Regression-based Negative Control of Homophily in Dyadic Peer Effect Analysis

Lan Liu\textsuperscript{1} and Eric Tchetgen Tchetgen\textsuperscript{2}

School of Statistics, University of Minnesota at Twin Cities\textsuperscript{1}
Department of Statistics of the Warton School, University of Pennsylvania\textsuperscript{2}

Abstract

A prominent threat to causal inference about peer effects over social networks is the presence of homophily bias, that is, social influence between friends and families is entangled with common characteristics or underlying similarities that form close connections. Analysis of social network data has suggested that certain health conditions such as obesity and psychological states including happiness and loneliness can spread over a network. However, such analyses of peer effects or contagion effects have come under criticism because homophily bias may compromise the causal statement. We develop a regression-based approach which leverages a negative control exposure for identification and estimation of contagion effects on additive or multiplicative scales, in the presence of homophily bias. We apply our methods to evaluate the peer effect of obesity in Framingham Offspring Study.

\textbf{Key words and phrases:} Causal inference; Collider; Exogeneity; Homophily; Negative Control Exposure.
1. INTRODUCTION

In social network studies, it is of great interest to assess the causal contagion effect of one individual on their social contacts. Classical causal inference was primarily developed within the potential outcome framework and typically involves a “no interference” assumption. Recently, causal inference research has extended the classical potential outcome framework to allow for interference, i.e., that an individual’s outcome may be affected by another’s exposure (Sobel, 2006; Hudgens and Halloran, 2008; VanderWeele and Tchetgen Tchetgen, 2011; Tchetgen Tchetgen and VanderWeele, 2012; Liu and Hudgens, 2014; Liu et al., 2016). However, inferring causation from networks remains challenging because correlation in outcomes between individuals with network ties may not only be due to social influence, but also to latent factors that influence network formation. The phenomenon that individuals tend to associate and bond with persons that they have most in common with is known as homophily (Shalizi and Thomas, 2011).

Different types of experimental designs and analytic methods have been developed to study network formation or to adjust for homophily bias. For example, Camargo et al. (2010) investigated friendship formation among randomly assigned roommates in college and concluded that randomly assigned roommates of different races are as likely to become friends as of the same race. In observational studies, Christakis and Fowler (2007) explored the spread of obesity to one individual (ego) from their friend or spouse (alter). Specifically, they included in a regression model for ego’s BMI, a time-lagged measurement of ego’s obesity status, the obesity status of alter, a time-lagged measurement of alter’s obesity status and some observed covariates. They found that obesity spreads through social ties. Using the same approach, Christakis and Fowler examined the evidence of social influence for smoking, happiness, loneliness, depression, drug use, and alcohol consumption (Christakis and Fowler 2007, 2008; Fowler and Christakis 2008; Christakis and Fowler 2013).

In recent years, published analyses by Christakis and Fowler have come under critical
scrutiny. For instance, Shalizi and Thomas (2011) argued that controlling for alter’s lagged obesity status may at best only partially account for homophily bias. They pointed out that if the latent factor influencing friendship formation affects current obesity status even after controlling for past obesity status, one may still observe an association between ego’s and alter’s obesity status using classical regression methods even if alter has no social influence on ego’s obesity status. Cohen-Cole and Fletcher (2009) argued that using the same method as Christakis and Fowler’s on traits unlikely to be transmitted over a network such as height, acne and headaches led to the same conclusion that they spread over the network. To account for both unmeasured confounding and homophily, O’Malley et al. (2014) leveraged multiple genes in an instrumental variables (IV) approach to identify peer effects under a linear model for the outcome and exposure. They assume that the causal relationship is non-directional and found a positive causal peer effect of BMI between ego and alter using this IV approach. However, the IV approach requires the exclusion restriction that none of the genes used to define the IV has a causal effect on any of the unmeasured factors that give rise to formation of social ties, an assumption which may be difficult to justify in social network problems (Fowler et al., 2009).

In this paper, we are also interested in evaluating the person-to-person spread of traits in a social network. We develop an alternative regression-based approach that explicitly accounts for the presence of homophily bias without requiring a valid IV or relying on linear exposure and outcome regression models. Instead of an IV based design, we consider a negative control design that one observes a variable associated with the unmeasured factor inducing homophily bias, unconfounded with the outcome, and that does not have a direct causal effect on the outcome in view. Such a variable is formally called a negative control exposure variable.

Negative control variables have primarily been used in epidemiological applications to detect and sometimes correct for unmeasured confounding (Lipsitch et al., 2010; Tchetgen Tchetgen, 2013; Sofer et al., 2016; Miao et al., 2018). Elwert and Christakis (2008) recently used a negative control exposure to detect homophily bias in the analysis of dyadic data, i.e., data with
pairs of two individuals. Specifically, they used the death of an ex-wife as a negative control variable to investigate the “widowhood effect”, i.e., the effect of the death of a spouse on the mortality of a widow. However, they do not provide a formal counterfactual approach for inference leveraging a negative control outcome to completely account for homophily bias. Partly inspired by this work, we develop theoretical grounds for the use of negative control exposures in peer influence settings. In order to illustrate our approach, we reconsider as running example the analysis performed by Christakis and Fowler (2007) to evaluate the contagion effect of obesity using dyadic data from the Framingham Study. In the Framingham study, we consider as negative control exposure, the alter’s BMI measurement from the subsequent visit. In contrast to the IV assumption which rules out any dependence between the IV and the unmeasured factor implicated in homophily mechanism, our method requires and leverages such dependence. We provide sufficient conditions under which our negative control exposure can be used to detect and account for homophily bias in order to recover the causal effect of primary interest. Moreover, it is worth noting that the proposed method accommodates both directional and mutual nameship in social influences.

The paper is organized as follows. In Section 2, we introduce notation. We propose a general regression-based framework to adjust for homophily bias with a negative control exposure variable in Section 3. Next, we illustrate our methods in estimating the spread of obesity in the Framingham Offspring Study in Section 4. We conclude with a discussion in Section 5.

2. PRELIMINARIES

In social-network dyadic analysis terminology, the key subjects of interest are called “egos” and any subjects to whom egos are linked are called “alters.” The roles of ego and alter are exchangeable depending on which person’s outcome is of interest. To simplify the problem, we only consider data where the study population can be partitioned into pairs, or “dyads” in social sociology terminology. Although the approach equally applies to overlapping dyads but
requires appropriately accounting for dependence across dyads as discussed in VanderWeele et al. (2012). Following the notation of O’Malley et al. (2014), we use subscript 1 to denote alter and 2 to denote ego for any given dyad. We focus on the spread of a trait between two time points. That is, we take the perspective of individual 2 and the goal is to estimate the effect of individual 1’s trait at baseline on the trait of individual 2 at follow-up. For example, in Framingham Offspring Study, we are interested in the effect of having an obese person as alter at baseline on ego’s BMI status at a subsequent study visit. Such information is important for clinical and public health interventions (Christakis and Fowler, 2007).

