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## Article (Accepted version) (Refereed)

Running Head: WITHIN-CLUSTER DYNAMIC EFFECTS

# A Multilevel Simultaneous Equations Model for Within-Cluster Dynamic Effects, with an Application to Reciprocal Parent-Child and Sibling Effects

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Fiona Steele and Jon Rasbash were supported by a research grant from the UK Economic and Social Research Council as part of the National Centre for Research Methods (RES-576-25-0003). Jennifer Jenkins was supported by a grant from Social Sciences and Humanities Research Council. Data came from the Avon Brothers and Sisters Study which was funded by the Medical Research Council, UK. We are grateful to Judy Dunn and Tom O'Connor for allowing the use of these data, and to Paul Clarke, the Associate Editor and the reviewers for their extremely helpful comments on earlier versions of the paper. Sadly, Jon Rasbash passed away in March 2010.

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Abstract

There has been substantial interest in the social and health sciences in the reciprocal causal influences that people in close relationships have on one another. Most research has considered reciprocal processes involving only two units, although many social relationships of interest occur within a larger group (families, work groups, peer groups, classrooms etc.). This article presents a general longitudinal multilevel modelling framework for the simultaneous estimation of reciprocal relationships amongst individuals with unique roles operating in a social group. We use family data for illustrative purposes but the model is generalizable to any social group in which measurements of individuals in the social group occur over time, individuals have unique roles and clustering of the data is evident. We allow for the possibility that the outcomes of family members are influenced by a common set of unmeasured family characteristics. The multilevel model we propose allows for residual variation in the outcomes of parents and children at the occasion, individual and family levels, and residual correlation between parents and children due to the unmeasured shared environment, genetic factors and shared measurement. Another advantage of this method over approaches used in previous family research is it can handle mixed family sizes. The method is illustrated in an analysis of maternal depression and child delinquency using data from the Avon Brothers and Sisters Study.

*Keywords*: reciprocal influences; autoregressive cross-lagged model; multilevel structural equations model; family effects; parent-child effects; sibling effects

# A Multilevel Simultaneous Equations Model for Within-Cluster Dynamic Effects, with an Application to Reciprocal Parent-Child and Sibling Effects

Social groups, made up of a set of individuals interacting together over time, represent complex phenomena to study. Individuals and groups influence one another through a range of social and biological mechanisms. Experimental designs allow one to isolate the mechanisms of influence unambiguously. Many social phenomena, however, are not amenable to experimental study, and as a consequence attempts have been made to investigate causal influences using longitudinal data. The extent to which individuals are influenced by other individuals or groups (as well as reciprocal relationships) has been examined across multiple domains of study. In the peer literature, adolescent peers have been shown to influence one another's delinquency (Dishion & Owen, 2002) as well as their depressive symptomatology (Connell & Dishion, 2006). In work settings, characteristics of the group (Jolivet et al., 2010) and dyadic (Yakovleva, Reilly, & Werko, 2010) environments have been found to be associated with the functioning of individuals. In classrooms, peer linguistic skills influence the language development of the individual (Mashburn, Justice, Downer, & Pianta, 2009). In the health literature, the obesity of family members and friends has an effect on the obesity of the individual (Christakis & Fowler, 2007). When possible, given the phenomena under study, reciprocal influences are also investigated. Thus we can examine the extent to which a mother influences her child, as well as the way the child influences the mother's behavior over time.

One of the challenges in the interpretation of correlational data is the isolation of influences that help us draw conclusions about causal mechanisms. For instance, the correlation in behavior between parents and children may be due to genes, environmental experiences, or a mixture of the two (Kaffman & Meaney, 2007; Plomin & Davis, 2009). Clustering of individuals within a work setting on feelings of depression may be attributable

to time-invariant factors (the physical plant or the organizational structure) or time-varying factors (the mood of the boss). In the present paper we present a statistical model for the investigation of mutual influence within a social group. We chose the family for our social group as there is already a substantial literature on statistical models that test reciprocal influences in the family domain (Cui, Donnellan, & Conger, 2007; Elgar, Curtis, McGrath, Waschbusch, & Stewart, 2003; Gross, Shaw, & Moilanen, 2008). We use this literature to illustrate the difference between existing approaches and our own. It is the case, however, that the models that we present are generalizable to any social group in which measurements of individuals in the social group occur over time, individuals have unique roles and clustering of the data is evident.

There are several goals to this article. First, we briefly outline the methods that have been used previously for examining reciprocal processes. Such methods are based on growth curve and autoregressive cross-lagged models. We discuss the limitations of these methods for conclusions about causal mechanisms. Second, we introduce an analytic framework that allows for a more comprehensive and flexible examination of reciprocal influences than has been presented to date. The model allows for the inclusion of social groups of different sizes (including groups with only one dyad). We differentiate between reciprocal influences between individuals in a power hierarchy (parents and their multiple children), as well as individuals at the same level of power (e.g. siblings). We allow effects to depend on characteristics of individuals with different roles (e.g. parent and child). Effects of unmeasured factors at the occasion, individual and group levels are included. Third, we illustrate our approach with an analysis of the reciprocal effects of maternal depression and children's behavioral problems, using a dataset that includes up to three children per family. Our approach can be implemented in existing multilevel modelling software.

## A Review of Methods for Estimation of Reciprocal Parent-Child and Sibling Effects

Previous studies into how the behavior of one family member affects another have been restricted to dyadic (or pairwise) relationships involving a parent-child (e.g. Elgar, Curtis, McGrath, Waschbusch, & Stewart, 2003), or sibling pair (e.g. Slomkowski, Rende, Conger, Simons, & Conger, 2001). These studies have involved separate analyses for parent-child and sibling relationships using growth curve models, autoregressive cross-lagged models, or a combination of the two. In the next section, we show how these approaches can be considered special cases within a more general framework that allows simultaneous modelling of parent and child outcomes for mixed family sizes. This generalization has considerable benefits for the framing and testing of complex hypotheses about complex systems. Before introducing our general framework in the next section, we first review the methods used in previous studies of parent-child and sibling relationships, highlighting the biases that may arise when these relationships are not considered simultaneously, and when the effects of unmeasured family characteristics are ignored.

### Bivariate Growth Curve Models

Growth curve models (GCM) are also known as latent trajectory models and are the standard approach for analysing changes in individuals' outcomes over time (Singer & Willett, 2003). In this approach, a curve is fitted to repeated measures of an outcome with individual variation in the level and rate of change in the outcome captured by individual-specific random effects attached to the intercept and slope parameters. Two or more growth processes can be modelled simultaneously using a multivariate GCM in which the random intercepts and slopes for each process are allowed to be correlated (MacCallum, Kim, Malarkey, & Kielcolt-Glaser, 1997; McArdle, 1988). The random effects covariance structure is of particular substantive interest in GCMs involving family data because a non-

zero covariance indicates an association between the underlying trajectories of family members (Gross, Shaw, & Moilanen, 2008; Laird, Pettit, Bates, & Dodge, 2003).

As described by Curran (2003), a GCM can be framed as a multilevel model (MLM) (Goldstein, 2003; Raudenbush & Bryk, 2002) or as a structural equation model (SEM) (Bollen & Curran, 2006), but one may be preferred over the other for certain types of application. For example, it is more straightforward in a multilevel model to accommodate individual variation in the timing of measurements at a given occasion and to allow for further levels of clustering. (Further discussion of the relative strengths of MLM and SEM approaches to growth curve modelling can be found in Ghisletta & Lindenberg (2004), MacCallum et al. (1997), Steele (2008) and Wu, West, & Taylor (2009).)

**Bivariate Autoregressive Cross-lagged Models**

While a bivariate GCM can be used to examine the association between the intercept and slopes of two family members' trajectories, it does not allow for *dynamic* reciprocal effects between individuals' outcomes. Cross-lagged models have become the standard technique for examining whether an earlier outcome of one individual affects a subsequent outcome of another, and have been used in several studies of reciprocal influences between family members. In this approach, the outcome at time $t - 1$ for one individual in a family pair is included as an explanatory variable in the model for the other's outcome at time $t$, and vice versa, with the coefficients of these cross-lags interpreted as reciprocal effects between individuals.

We now consider the form of the cross-lagged model: first for estimating reciprocal parent-child effects; second for estimating reciprocal sibling effects; and discuss the biases that may arise as a result of their use in previous family research. This also introduces notation we will use when specifying our general framework in the next section.

**Model for parent-child effects.** We begin by outlining a simple model for a parent-child dyad of the type already used in previous studies. Denote by $y_{tj}^{(P)}$ and $y_{tj}^{(C)}$ the responses at time $t$ for the parent and child in family $j$ ($t = 1, \ldots, T$; $j = 1, \ldots, J$). (Throughout the article, superscripts $P$ and $C$ are used to indicate variables, coefficients and residuals in the parent and child equations respectively.) Omitting covariates to simplify the exposition, a bivariate model for $y_{tj}^{(P)}$ and $y_{tj}^{(C)}$ (for $t > 1$) can be written

$$y_{tj}^{(P)} = \beta_0^{(P)} + \beta_1^{(P)} y_{t-1,j}^{(P)} + \beta_2^{(P)} y_{t-1,j}^{(C)} + \varepsilon_{tj}^{(P)} \qquad (1a)$$

$$y_{tj}^{(C)} = \beta_0^{(C)} + \beta_1^{(C)} y_{t-1,j}^{(C)} + \beta_2^{(C)} y_{t-1,j}^{(P)} + \varepsilon_{tj}^{(C)} \qquad (1b)$$

where ($\beta_0^{(P)}$, $\beta_0^{(C)}$) are intercepts, ($\beta_1^{(P)}$, $\beta_1^{(C)}$) are the autoregressive or lagged effects, ($\beta_2^{(P)}$, $\beta_2^{(C)}$) are the cross-lagged effects representing reciprocal parent-child effects, and ($\varepsilon_{tj}^{(P)}$, $\varepsilon_{tj}^{(C)}$) are occasion-specific, normally distributed residuals. The inclusion of the lagged terms means that the cross-lagged effects are interpreted as the effect of one individual's outcome at $t - 1$ on the change in the other individual's outcome between $t - 1$ and $t$.

