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A Reduced Social Relations Model for Dyad-Constant Dependent Variables

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Abstract Dyadic network data occur when each member in a group provides data about each other member in the group (e.g., how much they like each other person). Such data have a complex nesting structure, such that bivariate responses (e.g., Person A's liking of B and vice versa) are dependent upon out-going and in-coming random effects that are correlated within individuals. Dyadic network models for such data include the social relations model for normal data and the p_2 and j_2 models for dichotomous data, but we have seen no application or generalization to accommodate a rarely discussed type of variable from this framework: variables that are constant within a dyad. Dyad-constant variables could include background variables such as whether a dyad is same or opposite sex or how many years two friends have known each other, which require no special modification to use as predictors (Jorgensen et al., Soc Netw 54:26–40, 2018). But they could also be outcomes, such as the difference in a married couple's relationship satisfaction or the similarity in symptoms of a (set of) psychological disorder(s). We explore how such dyad-constant outcomes can be modeled, demonstrating on a data set from a clinic for patients with eating disorders.

Keywords Dyadic data · Social networks · Round-robin designs · Social relations model

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1 Introduction

In this paper we present a modified social relations model (SRM) for network-structured outcomes that are constant within a dyad. The SRM is traditionally applied to data gathered from a round-robin design (Warner et al., 1979), wherein each member of a group provides information about each other member of the group (e.g., how much person i likes person j). For a group of size N, this would yield $N^2 - N = N(N-1)$ unique observations—two observations for each pair (dyad) in the group, depicted by the vector $\mathbf{y}_{\{ij\}}$, where the braces indicate the ordering of members $i \neq j$ is arbitrary. Because each member belongs to multiple dyads, each bivariate dyadic observation is nested in the set of observations in which person i is a member, as well as in the set of observations in which person j is a member.

From a multilevel-modeling perspective (Snijders & Kenny, 1999), the bivariate dyadic (i.e., Level-1) observations $\mathbf{y}_{\{ij\}}$ are cross-classified under both ego and alter (i.e., Level-2) effects. However, round-robin data (also called sociometric data, relational data, interpersonal data, and network data) are more complexly structured than textbook examples of cross-classified data (e.g., students nested with schools and neighborhoods, whose effects are independent of each other). The nature of the network structure is explained by way of introducing the SRM and its extensions, followed by showing how the SRM can be specified to accommodate data that do not vary within a dyad (the focus of this chapter). Results are presented from an empirical clinical-psychology example, which was the motivation behind developing this innovation. The discussion includes comparison with related models and suggestions for future developments.

1.1 The Social Relations Model

The SRM can be depicted as a random-effects model (Nestler, 2016) that decomposes $\mathbf{y}_{\{ij\}}$ into person- and dyad-level components:

$$\mathbf{y}_{\{ij\}} = \begin{bmatrix} y_{ij} \\ y_{ji} \end{bmatrix} = \mu + \begin{bmatrix} E_i + A_j + R_{ij} \\ E_j + A_i + R_{ji} \end{bmatrix}, \tag{1}$$

where μ is the expected value of the observations (e.g., average amount of liking in the group). E_i and A_j are person-level ego (out-going) and alter (in-coming) effects, respectively—for example, E_i would represent how much person i likes others in general, and A_j would represent how much person j is generally liked by others (i.e., likeability). Each R is a dyad-level residual, which contains relationship-specific effects (e.g., how much i uniquely likes j beyond what is expected from their person-level effects) as well as measurement error. More descriptive terms have been used for E_i and A_j, such as actor and partner effects when $\mathbf{y}_{\{ij\}}$ are behavioral interactions (e.g., social mimicry; Salazar Kämpf et al., 2018) or perceiver and target

effects when $\mathbf{y}_{\{ij\}}$ are interpersonal perceptions (e.g., of personality traits; Kenny, 1994).

Each person's vector of ego and alter effects is assumed bivariate normally distributed:

$$\begin{bmatrix} E_i \\ A_i \end{bmatrix} \sim \mathcal{N}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \; \Sigma_{EA} = \begin{bmatrix} \sigma_E^2 & \\ \sigma_{EA} & \sigma_A^2 \end{bmatrix} \right), \tag{2}$$

where σ_E^2 and σ_A^2 are the variances of the random person-level effects, respectively, and $\rho_{EA} = \frac{\sigma_{EA}}{\sigma_E \sigma_A}$ is their correlation, termed *generalized reciprocity* (Kenny, 1994). Following from the liking example, positive generalized reciprocity would be observed when those who have a propensity to (dis)like people are also generally (un)likeable. Negative generalized reciprocity might be observed when measuring helpfulness in a collaborative work situation: those who receive the most help (e.g., because they have lower competence) could be expected to provide the least help to others.

