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Lower Level Mediation in Multilevel Models

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Multilevel models are increasingly used to estimate models for hierarchical and repeated measures data. The authors discuss a model in which there is mediation at the lower level and the mediational links vary randomly across upper level units. One repeated measures example is a case in which a person's daily stressors affect his or her coping efforts, which affect his or her mood, and both links vary randomly across persons. Where there is mediation at the lower level and the mediational links vary randomly across upper level units, the formulas for the indirect effect and its standard error must be modified to include the covariance between the random effects. Because no standard method can estimate such a model, the authors developed an ad hoc method that is illustrated with real and simulated data. Limitations of this method and characteristics of an ideal method are discussed.

Multilevel models for hierarchical and repeated measures data are becoming increasingly common (Diggle, Heagerty, Liang, & Zeger, 2001; Hox, 2002; Raudenbush & Bryk, 2002; Snijders & Bosker, 1999). These models assume that there are at least two levels in a data set, an upper level, or Level 2, and a lower level, or Level 1. The Level 1 units are nested within the Level 2 units. In some applications, the upper level refers to persons and the lower level refers to observations or repeated measurements. For example, a researcher might collect daily diary (repeated measures) data over several weeks on people's exposure to daily stressors, their coping efforts, and their emotional states (e.g., Bolger, Davis, & Rafaeli, 2003; Bolger & Zuckerman, 1995). In other applications, the upper level refers to groups and the lower level refers to persons who are members of those groups. For example, a researcher may collect demographic background, parenting practices, and educational achievement data on all schoolchildren in a sample of schools (e.g., Raudenbush & Bryk, 1986). In this example, schools are the groups, and schoolchildren are the persons nested within those groups.

A basic multilevel model for the repeated measures data might specify that at Level 1, the repeated measures level, a person's mood on a given day is a function of a baseline mood level that is common across all days, a stressor reactivity effect that reflects whether or not he or she has experienced a stressor that day, and a Level 1 residual effect that varies randomly from day to day but whose average value is zero. At Level 2, the between-person level, the model might specify that people differ in how reactive they are to daily stressors and that a given person's reactivity is a function of an effect that is common to all people, and a residual Level 2 effect that varies randomly from person to person but whose average value is zero.

Multilevel modeling has several advantages over traditional models for such data. For instance, unlike traditional models for repeated measures data, multilevel models can effectively manage unequal group sizes and missing data on the repeated measure. Multilevel modeling can also be used to examine simultaneously the effects of Level 2 (e.g., school level) and Level 1 (e.g., child level) variables in nested data sets. In doing so, multilevel models take account of (and adjust for) any bias in standard errors and statistical tests resulting from the nonindependence of observations that is typical in such data (Krull & MacKinnon, 2001). Because of these advantages and others, multilevel modeling has drawn substantial attention recently.

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Testing mediational hypotheses is a central activity in psychological science (Baron & Kenny, 1986; Kenny, Kashy, & Bolger, 1998; Shrout & Bolger, 2002), and mediational questions are as relevant to multilevel data as they are to traditional data structures. Kenny, Kashy, and Bolger (1998) introduced the topic of multilevel mediation and explained the distinction between upper level and lower level mediation. In *upper level mediation* the initial or putative causal variable whose effect is mediated is an upper level variable. An example of this type of mediation is where the effect of a school-level safer-sex intervention program (upper level initial variable) on students' intentions to engage in safer sex (lower level dependent variable) is mediated by students' motivations to engage in safer sex (lower level mediator).

In *lower level mediation,* the initial variable is at the lower level. In the repeated measures example described previously, this might be where the reactivity effect of a daily stressor (lower level initial variable) on a person's mood on a given day (lower level dependent variable) is mediated by the person's coping efforts that day (lower level mediator). In their discussion of lower level mediation, Kenny, Kashy, and Bolger (1998) considered cases where the lower level initial variable and mediator show random upper level variability in their effects. This variability can be thought of as a form of moderation (Baron & Kenny, 1986), a topic that we return to later. For instance, in the repeated measures example, the effect of stressors on coping and the effect of coping on mood may vary across persons, the upper level units. In prior work using daily diary reports of stressors, coping, and mood, Bolger and Zuckerman (1995) found evidence of random variability in these links.