Most previous applications of cross-lagged models in analyses of parent-child reciprocal effects allow for a non-zero correlation between $\varepsilon_{tj}^{(P)}$ and $\varepsilon_{tj}^{(C)}$ at a given occasion $t$, which means that (1a) and (1b) should be estimated jointly (Cui, Donnellan, & Conger, 2007; Elgar, Curtis, McGrath, Waschbusch, & Stewart, 2003; Gross, Shaw, & Moilanen, 2008). A residual correlation will arise if there are unobserved characteristics that affect both parent and child outcomes; for example genetic effects, attributes of the family environment or bias in measurement related to single informant data. The actor-partner interdependence model (APIM) for longitudinal designs (Cook & Kenny, 2005) is a special case of the model described by (1a) and (1b). The APIM is usually applied in situations where the same

outcome is measured on both members of the dyad, for example mother and child attachment security in relation to each other, and is described by Cook and Kenny for two time points. Using APIM terminology, the lags are 'actor' effects and the cross-lags are 'partner' effects.

**Model for sibling effects.** Turning to reciprocal sibling effects, previous research is based on analyses of two children per family. These studies are typically restricted to two-child families or, in larger families, the effects of other siblings are ignored. Moreover, it is common to focus only on the effect of an older sibling on a younger child and to allow only for cross-sectional associations between sibling outcomes (Slomkowski, Rende, Conger, Simons, & Conger, 2001; Snyder, Bank, & Burraston, 2005). Where reciprocal relationships are considered, some form of cross-lagged model is generally employed with the outcome of one child at time $t$ (or the change in their outcome between $t - 1$ and $t$) depending on their sibling's outcome at time $t - 1$ (e.g. Natsuaki, Ge, Reiss, & Neiderhiser, 2009). Denote by $y_{tij}^{(C)}$ the outcome of child $i$ ($i = 1, 2$) of family $j$ at time $t$, and define the younger sibling as child 1 and the older sibling as child 2. A simple autoregressive cross-lagged model for $y_{t1j}^{(C)}$ and $y_{t2j}^{(C)}$ (for $t > 1$) can be written

$$y_{t1j}^{(C)} = \beta_0^{(C)} + \beta_1^{(C)} y_{t-1,1j}^{(C)} + \beta_2^{(C)} y_{t-1,2j}^{(C)} + \varepsilon_{t1j}^{(C)} \qquad (2a)$$

$$y_{t2j}^{(C)} = \beta_0^{(C)} + \beta_1^{(C)} y_{t-1,2j}^{(C)} + \beta_2^{(C)} y_{t-1,1j}^{(C)} + \varepsilon_{t2j}^{(C)} \qquad (2b)$$

which has the same form as the parent-child reciprocal model of (1a) and (1b), and assumes common intercepts, autoregressive effects and cross-lagged effects across siblings . As before, the occasion-specific residuals ( $\varepsilon_{t1j}^{(C)}$ , $\varepsilon_{t2j}^{(C)}$ ) will usually be assumed to follow a bivariate normal distribution, allowing for correlation between sibling outcomes at the same measurement occasion.

Studies that consider only the effects of an older sibling on a younger child estimate

only (2a) using OLS regression, which is likely to lead to biased estimates of sibling effects. The problem with this approach is that the dependent variable $y_{t1j}^{(C)}$ and lagged sibling outcome $y_{t-1,2j}^{(C)}$ will in general have shared unmeasured risk factors (for example genetic and environmental characteristics) leading to correlation between the predictor $y_{t-1,2j}^{(C)}$ and $\varepsilon_{t1j}^{(C)}$ which will in turn result in a biased estimate of $\beta_2^{(C)}$. It is therefore important to model ($y_{t1j}^{(C)}$, $y_{t2j}^{(C)}$) jointly, allowing for correlation between $\varepsilon_{t1j}^{(C)}$ and $\varepsilon_{t2j}^{(C)}$.

Sibling effects models typically include a number of covariates including measures of sibling interaction or relationship quality (Rende, Slomkowski, Lloyd-Richardson, & Niaura, 2005; Slomkowski, Rende, Conger, Simons, & Conger, 2001; Snyder, Stoolmiller, Wilson, & Yamamoto, 2003) and parenting (Natsuaki, Ge, Reiss, & Neiderhiser, 2009; Snyder, Bank, & Burraston, 2005). Such analyses suffer from the same problem noted above when modelling only one side of the sibling relationship: some of the unmeasured family characteristics, both genetic and environmental, that affect child behavior may also influence sibling interactions and parent outcomes. Inappropriately treating measures of sibling interaction and parental behavior as exogenous with respect to child outcomes may lead to biased estimates not only of the effects of these variables, but also of the cross-lagged sibling effects of interest. A solution to the problem, and the approach taken in this article, is to model child and parent outcomes simultaneously allowing for residual correlation between outcomes.

Cross-lagged models, and bivariate growth curve models, are special cases of the bivariate latent difference score model and the more general bivariate dual change score model (DCSM) (Ghisletta & Lindenberger, 2003; McArdle, 2001). The DCSM is specified in terms of the 'true' (latent) scores which underlie the observed scores $y_{tj}^{(P)}$ and $y_{tj}^{(C)}$. Coefficients in a DCSM thus represent effects on outcomes that are purged of measurement

error. Another advantage of the DCSM is that it can straightforwardly handle incomplete data (under a missing at random assumption) and unequally spaced measurements; true scores can be defined for any time point regardless of whether an individual is observed at that time. As in the GCM and the multilevel model described in the next section, the model distinguishes between occasion-specific residuals (usually assumed to be measurement error) and time-invariant random effects.

**Motivation for a Joint Multilevel Model for Parent and Child Outcomes**

Previous studies of parent-child and sibling relationships have not allowed for dependency between outcomes for the same individual or between different family members that is due to unmeasured time-invariant factors. In the multilevel model described in the next section, the residual variation is partitioned into occasion and individual components with residual correlation at each level. Where the same outcome is measured on more than one individual per family, as in sibling studies, it is also possible to distinguish individual and family effects. This variance partitioning allows us to identify whether an association between parent and child outcomes, or between sibling outcomes, is due mainly to time-varying or time-invariant factors. For instance, a larger correlation between the individual-specific residuals for parent and child outcomes would suggest predominance of time-invariant family characteristics that are shared by parent and child, including genetic influences or environmental factors that are fixed over the study period.

The most important reason for separating out family effects is that an apparent causal effect from one individual to another, as measured by the cross-lagged terms in (1a) and (1b) and in (2a) and (2b), could be explained by a dependency of both individuals' outcomes on a common set of family-level unobservables. A multilevel model that allows for different sources of variation is also a closer representation of family structures and allows us to

identify the strength of family effects (which includes both unmeasured genetic and environmental influences) on child and parent outcomes relative to individual and time-varying effects.

## A General Multilevel Model for Sibling and Parent-child Reciprocal Effects

In this section, we describe a general modelling framework that allows simultaneous estimation of reciprocal parent-child effects and sibling effects, allowing for the possibility that parent and child outcomes may be influenced by a common or correlated set of unmeasured family characteristics. The multilevel model we propose also distinguishes between unmeasured risk factors that are time-varying and those that are operating at the individual or family level. A further advantage of our approach is that it accommodates families of different sizes, including one-child families who contribute information for the estimation of reciprocal parent-child effects, but not sibling effects. For simplicity, we consider the case where we have a single response for one parent and a single response for each child, but the proposed model can be extended to handle mixtures of single and dual-parent families and responses on more than one variable. We also present models without covariates, although these can be included straightforwardly and are considered in the application that follows.

As before we denote by $y_{tj}^{(P)}$ the response at occasion $t$ of the parent in family $j$, but we now allow for multiple children per family where $y_{tij}^{(C)}$ is the response at occasion $t$ of child $i$ in family $j$ ($i = 1, \ldots, n_j$). We begin by describing a basic multilevel simultaneous equations cross-lagged model for the parent and child responses. We then show how the model can be extended to allow child effects on parent responses – or parent effects on child responses – to depend on child or parent characteristics. Next, we consider a further

generalisation to include sibling effects that may depend on characteristics of the target child, his or her siblings or the sibling dyad. At each stage, we demonstrate how mixed family sizes can be handled. Finally we discuss approaches for addressing the 'initial conditions problem' which arises when measurement starts when the processes under study are already underway.

**Bivariate Autoregressive Cross-lagged Model for Reciprocal Parent-Child Effects**

A simultaneous equations model that allows for autoregressive effects and cross-lagged effects between parent and child can be written (for $t > 1$)

$$y_{tj}^{(P)} = \beta_0^{(P)} + \beta_1^{(P)} y_{t-1,j}^{(P)} + \beta_2^{(P)} y_{t-1,+j}^{(C)} + v_j^{(P)} + e_{tj}^{(P)} \qquad (3a)$$

$$y_{tij}^{(C)} = \beta_0^{(C)} + \beta_1^{(C)} y_{t-1,ij}^{(C)} + \beta_2^{(C)} y_{t-1,ij}^{(P)} + v_j^{(C)} + u_{ij}^{(C)} + e_{tij}^{(C)} \qquad (3b)$$

The model defined by (3a) and (3b) differs from that of (1a) and (1b) in two key respects. First, the extension to multiple children per family leads to a three-level structure for the child responses, with repeated measures (level 1) nested within children (level 2) within families (level 3). Equation (3b) therefore contains three residual terms or random effects: a family effect $v_j^{(C)}$ representing unmeasured time-invariant characteristics shared by siblings in family $j$, a child effect $u_{ij}^{(C)}$ capturing unmeasured time-invariant characteristics specific to child $i$ in family $j$, and an occasion-specific residual $e_{tij}^{(C)}$. The parent equation (3a) contains only two residuals – a parent or family effect $v_j^{(P)}$ and an occasion-specific residual $e_{tj}^{(P)}$ - because we observe only one parent per family. The family effects on the child and parent responses, $v_j^{(C)}$ and $v_j^{(P)}$, are assumed to follow a bivariate normal distribution to allow for residual correlation at the family level between child and parent responses owing to

unobserved time-invariant family-specific factors.[1] The occasion-specific residuals may also be correlated across parent and child and between siblings due to time-varying 'shocks' to the family system. Equations (3a) and (3b) should be estimated jointly to allow for non-zero residual correlations among family members. All other residuals are assumed to follow independent normal distributions.