We consider a study design where the dyads are based on nameship. As in Framingham Offspring Study, each study participant is required to name a single person of contact in an effort to mitigate loss to follow-up. A dyad is formed between two persons if at least one person names the second. Let \( R_1 = 1 \) if alter names ego as their contact person at baseline and otherwise \( R_1 = 0 \). Similarly, let \( R_2 \) denote whether ego names alter as their contact at baseline. We restrict nameship variables \( R_1 \) and \( R_2 \) within a dyad. Because both \( R_1 \) and \( R_2 \) are binary variables, there are four different nameship types, which we encode with \( S \): (a) null naming \( S = 0 \) if \((R_1, R_2) = (0, 0)\); (b) active naming \( S = 1 \) if \((R_1, R_2) = (0, 1)\); (c) passive naming \( S = 2 \) if \((R_1, R_2) = (1, 0)\) and (d) mutual naming \( S = 3 \) if \((R_1, R_2) = (1, 1)\). Active naming indicates ego names alter while the alter does not name the ego. Passive naming indicates alter names the ego while the ego does not name the alter. Null naming indicates neither individual names the other while mutual naming indicates both individuals name the other. Because dyad formation requires at least one person naming another, \( S \geq 1 \) in the observed sample of dyads.

Let \( Y_{ib}^i \) and \( Y_{i1}^i \) denote the observed traits of individual \( i \) at baseline and at follow-up \( i = 1, 2 \). The outcome of interest is ego’s trait at follow-up, i.e., \( Y_{21}^i \). For clarity sake, subscripts and superscripts are sometime suppressed, such as \( Y = Y_{21}^i \). Let \( A \) denote ego’s exposure value, i.e., that is, the indicator of alter’s trait at baseline. For example, in the case where obesity defines the trait of interest, \( A \) is alter’s obesity status, i.e., \( A = 1(\text{alter’s BMI } \geq 30) \). Our methods
apply more generally, whether $A$ is binary, continuous, polytomous or a count exposure. Let $a$ be a possible realization of $A$ (e.g., $a = 1$ for obese and $a = 0$ for no obese), and $Y(a)$ denote an ego’s potential outcome if her exposure were hypothetically set to $a$. Throughout, we make the consistency assumption that the observed outcome is $Y = Y(a)$ almost surely, when $A = a$.

Let $C_1, C_2$ denote the observed covariates at baseline for alter and ego respectively including exposure variables and let $C$ denote covariates for alter and ego excluding the exposure variable, i.e., $C = (C_1, C_2) \setminus A$. In Framingham Offspring study, we include in $C$: age, sex of both alter and ego, interaction between ego and alters’ age, and ego’s baseline BMI status. Let $U_1$ denote an unmeasured factor that affects not only past and current traits of the alter ($Y_1^b, Y_1^f$), but also the nameship variable $R_1$. Define $U_2$ similarly. The corresponding directed acyclic graph is given in Figure 1 (Shalizi and Thomas, 2011). The parameter of interest is $\gamma_{s,c} = E\{Y(1) - Y(0) | S = s, C = c\}$ for $s = 1, 2, 3$, which corresponds to the average treatment effect of the alter’s baseline trait on ego’s trait at the follow-up visit, given that the dyad is of type $s$ and covariates $C = c$.

Because for all observed dyads, $S \geq 1$, the DAG in Figure 1 represents the conditional distribution of $(Y, A, C)$ conditional on $S \geq 1$. Because $S$ is a descendant of both $U_1$ and $U_2$, in the terminology of graph theory, $S$ is called a collider\(^1\) (Pearl, 2009, Shalizi and Thomas, 2011). A direct consequence of this graphical structure is that a standard regression model for $Y$ conditional on $S, C$ and $A$, which fails to condition on either $U_1$ or $U_2$ will generally be subject to collider bias so that it may reveal a non-null association between $A$ and $Y$ even when $A$ fails to cause $Y$ and there is no unmeasured confounding of the effects of $A$ on $Y$ in the underlying population (see Figure 1). This specific type of collider bias is called homophily bias. Because $U_1$ and $U_2$ are unobserved and $S \geq 1$ is always conditioned on, homophily bias (Shalizi and Thomas, 2011) cannot be accounted for without an additional assumption. Next

\(^1\)Conditioning on collider $S$ or its descendant unblocks a back-door path $A - U_1 - R_1 - S - R_2 - U_2 - Y$ (Pearl, 2009).
we consider leveraging a negative control exposure to both detect and correct for collider bias.

Let $Z$ denote a negative control exposure variable that satisfies the following assumptions:

**Assumption 1.** $Z \not\perp S \mid A, C$;

**Assumption 2.** $Y(a, z) = Y(a)$ almost surely;

**Assumption 3.** $Z \perp Y(a, z) \mid A, C, S, U_2$,

where $\perp$ denotes independence between variables and $\not\perp$ denotes dependence. Assumption 1 states that $Z$ must be associated with $S$ given $A$ and $C$. This assumption is represented in the DAG of Figure 1, provided that the arrow between $U_1$ and $Z$ is known to be present. The assumption would also hold if $Z$ where a direct cause of $R_1$ even if $Z$ were independent of $U_1$. Assumption 2 is a form of exclusion restriction of no direct causal effect of $Z$ on $Y$ upon setting $A$ to $a$. Assumption 3 is an assumption of no unmeasured confounding between $Z$ and $Y$ conditional on $A, C, S,$ and $U_2$. Thus, the association between $Z$ and $Y$ given $A, C, S$ can be attributed completely to homophily bias. Hereafter, a negative control exposure for homophily bias control is a variable known to satisfy Assumptions 1-3.

Furthermore, we assume that the exposure variable is not subject to unmeasured confounding given $(C, S, U_2)$ as illustrated in the DAG in Figure 1:

**Assumption 4.** $A \perp Y(a) \mid C, S, U_2$.

Assumption 4 rules out residual confounding of the causal effect of $A$ on $Y$ upon conditioning on $C, U_2$ and nameship type $S$. However, $A$ is not independent of $Y(a)$ given $C$ and $S$ only and therefore, homophily may be interpreted as inducing a form of unmeasured confounding by $U_2$ upon conditioning on $S$, even though $U_2$ is not a common cause of $A$ and $Y$ in the overall population (i.e., upon marginalizing over $S$).