Each dyad's pair of residuals is also assumed bivariate normally distributed:

$$\begin{bmatrix} R_{ij} \\ R_{ji} \end{bmatrix} \sim \mathcal{N}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \; \Sigma_R = \sigma_R^2 \begin{bmatrix} 1 & \\ \rho_R & 1 \end{bmatrix} \right), \tag{3}$$

where variances σ_R^2 are constrained to equality for indistinguishable dyads (Kenny et al., 2006, ch. 8) and the (residual) correlation ρ_R between relationship effects is called *dyadic reciprocity* (Kenny, 1994). Following from the liking example, positive dyadic reciprocity implies that if person i particularly likes person j (i.e., more than would be expected given person i's general propensity for liking and person j's general likeability), then person j particularly likes person i, too (i.e., the feeling is mutual). Negative dyadic reciprocity might be observed if person i found person j particularly helpful during collaboration, in which case person j might not have received much help from person i.

A common goal of univariate SRM studies is to calculate the relative contributions of each level of analysis on the overall variability in $\mathbf{y}_{\{ij\}}$ (σ_y^2). This can be expressed as the proportion of variance accounted for by each orthogonal variance component:

$$\sigma_y^2 = \sigma_E^2 + \sigma_A^2 + \sigma_R^2. \tag{4}$$

Note that although E_i and A_i can be correlated *within person* i, the random effects of different persons i and j are assumed independent (and identically distributed). Thus, for a single observation y_{ij}, the ego and alter components are independent. The proportions of variance explained by each component can be used to compare their relative impact on the observed phenomenon. For example, is the degree to which person i likes person j influenced more by person i's propensity to like others, by person j's likeability, or is it primarily their personal chemistry with each

other? The decomposition of the full covariance matrix for $\mathbf{y}_{\{ij\}}$ (i.e., Σ_y) follows a similar logic:

$$\Sigma_y = \Sigma_{EA} + \Sigma'_{EA} + \Sigma_R\,, \tag{5}$$

$$\sigma_y^2 \begin{bmatrix} 1 & \\ \rho_{ij} & 1 \end{bmatrix} = \begin{bmatrix} \sigma_E^2 & \sigma_{EA} \\ \sigma_{EA} & \sigma_A^2 \end{bmatrix} + \begin{bmatrix} \sigma_A^2 & \sigma_{EA} \\ \sigma_{EA} & \sigma_E^2 \end{bmatrix} + \sigma_R^2 \begin{bmatrix} 1 & \rho_R \\ \rho_R & 1 \end{bmatrix}. \tag{6}$$

1.2 Extending the SRM with Covariates

Covariates can be added to the SRM, either as explicit predictors of random effects (e.g., Koster & Leckie, 2014; Lüdtke et al., 2013) or as auxiliary correlates (e.g., Brunson et al., 2016), which can alleviate the effects of missing data (Jorgensen et al., 2018). When person-level covariates ($\mathbf{x}$) are added as predictors of ego and alter effects, the distributional assumption in Eq. 2 applies to Level-2 residuals ε and δ:

$$\begin{bmatrix} E_i \\ A_i \end{bmatrix} = \begin{bmatrix} \sum_{p=1}^{P} \beta_p x_{i,p} + \varepsilon_i \\ \sum_{p=1}^{P} \alpha_p x_{i,p} + \delta_i \end{bmatrix}, \tag{7}$$

where P is the number of person-level predictors, β_p is the effect of predictor x_p on ego effects, and α_p is the effect of predictor x_p on alter effects. For example, personality traits ($\mathbf{x}$) such as openness to experience and extraversion could be used to predict general liking (E) and likability (A), respectively.

Likewise, dyad-level predictors $q = 1, \ldots, Q$ can be added to the Level-1 model:

$$\begin{bmatrix} y_{ij} \\ y_{ji} \end{bmatrix} = \beta_0 + \begin{bmatrix} E_i + A_j + \sum_{q=1}^{Q} \gamma_q w_{ij,q} + \sum_{q=1}^{Q} \lambda_q w_{ji,q} + R_{ij} \\ E_j + A_i + \sum_{q=1}^{Q} \gamma_q w_{ji,q} + \sum_{q=1}^{Q} \lambda_q w_{ij,q} + R_{ji} \end{bmatrix}, \tag{8}$$

where the intercept β_0 is a conditional mean that supplants the role of the grand mean μ in Eq. 1. The E and A terms in Eq. 8 can also incorporate predictors as in Eq. 7. Intrapersonal (γ) and interpersonal (λ) slopes can be distinguished (Nestler, 2016). For example, Salazar Kämpf et al. (2018) reported that person i especially liking person j was associated with person i especially mimicking person j (an intrapersonal effect) in subsequent interaction; however, after a time lag, person i especially mimicking person j was then associated with person j especially liking person i (an interpersonal effect).