**Child-Parent Effects for Mixed Family Sizes**

The second difference between the model of (3a) and (3b) and the models used in previous family research also arises from allowing for the possibility that a family may have more than one sample child. The parent equation (3a) now includes as a predictor the *total* of the lagged responses of all sampled children in the family,

$$y^{(C)}_{t-1,+j} = \sum_{i=1}^{n_j} y^{(C)}_{t-1,ij}$$

which implies that the effect of a child's lagged response on their parent's response is the same for all children in a family. To see this, consider a two-child family and write the contribution of the children's lags as

$$\beta^{(P)}_{2,1} y^{(C)}_{t-1,1j} + \beta^{(P)}_{2,2} y^{(C)}_{t-1,2j}$$

which equals $\beta^{(P)}_{2} y^{(C)}_{t-1,+j}$ if $\beta^{(P)}_{2,1} = \beta^{(P)}_{2,2} = \beta^{(P)}_{2}$. Under this assumption, child effects can be estimated by including the total of the lagged child responses for families of any size.

[1] When only one parent is considered, parent and family effects are confounded in $v^{(P)}_j$. Nevertheless, the *correlation* between $v^{(C)}_j$ and $v^{(P)}_j$ arises from unmeasured characteristics shared by the parent and all children, and may therefore be interpreted as a consequence of family effects.

While the above specification is a useful starting point for considering child-to-parent effects, it will usually be unrealistic to assume that in multiple-child families each child makes equal, and additive, contributions to their parent's outcome. The parent-child relationship is likely to vary across children, for example according to the child's age or gender, which may lead to differential child-to-parent (and indeed parent-to-child) effects. We therefore consider two ways of relaxing this assumption: (i) including interaction effects between the cross-lags and parent or child characteristics, and (ii) specifying random coefficients for the cross-lags. In the following, we describe how (3a) can be extended to allow the cross-lagged effect from child to parent to vary across children. We can extend (3b) in a similar way to allow cross-lagged parent-to-child effects to depend on child or parent characteristics, and this is demonstrated in our application.

We begin by specifying the cross-lagged effect of a child outcome on a parent outcome in (3a) as a function of *measured* characteristics of the child and perhaps of the parent, family or the parent-child dyad. Consider first the subset of families with one child, and denote by $z_{t-1,1j}^{(C)}$ a characteristic measured at time *t*-1 (e.g. the age) of the child from family *j*. We can replace the cross-lagged child-to-parent effect $\beta_{2,1}^{(P)}$ by an effect that depends linearly on $z_{t-1,1j}^{(C)}$:

$$(\beta_{2,1}^{(P)} + \gamma_1^{(P)} z_{t-1,1j}^{(C)}) y_{t-1,1j}^{(C)} \qquad (4)$$

A more flexible nonlinear dependency can be fitted by including dummy variables for categories formed from $z_{t-1,1j}^{(C)}$.

Next consider a mixture of one and two-child families. Denote by $n_j$ the size of family $j$ and define an indicator variable for two children, $I(n_j = 2)$. An extension of (4) that allows the cross-lagged effect of child *i* from family *j* to depend on lagged characteristic $z_{t-1,ij}^{(C)}$

is

$$\beta_2^{(P)} y_{t-1,+j}^{(C)} + \gamma_1^{(P)} z_{t-1,1j}^{(C)} y_{t-1,1j}^{(C)} + \gamma_2^{(P)} I(n_j = 2) z_{t-1,2j}^{(C)} y_{t-1,2j}^{(C)} \qquad (5)$$

The expression in (5) follows from the earlier assumption that $\beta_{2,1}^{(P)} = \beta_{2,2}^{(P)} = \beta_2^{(P)}$. In addition we typically assume $\gamma_1^{(P)} = \gamma_2^{(P)} = \gamma^{(P)}$. These assumptions together imply that, conditional on $z_{t-1,ij}^{(C)}$, children are exchangeable in terms of their effects on the mother's response. Larger families can be included by replacing $I(n_j = 2)$ by an indicator for whether family *j* has two or more children, $I(n_j \geq 2)$, and defining further family size indicators which are also interacted with $z_{t-1,ij}^{(C)} y_{t-1,ij}^{(C)}$. For example, we would add the term $\gamma^{(P)} I(n_j = 3) z_{t-1,3j}^{(C)} y_{t-1,3j}^{(C)}$ to (5) to accommodate three-child families. The effect of the lagged child response is then $\beta_2^{(P)} + \gamma^{(P)} z_{t-1,ij}^{(C)}$ for any family size.

The inclusion of interactions $z_{t-1,ij}^{(C)} y_{t-1,ij}^{(C)}$ in (3a) permits the lagged child-to-parent effect to vary according to a child characteristic, and the model can be extended to include additional characteristics of children, their parents or the parent-child dyad (e.g. measures of relationship quality). Nevertheless, it is possible that variation in child-to-parent effects across children may not be fully captured by observed variables. We can allow for between-child variation in the child-to-parent effect due to unobserved characteristics by specifying a random coefficient for $y_{t-1,ij}^{(C)}$. The effect of the lagged child response for child *i* in family *j* can then be expressed as $\beta_2^{(P)} + u_{2ij}^{(C)}$ where $u_{2ij}^{(C)}$ is a child-level random effect. Identification of the variance of $u_{2ij}^{(C)}$ requires at least three waves of measurement and, in practice, more will usually be needed especially if there is little within-person variation in either parent or child outcomes.

**Introducing Sibling Effects on Child Outcomes**

We now consider an extension of the child equation (3b) to include sibling effects. We begin by including in (3b) the effect of the lagged response of child 2 on the response of child 1

$$\beta_3^{(C)} y_{t-1,2j}^{(C)} \qquad (6)$$

where, as before, children are labelled arbitrarily within families. An extension of (6) that allows the effect of child 2 on child 1 to depend linearly on a characteristic of the dyad (12), $z_{t-1(12)j}^{(C)}$, for example their age difference, is

$$(\beta_3^{(C)} + \gamma^{(C)} z_{t-1(12)j}^{(C)}) y_{t-1,2j}^{(C)} . \qquad (7)$$

For a three-child family, under the assumption of equality of effects across siblings, the contributions of children 2 and 3 to the response of child 1 can be written

$$\beta_3^{(C)} \tilde{y}_{t-1,1j}^{(C)}$$

where $\tilde{y}_{t-1,1j}^{(C)} = y_{t-1,2j}^{(C)} + y_{t-1,3j}^{(C)}$, i.e. the total lagged response for all of child 1's siblings. Moreover, (7) can be extended to allow for interactions between $y_{t-1,lj}^{(C)}$ and dyad characteristics $z_{t-1(1l)j}^{(C)}$ $(l = 2,3)$ to give

$$\beta_3^{(C)} \tilde{y}_{t-1,1j}^{(C)} + \gamma_2^{(C)} z_{t-1(12)j}^{(C)} y_{t-1,2j}^{(C)} + \gamma_3^{(C)} z_{t-1(13)j}^{(C)} y_{t-1,3j}^{(C)} \qquad (8)$$

with the constraint $\gamma_2^{(C)} = \gamma_3^{(C)}$.

In samples with a mixture of two- and three-child families, the contribution from child 3 can be "switched on or off" according to family size by interacting the third term in (8) with an indicator $I(n_j \geq 3)$ of whether the family has three or more children, i.e.

$$\beta_3^{(C)} \tilde{y}_{t-1,1j}^{(C)} + \gamma_2^{(C)} z_{t-1(12)j}^{(C)} y_{t-1,2j}^{(C)} + \gamma_3^{(C)} I(n_j \geq 3)\, z_{t-1(13)j}^{(C)} y_{t-1,3j}^{(C)}$$

**A Bivariate Autoregressive Cross-lagged Growth Curve Model for Reciprocal Parent-**

**Child Effects**

Equations (3a) and (3b) can both be extended to include a growth component as in Bollen & Curran's (2004) bivariate autoregressive latent trajectory (ALT) model. In an ALT model, the current value of $y$ for an individual is determined both by their lagged $y$ value and the individual-specific intercept and slope of their latent trajectory. For example, the child equation in a three-level bivariate ALT model, a generalisation of (3b), can be written

$$y_{tij}^{(C)} = \beta_0^{(C)} + \beta_1^{(C)} y_{t-1,ij}^{(C)} + \beta_2^{(C)} y_{t-1,ij}^{(P)} + \theta^{(C)} a_{tij}^{(C)} + v_j^{(C)} + u_{0ij}^{(C)} + u_{1ij}^{(C)} a_{tij}^{(C)} + e_{tij}^{(C)} \quad (9)$$

where $\beta_0^{(C)} + \theta^{(C)} a_{tij}^{(C)} + u_{0ij}^{(C)} + u_{1ij}^{(C)} a_{tij}^{(C)}$ defines a growth trajectory for child $i$ in family $j$ as a linear function of $a_{tij}$, the child's age at occasion $t$. While age is usually the most appropriate time metric for child developmental trajectories, other metrics may be used such as calendar time or simply wave of measurement (see Bollen & Curran, 2006, Section 3.2). In a multilevel model, growth curves are fitted by including some functions of time as explanatory variables and allowing the coefficients of these functions to vary randomly across individuals. In the linear specification (9), for example, age is treated as an explanatory variable with a random coefficient at the child level $\theta^{(C)} + u_{1ij}^{(C)}$. Particular advantages of the multilevel approach to growth curve modelling are that it is straightforward to allow for nonlinear trajectories (e.g. polynomials or splines) and for between-individual variation in the timing of measurement at a particular occasion *t* (Singer & Willett, 2003; Steele, 2008).