The following two examples provide choices of negative control exposures that have been considered in social studies.
Example 1. Elwert and Christakis (2008) investigated the potential presence of homogamy bias (homophily bias due to spousal similarity) in making inference about the widowhood effect. Specifically, they proposed to use the potential death of an ex-wife as a negative control exposure of the widowhood effect on the mortality of their ex-husband to test for homogamy bias. They found a significant effect of a current wife’s death on her husband’s mortality but no significant effect of an ex-wife’s death on her ex-husband’s mortality. These results support the existence of a causal widowhood effect, which cannot be explained away by homogamy bias.

Example 2. Cohen-Cole and Fletcher (2009) applied the regression methods in Christakis and Fowler (2007) and Christakis and Fowler (2008) to traits that are unlikely to be transmitted via social networks including acne, headaches, and height. They found that these traits are significantly associated among friends and thus conclude the existence of homophily bias in the network effects in the literature. Technically, these analyses may be viewed as double negative control analyses as they incorporate both negative control exposure and outcome variables (Miao and Tchetgen Tchetgen, 2017; Miao et al., 2018).

We reanalyze the Framingham data considered by Christakis and Fowler (2007) using our proposed methodology taking as negative control exposure variable, the ego’s BMI measure at follow-up $Z = Y^1$. Ego and alter’s contemporaneous BMI measures cannot be causally related, therefore fulfilling Assumption 2. Furthermore, it is clear that such a choice of $Z$ is guaranteed to satisfy Assumption 1 because any unmeasured cause of ego’s baseline BMI (and S) is likely also a cause of his or hers BMI at follow-up. In Section 3, we provide conditions under which Assumption 3 is also credible for this choice of negative control exposure.
3. REGRESSION BASED APPROACH

3.1 Identification

We first discuss the case where \( Y \) is continuous. Suppose the data generating mechanism satisfies

\[
E(Y|S = s, A, C, Z, U_2) = U_2 + b^s(A, C) + \tau^s(C),
\]

(1)

where \( b^s(0, C) = 0 \), \( b^s(A, C) \) and \( \tau^s(C) \) are otherwise unrestricted. The outcome regression model (1) assumes that the effect of \( U_2 \) on ego’s trait does not interact with \( A \). Under Assumptions 2–3 encoded in the model, the right-hand side of Model (1) does not depend on \( Z \). Furthermore, under Assumption 4, the conditional causal effect of interest under Model (1) is

\[
E\{Y(1) - Y(0)|S = s, U_2, C, Z\} = E\{Y(1) - Y(0)|S = s, C\} = b^s(1, C) - b^s(0, C).
\]

For example, in Framingham Offspring Study, the parameter of interest can be interpreted as the contagion effect in nameship \( s \) of alter’s obesity status at baseline on ego’s BMI at the follow-up visit within levels of \( C \). A detailed derivation of the causal contagion effect is given in the Appendix. The standard linear structural model is a special case corresponding to \( b^s(A, C; \beta^s_A) = \beta^s_A A \), \( \tau^s(C; \beta^s_C) = \beta^s_C T C \), where \( T \) denotes matrix transpose.

However, because \( U_2 \) is unobserved, an additional assumption is needed for identification. We consider the following generalized polytomous logit model for \( S|A, C, Z \) and \( U_2 \)

\[
\log \frac{\Pr(S = s|A, C, Z, U_2)}{\Pr(S = 0|A, C, Z, U_2)} = \alpha^s U_2 + \gamma^s(A, C, Z),
\]

(2)

where \( \gamma^s(A, C, Z) = \log \Pr(S = s|A, U_2 = 0, C, Z)/\Pr(S = 0|A, U_2 = 0, C, Z) \) is the baseline log odds function of \( S = s \) when \( U_2 \) is set to its reference value 0. Equation (2) specifies a log linear odds ratio association between \( U_2 \) and \( S \) conditional on \( A \), \( C \) and \( Z \) while leaving \( \gamma^s(A, C, Z) \) unrestricted. An important example within this class of models we will primarily focus on is given by a multinomial logistic regression \( \log \{\Pr(S = s|A, C, Z, U_2)/\Pr(S = 9 \}


Additionally, we assume that in the population, $U_2$ and $(A, Z)$ are mean independent conditional on $C$:

$$E(U_2|A, C, Z) = E(U_2|C).$$

Assumption (3) is consistent with the causal diagram in Figure 1 because $U_2$ and $A, Z$ are marginally independent for any pair of individuals in the underlying population, i.e. in absence of collider bias induced by conditioning on $S$.

Finally, we assume that

$$\Delta_s \perp \perp (A, Z)|S, C,$$

where $\Delta_s = U_2 - E(U_2|S = s, A, C, Z)$. Assumption (4) states that conditional on $C$ and $S$, the association between $U_2$ and $(A, Z)$ is entirely due to a location shift. This assumption would hold if $U_2$ were normally distributed with homoscedastic error, conditional on $S = s, A, C, Z$.

In principle, as apparent in proving our main results, equation (4) only needs to hold for $S = 0$, and therefore selection bias may in fact be more severe for dyads with $S \neq 0$ so that association between $\Delta_s$ and $(A, C)$ may manifest itself beyond the mean in these dyads, e.g., with the shape and spread of $U_2$.

Assumptions (1)–(4) are not testable without an additional restriction. The following example illustrates a familiar shared random effect model under which equations (1)–(4) hold.

**Example 3.** Suppose that $E(Y|S = s, A, C, Z, U_2) = U_2 + \beta_s^a(C)A + \beta_s^T C$,

$$\log \frac{\Pr(S = s|A, C, Z, U_2)}{\Pr(S = 0|A, C, Z, U_2)} = \alpha^sU_2 + \gamma_1^sA + \gamma_2^sT C + \gamma_3^sZ$$

and $U_2$ is the random effect shared between models for $Y$ and $S$ to encode a latent association between them with $U_2|S = s, A, C, Z \sim N(\eta^T C, \sigma^2)$, $s = 0, \ldots, 3$, then Assumptions (1)–(4) hold.
We now give our main identification result under Model (1).