Several researchers have focused on upper level mediation. For example, Krull and MacKinnon (1999) described and evaluated methods used to test upper level mediated effects. Using both simulated and real data, Krull and MacKinnon compared single level and multilevel mediation analyses, two ways to calculate the effect of the mediator, and two coefficient estimation methods, which coincided with the types of mediation analyses. Raudenbush and Sampson (1999) also focused on upper level mediation. They demonstrated how the computer program HLM5 (Raudenbush, Bryk, Cheong, & Congdon, 2000) can be used to estimate and test upper level mediational models. Raudenbush and Sampson demonstrated this approach using a multilevel design with latent variables in which measurement error is represented as a level within the model. Lastly, Krull and MacKinnon (2001) compared the appropriateness of single level and multilevel data analysis procedures to test mediation effects in nested data. Two of the three models that they considered were upper level mediational models.

By comparison, discussion of lower level mediation in multilevel modeling has been sparse, and to date there has been no discussion of the case in which the mediational links show random upper level variability. Judd, Kenny, and McClelland (2001) discussed lower level mediational analysis but only in the limited case where the problem could be recast in terms of standard fixed-effects analysis of variance. Krull and MacKinnon (2001) also considered lower level mediation in nested data but only to compare the appropriateness of single level and multilevel data analysis procedures to test a fixed-effects mediational model.

Thus, although mediation in multilevel models has been the focus of increasing attention recently, there has been no discussion of analysis methods for cases where the putative causal variable is at the lower level. As noted, with lower level mediation, all the mediational links may vary randomly across the upper level units, and so, it is much more complicated than upper level mediation. The present article provides a complete overview of lower level mediational analysis in a fully random model using a multilevel framework, and it shows that interesting, unexpected, and important complications arise in this case.

The Lower Level Mediational Model

Consider a Level 1 variable, *Y,* that is assumed to be caused by two other Level 1 variables, *X* and *M*. Of key interest in this article is the possibility that the variable *M* may mediate the effect of *X* on *Y,* lower level mediation. In this article, variable *X* is called the *initial* or *putative causal variable, M* is the *mediator,* and *Y* is the *outcome*. Ideally *X* is a variable that is experimentally manipulated, but it need not be. For ease of understanding, it might help to think of the example given previously where *X* is the occurrence of a stressor on a given day, *M* is the person's coping efforts that day, and *Y* is the person's mood that day. The upper level unit would be person, and the lower level unit would be day.

Equations

Figure 1 presents the basic mediational model as a path diagram. For the moment, the reader should

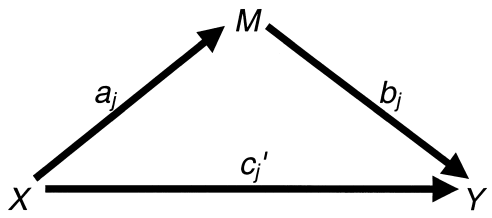

Figure 1. The Level 1 mediation model in which the effect of *X* on *Y* is partially mediated by *M;* for Level 2 unit *j, X* causes *M* (path a_j), *M* causes *Y* (path b_j), and *X* causes *Y* (path c_j').

ignore the subscripts in the coefficients. The variable *X* causes *M,* which in turn causes *Y.* For the example, stressors cause coping, which in turn causes mood. The direct effect of *X* on *Y* would be c', and the indirect effect would be *ab.* The total effect, the direct effect plus indirect effect, would equal $c' + ab$, which we denote as *c.* The reader should note that we do not use Greek letters for these effects, even though they are population values. Whenever they are sample estimates, we will make it clear.

In a multilevel model, lower level effects may vary by upper level unit. So, all of the effects, *a, b,* c', and *c* might vary by the upper level unit. That is why they are subscripted by the upper level unit, denoted in Figure 1 as *j*. The figure presents the mediational model for upper level unit *j*. For the example, the mediation of the stressor–mood relationship by coping might vary by person, and so the direct effect of stressor on mood, path c_j', might be different for different persons.