Like dyad-level outcomes, dyad-level predictors can differ for each member of the dyad (i.e., $w_{ij} \neq w_{ji}$)—for example, how attractive or agreeable each person thinks the other person is. However, predictors could also be constant within a dyad ($w_{\{ij\}} = w_{ij} = w_{ji}$), which is often (but not necessarily) a function of person-

level variables. For example, a dummy code indicating same- or opposite-sex dyads is a function of the members' sexes. Alternatively, how many months or years the members of a dyad have been acquainted is not a function of their person-level characteristics, but it is nonetheless constant within a dyad. Note that when $w_{ij,q} = w_{ji,q}$, the intrapersonal and interpersonal effects in Eq. 8 cannot be distinguished ($\gamma_q = \lambda_q$), so the predictor $w_{\{ij\},q}$ should only be included once. But even when a predictor W varies within a dyad, intrapersonal (γ) and interpersonal effects (λ) are each constrained to equality across the bivariate observations. These equality constraints hold for indistinguishable dyads, for the same reason there is an equality constraint on the residual variances (i.e., the order within dyad $\{ij\}$ is arbitrary).

2 Reducing the SRM for Dyad-Constant Outcomes

Dependent relational/network variables can also be constant within a dyad ($y_{ij} = y_{ji}$). For example, we might be interested in explaining why friends differ in how much they like each other. No method has been formally defined for accommodating such data, so introducing such a method is the primary goal of this paper. We will begin by focusing only on the basic SRM in Eq. 1, which suffices to discuss the relevant issues. We then consider covariate effects after resolving the issues.

A dyad-constant outcome $y_{\{ij\}}$ is equivalent to equating bivariate observations on the left-hand side of Eq. 1 ($y_{ij} = y_{ji}$), which implies the equality of the summed components on the right-hand side of Eq. 1:

$$E_i + A_j + R_{ij} = E_j + A_i + R_{ji} \text{ ; thus,} \tag{9}$$

$$R_{ij} = R_{ji} \quad \text{and} \tag{10}$$

$$E_i + A_j = E_j + A_i \text{ ; furthermore,} \tag{11}$$

$$E_i = A_i \quad \text{and} \tag{12}$$

$$A_j = E_j \text{ .} \tag{13}$$

Because the person- and dyad-level components of Eq. 9 are independent, this further implies the equivalence of the relationship components in Eq. 10 and of the sum of person components in Eq. 11. Finally, person i's random effects are independent of person j's effects, implying the ego and alter effects are equivalent for persons i (Eq. 12) and j (Eq. 13).

Returning to the social-mimicry example (Salazar Kämpf et al., 2018), we might be interested in the degree to which persons i and j differ in how frequently they (un)consciously imitate each other during a conversation. Larger absolute values of this discrepancy ($y_{\{ij\}} = |y_{ij} - y_{ji}|$) could be interpreted as evidence of social dominance within a dyad, whereas smaller absolute values might indicate more equity among conversation partners.

The equivalence of person-level effects ($E_i = A_i$) implies equivalence of their variance components in Eq. 2 ($\sigma_E = \sigma_A$) and a correlation of $\rho_{EA} = 1$. The equivalence of relationship effects (Eq. 10) also implies a correlation of $\rho_R = 1$:

$$\begin{bmatrix} E_i \\ A_i \end{bmatrix} \sim \mathcal{N}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \sigma_E^2 \begin{bmatrix} 1 & \\ 1 & 1 \end{bmatrix} \right) \text{ and } \begin{bmatrix} R_{ij} \\ R_{ji} \end{bmatrix} \sim \mathcal{N}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \sigma_R^2 \begin{bmatrix} 1 & \\ 1 & 1 \end{bmatrix} \right). \tag{14}$$

This simplifies the distributional assumptions from bivariate normality (Eqs. 2 and 3) to univariate normality:

$$E_i \sim \mathcal{N}(0, \sigma_E) \text{ and } R_{\{ij\}} \sim \mathcal{N}(0, \sigma_R), \tag{15}$$

where the use of E or A subscript is arbitrary given their equivalence, and again the braces around $\{ij\}$ indicate the order is arbitrary. Thus, the dyad-constant outcome $y_{\{ij\}}$ can be expressed as a univariate function of person- and dyad-level effects:

$$y_{\{ij\}} = \mu + E_i + E_j + R_{\{ij\}}. \tag{16}$$

In the social-dominance (discrepancy in mimicry) example, the person-level effect E_i would represent person i's general tendency to dominate ($y_{ij} - y_{ji} > 0$) or defer ($y_{ij} - y_{ji} < 0$) control of a conversation, and the residual $R_{\{ij\}}$ continues to capture relationship-specific tendencies along with other sources of error.