**Initial Conditions**

The 'initial conditions' problem comes about because the start of measurement does not usually coincide with the start of the process under study. Conditioning on the outcome at $t$=1 ($y_1$) is not generally sufficient in itself to control for what happened prior to the first measurement occasion. Generally, this means it is likely that unmeasured time-invariant

characteristics influencing $y_2, \ldots, y_T$ will also influence $y_1$, i.e. $y_1$ is endogenous. In an autoregressive model, $y_{t-1}$ is included as a predictor of $y_t$ and, by repeated substitution for $y_{t-1}$, it can be shown that the dependence of $y_t$ on previous $y$ operates entirely through $y_1$. Hence, endogeneity of $y_1$ implies correlation between $y_1$ and the random effects that represent unmeasured family and child family characteristics, which invalidates the standard assumption that the random effects are uncorrelated with the predictors. Failure to properly account for the initial conditions will lead to biased estimates of the lag effects and, in bivariate models, cross-lag effects. It is especially important to take account of endogeneity in short panels because, while the influence of $y_1$ on $y_t$ diminishes with $t$, all subsequent measures will be strongly associated with $y_1$ if $t$ is small (Bhargava & Sargan, 1983).

Methods for handling the initial conditions problem have received a great deal of attention in the econometrics literature, although mainly for the univariate case; Kazemi and Crouchley (2006) review and compare approaches for univariate autoregressive models with random effects. A discussion of the treatment of initial conditions in bivariate autoregressive models and the generalisation to autoregressive latent trajectory (ALT) models can be found in Bollen and Curran (2004). Approaches to the problem generally involve specifying a model for $y_1$ which is estimated jointly with the model for $y_2, \ldots, y_T$. A general model for the initial outcome for individual $j$ is

$$y_{1j} = \alpha_0 + w_j + e_{1j}, \qquad (10)$$

where $\alpha_0$ is the intercept, $w_j$ is a random term representing time-invariant individual characteristics, and $e_{1j}$ is a residual specific to the first measurement. Estimation of (10) requires some constraint to be placed on $\text{var}(w_j)$ or $\text{var}(e_{1j})$, which involves some assumption about the distribution of $y_{1j}$. One approach is to assume the same process that generated the observed $y$ was also functioning before the first measurement at $t$=1. In the

case of a first-order autoregressive process, this leads to

$$y_{1j} = \alpha_0 + \lambda_v v_j + e_{1j} \qquad (11)$$

where $v_j$ is the individual-specific random effect in the model for $y_{2j}, \ldots, y_{Tj}$, and the loading $\lambda_v$ and $\text{var}(e_{1j})$ are nonlinear functions of the coefficient of $y_{t-1,j}$ in the model for $y_{2j},\ldots, y_{Tj}$ and $\text{var}(v_j)$; see Bhargava and Sargan (1983) for full details and Bollen and Curran (2004) for the extension to the bivariate ALT model. The main advantage of this approach is that only one additional parameter, the intercept $\alpha_0$, is required to handle the initial condition because all other parameters are functions of parameters in the model for $y_{tj}$ ($t > 1$). It is therefore an attractive option for identification of random coefficient models fitted to short panels. (See Bollen and Curran (2004) for a derivation of the exact form of the nonlinear constraints required for identification of the bivariate ALT model for three waves.) However, the caveat is that in some applications it may be unrealistic to assume that an AR(1) process was at work prior to the start of measurement (sometimes referred to as the stationarity assumption).

In a more general form of (11), referred to as a one-factor decomposition model, $\lambda_v$ is freely estimated (Crouchley, Stott, & Pritchard, 2009). Thus a common set of unmeasured individual characteristics are assumed to influence $y_{1j}$ and $y_{tj}$ given $y_{t-1,j}$ ($t > 1$), but $\lambda_v$ allows their effects to differ for $y$ at $t = 1$ and for the change in $y$ between $t$-1 and $t$ (for $t > 1$). A constraint must be imposed on $\text{var}(e_{1j})$ because only one variance parameter can be estimated, i.e. $\lambda_v$ and $\text{var}(e_{1j})$ cannot both be free to vary. Possible identification constraints are $\text{var}(e_{1j}) = 1$ or $\text{var}(e_{1j}) = \text{var}(e_{2j})$.

In the model for parent and child outcomes, the initial condition equation (11) is replaced by

$$y_{1j}^{(P)} = \alpha_0^{(P)} + \lambda_v^{(P)} v_j^{(P)} + e_{1ij}^{(P)} \qquad (12a)$$

$$y_{1ij}^{(C)} = \alpha_0^{(C)} + \lambda_v^{(C)} v_j^{(C)} + \lambda_u^{(C)} u_{ij}^{(C)} + e_{1ij}^{(C)} \qquad (12b)$$

which are jointly estimated with (3a) and (3b) for $t > 1$. This is achieved by specifying indicator variables for whether an observation is measured at $t = 1$ or $t > 1$ and interacting these with explanatory variables and (for $t > 1$) the lags and cross-lags. Further details can be found in the Supplementary Appendix; see also Alfò and Aitkin (2006). In the application that follows, we also consider simplified forms of (12a) and (12b) where one or more of the three random effects loadings are constrained to equal 1.

**Estimation and Identification**

The multilevel simultaneous equations model defined by (3a) and (3b), and the various extensions described above, can be estimated by maximum likelihood in any statistical software package that can handle multivariate response data where different responses have a different hierarchical structure (i.e. single-level for the parent's initial conditions, two-level for parent responses for $t > 1$ and children's initial conditions, and three-level for child responses for $t > 1$). Software packages that can handle such general multivariate structures include PROC NLMIXED in SAS (SAS Institute Inc., 2008), MLwiN (Rasbash, Charlton, Browne, Healy, & Cameron, 2009) and aML (Lillard & Panis, 1998-2003). Options for estimating the one-factor decomposition model for initial conditions (with random effect loadings) are more limited, but include PROC NLMIXED and aML. All analyses presented in this article were carried out in aML. Further details, with examples of the required data structure, are given in the Supplementary Appendix.

Bollen and Curran (2004) discuss conditions for identification of the closely related ALT model, and consider in detail the special cases of three and four waves of measurement.

For example, identification with fewer than five waves requires the assumption that the coefficients of the lags and cross-lags are constant over time. The complexity of the growth component in (9) that can be fitted will also be limited in short panels; for example, at least three waves are required to estimate quadratic curves. The decomposition of the residual variance in the multilevel extension to the ALT model proposed here does not introduce any major new considerations. However, random coefficient models – for example to allow for between-sibling variation in child-to-parent effects, as discussed earlier – will generally be weakly identified in short panels. Furthermore, child and family effects may be confounded if there are few families with more than one child or in cases where there is little within-family variation in child outcomes.

**Missing Data**

Multiple imputation is now a commonly used method for handling the problem of non-response bias resulting from data which are 'missing at random' (MAR) (Little & Rubin, 2002). While multiple imputation methods suitable for multilevel data structures have recently been developed (Browne, 2009; Goldstein, Carpenter, Kenward, & Levin, 2009), we use an alternative method closely related to full information maximum likelihood (Arbuckle, 1996; Rubin, 1976). Each family member must have responses for at least two consecutive occasions to contribute to estimation of lagged and cross-lagged effects. Individuals who are observed only at $t = 1$ may also be included as they contribute information for estimation of the initial conditions equations. Provided that, at occasion $t$, the probability of $\mathbf{y}_{tj} = (y_{tj}^{(P)}, y_{t1j}^{(C)}, \ldots, y_{tm_j j}^{(C)})$ being missing does not depend on $\mathbf{y}_{tj}$ itself (or future $\mathbf{y}_{t+1,j}$, $\mathbf{y}_{t+2,j}$, etc.) given the covariates, then our estimator is consistent. A minor drawback, however, is that our estimator is not fully efficient because it excludes information from individual

contributions at waves where there are missing lags.

## Application to Maternal Depression and Child Delinquency

### Sample

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a longitudinal, prospective study of women, their partners, and a target child. The study design included all pregnant women living in the health district of Avon, England, who were to deliver their baby between April 1991 and December 1992 (Golding, 1996). It was estimated that 85% to 90% of the eligible population took part (n = 14,000). The current study, the Avon Brothers and Sisters Study (ABSS), is a sub-study of 235 families, within ALSPAC, that capitalized on the community nature of the ALSPAC sample and used it as a sampling frame to select a representative group of non-stepfamilies, single-parent families, and stepfamilies (Dunn, Davies, O'Connor, & Sturgess, 2000) that included multiple children per family, under the age of 17 years old. The average age at $t = 1$ of the youngest child (the ALSPAC target child) was 4.8 years ($SD = .38$) and the age range of the siblings was 6 to 17 years ($M = 10.2$, $SD = 2.9$). Three waves of data were collected with families being visited at intervals of approximately two years.

The following analysis is based on families with a mother and two or three children. The analysis sample contains families with information available on each family member for at least two consecutive waves as well as individuals who were observed only at $t = 1$. The final sample contains 177 families and 418 children who together contribute 1389 observations over the course of the study. Table 1 shows the number of families, mothers and children observed at each wave. The declining proportion of three-child families suggests that mothers with more children were more likely to drop out of the study.

