is uninformative of x relative to θ, and therefore the conditional expectation $E(x \mid \theta, a, y)$ places a low weight on y. As a result, the DM's erroneous treatment of y does not matter much, such that the DM's belief ends up being approximately correct. In contrast, when τ is large, θ is uninformative of x relative to y, and therefore the conditional expectation $E(x \mid \theta, a, y)$ places a large weight to y. Since the DM regards y as an exogenous variable that is independent of θ, the DM ends up attaching low weight to the realized value of θ when evaluating $E_G(x \mid \theta, a)$.

The role of λ

Recall that the parameter λ measures the direct effect of a on y, which is the outcome variable that the DM erroneously regards as an independent cause of x. The case of $\lambda = 0$ corresponds to a DM who is a "spectator" as far as the outcome variable y is concerned. At the other extreme, the case of $\lambda = 1$ corresponds to a DM whose action fully offsets the effect of x on y.

It is easy to see from (9) that the departure of the DM's subjectively optimal action from the correct-specification benchmark becomes smaller as λ grows larger. Accordingly, the DM's welfare loss due to his reverse-causality error becomes smaller. When $\lambda = 1$, the DM's action *coincides* with the correct-specification benchmark. In this case, the DM's reverse-causality error inflicts no welfare loss. In other words, the fact that the DM is not a spectator but an actor who influences y protects him from the consequences of his erroneous view of y as a cause of x. Of course, this conclusion relies on the particular restrictions on the value of λ, and should not be taken as a general lesson.

The intuition for this effect is as follows. When $\lambda = 1$, $y = x - a + \eta$. This means that given θ,

$$y = x - E_G(x \mid a, \theta) + \eta$$

In other words, y is equal to the difference between the realization of x and its point forecast, plus independent noise. The point forecast can be viewed as an OLS estimate based on some linear regression (where x is regressed on θ, a and y, and then y is integrated out). From this point of view, y is an OLS residual (plus independent noise). By definition, this residual is independent of the regression's variables, and therefore its incorrect inclusion has no distorting effect on the estimation of x.

Example 2.3 revisited
Consider the pure-prediction case of $\gamma = 0$, and recall Example 2.3. This example concerns inflation forecasts based on a wrong Phillips curve that inverts the direction of causality between inflation and employment. The case of $\lambda = 1$ corresponds to the "new classical" monetary theory that anticipated inflation has no real effects. As it turns out, in this case the private sector forms *correct inflation forecasts* despite its wrong model. A discrepancy with the rational-expectations forecast – in the direction of more rigid forecasts that only partially react to fluctuations in θ – arises when $\lambda < 1$ – i.e. when anticipated inflation has real effects.

4 Two Variations

This section explores two variations on the basic model of Section 2. Each variation relaxes one of the twin assumptions that G inverts the link between x and y and treats y as an exogenous variable.

4.1 Belief in Exogeneity without Reverse Causality

Let the DM's subjective DAG be the rightward DAG in Figure 1, and modify the true model G^* (given by the leftward DAG in Figure 1) with the following

DAG G^{**}:

$$\begin{array}{ccccc} \theta & \rightarrow & a & & \\ & \searrow & \downarrow & \searrow & \\ & & x & \leftarrow & y \end{array}$$

Specifically, the equations for x and y are

$$\begin{aligned} y &= \delta a + \eta \\ x &= \theta - \kappa a + \alpha y + \varepsilon \end{aligned}$$

where α, δ, κ are constants in $(0,1)$, and $\varepsilon \sim N(0, \sigma_\varepsilon^2)$ and $\eta \sim N(0, \sigma_\eta^2)$ are independently distributed noise variables.

The DM's subjective model G departs from G^{**} by removing the link $a \rightarrow y$, but otherwise the DAGs are identical. Thus, the only causal misperception that G embodies relative to the true model G^{**} is a belief that y is an exogenous, independent variable. The following example provides an illustration of this specification of G and G^{**}.

Example 4.1: Public health
Suppose that θ represents initial conditions of a public health situation; the DM is a public-health authority and a represents the intensity of some mitigating public-health measure; y represents the population's behavioral personal-safety response (higher y corresponds to more lax behavior); and x represents the eventual public health outcome. The authority's error is that it takes the likelihood of various scenarios of the population's behavior as given without taking into account the fact that it responds to the intensity of the public-health measure.