**Proposition 1.** Under Model (1), Assumptions 1–4 and equations (2)–(4), we have that

\[
E(Y|A, C, Z, S = s) = \sum_{\tilde{s} \neq s} \beta^{\tilde{s}}(C) \Pr(S = \tilde{s}|A, C, Z) + b^s(A, C) + \bar{\tau}^s(C),
\]

(5)

where \(\bar{\tau}^s(C)\) is an unrestricted function of \(C\), \(\beta^{\tilde{s}}(C) = E(U_2|A, C, Z = s) - E(U_2|A, C, Z, S = \tilde{s})\). Equation (5) highlights the important role of the negative control variable \(Z\) which appears on the right hand side of the equation only through its association with \(S\) in \(\Pr(S = \tilde{s}|A, C, Z)\). Note that equation (5) would continue to hold even if \(Z\) were not conditioned on (or the edge from \(Z\) to \(U_1\) were removed in Figure 1, such that \(Z\) were independent of \(U_1\) given \(A, C, S\), with \(\Pr(S = \tilde{s}|A, C)\) in for \(\Pr(S = \tilde{s}|A, C, Z)\), in which case it would generally not be possible to tease apart this latter term which captures selection bias from structural part of the equation \(b^s(A, C)\) as both are unrestricted function of \((A, C)\), thus rendering the causal effect non-identified. Identification of the causal contagion effect now depends on identification of \(\Pr(S = \tilde{s}|A, C, Z)\) given dyadic study design. Below, we provide sufficient conditions under which such identification is possible.

According to Proposition 1, the coefficient \(\beta^{\tilde{s}}(C) = E(U_2|A, C, Z, S = s) - E(U_2|A, C, Z, S = \tilde{s})\). Hence, \(\beta^{\tilde{s}}(C)\) encodes the association between \(S\) and \(U_2\) and therefore is zero if either \(U_2\) does not predict \(S\), i.e., \(\alpha^s\) is the same for all \(s\), or if \(U_2\) is degenerate in the sense that it does not predict \(Y\). In the Gaussian case of Example 3, we show in the Appendix that \(\beta^{\tilde{s}}(C) = \sigma^2(\alpha^s - \alpha^{\tilde{s}})\) making explicit the aforementioned interpretation. An important advan-
tage of the proposed approach is that it provides a framework to formally test the null hypothesis of no homophily bias as a test of the null hypothesis that $\beta_{s\bar{s}} = 0$ for all $s, \bar{s}$.

Proposition 1 assumes the identity link function for the outcome model. Similar results can be obtained for a multiplicative model (i.e. log link) which is more appropriate for binary or count outcomes. For instance, when the response is binary, the following conditional causal risk ratio may be of interest $P\{Y(1) = 1|S, C\}/P\{Y(0) = 1|S, C\}$ for $s = 1, 2, 3$. To ground ideas, suppose that

$$\log E(Y|S = s, A, C, Z, U_2; \beta^s) = U_2 + b^s(A, C) + \tau^s(C).$$

(6)

Because $U_2$ is conditioned on in (6), suppose Assumption 4 holds, $\exp\{b^s(1, C) - b^s(0, C)\}$ can be interpreted as the causal contagion effect of alter on ego on the multiplicative scale, e.g. on the risk ratio scale for binary $Y$. A similar effect can be defined when the treatment $A$ is continuous. We have the following result for the multiplicative model, the proof of which is given in the Appendix. With a slight abuse of notation, we use the same notation for parameters as in the case of the additive model.

**Proposition 2.** Under Model (6), Assumptions 1–4 and equations (2)–(4), we have

$$\log E(Y|S = s, A, C, Z; \beta^{s\bar{s}}(C)) = \sum_{\bar{s} \neq s} \beta^{s\bar{s}}(C) \Pr(S = \bar{s}|A, C, Z) + b^s(A, C) + \tau^s(C),$$

(7)

where $\tau^s(C)$ is an unrestricted function of $C$.

Propositions 1 and 2 are only useful to the extent that one can identify the selection mechanism $Pr(S|A, C, Z)$ from observed dyadic sample. Because the sample implicitly conditions on $S \geq 1$, nonparametric identification is in general not an option, and therefore one must impose a restriction in order to make progress. In this vein, we propose to posit a model of form $Pr(S|A, C, Z; \theta)$ with finite dimensional unknown parameter $\theta$.  

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3.2 Estimation and Inference

Specifically, we make the following assumption to simplify the estimation procedure.

**Assumption 5.** $R_1 \perp \perp R_2|(A, C, Z)$.

Under Assumption 5, we may specify the following parametric model

$$
Pr(S = \tilde{s}|A, C, Z; \theta) = Pr(R_1|C_1, Z; \theta_1) Pr(R_2|C_2; \theta_2),
$$

where $\theta = (\theta_1^T, \theta_2^T)^T$, $R_1$ is independent of $C_2$ given $C_1, Z$ and $R_2$ is independent of $C_1, Z$ given $C_2$, $Pr(R_i = 1|C_i, Z; \theta_i)$ follows a logistic regression with mean $\exp(\theta_i^T \tilde{C}_i)/(1 + \exp(\theta_i^T \tilde{C}_i))$, where $\tilde{C}_1 = (C_1, Z)$ and $\tilde{C}_2 = C_2$. Then, we have

$$
Pr(R_1, R_2|A, C, Z; \theta) = \frac{\exp(\theta_1^T \tilde{C}_1 R_1 + \theta_2^T \tilde{C}_2 R_2)}{\{1 + \exp(\theta_1^T \tilde{C}_1)\}\{1 + \exp(\theta_2^T \tilde{C}_2)\}}.
$$

It follows that

$$
Pr(S|A, C, Z; \theta) = \frac{1(S = 1) \exp(\theta_2^T \tilde{C}_2) + 1(S = 2) \exp(\theta_1^T \tilde{C}_1) + 1(S = 3) \exp(\theta_1^T \tilde{C}_1 + \theta_2^T \tilde{C}_2)}{\{1 + \exp(\theta_1^T \tilde{C}_1)\}\{1 + \exp(\theta_2^T \tilde{C}_2)\}}.
$$

Because the observed sample space conditions on $R_1 + R_2 \geq 1$, the observed likelihood function for nameship mechanism for a given dyad is given by

$$
Pr(R_1, R_2|R_1 + R_2 \geq 1, A, C, Z; \theta) = \frac{\exp(\theta_1^T \tilde{C}_1 R_1 + \theta_2^T \tilde{C}_2 R_2)}{\exp(\theta_1^T \tilde{C}_1) + \exp(\theta_2^T \tilde{C}_2) + \exp(\theta_1^T \tilde{C}_1 + \theta_2^T \tilde{C}_2)},
$$

the conditional log-likelihood is,

$$
l(\theta) = \sum_{j=1}^{J} \log Pr(S_j|S_j \geq 1, \tilde{C}_{j1}, \tilde{C}_{j2}; \theta)
$$

$$
= \sum_{j=1}^{J} \theta_1^T \tilde{C}_{j1} 1(S_j = 1) + \theta_2^T \tilde{C}_{j2} 1(S_j = 2) + (\theta_1^T \tilde{C}_{j1} + \theta_2^T \tilde{C}_{j2}) 1(S_j = 3)
$$

$$
- \log \{\exp(\theta_1^T \tilde{C}_{j1}) + \exp(\theta_2^T \tilde{C}_{j2}) + \exp(\theta_1^T \tilde{C}_{j1} + \theta_2^T \tilde{C}_{j2})\},
$$