The following analyses are used to illustrate the application of the proposed

simultaneous equations approach and its advantages over methods used in previous research rather than for detailed substantive consideration. A limitation of the data source is that child behavior is reported by the mother. It is well known that single informant data are subject to measurement error, inflating associations between predictor and outcome variables.

## Measures

**Maternal depression.** Mothers' ratings of their own depressive and anxious symptomatology were obtained using the Malaise Inventory (Rutter, Tizard, & Whitmore, 1970) at $t = 1$. The Malaise Inventory is a 24-item questionnaire that assesses both emotional (e.g. 'do you often feel miserable or depressed?') and physical (e.g. 'do you suffer from an upset stomach?') stress. Mothers responded to items by indicating whether or not a symptom was present (1) or absent (0) and a mean depression/anxiety score was calculated by dividing the number of items endorsed by the total number of items. Given that this scale was utilized to assess mothers' present levels of depressive/anxious symptomatology, the item 'have you ever had a nervous breakdown?' was dropped. The full Malaise Inventory has been shown to have good reliability and validity (McGee, Williams, & Silva, 1986). In the present study, the internal consistency for the scale at all waves was above $\alpha = .79$. We refer to this scale as maternal depression for brevity. The mean depression score is similar for waves 1 and 2 but lower for wave 3 (see Table 1), which may be due to mothers with a higher score being more likely to drop out of the study.

**Child delinquency.** At each wave, mothers completed the Child Behavior Checklist (Achenbach, 1991) on each of their children. The delinquency scale used for analysis was made up of 11 items. Mothers were asked to say how frequently the behavior occurred in the last 6 months using a 3 point scale: *not at all true* (0), *somewhat true* (1), or *very true* (2). Sample items from the delinquency subscale included lack of guilt, lying or cheating, truancy

and stealing. The mean delinquency score is similar for each wave (Table 1) and internal consistency was above $\alpha = .75$ for all waves.

**Demographics.** Mothers reported on children's age in years and their gender. The mean age and proportion of girls at each wave are shown in Table 1.

## Analytic Plan

Figure 1 shows the lagged and cross-lagged effects in the simplest model for reciprocal parent-child and sibling effects, as given by (3a) and (3b). We considered various extensions to this model to allow for interactions between the cross-lags and child characteristics. Specifically, we tested whether the effect of lagged child's delinquency on maternal depression – and the reciprocal effect of lagged depression on delinquency – depended on the child's age and gender. In the model for child delinquency, we additionally tested for an interaction between lagged sibling delinquency and both the gender of each child and the age difference between children. We considered the age difference in years, centred around 3 years so that the main effect of lagged sibling delinquency is interpreted as the sibling effect at a 3-year difference. Furthermore, we allowed the sibling effect to depend linearly on both the direction and the magnitude of the age difference in years by fitting separate terms for the main effects of lagged sibling delinquency and interaction with age difference effects according to whether the sibling is older or younger. Previous research on sibling effects has considered only the effect of an older child on their younger sibling.

While the effects of the cross-lags and their interactions with child sex and age and sibling age difference were the major focus of our analysis, we adjusted for the effects of family size (three versus two children) and the wave of data collection. We allowed for a dependency of child delinquency on age using a linear growth curve component (9). A linear model with a random slope for age was considered initially, but a simpler random intercept

model was selected after finding little evidence of between-child variation in slopes (possibly due to the small number of measurements and the inclusion of lags). Temporal changes in maternal depression were captured by dummy variables for wave of measurement.

Four broad model specifications were considered which differ with respect to the assumptions made about the residual structure. The first, simplest model includes uncorrelated occasion-specific residuals for mother and children and no child or family-level residuals. This model assumes that the association between the outcomes of different family members is fully explained by the cross-lags. It also assumes that the parent and child cross-lags are uncorrelated with the residuals, i.e. the cross-lags are *exogenous*. The equations for mother and child outcomes can be estimated in two separate steps as in recent studies of reciprocal parent-child effects (Hipwell et al., 2008; Pardini, Fite, & Burke, 2008) or, equivalently, estimated jointly with zero constraints imposed on all residual correlations. This approach will lead to biased estimates of reciprocal parent-child and sibling effects if there are omitted variables influencing both maternal depression and siblings' behaviour, i.e. the cross-lags are *endogenous*. In the second model specification, residual correlations are introduced between pairs of family members at a given occasion. This approach is closest to previous studies of parent-child reciprocal influences using SEM, but with the addition of between-sibling correlations. We refer to both models as single-level because all unobserved influences on parent and child outcomes are represented by a single residual term: no distinction is made between occasion, child and family-level unmeasured variables. The third specification is a multilevel extension of the second model with residuals at the occasion, child and family levels with the correlation at the family level fixed at zero. Finally, the fourth model allows for shared dependency of mother and child outcomes on unmeasured time-invariant family characteristics by explicitly modelling a non-zero correlation between the mother and child family-level random effects.

With up to four family members observed on three occasions, it was necessary to impose some restrictions on the 12 × 12 covariance matrix for the occasion-level residuals $(e_{tj}^{(P)}, e_{tij}^{(C)})$. This was achieved by assuming exchangeability between children within a family, conditional on covariates. Thus we assumed equal residual correlation for any mother-child pair and for any sibling pair within a family at a given occasion *t*, leading to six correlation parameters. Occasion-specific residual variances were estimated for both mother and children, resulting in a further six parameters. Based on significance tests of each variance and correlation, the following further simplifications were made in subsequent analysis: sibling correlations at occasions 1 and 3 were fixed at zero, and equality constraints were imposed on the mother-child correlations and mother and child variances at occasions 2 and 3. When child and family random effects were added, the between-child (within-family) variance was estimated as zero, possibly due to the small sample size and few measurement occasions. We therefore restricted our attention to two-level models with family effects for mother and child, $v_j^{(P)}$ and $v_j^{(C)}$.

As described earlier, we considered two alternative specifications for the initial conditions, i.e. equations (12a) and (12b) for maternal depression and child delinquency at *t* = 1 (but without the child-level residual $u_{ij}^{(C)}$). The first specification included a common random effect for all occasion *t* (i.e. $\lambda_v^{(P)} = \lambda_v^{(C)} = 1$), while in the second the loadings were freely estimated. As neither loading differed significantly from 1, the simpler model with a common random effect was selected.

## Results

Table 2 shows the parameter estimates for the four models described above, with increasingly complex residual structure. All models include the same set of explanatory

variables. M1 and M2 are single-level models with occasion-level residuals, uncorrelated in M1 and correlated (with restrictions) in M2; M3 is an extension of M2 with uncorrelated family-level random effects; and M4 extends M3 to allow for a non-zero residual correlation at the family level. Estimates of the residual standard deviations and correlations for M2 and M4 are presented in Table 3. Based on a comparison of M1 and M2, there is strong evidence of correlation between the outcomes of pairs of family members that is not explained by the cross-lagged effects (-2 Δ log-likelihood = 43.78, 3 *df*, $p < .001$). The addition of uncorrelated family-level random effects further improves model fit (M3 vs M2: -2 Δ log-likelihood = 4.64, 2 *df*, $p = .049$).[2] However, there is also evidence of a family-level residual correlation (M4 vs M3: -2 Δ log-likelihood = 5.48, 1 *df*, $p = .019$). This strong, positive correlation between unmeasured time-invariant family influences on maternal depression and child delinquency is likely to be due in part to shared informant measurement. After accounting for this family-level correlation, the occasion-level mother-child correlations become non-significant (Table 3).

As there was evidence of correlated family effects for maternal depression and child behaviour we focus on the interpretation of the full multilevel model (M4), but we note where assuming a simpler residual structure (M1 to M3) would have led to misleading conclusions. The estimated coefficients for M1 and M2 (Table 2) were very similar; differences only emerged after accounting for family effects (M3 and M4). We also considered a sequence of models including only one of the three types of cross-lagged effects at a time, for example a parent-to-child effect in the absence of child-to-parent or sibling effects, but the substantive

[2] The null hypothesis that the standard deviations of the family-level random effects are both zero was tested against a one-sided alternative because a standard deviation must be non-negative (see Snijders & Bosker, 1999).

conclusions were identical to those obtained from considering all reciprocal effects simultaneously as in M1-M4 (results not shown).

**Mother-Child Effects**

There was no evidence in any model of an association between child delinquency at occasion $t - 1$ and change in maternal depression between $t - 1$ and $t$. There was evidence of a cross-lagged effect in the opposite direction, but the effect depended on the child's gender. While lagged maternal depression did not affect boys' delinquency (the main effect of the lag is estimated as 0.022 in M4 and is non-significant), a higher maternal depression score was associated with a lower delinquency score among girls two years later, controlling for the child's prior level of delinquency (0.022 – 0.256 = -0.234). In preliminary analysis (not shown) we also tested whether mother-to-child and child-to-mother effects varied by child's age, but neither were found to be significant. Estimates of reciprocal mother-child effects were markedly different according to whether correlated family residuals for mother and child outcomes were included. Most importantly, failure to account for correlated family effects (M1 – M3) led us to conclude that higher levels of maternal depression at one occasion were *positively* and *significantly* associated with higher levels of subsequent delinquency among *boys* (controlling for prior delinquency), but not associated with girls' delinquency. This change in the main effect of lagged maternal depression was due to a large positive residual correlation ($\hat{\rho}_v$ = 0.869) between the parent and child outcomes (Table 3). The apparent causal effect of maternal depression on boys' delinquency found when we did not account for correlated family effects was in fact due to the presence of unobserved family circumstances that were positively associated with both maternal depression and child delinquency: the higher mean delinquency observed among children of depressed mothers was explained by a dependency of both outcomes on unobserved time-invariant family

characteristics.