The reduced SRM in Eq. 16 still contains components from both members of the dyad because the observations $y_{\{ij\}}$ are still nested under (a) all the dyadic observations in which person i is a member, as well as (b) all the dyadic observations in which person j is a member. That is, the observations are still cross-classified, but there is simply no way to distinguish between out-going ego effects and in-coming alter effects. There is simply a single vector of person-level effects, two of which (E_i and E_j) are components of any dyadic observation $y_{\{ij\}}$. Thus, the variance decomposition in Eq. 4 becomes

$$\sigma_y^2 = 2\sigma_E^2 + \sigma_R^2. \tag{17}$$

In practice, the proportion of variance in dyad-constant $y_{\{ij\}}$ attributable to person-level characteristics (e.g., individuals' general tendencies to seize or surrender control of a conversation) should therefore be calculated by doubling the estimated variance of person-level effects: $2\hat{\sigma}_E^2 / (2\hat{\sigma}_E^2 + \hat{\sigma}_R^2)$.

The reduced SRM presented in Eqs. 16 and 15 can be applied to variables $y_{\{ij\}}$ that are identical within a dyad ($y_{ij} = y_{ji}$) or are of equal magnitude but opposite signs ($y_{ij} = -y_{ji}$), which would still be redundant information with the same variance decomposition. Dyad-constant variables could be defined independently, such as the number of things persons i and j found in common when getting acquainted, or $y_{\{ij\}}$ could be a function of person- or dyad-level variables. For example, differences in personality traits (person-level characteristics) would be

dyad-specific but constant in absolute magnitude, as would differences in how much each person likes the other (dyad-level characteristics).

Beyond questions of whether network-level phenomena are driven primarily by person- versus dyad-level characteristics, the reduced SRM provides no information about qualitatively distinct person-level variances ($\sigma_E^2 = \sigma_A^2$), nor does it provide information about generalized or dyadic correlations (both $\rho = 1$ because $E_i = A_i$ and $R_{ij} = R_{ji}$). Thus, the reduced SRM might not garner much interest for substantive applications. When network-structured variables are constant within each dyad, we imagine substantive interest would lie primarily in explaining individual or relationship differences at each level of analysis.

We now extend the reduced SRM to include covariates, which was what motivated its development (see Sect. 3). Given $E_i = A_i$, Eq. 7 reduces to

$$E_i = \sum_{p=1}^{P} \beta_p x_{i,p} + \varepsilon_i. \tag{18}$$

Substituting the Level-2 model (Eq. 18) into the reduced SRM (Eq. 16) yields an interesting result:

$$\begin{aligned} y_{\{ij\}} &= \beta_0 + E_i + E_j + R_{\{ij\}} \\ &= \beta_0 + \left(\sum_{p=1}^{P} \beta_p x_{i,p} + \varepsilon_i \right) + \left(\sum_{p=1}^{P} \beta_p x_{j,p} + \varepsilon_j \right) + R_{\{ij\}} \\ &= \beta_0 + \sum_{p=1}^{P} \beta_p (x_{i,p} + x_{j,p}) + \varepsilon_i + \varepsilon_j + R_{\{ij\}}, \end{aligned} \tag{19}$$

in that the slope β_p can be multiplied by the sum of person i's and j's values on predictor x_p.

Similarly, dyad-level predictors can be added, but when $y_{ij} = y_{ji}$, intra- and interpersonal effects cannot be distinguished ($\gamma = \lambda$), resulting in a similar result as in Eq. 19:

$$\begin{aligned} y_{\{ij\}} &= \beta_0 + E_i + E_j + \sum_{q=1}^{Q} \gamma_q w_{ij,q} + \sum_{q=1}^{Q} \gamma_q w_{ji,q} + R_{\{ij\}} \\ &= \beta_0 + E_i + E_j + \sum_{q=1}^{Q} \gamma_q (w_{ij,q} + w_{ji,q}) + R_{\{ij\}}. \end{aligned} \tag{20}$$

When a predictor is also constant within dyads ($w_{ij,q} = w_{ji,q}$), the slope is effectively a weight for $2 \times w_{\{ij\},q}$. In this case, it would be more intuitive to either (a) divide $w_{\{ij\},q}$ by 2 prior to analysis or (b) only include one "copy" of

$w_{\{ij\},q}$ in Eq. 20, so that $\hat{\gamma}_q$ could be interpreted as expected difference in $y_{\{ij\}}$ per unit-increase in $w_{\{ij\},q}$. In Sect. 3, we use option (b) to estimate the effect of a three-category dyad-constant predictor, represented by two dummy codes.