More formally, we consider a two-level model in which the first subscript, *i,* refers to the lower level, and the second subscript, *j,* as in the figure, refers to the upper level. The Level 1 equation for *Y* without the mediator is

$$Y_{ij} = d_{0j} + c_j X_{ij} + r_{ij}. \quad (1)$$

In this equation, *Y* is the outcome, d_{0j} is the intercept for each upper level unit, c_j represents the effect of *X* on *Y* for each upper level unit, and r_{ij} is an error term. The two mediational equations, implied in Figure 1, are

$$M_{ij} = d_{1j} + a_j X_{ij} + e_{ij} \quad (2)$$

and

$$Y_{ij} = d_{2j} + c_j' X_{ij} + b_j M_{ij} + f_{ij}. \quad (3)$$

Equations 1, 2, and 3 are sometimes called the *lower level* or *Level 1 equations*. We refer to path *c* (from Equation 1) as the total effect of *X* on *Y,* path c' (Equation 3) as the direct effect, path *a* (Equation 2) as the effect of *X* on *M,* and path *b* (Equation 3) as the effect of *M* on *Y*. All of these coefficients may vary across the upper level units, which is why they each have the subscript *j*. The two intercepts in Equations 2 and 3, denoted by *d,* are not central to our mediational model.

The Level 2 equation for the total effect, when there are no Level 2 effects, is

$$c_j = c + u_{0j}, \quad (4)$$

which simply says that the coefficient for unit *j* equals the average coefficient (the population mean) plus a deviation from that average for each upper level unit. So, *c* would represent the typical level of the total effect, and u_{0j} would represent a deviation from that typical level for upper level unit *j*. In the same way, we can write upper level equations for the other coefficients in the lower level mediational model. The following are the upper level equations for the other lower level coefficients when there are no Level 2 effects:

$$a_j = a + u_{1j}, \quad (5)$$

$$b_j = b + u_{2j}, \quad (6)$$

and

$$c_j' = c' + u_{3j}. \quad (7)$$

Thus, we denote the unsubscripted parameter as the *average parameter value.* (The intercepts also have similar Level 2 equations, but as noted, they are irrelevant to our discussion.) Returning to the stressor–coping–mood example, we would allow for individual differences in the effect of stressors on mood (*c* and c_j'; i.e., for some persons, stressors have more of an effect on mood than for others). There might also be individual differences in the effect of stressors on coping (path *a*; i.e., a stressor might activate a coping strategy for some persons but not for others). Finally, the effect of coping on mood (path *b*) might also vary by person (i.e., the coping strategy is effective for some people but not for others).

Decomposition of Effects

The single level equation for the decomposition of mediations effects (MacKinnon, Warsi, & Dwyer, 1995) is

$$c = c' + ab. \quad (8)$$

The equation states that the total effect (c) equals the direct effect (c') plus the indirect effect (ab). However, Equation 8 presumes that a and b are fixed parameters; that is, a and b do not vary across upper-level units.

If we assume that mediational effects a and b are random variables that have a bivariate normal distribution, a standard assumption within multilevel modeling, then it can be shown that the expected value of $a_j b_j$ does not equal ab but rather equals $ab + \sigma_{ab}$ (Goodman, 1960). The expected value of a product or $E(a_j b_j)$ does not equal the product of the expected values of the components of that product or $E(a_j)E(b_j)$ when a_j and b_j are correlated. Thus, given multivariate normality, the total effect in fully random lower level mediated multilevel models is decomposed as

$$c = c' + ab + \sigma_{ab}, \tag{9}$$

where σ_{ab} is the population covariance of a_j with b_j and refers to a possible correlation of a_j and b_j effects. A positive value for σ_{ab} implies that those upper level units that have large values of a_j also have large values of b_j. Conversely, a negative value for σ_{ab} implies that those upper level units that have large values of a_j also have small values of b_j. For the example, σ_{ab} refers to the covariance between the effect of stressors on coping and the effect of coping on mood.

We should note that even if a and b were not random, then Equation 9 would not exactly hold for estimates obtained from multilevel models (Krull & MacKinnon, 1999). Because maximum-likelihood estimation, not ordinary least squares (OLS), is used, the equation is not exact, only approximate. Alternatively, we can avoid obtaining an independent estimate of the total effect c by instead estimating it indirectly by calculating c', ab, and σ_{ab} and summing them. Thus, the total effect would be defined as $c' + ab + \sigma_{ab}$.