With respect to the directionality of the final cross-lagged coefficients (negative rather than positive as expected intuitively) it is important to note two issues that may explain this. First, aggregation on the psychopathology of family members is well established. Children show higher levels of psychopathology when their parents show higher levels of psychopathology, resulting in positively valenced coefficients. This is true when both parents and children are measured on the same outcome (Bolton et al., 2006) and also true when they are assessed on different outcomes (O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998). Utilizing genetically sensitive designs, however, this aggregation is found to be attributable mainly to genetic factors. Second, previous investigations of cross-lagged influences between parent and child have often included the contemporaneous residual correlation between mother and child behavior in the model (Elgar, Curtis, McGrath, Waschbusch, & Stewart, 2003), but have not separated the effects of unmeasured time-varying and time-invariant family characteristics. The implication of both of these issues is that once we account for the *unobserved* family circumstances that are positively associated with both maternal depression and child delinquency, we have no basis for a prediction about the directionality of cross-lagged effects between mothers and children.

Although such findings need replication within other family studies there is a perspective within the family systems literature that can explain the negative coefficients on the parent-child lags (Nichols, 2007). It has been suggested that families operate as a homeostatic system, with complementary roles of individuals keeping overall functioning of the family within acceptable limits (i.e. as one person becomes worse the other become better). A child's delinquency may activate a mother towards better functioning as she works to reduce her child's problems. Similarly, children may help their parents. Empathy has been found to be stronger in girls than in boys and girls exposed to maternal depression show the

highest levels of empathy (Radke-Yarrow, Zahn-Waxler, Richardson, & Susman, 1994)

**Sibling Effects**

We tested for effects of sibling delinquency on a child's delinquency, allowing for sibling effects to vary according to whether he or she was older or younger than the child *i* and their age difference. We found that the results for the single-level models (M1 and M2) supported a sibling training effect with a significant, positive effect of an older child's behavior at $t - 1$ on their younger sibling's behavior at $t$. In M1 there was also weak evidence that the training effect diminishes as the age difference increases. This is in line with previous research showing a training effect of older siblings' delinquency on the delinquency of younger siblings' (Slomkowski, Rende, Conger, Simons, & Conger, 2001). Earlier work considered only younger child outcomes, rather than jointly modelling sibling outcomes, and is therefore based on assumptions similar to those of M1. We found this training effect weakened and became non-significant when we accounted for unmeasured time-invariant family influences in the multilevel models (M3 and M4). Thus we found that the apparent training effect from older to younger children was explained by a shared dependency of both siblings' behavior on unobserved family characteristics (M3). Estimates of sibling effects are, however, unaffected by allowing for residual correlation between parent and child outcomes at the family level (M4). This might be expected because, in both multilevel models, the inclusion of a family-level random effect allows for dependency among children in the same family. The family-level residual correlation between parent and child outcomes is only important for estimation of parent-child reciprocal effects.

**Effects of Lagged Outcomes and Covariates**

The significant lagged effects suggest that mothers with higher (lower) depression

scores at a given occasion $t - 1$ tended to have higher (lower) scores two years later at $t$, and similar lagged effects were also found for child delinquency. However, the magnitude of the estimates for the lags is substantially reduced after accounting for time-invariant mother effects (M3 and M4).

After conditioning on lagged depression and delinquency, the estimated coefficients of the covariates can be interpreted as effects on the *change* in each outcome between occasions $t$ - 1 and $t$ (for $t$ = 2, 3). Covariate effects on the baseline outcome (at $t$ = 1) are also shown in Table 2. We found weak evidence of higher maternal depression at $t$ = 1 in families with three rather than two children, but no effect of family size or of measurement occasion on the change in depression across subsequent time points. There was no effect of family size on either baseline child delinquency or change in delinquency. However, there was evidence that delinquency scores were higher among older children and boys at $t$ = 1, but with girls showing a greater change in delinquency over time.

## Discussion

In the present study we present a statistical model that allows for the differentiation of group and individual-specific influences in group settings as well as the differentiation between time-varying and time-invariant unmeasured influences on outcomes. Although we focussed primarily on the family, as we discuss the value of the statistical model that we present, we consider the way in which the model can be generalized to other social groups. The statistical model is flexible enough to allow for reciprocal relationships between *all* members of a social group simultaneously, accommodating different sizes of groups in the analysis and allowing for unmeasured factors that reflect the similarity between members of the group, that might either be genetic (when the group is genetically related) or environmental. This enables the testing of complex hypotheses regarding the ways in which

individuals influence one another, and allows some spurious effects that would otherwise confound causal inferences to be put aside.

Many social groups are hierarchically structured with different roles for individuals. In the illustration of our model we discussed mothers and children but the same structure is applicable to boss/employee, gang leader/gang members, teacher/students. One of the strengths of our model was the simultaneous estimation of equations for individuals in the group with different roles (e.g. parents and children in our illustration). Although this has been done by most investigators examining reciprocal influences (Cui, Donnellan, & Conger, 2007; Elgar, Curtis, McGrath, Waschbusch, & Stewart, 2003; Gross, Shaw, & Moilanen, 2008), it has not been utilized by all (Hipwell et al., 2008; Pardini, Fite, & Burke, 2008).

Because of the simultaneous estimation of equations for individuals, and the fact that our model can include more than two individuals potentially affecting one another over time, we were able to estimate the group-level random effects, including the correlation of the residuals for the people with different roles (mother and children in our illustration) as well as the correlation of occasion-level residuals. The group-level random effect is important in several ways. This quantifies the similarities between members of the group, that are not accounted for by measured predictors. In families, such similarity may be related to genetic influence, an aspect of the unmeasured environment that influences all family members (e.g. living in damp housing) or measurement bias because the same person has provided information about all members of the group. In groups in which there is no biological relatedness among members, similarity can either be explained by unmeasured influences held in common by the group (for instance an unstable economic climate) or shared informant bias contributing to measurement error (different consultants observe all the members of a work setting). Allowing for correlated occasion-specific residuals is also important because it may be that an unmeasured event at a specific time point affected

everyone in the group. The estimation of these group and occasion-level random effects results in cleaner and more precise estimates for the cross-lags. As the cross-lags provide an indication of the degree to which one person's earlier behaviour has an influence on another person's subsequent behavior this 'cleaning' is vital to our conclusions about causal influence. This was seen in the illustration. On the basis of previous studies we expected older siblings to influence the delinquency of their younger siblings. A significant effect of older siblings' delinquency on younger siblings' delinquency was found before we included a random effect at the family level in the model. Thus if we just fit a reciprocal effects model, ignoring the multilevel structure of the data, it appears that the lag of older siblings' delinquency predicts a change in younger siblings' delinquency over time, i.e. older siblings are training younger siblings. This approach, however, fails to take account of the fact that the behavior of siblings (and their mother) is likely to be influenced by a common set of unmeasured family-specific characteristics. When this clustering was accounted for, the 'environmental' sibling effect disappeared. This suggests, using the present data, that whatever the influences are that make siblings more similar to one another on delinquency, they do not involve a lagged effect of the older sibling training the younger. Several unmeasured family processes, either genetic or environmental may be operating. As most longitudinal datasets are not based on genetically-sensitive designs (that allow for a differentiation of genetic and environmental influence through the comparison of sibling similarity in siblings that share different degrees of genetic relatedness) it is important in 'environmental' studies to control for unmeasured risk factors that may indicate genetic influence.

Finally, consider an extension of this model to another social group. In the work setting we might be interested in the effect of the boss's expressed anger at *t*-1 on the depression of employees at *t*. We need to fit the group-level random effect to rule out the

possibility that the workplace climate has influenced both the boss and the employees, without the anger of the boss having any causal effect on employees.

Allowing the residual effects of the group members to correlate is also essential to improving the estimation of the cross-lags. In some types of psychological data shared informant bias is inevitable. This happens when the measurement for a group relies on one person (e.g. if only one coder can go into a work setting or only one adult is acquainted with all the children in a group). In our illustration, in which single informant methodology was used, if we had not allowed the residual effects of the group members to correlate, we would have drawn an erroneous conclusion about the causal effect of mothers' depression on boys' delinquency. After allowing the residual effects of mothers and children to correlate, earlier maternal depression was found to predict a decrease in the delinquency of girls.

Although our analysis was presented for illustrative rather than substantive purposes it is worth considering several limitations of our dataset. First, the dataset is small which may have limited our ability to identify cross-lag influences that operate in families. Second, single informant methodology was used. Third, measurement occasions were few. More occasions of measurement would have enabled us to better differentiate between unobserved heterogeneity between individuals and state-dependence (lagged effects), and between residual correlation between outcomes for different family members and cross-lagged effects. The data presented, however, are valuable for demonstrating that exclusions to the model, evident in previous analyses of reciprocal processes in families, may result in unwarranted conclusions about environmental influences in families.

## References

Achenbach, T. (1991). *Manual for the Child Behavior Checklist/4-18*. Burlington, VT: Department of Psychiatry, University of Vermont.

Alfò, M., & Aitkin, M. (2006). Variance component models for longitudinal count data with baseline information: epilepsy data revisited. *Statistical Computing, 16*, 231-238.

Arbuckle, J. L. (1996). Full information estimation in the presence of incomplete data. In G. A. Marcoulides & R. E. Schumacker (Eds.), *Advanced Structural Equation Modeling: Issues and Techniques*. Mahwah, NJ: Lawrence Erlbaum Associates.

Bhargava, A., & Sargan, J. D. (1983). Estimating dynamic random effects models from panel data covering short time periods. *Econometrica, 51*(6), 1635-1659.

Bollen, K. A., & Curran, P. J. (2004). Autoregressive latent trajectory (ALT) models a synthesis of two traditions. *Sociological Methods & Research, 32*(3), 336-383.

Bollen, K. A., & Curran, P. J. (2006). *Latent Curve Models: A Structural Equation Perspective*. Hoboken, New Jersey: John Wiley & Sons, Inc.