We will now see that although this error has non-null behavioral implications, those are quite different from what we saw in Section 3. The analysis here is much simpler. As in the main model, the DM chooses a such that

$$a = E_G(x \mid \theta, a) = \int_y p(y) E(x \mid \theta, a, y)$$

The difference is that now, $E(x \mid \theta, a, y)$ is straightforward to calculate be-

cause x is conditioned on its actual direct causes:

$$E(x \mid \theta, a, y) = \theta - \kappa a + \alpha y$$

I take it for granted that $E(y) = 0$. Therefore,

$$a = \theta - \kappa a$$

such that the DM's subjectively optimal action is

$$a_G(\theta) = \frac{\theta}{1 + \kappa}$$

Note that personal-equilibrium subtleties are irrelevant in this derivation.

By comparison, the correct-specification action would satisfy

$$a = E(x \mid \theta, a) = \theta - \kappa a + \alpha\delta a$$

such that

$$a_{G^{**}}(\theta) = \frac{\theta}{1 + \kappa - \alpha\delta}$$

We see that as in the model of Section 2, treating y as an independent exogenous variable leads to a more rigid response to θ. Here the reason is that since $E(y) = 0$, the average unconditional effect of y on x is null, whereas conditional on $a > 0$ it is positive. Therefore, failing to treat y as an intermediate consequence of a leads the DM to neglect a causal channel that affects x, which means that he ends up underestimating the total effect of a on x.

However, unlike the basic model of Section 2, the variances of the noise parameters play no role in the solution: a change in the precision of the equations for x or y will have no effect on the outcome. Finally, unlike the basic model, the decision context does not protect the DM from his own error. Indeed, a larger δ only $magnifies$ the discrepancy between $a_G(\theta)$ and $a_{G^{**}}(\theta)$. The conclusion is that the mere failure to recognize the endogeneity of y leads to very different effects than when this failure is combined with the reverse-causality error.

4.2 Reverse Causality without Belief in Exogeneity

In this sub-section I consider a variation on the model that retains the reverse causality aspect of G, while relaxing the assumption that the DM regards y as an independent exogenous variable. Thus, assume that the true DAG is G^*, as given by Figure 1, while the DM's subjective DAG G is

$$\begin{array}{ccccc} \theta & \rightarrow & a & & \\ & \searrow & \downarrow & \searrow & \\ & & x & \leftarrow & y \end{array} \qquad \text{(Figure 2)}$$

The subjective model departs from the true model by inverting the link between x and y, but otherwise it is identical to G^*.

The DM's misperception can be viewed in terms of the conditional-independence properties that it gets wrong. While G postulates that $y \perp \theta \mid a$, G^* violates this property because of the causal channel from θ to y that passes through x.

Example 4.2: More parenting
Suppose that θ represents an adolescent's initial conditions. The DM is the adolescent's parent. The variable y represents the amount of time that the adolescent spends online, and a represents the intensity of the parent's attempts to limit this online exposure. The variable x represents the adolescent's mental health. Thus, according to the true model, online exposure is not a cause but a consequence of mental health. The parent's intervention may have a direct effect on mental health, possibly because it includes spending more time with his child, which may have direct effects on x, independently of the activity it substitutes away from. The parent believes that online exposure has a direct causal effect on the adolescent's mental health.

Let us analyze personal equilibrium in linear strategies under this specification of G^* and G.

Proposition 2 *There is a unique personal equilibrium in linear strategies. The DM plays the rational-expectations strategy given by (5).*

Proof. The DM chooses a to minimize

$$\int_x p_G(x \mid \theta, a)(x-a)^2 = \int_y p(y \mid a) \int_x p(x \mid \theta, a, y)(x-a)^2 \tag{19}$$

Using essentially the same argument as in the proof of Proposition 1, the DM's strategy satisfies:

$$a = E_G(x \mid \theta, a) \tag{20}$$

where

$$E_G(x \mid \theta, a) = \int_y p(y \mid a) E(x \mid \theta, a, y)$$

Calculating $E(x \mid \theta, a, y)$ proceeds exactly as in the proof of Proposition 1 because the true process that links these four variables is the same. Therefore,

$$E(x \mid \theta, a, y) = (1-\beta)\theta + (\beta\lambda + \beta\gamma - \gamma)a + \beta y$$

where β is given by (18). It follows that

$$\begin{aligned} E_G(x \mid \theta, a) &= \int_y p(y \mid a)[(1-\beta)\theta + (\beta\lambda + \beta\gamma - \gamma)a + \beta y] \\ &= (1-\beta)\theta + (\beta\lambda + \beta\gamma - \gamma)a + \beta \int_y p(y \mid a) y \end{aligned}$$