3 Motivating Example

3.1 Background

Eating disorders are serious psychiatric conditions characterized by overconcern with weight and shape and by problematic behaviors such as fasting, binge-eating, and self-induced vomiting that contribute to increased morbidity and elevated risk of death (van Hoeken and Hoek, 2020). Risk models for eating disorders, such as the Tripartite Influence Model, posit that appearance pressures from peers contribute to the development of eating disorder symptoms (Thompson et al., 1999). Existing research supports the importance of both socialization (i.e., contagion) of some disordered eating behaviors (i.e., binge-eating like behaviors; Zalta and Keel, 2006), and selection effects, that is, individuals forming friendships with those who share similar attitudes (Rayner et al., 2013). If peer attitudes and behaviors increase risk for eating-disorder attitudes and behaviors, then peers might also be able to help mitigate risk or clinically significant eating-disorder symptoms. Indeed, changes in perceptions of peer norms predict decreases in disordered eating attitudes in prevention settings (Cruwys et al., 2015).

While peers are prominent in sociocultural models of eating disorder risk, less is known about the role of peers in the maintenance or treatment of clinically diagnosed eating disorders. Outpatient treatment models rely on one-to-one interactions between patients and clinicians. Augmenting this standard care with mentorship from a recovered peer improves some treatment outcomes (Ranzenhofer et al., 2020). More intensive treatments, such as partial hospitalization programs, occur in group settings for significant periods of time (e.g., 30 hours/week). By the nature of the group setting, peers with eating disorders are an integral part of treatment in partial hospitalization programs. Preliminary work suggests that patients who develop quality friendships in treatment have greater motivation to change (Malmendier-Muehlschlegel et al., 2016). However, engagement in relationships developed during treatment is associated (after discharge) with both positive and negative outcomes, depending on the types of interactions that take place (Saffran et al., 2016).

Taken together, the literature suggests that friendships—and the disordered eating attitudes and behaviors of those friends—may a play a role in the maintenance of eating disorder behaviors and thus may also play a role in facilitating positive treatment outcomes. Existing literature has been limited by reliance on perceptions of peers, but social network data can overcome these limitations by "objectively" measuring peer eating-disorder symptoms (Jorgensen et al., 2018). The current

study represents the first step in understanding how friendships formed in treatment may contribute improved treatment outcomes via the modeling of socialization and recovery-oriented attitudes and behaviors. We sought to understand whether similarity in the severity of eating disorder symptoms was associated with reported friendships. We hypothesized that eating disorder symptoms would be more similar among friends than non-friends.

3.2 Method

Participants and Procedure Participants were recruited from a nonprofit partial hospitalization program for eating disorders in the midwestern United States. Patients were enrolled in 30 hours of evidence-based treatment per week, with most treatment occurring in a group setting. As part of routine care, patients completed assessments on a weekly basis. For the current study, participants were provided a letter from the second author inviting the participant to participate in a study of social influences on eating-disorder treatment outcomes. A graduate research assistant was available to answer any questions that patients had about the research study. After having questions answered, participants provided written informed consent. All study procedures were approved by the local Institutional Review Board. Once enrolled, participants were asked to complete a weekly assessment of eating disorder symptoms and a social network assessment. In addition, participants were asked to provide permission to access their medical record to extract diagnosis, demographic information, and weekly assessments of depressive and anxiety symptoms. Data collection occurred over an 8-week period. Over the 8-week period, 18 individuals were invited to participate, 13 individuals provided informed consent, and 12 individuals provided data on their eating disorder symptoms and social network. Because patients both began and ended treatment during the course of the study, participation by week ranged from 3 to 7. The current study focuses on the three consecutive weeks with highest absolute participation. Among those who provided data, mean age was 27.25 years ($SD = 14.59$). All participants identified as non-Hispanic, White females. The modal eating-disorder diagnosis was "Other Specified Feeding or Eating Disorder," and all patients had a comorbid mood or anxiety disorder.