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where \( j = 1, \ldots, J \) is the index for dyad and \( J \) is the total number of dyads in the study. The maximum likelihood estimator \( \hat{\theta} \) for \( \theta \) is defined as \( \hat{\theta} = \arg \max \theta l(\theta) \). Once \( \hat{\theta} \) has been obtained, we proceed by fitting Model (5) with the plug-in estimate \( \Pr(S|A, C, Z; \hat{\theta}) \) to obtain the least square estimates for parameters in the model of \( Y \) on regressors \( \{b^s(A, C), \tau^s(C), \Pr(S = \tilde{s}|A, C, Z) : \tilde{s} \neq s\} \). The two-step estimation procedure is summarized in Algorithm 1.

**Algorithm 1** Two step estimation procedure with a negative control variable

1. Choose a negative control variable \( Z \) that satisfies Assumptions 1–3. Specify the nameship model \( \Pr(S|A, C, Z; \theta) \). Obtain an estimate \( \hat{\theta} \) of \( \theta \) by fitting \( \Pr(S|S \geq 1, A, C, Z; \theta) \) to the data.

2. Estimate coefficients in the additive Model (5) (or the multiplicative Model (7)) using the estimated \( \Pr(S|A, C, Z; \hat{\theta}) \).

One could in principle relax Assumption 5 by allowing dependence between \( R_1 \) and \( R_2 \). A natural approach could involve specifying a random effects model \( \Pr(R_1, R_2|A, C, Z) = \int_b \Pr(R_1|C_1, Z, b) \Pr(R_2|C_2, b) f(b) db \) where \( f(b) \) follows a normal distribution with mean zero variance \( \sigma_b^2 \) and \( \Pr(R_1|C_1, Z, b) \) and \( \Pr(R_2|C_2, b) \) are logistic regressions with random intercept \( b \). All parameters could be estimated by maximizing observed data likelihood as described above, which in this case would involve numerical integration to evaluate the likelihood contribution of each dyad.

To derive the asymptotic distribution of the contagion effect estimator, we assume dyads are non-overlapping and people from different dyads are independent. Let \( \rho = (\theta, \beta) \) denote the vector of the parameters in the nameship mechanism and the outcome regression. Let \( G_\theta(O; \theta) \) and \( G_\beta^s(O; \rho) \) denote the estimating functions corresponding to \( \hat{\theta} \) and \( \hat{\beta} \) in nameship type \( s \), such that \( \hat{\rho} = \{\hat{\theta}, \hat{\beta}\} \) is the solution to the vector equation \( \sum_{i=1}^n G^s(O; \rho) = 0 \), where \( G^s(O; \rho) = \{G_\theta(O; \theta), G_\beta^s(O; \rho)\}^T \). Let \( A \otimes 2 = A \otimes A^T \) denote the Kronecker product of \( A \) and \( A^T \) and let \( \overset{\cdots}{\rightarrow} \) denote convergence in distribution. The following proposition gives the
asymptotic distribution of $\hat{\rho}$. A proof can be directly obtained from standard estimating equation theory (Stefanski and Boos, 2002). The resulting variance estimator with plugin parameter estimates is typically known as the sandwich estimator.

**Proposition 3.** Under Model (1), suppose that Assumptions 1–4 hold and that modeling Assumptions (2)–(4) hold, then $n^{1/2}(\hat{\rho} - \rho) \xrightarrow{d} N(0, \Sigma_{\rho}^s)$ as $n \to \infty$, where $\Sigma_{\rho}^s = U^{-1}VU^{-T}$, $U = -E\{\partial G^s(O_i; \rho)/\partial T \rho\}$, $V = E\{G^s(O_i; \rho)^{\otimes 2}\}$.

Similar to the additive model, the multiplicative model (7) only involves observed variables, and thus it could be fit to the data. We also carry out estimation in a two-step fashion: the nameship mechanism is estimated at the first stage and the estimated propensity is used at the second stage to obtain parameter estimates in the regression model. Asymptotic distribution of the parameter estimates under model (7) can be obtained similar to that in Proposition 3.

4. FRAMINGHAM OFFSPRING STUDY

The Framingham Offspring Study was initiated in 1971 and the study population consists of most of the offsprings of the original Framingham Heart Study cohort and the spouses of the offsprings. Clinical exams were offered every four years. During each clinical exam, the participants underwent a detailed examination including physical examination, medical history, laboratory testing, and electrocardiogram. At the end of each exam, each participant was asked to name a single friend, sibling or spouse, which was likely to be the one with the most influence. The original purpose of the naming process was to record a person of contact, but such information also revealed network ties and thus has been used to assess the social influence (Christakis and Fowler, 2007; O’Malley et al., 2014). Among the network ties provided, approximately 50% of the nominated friend contacts were also participants in the FHS and thus they had the same information, including BMI collected. Most spouses of FHS participants were also FHS participants.
Therefore, by design, the Framingham Offspring Study population could be partitioned into dyads. We estimated our model with unique dyads of spousal and nearly disjoint friendship. Occasional overlap of dyads when the same person was named by multiple individuals was ignored similar to O’Malley et al. (2014). Because later visits suffered from severely low attenuation rate, we focused on the spread of obesity between baseline and the first follow-up.

We carried out a peer effect analysis for 4849 distinct dyads for which alters are spouses, siblings, or friends of egos. The status of ego and alter was randomly assigned. In principle, one can use both assignments in single analysis, however, that required clustering analysis at the level of dyad to account for correlation within dyad. For the purpose of illustration, we considered a single contribution per dyad. Obesity status was defined as a binary variable that takes value 1 if BMI is over 30, and 0 if otherwise. Let \( A = 1(Y_1 > 30) \) denote the exposure of ego, that is, the indicator of alter being obese at baseline. We were interested in the causal effect of alter’s obesity status at baseline on the ego’s BMI at follow-up. Covariates \( C \) included sex (1 for Female and 0 for Male), age of both ego and alter, two-way interactions between ages of ego and alter, and ego BMI at baseline. Ages were mostly between 19 to 52 (5% and 95% quantile respectively). We mean centered age for both ego and alter for numerical stability.