Our task now is to calculate the last term of this expression: First, plugging (1)-(2), we obtain

$$\begin{aligned} \beta \int_y p(y \mid a) y &= \beta \int_{\theta'} p(\theta' \mid a) \int_y p(y \mid \theta', a) y \\ &= \beta \int_{\theta'} p(\theta' \mid a)[\theta' - \gamma a - \lambda a] \\ &= \beta E(\theta' \mid a) - \beta(\gamma + \lambda) a \end{aligned}$$

where $E(\theta' \mid a)$ is determined by the DM's equilibrium strategy, which we have yet to derive.

Plugging this in the expression for $E_G(x \mid \theta, a)$, we obtain

$$\begin{aligned} E_G(x \mid \theta, a) &= (1-\beta)\theta + (\beta\lambda + \beta\gamma - \gamma - \beta\gamma - \beta\lambda)a + \beta E(\theta' \mid a) \\ &= (1-\beta)\theta - \gamma a + \beta E(\theta' \mid a) \end{aligned}$$

Plugging in (20) and rearranging, we obtain

$$\theta = \frac{(1+\gamma)a - \beta E(\theta' \mid a)}{1-\beta}$$

This equation defines θ as a deterministic function f of a, such that $E(\theta' \mid a) = f(a)$. It follows that

$$f(a) = \frac{(1+\gamma)a - \beta f(a)}{1-\beta}$$

or

$$f(a) = (1+\gamma)a$$

Inverting the function, we obtain

$$f^{-1}(\theta) = \frac{\theta}{1+\gamma}$$

which is the DM's equilibrium strategy. ■

Thus, when the DM correctly perceives that a has a direct causal effect on y, the fact that he inverts the causal link between x and y is immaterial for his equilibrium behavior, which coincides with the correct-specification benchmark. It follows that the DM's belief that y is independent of θ is essential for the anomalous effects of our main model. The same conclusion would be obtained if we assumed instead that the DM's subjective DAG does not contain a direct $a \to y$ link but instead contains the link $\theta \to y$ (or an indirect link $\theta \to \phi \to y$, where ϕ is a variable that is objectively correlated with θ but independent of all other variables conditional on θ).

5 Conclusion

This paper explored behavioral consequences of a certain reverse-causality error – inverting the causal link between two outcome variables and deeming one of them exogenous – in an example of a quadratic-normal environment. Two novel qualitative effects emerge from our analysis. First, the DM's behavior is sensitive to the variances of the noise terms in the equations for the two outcome variables x and y – something that would not arise in the quadratic-normal setting under a correctly specified subjective model. Second, the anomalous effects vanish when the DM's direct effect on the final outcome variable y fully offsets the effect that the intermediate outcome variable x has on y. In this sense, the decision context protects the DM from his reverse-causality error.

As we saw in Section 4, these novel effects crucially rely on the combination of the two aspects of the DM's model misspecification – namely, his inversion of the causal link between the two outcome variables and his belief in the exogeneity of y. When one of these assumptions is relaxed, the DM's behavior ceases to display these effects and can even revert to the correct-specification benchmark. This should not be construed as "fragility" of our analysis, but rather as further demonstration that in Gaussian environments, rational-expectations predictions tend to be robust to many causal misspecifications (see Spiegler (2020b)). The same causal misperceptions would be less innocuous under a non-Gaussian objective data-generating process.

Of course, the specification of the true and subjective DAGs in Figure 1 does not exhaust the range of relevant reverse-causality errors. Spiegler (2016) gives an example in which the true DAG is $a \to x \leftarrow y$, the DM's wrong model is $a \to x \to y$, and y (rather than x) is the payoff-relevant variable. That is, in reality y is exogenous and the DM's reverse-causality error leads him to believe that a causes y – the exact opposite of the situation in our main model. Eliaz et al. (2021) quantify the maximal possible distortion of the estimated correlation between x and y due to this error (as well as more general versions of it).

Thus, I am not claiming that the results in this paper are universal fea-

tures of situations in which the true and subjective DAGs disagree on the direction of a certain link. That would be analogous to claiming that subgame perfect equilibrium in an extensive-form game is robust to changes in the game form. Nevertheless, as the examples in this paper demonstrated, many real-life situations fit the mold of Figure 1 and its variations, such that hopefully the analysis in this paper has provided relevant insights into the consequences of reverse-causality errors for agents who act on them.

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