Measures Eating-disorder symptoms were measured using the Eating Disorder Examination Questionnaire Short Form (Gideon et al., 2016), an adapted version of the well-established Eating Disorder Examination Questionnaire (Fairburn and Beglin, 1994). Twelve questions assess eating-disorder attitudes and behaviors over the previous 7 days; possible scores ranged 0–36. Scale reliability ranged from $\alpha = 0.89$–0.95 across weeks.

Participants were provided a roster of all patients in the partial hospitalization program. They were asked to identify "your friends, that is, the group members you hang around with the most or are closest to. You are welcome to list as many friends

as appropriate." They were also given the option to identify group members whom they looked to as a role model for recovery.

3.3 Estimating a Reduced SRM

Our outcome of interest was the absolute difference in eating-disorder symptoms between patients i and j (i.e., $y_{\{ij\}}$ was a function of person-level characteristics). Higher values indicated greater dissimilarity in a dyad, and values closer to zero indicated greater similarity. Our focal predictor was whether patients i and j considered each other friends, which is a dyad-level variable that can vary within dyads. Using mutual nonfriendship as the reference category, we used a dummy code to indicate dyads with reciprocated friendship ($w_{\{ij\},2}$). Because the outcome did not vary with dyads, we could not distinguish between asymmetry in one direction (ij) or the other (ji), so a single dummy code ($w_{\{ij\},1}$) represented nonreciprocal, asymmetric friendship. Thus, in our fitted model:

$$y_{\{ij\}} = |y_i - y_j| = \beta_0 + \gamma_1 w_{\{ij\},1} + \gamma_2 w_{\{ij\},2} + E_i + E_j + R_{\{ij\}}, \tag{21}$$

β_0 represents the average dissimilarity in eating disorder symptoms among mutual nonfriends. The slopes for dummy codes in Eq. 21 represent how different from nonfriends the average (dis)similarity was among asymmetric (γ_1) and mutual (γ_2) friends. Our fitted model does not include person-level effects.

Given the documented limitations of two-step estimation approaches (Nestler, 2016; Nestler et al., 2020; Lüdtke et al., 2018), we only considered options to estimate the whole model simultaneously. Although maximum likelihood estimation (MLE) is available for round-robin data in the R package `srm` (Nestler et al., 2019), the software is not set up to accommodate dyad-constant outcome variables (Nestler et al., 2020). Instead, we used Markov chain Monte Carlo (MCMC) estimation (Hoff, 2005; Lüdtke et al., 2013; Jorgensen et al., 2018) using the general Bayesian modeling software Stan (Carpenter et al., 2017) via the R package `rstan`. Stan uses a modified Hamiltonian Monte Carlo (HMC) algorithm called the No-U-Turn Sampler (NUTS) that simultaneously samples the entire vector of estimates from the parameter space, as opposed to iterating one parameter at a time like Gibbs sampling.

The unknown quantities (parameters) in Eq. 21 include β_0, the fixed effects (γ_1 and γ_2), the vector of person-level random effects (E), and the variance components (σ^2_E and σ^2_R). In our Stan program (available online[1] and in the Appendix), we specified a standard-normal prior distribution for random effects:

[1] Data and software scripts are available on the Open Science Framework (OSF): https://osf.io/j53n8/. No person-level variables are provided, and person-level IDs are randomized within each week to preserve anonymity (i.e., IDs in Week 6 do not correspond to IDs in Weeks 7 or 8).

$$E^* \sim \mathcal{N}(\mu = 0, \sigma = 1). \tag{22}$$

Priors for other parameters were selected based on descriptive statistics to be minimally informative without placing undue weight on values far outside the range of data (Smid et al., 2020). We specified half-normal priors (i.e., normal distributions truncated below 0) for SDs rather than variances (σ_E and σ_R):

$$\sigma_{(E \text{ or } R)} \sim \text{half-}\mathcal{N}(\frac{h}{5}, \frac{h}{5}), \tag{23}$$

where $h = \frac{\max(y_{\{ij\}}) - \min(y_{\{ij\}})}{2}$ (i.e., half the empirical range of the outcome). The intercept and slopes were also specified to include the range of plausible values without being strongly informative:

$$\beta_0 \sim \mathcal{N}(\text{median}(y_{\{ij\}}), h) \tag{24}$$

$$\gamma_{(1 \text{ or } 2)} \sim \mathcal{N}(0, h) \tag{25}$$

To calculate each dyad's expected values $\hat{y}_{\{ij\}}$ each time parameters were sampled from the posterior (indexed below with superscript m), we added scaled random effects ($E = E^* \times \sigma_E$) to the intercept and fixed effects (i.e., Eq. 21 but with the residual $R_{\{ij\}}$ omitted).