We first carried out a standard regression-based analysis which did not adjust for the potential homophily bias. More specifically, we first fitted a naive model without distinction among different nameships \( E(Y|S = s, A, C; \beta_0, \beta_a, \beta_c) = \beta_0 + \beta_a A + \beta_c T C \) to the data. Results are given in Table 1. Ego BMI at baseline was significantly associated with ego BMI at the follow-up. Adjusting for ego and alter’s gender and age, alter’s obesity status had a significant positively association with the ego’s BMI at follow-up (\( \hat{\beta}_a = 0.24 \), with standard error 0.10 and \( p \)-value 0.01). This effect was subject to homophily bias. Next, we fitted a naive model stratifying by different nameship types, i.e., we fitted \( E(Y|S = s, A, C; \beta_0^s, \beta_a^s, \beta_c^s) = \beta_0^s + \beta_a^s A + \beta_c^s T C \) to the data. Results are given in Table 2. Alter’s obesity status at baseline had a significant positive association on ego’s current BMI in a mutual nameship (\( \hat{\beta}_a^3 = 0.33 \) with standard error 0.13.
and \(p\)-value 0.01). Although this model is more informative than the naive model which does not condition on nameship type, such an effect still may not have causal interpretation due to possible homophily bias.

Next, we carried out a negative control regression adjustment for homophily bias. We selected alter’s weight at follow-up as a negative control variable, i.e., \(Z = Y_i^1\). Alter’s follow-up weight is an appropriate choice of negative control exposure because it cannot be causally related to ego’s contemporaneous weight, therefore satisfying Assumptions 2–3. Such assumptions presume absence of any feedback in alter and ego weight change between baseline and follow-up, which is certainly expected under the sharp null of no contagion effect of weight, but may be violated under the alternative, as discussed in conclusion. Because \(U_1\) is associated with ego’s baseline weight, it may be reasonable to expect that it would also be associated with ego’s weight at follow-up \(Z\), therefore fulfilling assumption 1. The parameter estimates of the nameship process are given in Table 3. Negative control variable, the alter BMI at follow-up, was significantly associated with nameship process. The estimated nameship mechanisms were then included as predictors in the outcome regression model

\[
E(Y|A, C, Z, S = s; \beta_0^s, \beta_a^s, \beta_c^s, \beta_{cs}^s) = \beta_0^s + \sum_{\tilde{s} \neq s} \beta_{cs}^s \Pr(S = \tilde{s}|A, C, Z) + \beta_a^s A + \beta_c^s C
\]

under an assumption that \(\beta_{cs}^s(C)\) does not depend on \(C\). Outcome regression model estimates were given in Table 4. Standard errors were estimated following Proposition 3. Therefore, uncertainty from both stages of estimation is reflected in both estimated standard errors and \(p\)-values. Our analysis provides formal evidence that homophily bias may be operating in these data. Specifically, a subset of homophily coefficients \(\beta_{cs}^s\) were marginally statistically significant (for example, \(\hat{\beta}_{12} = -20.67\) with standard error 10.57 and \(p\)-value 0.05) indicating at least part of the association between ego and alter’s weight within each dyad was indeed subject to homophily bias and therefore not causal. Alter’s obesity status at baseline had a positive association with ego’s BMI at the follow-up for passive-nameship and mutual nameship but not for active-nameship. However, no contagion effect remained statistically significant upon
accounting for homophily bias.

5. DISCUSSION

In this paper, we have proposed a simple regression-based adjustment for homophily bias with a negative control exposure variable $Z$. The unmeasured variables $U_1$ and $U_2$ could in principal also directly affect $R_2$ and $R_1$ respectively, in which case, under our negative control assumptions the proposed approach may still apply.

A reviewer noted that our choice of negative control exposure in Framingham application, ego BMI at follow-up is only applicable as a negative control variable if contagion only occurs at discrete times which are directly observed, i.e. ruling out feedback effects alluded to in Section 4. To illustrate this, consider a situation where there is an intermediate time $t = 0.5$ in between baseline and follow-up (shown in Figure 3). Ego and alter BMI can affect the other person’s BMI at a follow-up visit. The dashed line denotes effects between individuals. Although alter BMI at follow-up is unlikely to have a direct causal effect on ego BMI at follow-up, they are both confounded by ego BMI at the intermediate time, $Y_{1^{0.5}}$. Such confounding could potentially invalidate the negative control assumption 3. This point has also been suggested in Ogburn and VanderWeele (2014): estimation of contagion effects at multiple time points may be complicated by the feedback issue as the entire evolution history need to be considered. The problem of potential uncontrolled confounding may also persist when we have multiple time points as compared with continuous time points. Because the Framingham Offspring Study follow-up was at 4 years post baseline, it is possible that causal contagion effects exist at some intermediate time between the two visits. The assumption of no unmeasured intermediate time with contagion effects is more plausible in the setting where individuals only interact during visits not in between, e.g., patients usually interact with their doctors at clinic visits. It is still notable as suggested in Section 4 that such complication will not occur even in Framingham Offspring Study under the sharp null hypothesis of no contagion effect, in which case, our ap-
proach would provide a valid test of the sharp null hypothesis of no contagion within 4 year window between baseline and follow-up.

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A.

Proof of Proposition 1

Under Model (1), we have

\[
E(Y|S = s, A, C, Z) = E(U_2|S = s, A, C, Z) + b^s(A, C) + \tau^s(C)
\]

\[
= E(U_2|A, C, Z) + \sum_{\tilde{s} \neq s} \{E(U_2|A, C, Z, S = s) - E(U_2|A, C, Z, S = \tilde{s})\} \Pr(S = \tilde{s}|A, C, Z)
\]

\[
+ b^s(A, C) + \tau^s(C)
\]

\[
= E(U_2|A, C, Z) + \sum_{\tilde{s} \neq s} \{E(U_2|A, C, Z, S = s) - E(U_2|A, C, Z, S = \tilde{s})\} \Pr(S = \tilde{s}|A, C, Z)
\]

\[
+ b^s(A, C) + \tau^s(C),
\]

where the last equation follows from assumption (3). Because by assumption (2),

\[
\exp(\alpha^s U_2) = \frac{\Pr(S = s|A, C, Z, U_2) \Pr(S = 0|A, C, Z, U_2 = 0)}{\Pr(S = 0|A, C, Z, U_2) \Pr(S = s|A, C, Z, U_2 = 0)}
\]

\[
= \frac{f(U_2|A, C, Z, S = s) \Pr(U_2 = 0|A, C, Z, S = 0)}{f(U_2|A, C, Z, S = 0) \Pr(U_2 = 0|A, C, Z, S = s)},
\]