Bolton, D., Eley, T., O'Connor, T. G., Perrin, S., Rabe-Hesketh, S., Rijsdijk, F., et al. (2006). Prevalence and genetic and environmental influences on anxiety disorders in 6-year-old twins. *Psychological Medicine, 36*, 335-344.

Browne, W. J. (2009). *MCMC Estimation in MLwiN v2.10*. University of Bristol: Centre for Multilevel Modelling.

Christakis, N. A., & Fowler, J. H. (2007). The spread of obesity in a large social network over 32 years. *The New England Journal of Medicine, 357*, 370-379.

Connell, A. M., & Dishion, T. J. (2006). The contribution of peers to monthly variation in adolescent depressed mood: a short-term longitudinal study with time-varying predictors. *Development and Psychopathology, 18*(1), 139-154.

Cook, W. L., & Kenny, D. A. (2005). The Actor-Partner Interdependence Model: A model of bidirectional effects in developmental studies. *International Journal of Behavioral Development, 29*(2), 101-109.

Crouchley, R., Stott, D., & Pritchard, J. (2009). *Multivariate Generalised Linear Mixed*

*Models via sabreStata (Sabre in Stata)*: University of Lancaster, UK.

Cui, M., Donnellan, M. B., & Conger, R. D. (2007). Reciprocal influences between parents' marital problems and adolescent internalizing and externalizing behaviour. *Developmental Psychology, 43*(6), 1544-1552.

Curran, P. J. (2003). Have multilevel models been structural equation models all along? *Multivariate Behavioral Research, 38*(4), 529-568.

Dishion, T. J., & Owen, L. D. (2002). A longitudinal analysis of friendships and substance use: bidirectional influence from adolescence to adulthood. *Developmental Psychology, 38*(4), 480-491.

Dunn, J., Davies, L., O'Connor, T. G., & Sturgess, W. (2000). Parents' and partners' life course and family experiences: Links with parent-child relationships in different family settings. *Journal of Child Psychology and Psychiatry, 41*(8), 955-968.

Elgar, F. J., Curtis, L. J., McGrath, P. J., Waschbusch, D. A., & Stewart, S. H. (2003). Antecedent-consequence conditions in maternal mood and child adjustment: a four-year cross-lagged study. *Journal of Clinical Child and Adolescent Psychology, 32*(3), 362-374.

Ghisletta, P., & Lindenberger, U. (2003). Age-based structural dynamics between perpetual speed and knowledge in the Berlin Aging Study: Direct evidence for ability dedifferentiation in old age. *Psychology and Aging, 18*(4), 696-713.

Ghisletta, P., & Lindenberger, U. (2004). Static and dynamic longitudinal structural analyses of cognitive changes in old age. *Gerontology, 50*, 12-16.

Golding, J. (1996). Children of the nineties: A longitudinal study of pregnancy and childhood based on the population of Avon (ALSPAC). *Contemporary Reviews in Obstetrics and Gynaecology 8*, 89-92.

Goldstein, H. (2003). *Multilevel Statistical Models* (3rd ed.). London: Arnold.

Goldstein, H., Carpenter, J., Kenward, M. G., & Levin, K. A. (2009). Multilevel models with multivariate mixed response types. *Statistical Modelling, 9*(3), 173-197.

Gross, H. E., Shaw, D. S., & Moilanen, K. L. (2008). Reciprocal associations between boys' externalising problems and mothers' depressive symptoms. *Journal of Abnormal Child Psychology, 36*, 693-709.

Hipwell, A., Keenan, K., Kasza, K., Loeber, R., Stouthamer-Loeber, M., & Bean, T. (2008). Reciprocal influences between girls' conduct problems and depression, and parental punishment and warmth: a six year prospective analysis. *Journal of Abnormal Child Psychology, 36*, 663-677.

Jolivet, A., Caroly, S., Ehlinger, V., Kelly-Irving, M., Delpierre, C., Balducci, F., et al. (2010). Linking hospital workers' organizational work environment to depressive symptoms: a mediating effect of effort-reward imbalance? The ORSOSA study. *Social Science and Medicine, 71*, 534-540.

Kaffman, A., & Meaney, M. J. (2007). Neurodevelopment sequelae of postnatal maternal care in rodents: clinical and research implications of molecular insights. *Journal of Child Psychology and Psychiatry, 48*, 224-244.

Kazemi, I., & Crouchley, R. (2006). Modelling the initial conditions in dynamic regression models of panel data with random effects. In B. H. Baltagi (Ed.), *Panel data econometrics: theoretical contributions and empirical applications*. Amsterdam: Elsevier.

Laird, R. D., Pettit, G. S., Bates, J. E., & Dodge, K. A. (2003). Parents' monitoring knowledge and adolescents' delinquent behavior: evidence of correlated developmental changes and reciprocal influences. *Child Development, 74*, 752-768.

Lillard, L. A., & Panis, C. W. A. (1998-2003). *aML User's Guide and Reference Manual, Version 2*. Los Angeles: EconWare.

Little, R. J. A., & Rubin, D. B. (2002). *Statistical Analysis with Missing Data* (2nd ed.). New York: Wiley.

MacCallum, R. C., Kim, C., Malarkey, W., & Kielcolt-Glaser, J. (1997). Studying multivariate change using multilevel models and latent curve models. *Multivariate Behavioral Research, 32*, 215-253.

Mashburn, A. J., Justice, L. M., Downer, J. T., & Pianta, R. C. (2009). Peer effects on children's language achievement during pre-kindergarten. *Child Development, 80*, 686-702.

McArdle, J. J. (1988). Dynamic but structural equation modeling of repeated measures data. In J. R. Nesselroade & R. B. Cattell (Eds.), *Handbook of Multivariate Experimental Psychology* (Vol. 2, pp. 561-614). New York: Plenum Press.

McArdle, J. J. (2001). A latent difference score approach to longitudinal dynamic structural analysis. In R. Cudeck, S. du Toit & D. Sörbom (Eds.), *Structural Equation Modeling: Present and Future. A Festschrift in Honor of Karl Jöreskog* (pp. 1-40). Lincolnwood, IL: Scientific Software International, Inc.

McGee, R., Williams, S., & Silva, P. A. (1986). An evaluation of the Malaise inventory. *Journal of Psychosomatic Research, 30*(2), 147-152.

Natsuaki, M. N., Ge, X., Reiss, D., & Neiderhiser, J. M. (2009). Aggressive behavior between siblings and the development of externalising problems: evidence from a genetically sensitive study. *Developmental Psychology, 45*(4), 1009-1018.

Nichols, M. (Ed.). (2007). *Family Therapy: Concepts and Methods* (8th ed.). Boston: Allyn and Bacon.

O'Connor, T. G., Neiderhiser, J. M., Reiss, D., Hetherington, E. M., & Plomin, R. (1998). Genetic contributions to continuity, change, and co-occurrence of antisocial and depressive symptoms in adolescence. *Journal of Child Psychology and Psychiatry,*

*39*, 323-336.

Pardini, D. A., Fite, P. J., & Burke, J. D. (2008). Bidirectional associations between parenting practices and conduct problems in boys from childhood to adolescence: the moderating effect of age and African-American ethnicity. *Journal of Abnormal Child Psychology, 36*, 647-662.

Plomin, R., & Davis, O. (2009). The future of genetics in psychology and psychiatry: microarrays, genome-wide association, and non-coding RNA. *Journal of Child Psychology and Psychiatry, 50*, 63-71.

Radke-Yarrow, M., Zahn-Waxler, C., Richardson, D., & Susman, A. (1994). Caring behavior in children of clinically depressed and well mothers. *Child Development, 65*, 1405-1414.

Rasbash, J., Charlton, C., Browne, W. J., Healy, M. J. R., & Cameron, B. (2009). MLwiN Version 2.1: Centre for Multilevel Modelling, University of Bristol.

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical Linear Models*. Newbury Park: Sage.

Rende, R., Slomkowski, C., Lloyd-Richardson, E., & Niaura, R. (2005). Sibling effects on substance use in adolescence: social contagion and genetic relatedness. *Journal of Family Psychology, 19*(4), 611-618.

Rubin, D. B. (1976). Inference and missing data. *Biometrika, 63*, 581-592.

Rutter, M., Tizard, J., & Whitmore, K. (1970). *Education, Health and Behaviour*. London: Longman.

SAS Institute Inc. (2008). *SAS/STAT® 9.2 User's Guide*. Cary, NC: SAS Institute Inc.

Singer, J. D., & Willett, J. B. (2003). *Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence*. New York: Oxford University Press.

Slomkowski, C., Rende, R., Conger, K. J., Simons, R. L., & Conger, R. D. (2001). Sisters, brothers, and delinquency: evaluating social influence during early and middle

adolescence. *Child Development, 72*(1), 271-283.

Snijders, T. A. B., & Bosker, R. J. (1999). *Multilevel Analysis: An Introduction to Basic and Advanced Multilevel Modeling*. London: Sage Publications Ltd.

Snyder, J., Bank, L., & Burraston, B. (2005). The consequences of antisocial behaviour in older male siblings for younger brothers and sisters. *Journal of Family Psychology, 19*(4), 643-653.

Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays and early child antisocial behavior. *Social Development, 12*, 335-360.

Steele, F. (2008). Multilevel Models for Longitudinal Data. *Journal of the Royal Statistical Society, Series A, 171*(1), 5-19.

Wu, W., West, S. G., & Taylor, A. B. (2009). Evaluating model fit for growth curve models: integration of fit indices from SEM and MLM frameworks. *Psychological Methods, 14*(3), 183-201.

Yakovleva, M., Reilly, R., & Werko, R. (2010). Why do we trust? Moving beyond individual to dyadic perceptions. *Journal of Applied Psychology, 95*, 79-91.