$$\hat{y}^m_{\{ij\}} = \beta^m_0 + \gamma^m_1 w_{\{ij\},1} + \gamma^m_2 w_{\{ij\},2} + (E^{*,m}_i + E^{*,m}_j)\sigma^m_E, \tag{26}$$

The likelihood was thus specified as:

$$y_{\{ij\}} \sim \mathcal{N}(\hat{y}_{\{ij\}}, \sigma_R). \tag{27}$$

After 250 burn-in iterations on each of 4 Markov chains, we saved 250 samples from each chain's estimated posterior distribution. Convergence was assessed visually by verifying proper mixing in traceplots, as well as numerically using the potential scale-reduction factor ($\widehat{R} < 1.05$; Gelman and Rubin, 1992) and effective sample size ($N_{\text{eff}} > 100$; Vats et al., 2019), both of which are reported with results in Table 1. The combined 1000 samples from the posterior were used to calculate point (posterior mean) and SE (posterior SD) estimates, as well as empirical 95% credible intervals (CIs) for each (function of) parameter(s). The difference between slopes ($\gamma_2 - \gamma_1$) was calculated at each iteration to capture the mean difference between reciprocal and asymmetric friendships. The 95% CIs were used to infer whether differences between groups were (non)zero.

3.4 Results and Discussion

Variability of friendship nominations confirmed that friendships form in partial hospitalized programs, although friendship nominations were not always reciprocated. Estimated group mean differences in dissimilarity of eating-disorder symptoms are presented in Table 1. Recall that symptoms were measured on a 0–36 scale, so absolute differences between subjects could be as large as 36, although they tended to be smaller because all patients had relatively higher symptom-scores than would be expected in the general population. The top row indicates that due to autocorrelation, the 1000 samples from the posterior have an effective sample size of 421 independent samples from the posterior, which is more than sufficient to minimize Monte Carlo sampling error. The small $\widehat{R} = 1.01$ shows no evidence of convergence problems, and the traceplots (not shown here, but available using the R script on OSF) show evidence of good mixing across chains. Similar convergence diagnostics were found across parameters (all $\widehat{R} \leq 1.01$), and all other effective sample sizes in Table 1 exceed 500.

Again focusing on the top row of Table 1, the average dissimilarity between patients i and j was 2.46 points higher ($SE = 2.42$) among asymmetric friends than among nonfriends. The corresponding CI indicates that given the observed data, there is a 95% posterior probability that the true mean difference is between −2.12 and 7.27, which is a very wide margin of error. Likewise, the second row shows 1.92 points higher average dissimilarity ($SE = 2.09$) among mutual friends than among nonfriends, also with a large margin of error: 95% CI [−2.27, 5.73]. These results contradict our hypothesis that friends would manifest more similar symptoms, but because both CIs include 0, we cannot reject the H_0 that there is simply no effect of friendship on (dis)similarity. The third row compares asymmetric to mutual friends

Table 1 MCMC summaries from fitting reduced SRM to cross-sectional samples from the 3 weeks with the largest sample sizes (21 dyads). Pairwise comparisons are made between groups of dyads that indicated no friendship (0), asymmetric friendship (1), or reciprocated friendship (2). EAP = expected a posteriori (posterior mean), SD = posterior standard deviation, CI = credible interval calculated from posterior percentiles, N_{eff} = effective number of posterior samples (given autocorrelation), $\widehat{R}$ = potential scale-reduction factor

Week	Groups	EAP	SD	95% CI	N_{eff}	$\widehat{R}$
6	1 vs. 0	2.46	2.42	[−2.12, 7.27]	421.06	1.01
	2 vs. 0	1.92	2.09	[−2.27, 5.73]	501.27	1.01
	2 vs. 1	−0.54	2.18	[−5.14, 3.95]	768.25	1.00
7	1 vs. 0	3.10	5.53	[−7.99, 13.48]	1176.77	1.00
	2 vs. 0	−1.00	3.60	[−8.10, 6.16]	961.92	1.00
	2 vs. 1	−4.10	6.54	[−16.35, 9.10]	1304.70	1.00
8	1 vs. 0	−3.35	5.23	[−13.26, 7.24]	722.00	1.00
	2 vs. 0	−3.00	3.79	[−10.73, 4.31]	602.52	1.01
	2 vs. 1	0.35	5.81	[−11.42, 11.17]	1121.66	1.00

in Week 6, and the remaining rows of Table 1 show the estimated mean differences between types of dyad for Weeks 7 and 8.