we have

\[
E(U_2|A, C, Z, S = s) = \frac{E\{U_2 \exp(\alpha^s U_2)|A, C, Z, S = 0\}}{E\{\exp(\alpha^s U_2)|A, C, Z, S = 0\}}
\]

\[
= \frac{\partial}{\partial \alpha^s} \log E\{\exp(\alpha^s U_2)|A, C, Z, S = 0\}
\]

\[
= \frac{\partial}{\partial \alpha^s} \log E[\exp\{\alpha^s(E(U_2|A, C, Z, S = 0) + \Delta_0)\)|A, C, Z, S = 0]
\]

\[
= E(U_2|A, C, Z, S = 0) + \frac{\partial}{\partial \alpha^s} \log E[\exp(\alpha^s \Delta_0)|A, C, Z, S = 0]
\]

\[
= E(U_2|A, C, Z, S = 0) + \beta^s(C),
\]
where $\beta^s(C) = \partial \log E[\exp(\alpha^s\Delta_s)|A, C, Z, S = 0]/\partial \alpha^s = \partial \log E[\exp(\alpha^s\Delta_0)|C, S = 0]/\partial \alpha^s$ and the last equation holds due to assumption (4). Thus, $E(Y|s, A, C, Z) = \sum_{\tilde{s} \neq s} \beta^{s\tilde{s}}(C) \Pr(S = \tilde{s}|A, C, Z) + b^s(A, C) + \tau^s(C)$, where $\beta^{s\tilde{s}}(C) = \beta^s(C) - \beta^{\tilde{s}}(C)$ and $\tau^s(C) = E(U_2|C) + \tau^s(C)$.

Now, we show the identification of the causal effect. Under Model (1), we have

$$E(Y|A = 1, C, S, Z, U_2) - E(Y|A = 0, C, S, Z, U_2) = b^s(1, C) - b^s(0, C).$$

We also have

$$E(Y|A = 1, C, S, Z, U_2) - E(Y|A = 0, C, S, Z, U_2)$$

$$= E(Y|A = 1, C, S, U_2) - E(Y|A = 0, C, S, U_2)$$

$$= E(Y(1)|A = 1, C, S, U_2) - E(Y(0)|A = 0, C, S, U_2)$$

$$= E(Y(1)|C, S, U_2) - E(Y(0)|C, S, U_2),$$

where the first equation is due to Assumption 3, the second and third are due to causal consistency assumption and Assumption 4, respectively. Thus, $E(Y(1)|C, S, U_2) - E(Y(0)|C, S, U_2) = b^s(1, C) - b^s(0, C)$. Integrating over $U_2$ yields that $E(Y(1)|C, S) - E(Y(0)|C, S) = b^s(1, C) - b^s(0, C)$. By Assumption 1, the nameship mechanism $\Pr(S|A, C, Z)$ is a function of the negative control exposure $Z$. Additionally, the negative control exposure variable $Z$ only appears in the nameship mechanism model, thus, the term that involves $Z$, $\sum_{\tilde{s} \neq s} \beta^{s\tilde{s}}(C) \Pr(S = \tilde{s}|A, C, Z)$; and the term that does not involve $Z$, $b^{ss}(A, C) = b^s(A, C) + \tau^s(C)$ can be identified. Hence, the causal effect can be identified.

**Derivation of $\beta^{s\tilde{s}}(C)$ under Example 3**

Under the assumptions of Example 3, $\Delta_s \sim N(0, \sigma^2)$. Using the moment generating function, $\beta^s(C) = \partial \log \exp(\sigma^2\alpha^s_2/2)/\partial \alpha^s = \sigma^2\alpha^s_2$. Thus, $\beta^{s\tilde{s}}(C)$ can be derived.
Proof of Proposition 2

As shown in the proof of Proposition 1, assuming (2)–(4), we have

\[
E(U_2|A, C, Z, S = s) = E(U_2|A, C, Z) + \sum_{\tilde{s} \neq s} \{E(U_2|A, C, Z, S = \tilde{s}) - E(U_2|A, C, Z, S = s)\} \Pr(S = \tilde{s}|A, C, Z)
\]

\[
= E(U_2|C) + \sum_{\tilde{s} \neq s} \{E(U_2|C, S = s) - E(U_2|C, S = \tilde{s})\} \Pr(S = \tilde{s}|A, C, Z)
\]

\[
= E(U_2|C) + \sum_{\tilde{s} \neq s} \beta^{s\tilde{s}}(C) \Pr(S = \tilde{s}|A, C, Z).
\]

Under Model (6), we have

\[
E(Y|S = s, A, C, Z) = E\{E(Y|U_2, S = s, A, C, Z)|S = s, A, C, Z\}
\]

\[
= E[\exp\{U_2 + b^s(A, C) + \tau^s(C)\}]|S = s, A, C, Z
\]

\[
= \exp\{E(U_2|S = s, A, C, Z) + b^s(A, C) + \tau^s(C)\} E\{\exp(\Delta_s)|S = s, A, C, Z\}
\]

\[
= \exp\{E(U_2|C) + \sum_{\tilde{s} \neq s} \beta^{s\tilde{s}}(C) \Pr(S = \tilde{s}|A, C, Z) + b^s(A, C) + \tau^s(C)\} E\{\exp(\Delta_s)|S = s, C\}
\]

\[
= \exp\{\sum_{\tilde{s} \neq s} \beta^{s\tilde{s}}(C) \Pr(S = \tilde{s}|A, C, Z) + b^s(A, C) + \tilde{\tau}^s(C)\},
\]

where \(\tilde{\tau}^s(C) = \tau^s(C) + \log E\{\exp(\Delta_s)|S = s, C\} + E(U_2|C)\). To identify the causal contagion effect on the risk ratio scale, we have

\[
\log \{\Pr(Y(1) = 1|S, A = 1, C, Z, U_2)/\Pr(Y(0) = 1|S, A = 1, C, Z, U_2)\} = b^s(1, C) - b^s(0, C),
\]

which is equivalent to \(\{\Pr(Y(1) = 1|S, A = 1, C, Z, U_2)/\Pr(Y(0) = 1|S, A = 0, C, Z, U_2)\} = \exp\{b^s(1, C) - b^s(0, C)\}\), hence under Assumption 4 we have

\[
\{\Pr(Y(1) = 1|S, C)/\Pr(Y(0) = 1|S, C)\} = \exp\{b^s(1, C) - b^s(0, C)\},
\]

which is the causal contagion effect. Under Assumption 1, the nameship mechanism model
Figure 1: Causal diagram illustrating homophily bias.