In single level mediational analysis, the formula for the sampling variance of the mediated effects, denoted as $\sigma_{(ab)}^2$, is

$$\sigma_{(ab)}^2 = b^2\sigma_a^2 + a^2\sigma_b^2 + \sigma_a^2\sigma_b^2. \tag{10}$$

The formula for the variance of indirect effects given by Sobel (1982) is an approximation and does not include the last term of the above equation (Aroian, 1947; Baron & Kenny, 1986). Equation 10 presumes that a and b are fixed; that is, they do not vary across upper level units. If, however, we presume that these parameters are random, the equation for the variance of mediated effects assuming multivariate normality of effects is

$$\sigma_{(ab)}^2 = b^2\sigma_a^2 + a^2\sigma_b^2 + \sigma_a^2\sigma_b^2 + 2ab\sigma_{ab} + \sigma_{ab}^2 \tag{11}$$

(see Kendall & Stuart, 1958). The two terms in the right side of the equation that are not included in the single level formula (see Equation 10) involve σ_{ab}, the covariance of a and b, which is critical in the estimation and testing of lower level mediation in multilevel models with random effects.

The variance of the total effect σ_c^2, given the assumption that all mediational links are random variables whose joint distribution is multivariate normal, is

$$\sigma_c^2 = \sigma_{c'}^2 + b^2\sigma_a^2 + a^2\sigma_b^2 + \sigma_a^2\sigma_b^2 + 2ab\sigma_{ab} + \sigma_{ab}^2 + 2b\sigma_{ac'} + 2a\sigma_{bc'}. \tag{12}$$

Note that there are two new terms in this equation; one is the covariance between a and c' and the other is the covariance between b and c'. We consider the term σ_c^2 in more detail in the next section when we discuss moderation.

Mediation and Moderation

In this section we consider both the mediation and moderation of the effect of X on Y. We see that with multilevel data in which both X and Y are Level 1 variables, considerable detail can be obtained for both mediation and moderation, much more than with single level models. For ease of presentation, we assume in this section that a, b, and c are all positive.

Controlling for M might reduce the overall effect of X on Y (i.e., c). This is classical mediation. In the fully random-effects multilevel model, there are two ways in which c' might be less than c. First, there is reduction if ab is nonzero, or second, if σ_{ab} is nonzero. Note that ab might equal zero, but c' can still be less than c because σ_{ab} can be nonzero.

In the fully random-effects model, there is potentially Level 2 variation in the effect that X has on Y. This variation designated as σ_c^2 reflects evidence of moderation of the X-to-Y relationship, though the identity of the moderating variable has yet to be discovered. We can also determine how much of this moderation is reduced or even eliminated by controlling for M. Note that if σ_c^2 were zero, then X would affect Y to the same degree for all upper level units; that is, there would be no moderation. Stated differently, if σ_c^2 equals zero, the effect of X on Y does not vary, and so, it does not make sense to search for

Level 2 factors that would explain any variation. In summary, if the effect of *X* on *Y* is fully explained by *M,* then (a) the overall effect, *c,* should be explained, and c' would be zero, and (b) the variance of the effects, σ_c^2, should be explained, and, therefore, $\sigma_{c'}^2$ would be zero.