Table 1

*Descriptive Statistics for the Analysis Sample, Avon Brothers and Sisters Study*

| | Measurement occasion (2 years apart) | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Number of families | 172 | 143 | 108 |
| Percent with 3 (rather than 2) children | 41.3 | 37.8 | 34.3 |
| Number of mothers | 172 | 141 | 104 |
| Mean depression score (SD) | 0.185 (0.152) | 0.189 (0.165) | 0.173 (0.149) |
| Number of children | 402 | 328 | 242 |
| Mean delinquency score (SD) | 0.101 (0.180) | 0.116 (0.184) | 0.110 (0.194) |
| Mean age in years (SD) | 8.18 (3.33) | 10.15 (3.28) | 12.46 (3.21) |
| Percent girls | 46.0 | 43.6 | 45.0 |

Table 2

*Estimated Coefficients from Alternative Models of Maternal Depression and Child Delinquency*

| | M1 | | M2 | | M3 | | M4 | |
|---|---|---|---|---|---|---|---|---|
| Maternal depression equation ( $y_t^{(P)}$ ) | | | | | | | | |
| $t$ = 1 | | | | | | | | |
| Constant | 0.166*** | (0.015) | 0.166*** | (0.015) | 0.167*** | (0.015) | 0.166*** | (0.015) |
| 3-child family (versus 2-child) | 0.045* | (0.023) | 0.045* | (0.023) | 0.045* | (0.023) | 0.045* | (0.023) |
| $t$ = 2, 3 | | | | | | | | |
| Constant | 0.057*** | (0.014) | 0.056*** | (0.014) | 0.124*** | (0.028) | 0.150*** | (0.023) |
| Occasion $t$ = 3 (versus $t$ = 2) | -0.008 | (0.014) | -0.006 | (0.014) | -0.005 | (0.012) | -0.001 | (0.012) |
| 3-child family (versus 2-child) | -0.005 | (0.016) | -0.005 | (0.016) | -0.004 | (0.020) | 0.019 | (0.023) |
| Lag maternal depression | 0.724*** | (0.048) | 0.723*** | (0.048) | 0.310** | (0.144) | 0.257** | (0.126) |
| *Child-to-mother effects* | | | | | | | | |
| Lag child delinquency | 0.001 | (0.024) | 0.001 | (0.024) | 0.029 | (0.028) | -0.072 | (0.056) |
| Child delinquency equation ( $y_t^{(C)}$ ) | | | | | | | | |
| $t$ = 1 | | | | | | | | |
| Constant | 0.006 | (0.025) | 0.003 | (0.025) | 0.0002 | (0.025) | 0.010 | (0.025) |
| 3-child family (versus 2-child) | 0.025 | (0.018) | 0.024 | (0.018) | 0.025 | (0.018) | 0.026 | (0.019) |
| Age in years at $t$ = 1 | 0.012*** | (0.003) | 0.012*** | (0.003) | 0.012*** | (0.003) | 0.012*** | (0.003) |
| Girl | -0.036** | (0.017) | -0.034** | (0.017) | -0.032* | (0.017) | -0.038** | (0.017) |
| $t$ = 2, 3 | | | | | | | | |
| Constant | -0.022 | (0.025) | -0.025 | (0.025) | -0.021 | (0.025) | 0.015 | (0.030) |
| 3-child family (versus 2-child) | -0.020 | (0.013) | -0.019 | (0.014) | -0.008 | (0.017) | -0.006 | (0.017) |
| Age in years at $t$ | 0.004* | (0.002) | 0.003* | (0.002) | 0.004* | (0.002) | 0.004* | (0.002) |
| Girl | 0.029 | (0.018) | 0.038** | (0.017) | 0.036** | (0.018) | 0.036** | (0.018) |
| Lag child delinquency | 0.663*** | (0.040) | 0.670*** | (0.038) | 0.610*** | (0.051) | 0.620*** | (0.051) |
| *Mother-to-child effects* | | | | | | | | |
| Lag maternal depression | 0.176*** | (0.054) | 0.204*** | (0.057) | 0.232*** | (0.061) | 0.022 | (0.110) |
| Lag maternal depression × girl | -0.202** | (0.078) | -0.253*** | (0.074) | -0.256*** | (0.075) | -0.256*** | (0.074) |
| *Sibling effects* | | | | | | | | |
| Lag younger sib delinquency (age difference=3 yrs) | 0.094 | (0.062) | 0.113* | (0.059) | 0.036 | (0.071) | 0.049 | (0.073) |
| Lag younger sib × \| age difference \| | 0.003 | (0.022) | 0.001 | (0.021) | 0.002 | (0.021) | 0.002 | (0.021) |
| Lag older sib delinquency (age difference=3 yrs) | 0.108** | (0.043) | 0.097** | (0.040) | 0.034 | (0.052) | 0.046 | (0.052) |
| Lag older sib × \| age difference \| | -0.017* | (0.009) | -0.014 | (0.008) | -0.013 | (0.008) | -0.013 | (0.008) |

| | | | | |
|---|---|---|---|---|
| - log likelihood | 714.11 | 736.00 | 738.32 | 741.06 |

*Note*. Standard errors are in parentheses. M1 includes uncorrelated occasion-level residuals, M2 extends M1 to include residual correlations, M3 extends M2 to include family effects, M4 includes a family-level residual correlation.
* $p < .1$. ** $p < .05$. *** $p < .001$.

Table 3

*Residual Standard Deviations and Correlations from Selected Joint Models of Maternal Depression and Child Delinquency, with and without Family-level Random Effects*

| | M2 (without family) | | M4 (with family) | |
|---|---|---|---|---|
| Maternal depression equation ( $y_t^{(P)}$ ) | | | | |
| Occasion level | | | | |
| SD of $e_{tj}^{(P)}$ at $t$ = 1 | 0.150*** | (0.008) | 0.104*** | (0.012) |
| SD of $e_{tj}^{(P)}$ at $t$ = 2, 3 | 0.111*** | (0.005) | 0.090*** | (0.007) |
| Family level | | | | |
| SD of $v_j^{(P)}$ | 0† | | 0.106*** | (0.015) |
| Child delinquency equation ( $y_t^{(C)}$ ) | | | | |
| Occasion level | | | | |
| SD of $e_{tij}^{(C)}$ at $t$ = 1 | 0.171*** | (0.006) | 0.163*** | (0.007) |
| SD of $e_{tij}^{(C)}$ at $t$ = 2, 3 | 0.141*** | (0.004) | 0.138*** | (0.005) |
| Between-sibling correlation at $t$ = 1 | 0† | | 0† | |
| Between-sibling correlation at $t$ = 2 | 0.340*** | (0.066) | 0.296*** | (0.079) |
| Between-sibling correlation at $t$ = 3 | 0† | | 0† | |
| Family level | | | | |
| SD of $v_j^{(C)}$ | 0† | | 0.052*** | (0.012) |
| Mother-child correlations | | | | |
| Occasion level | | | | |
| $\text{corr}(e_{tj}^{(P)}, e_{tij}^{(C)})$ at $t$ = 1 | 0.160*** | (0.042) | -0.014 | (0.116) |
| $\text{corr}(e_{tj}^{(P)}, e_{tij}^{(C)})$ at $t$ = 2, 3 | 0.131*** | (0.044) | 0.042 | (0.089) |
| Family level | | | | |
| $\text{corr}(v_j^{(P)}, v_j^{(C)})$ | 0† | | 0.869** | (0.345) |
| -log likelihood | 736.00 | | 741.06 | |

*Note*. Standard errors are in parentheses. † Parameter constrained to zero.
* $p < .1$. ** $p < .05$. *** $p < .001$.

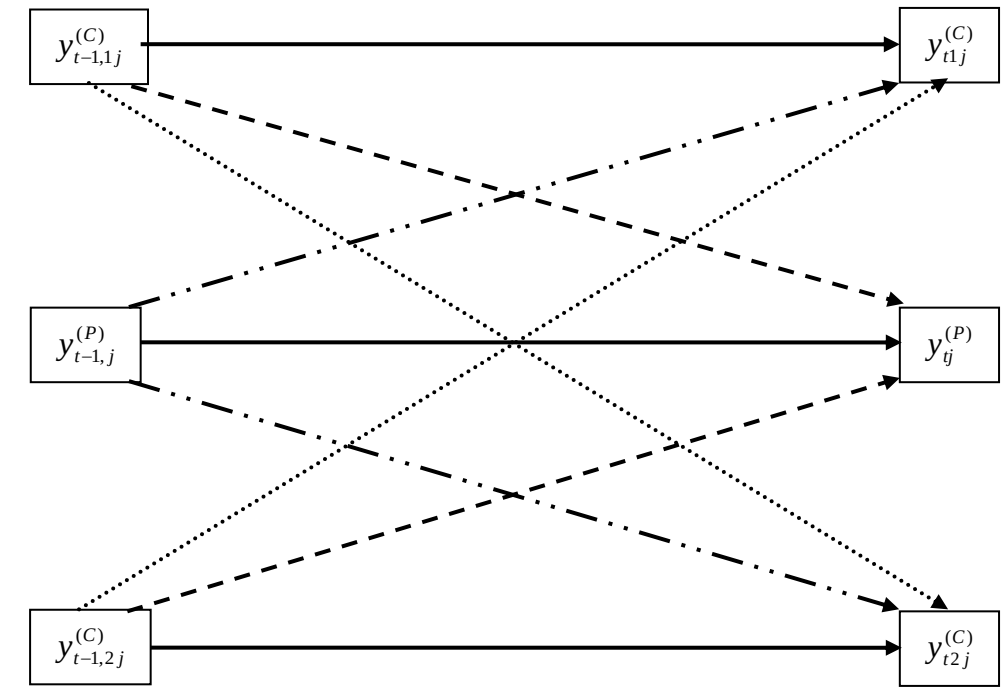


*Figure 1*. Lags and cross-lags in multilevel simultaneous equations model for parent-child reciprocal effects and sibling effects on responses at time *t*-1 and *t* in a two-child family *j*