Figure 2: The parameter of interest is the effect of the obesity status of alter (individual 1) at baseline on ego BMI (individual 2) at follow-up, i.e., $A = Y_{1}^{b}$, $Y = Y_{2}^{1}$. We use $Y_{i}^{b}$ and $Y_{i}^{1}$ to denote the observed weight information on individual $i$ baseline and follow-up, $U_{i}$ is the unmeasured factor that affects both the nameship and the weight of individual $i$, $R_{i}$ is the nameship variable for individual $i$ and $S$ is the summary of nameship type. We omit observed covariates $C_{i}$ for simplicity. In our empirical example, we use $Y_{1}^{1}$ as the negative control exposure $Z$.

Pr$(S | A, C, Z)$ depend on $Z$, hence, following a similar argument as in the proof of Proposition 1, $b^{s}(A, C)$ and the contagion effect on the risk ratio scale can be identified.
Table 1: Estimates, standard error and p-values of coefficients in a naive analysis without distinction among relationships

|                          | Est | SE   | p   |
|--------------------------|-----|------|-----|
| ego’s BMI<sub>b</sub>    | 0.94| 0.01 | <0.01|
| alter’s obese<sub>b</sub>| 0.24| 0.10 | 0.01 |
| ego’s sex                | 0.30| 0.07 | <0.01|
| ego’s age                | -0.18| 0.06| <0.01|
| alter’s sex              | -0.03| 0.07| 0.64 |
| alter’s age              | 0.17 | 0.06 | <0.01|
| age:alter’s age          | 0.02 | 0.02 | 0.30 |
Table 2: Estimates, standard error and p-values of coefficients in a naive analysis across different nameships: active naming ($S = 1$), passive naming ($S = 2$) and mutual naming ($S = 3$)

|                      | $S = 1$ |             | $S = 2$ |             | $S = 3$ |             |
|----------------------|---------|-------------|---------|-------------|---------|-------------|
|                      | Est     | SE          | $p$     | Est         | SE      | $p$         | Est     | SE      | $p$     |
| ego’s BMI$_b$        | 0.98    | 0.02        | <0.01   | 0.94        | 0.02    | <0.01       | 0.92    | 0.01    | <0.01   |
| alter’s obesity$_b$ | -0.16   | 0.22        | 0.47    | 0.37        | 0.24    | 0.11        | 0.33    | 0.13    | 0.01    |
| sex                  | 0.39    | 0.17        | 0.02    | 0.39        | 0.17    | 0.03        | 0.25    | 0.08    | <0.01   |
| age                  | 0.00    | 0.12        | 0.97    | -0.25       | 0.15    | 0.09        | -0.17   | 0.09    | 0.07    |
| alter’s sex          | -0.12   | 0.16        | 0.48    | -0.10       | 0.17    | 0.54        | 0.00    | 0.08    | 0.96    |
| alter’s age          | -0.04   | 0.14        | 0.78    | 0.13        | 0.14    | 0.35        | 0.27    | 0.09    | <0.01   |
| age:alter’s age      | 0.04    | 0.05        | 0.45    | 0.03        | 0.05    | 0.50        | 0.04    | 0.04    | 0.31    |
Table 3: Nameship mechanism estimates adjusted for alter’s age gender and $Z$.

|                | Est  | SE  | $p$  |
|----------------|------|-----|------|
| Ego model      |      |     |      |
| alter’s BMI    | 0.01 | 0.01| 0.39 |
| ego’s sex      | 0.09 | 0.08| 0.26 |
| ego’s age      | -0.24| 0.07| <0.01|
| alter’s sex    | -0.08| 0.09| 0.35 |
| alter’s age    | -0.41| 0.07| <0.01|
| Alter model    |      |     |      |
| ego’s BMI      | 0.02 | 0.01| 0.08 |
| alter’s sex    | 0.06 | 0.09| 0.46 |
| alter’s age    | -0.48| 0.07| <0.01|
| ego’s sex      | 0.21 | 0.09| 0.02 |
| ego’s age      | -0.18| 0.07| 0.01 |
| $Z$            | -0.03| 0.01| <0.01|
Table 4: Estimates, sandwich standard error and p-values of coefficients in homophily-adjusted analysis with an negative control exposure variable $Z$ across different nameships: active naming ($S = 1$), passive naming ($S = 2$) and mutual naming ($S = 3$)

|                      | $S = 1$ |              |         | $S = 2$ |              |         | $S = 3$ |              |         |
|----------------------|---------|--------------|---------|---------|--------------|---------|---------|--------------|---------|
|                      | Est     | SE           | $p$     | Est     | SE           | $p$     | Est     | SE           | $p$     |
| ego’s BMI$_b$        | 1.00    | 0.03         | $<0.01$ | 0.93    | 0.03         | $<0.01$ | 0.93    | 0.02         | $<0.01$ |
| alter’s obesity$_b$ | -0.24   | 0.33         | 0.47    | 0.52    | 0.34         | 0.13    | 0.21    | 0.19         | 0.27    |
| sex                  | 0.82    | 0.35         | 0.02    | 0.30    | 0.36         | 0.39    | 0.22    | 0.18         | 0.23    |
| age                  | -0.74   | 0.58         | 0.21    | -0.07   | 0.74         | 0.93    | 0.30    | 0.55         | 0.58    |
| alter’s sex          | -0.25   | 0.33         | 0.45    | -0.17   | 0.27         | 0.52    | 0.11    | 0.13         | 0.37    |
| alter’s age          | -1.42   | 0.92         | 0.12    | 0.37    | 1.19         | 0.76    | 0.80    | 0.76         | 0.29    |
| age:alter’s age      | 0.08    | 0.17         | 0.63    | 0.14    | 0.24         | 0.57    | 0.23    | 0.17         | 0.18    |
| $\beta_{s1}$        | -10.93  | 5.88         | 0.06    | -2.93   | 14.91        | 0.84    | -10.97  | 9.64         | 0.26    |
| $\beta_{s2}$        | -8.81   | 11.67        | 0.45    | 3.45    | 12.49        | 0.78    | -3.22   | 7.00         | 0.65    |
| $\beta_{s3}$        | -20.67  | 10.57        | 0.05    | 1.02    | 4.66         | 0.83    | 3.80    | 2.98         | 0.20    |
Figure 3: Causal diagram illustrating homophily bias for multiple time points.

Figure 4: The parameter of interest is the effect of the obesity status of alter (individual 1) at baseline ($A = 1(Y^b_1 > 30)$) on ego BMI (individual 2) at time 1 ($Y^1_2$). We use $Y_{i.5}$ to denote the observed weight information on individual $i$ at a time point between baseline and follow-up. The dashed line denotes causal effects between individuals. We take $Z = Y^1_1$ as the negative control exposure variable.