Across weeks, no clear pattern emerged in eating disorder symptoms; friends were not consistently more or less similar in regards to their eating disorder symptoms than nonfriends. Whereas Week-6 results descriptively indicate that asymmetric (1 vs. 0) and mutual friendship (2 vs. 0) led to greater dissimilarity, Week-8 results showed that asymmetric and mutual friendship led to greater similarity; Week-7 results were mixed. However, all 95% CIs revealed quite a lack of precision, so the none of the differences could be distinguished statistically from zero.

Friendship formation may be more strongly related to other traits, such as personality factors (Forney et al., 2019) or other aspects of psychopathology, rather than to eating disorder symptoms. Indeed, a more consistent pattern was observed such that friends tended to be more similar on depressive symptoms than nonfriends (see the file "SupplementalResults.pdf" on OSF). Additionally, prior work supports that the make-up of a therapy group (i.e., between-group effects) has a moderate effect on treatment outcomes (Kivlighan et al., 2020). Thus, the lack of consistent findings from week to week may reflect changes in the group make-up as patients entered or left treatment or got to know one another better. Future work may wish to examine closeness as a moderator of any similarity effects. Collecting and combining data from multiple, independent partial hospitalization programs will allow for a better understanding of factors that influence whether or how eating disorder symptoms are related to friendship formation and the converse: whether or how friendship may be related to improvements in eating disorder symptoms.

4 General Discussion

This chapter presents a reduced SRM with covariates designed to enable modeling of dyad-level outcomes that do not vary within a dyad. The real-data application showed that the model is estimable with real data, and converges quickly on a solution, even with relatively little data. However, the low sample size each week did not provide much power to detect any of the estimated effects. Future research should verify the practical feasibility of this model via Monte Carlo simulation studies.

Although it is difficult to anticipate the demand for this model development, it is noteworthy that similar network models for binary outcomes—the p_2 (Van Duijn et al., 2004; Zijlstra et al., 2006) and j_2 (Zijlstra, 2017) models—have also been adapted for symmetric (dyad-constant) outcomes (Blanken et al., 2021), as implemented in the `b2ML()` function of the R package `dyads` (Zijlstra, 2021). Future research might compare this implementation to the reduced SRM presented here, but with a probit link (Koster & Aven, 2018) to accommodate a binary outcome.

Appendix

Annotated Stan (Carpenter et al., 2017) syntax for the SRM fitted to the motivating-example data in Sect. 3.2 is provided below. The *.stan file can be found on the OSF, along with the data and an R script to fit the model using the R package `rstan`: https://osf.io/j53n8/

```
data {
  // sample sizes
  int<lower=0> Nd;         // number of dyads   (Level 1)
  int<lower=0> Np;         // number of persons (Level 2, cross-classified)
  // observed data
  vector[Nd] Y;            // observed round-robin outcome
  vector[Nd] one;          // dummy codes: one-way friend nomination
  vector[Nd] both;         //              reciprocal friend nomination
  // ID variables above Level 1
  int IDp[Nd, 2];          // person-level IDs (cross-classified)
}
transformed data {
  // save limits for default priors
  vector[2] yLimits;
  real halfRange;

  // calculate observed limits
  yLimits[1] = min(Y);
  yLimits[2] = max(Y);
  halfRange = (yLimits[2] - yLimits[1]) / 2;
}
parameters {
  // means and SDs
  vector[3] BETA;         // intercept + 2 slopes
  real<lower=0>  s_d;     // dyad-level residual SD
  real<lower=0>  s_p;     // person-level random-effect SD

  vector[Np] e_p;         // vector of person-level random effects (unit scale)
}
transformed parameters {
  vector[Nd] Yhat;        // expected values, given random effects
  for (n in 1:Nd) {
    Yhat[n] = BETA[1] + BETA[2]*one[n] + BETA[3]*both[n] +
              s_p*e_p[ IDp[n,1] ] + s_p*e_p[ IDp[n,2] ];
  }
}
model {
  // priors for means/slopes and SDs, based on empirical ranges:
  BETA[1] ~ normal(yLimits[1] + halfRange, halfRange);
  BETA[2] ~ normal(0, halfRange);
  BETA[3] ~ normal(0, halfRange);
  s_d ~ normal(halfRange / 5, halfRange / 5) T[0, ]; // residual SD
  s_p ~ normal(halfRange / 5, halfRange / 5) T[0, ]; // person-level SD
  // random effects (sample on unit scale)
  e_p ~ std_normal();

  // likelihood
  Y ~ normal(Yhat, s_d);
}
generated quantities{
  // mean difference between groups with dummy codes
  real recip;
  recip = BETA[3] - BETA[2];
}
```

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