For instance, assume that the population values are $a = 0.50$, $b = 1.00$, and $c' = 0.25$. We set $\sigma_a^2 = 1.00$, $\sigma_b^2 = 1.00$, $\sigma_{c'}^2 = 0.25$, $\sigma_{ab} = 0.50$, and $\sigma_{ac'} = \sigma_{bc'} = 0.00$. When we use the single level model (Equation 8), $c = 0.25 + 0.50 = 0.75$. Then, 67%, or (0.75 − 0.25)/0.75, of the overall effect of *X* on *Y,* or 0.75, is mediated. However, using the formulas for a fully random-effects model, it follows that $c = 0.25 + 0.50 + 0.50 = 1.25$ (Equation 9) and that $\sigma_c^2 = 0.25 + (1.00)^2(1.00) + (0.50)^2(1.00) + (1.00)(1.00) + 2(0.50)(1.00)(0.50) + (0.50)^2 + 2(1.00)(0.00) + 2(0.50)(0.00) = 3.25$ (Equation 12). Consequently, it is really that 80%, or (1.25 − 0.25)/1.25, of the overall effect of *X* on *Y,* or 1.25, is mediated. It also follows that 92% of the variance of the *X*-to-*Y* effect is explained by variation in *M,* (3.25 − 0.25)/3.25. These proportions of the total effect, which in the current example are calculated using population values, illustrate the importance of considering σ_{ab} when estimating the amount of mediation in a fully random model. Note too that the correct value for $\sigma_{(ab)}^2$ would be 3.00 as estimated by the formula for the fully random-effects model (Equation 11), not 2.25 as would be estimated using the single level formula (Equation 10). The proportions of the total effect can also be calculated using sample estimates, though these proportions can be very unstable.

Even more surprising, consider the case where the population values are $a = 0.00$, $b = 0.00$, $c' = 0.00$. In this model, there is no overall effect of *X* on *Y,* either direct or indirect. Using the single level model formula (Equation 8), we would think that the total effect would have to be zero. However, it is not. If we set $\sigma_a^2 = 1.00$, $\sigma_b^2 = 1.00$, or $\sigma_{c'}^2 = 0.00$, $\sigma_{ab} = 1.00$, and $\sigma_{ac'} = \sigma_{bc'} = 0.00$, using the random effects formulas, it follows that $c = 0.00 + (0.00)(0.00) + 1.00 = 1.00$ (Equation 9) and that $\sigma_c^2 = 2.00$ (Equation 12). Thus, we find that the total effect for the average Level 2 unit is nonzero and is entirely due to the covariance between *a* and *b*. To understand why this is the case, we need to consider that in this example, although the average of *a* and the average of *b* both equal zero, the average of *ab* tends to be positive because positive *a*s are paired with positive *b*s and negative *a*s are paired with negative *b*s.

As the above examples illustrate, when σ_{ab} is the same sign as *ab* and is not considered when determining the amount of the mediation, the amount of mediation is underestimated. However, when σ_{ab} is not considered, the amount of mediation could be overestimated. This is the case when σ_{ab} is opposite in sign to *ab*. Consider the following case: $a = 0.50$, $b = 1.00$, $c' = 0.25$, and $\sigma_{ab} = -0.50$. When we use the single level approach (Equation 8), $c = 0.25 + (0.50)(1.00) = 0.75$. Then, 67%, or (0.75 − 0.25)/0.75, of the overall effect of *X* on *Y*, or 0.75, is mediated. However, when we use the correct equation for a random effects model (Equation 9), it follows that $c = 0.25 + (0.50)(1.00) - 0.50 = 0.25$. Consequently, it is really that there is no mediation; that is, 0%, (0.25 − 0.25)/0.25, of the overall effect of *X* on *Y,* or .25, is mediated.

The Substantive Meaning of the ab *Covariance*

The interpretation of σ_a^2 and σ_b^2 is straightforward. The variance σ_a^2 represents differences in the effectiveness of *X* in causing the mediator *M*. The variance σ_b^2 represents differences in the effectiveness of *M* in causing the outcome *Y*. How might σ_{ab} be interpreted? First, note that there must be some variation in *a* and *b* for there to be any covariance between the two. Such variation should be first established before σ_{ab} is interpreted.

The exact interpretation of σ_{ab} depends on the particular application. Consider again the example of the effect of stressors on mood. The mediator might be a coping style. It might be the case that the coping style is more effective in relieving distress for some individuals than for others and that variation is captured by σ_b^2. For persons who have a large *b* (i.e., the coping style is effective), it seems reasonable to expect that stressors would induce more coping; therefore, their *a* parameter would be large. By the same token, for those whose *b* path was small meaning that the coping style was ineffective, it is expected that the *a* path would be small indicating that stressors would not affect amount of coping. Thus, it seems plausible that σ_{ab} would be positive and would be theoretically interesting, although theoretical importance is not required for consideration of σ_{ab}; the term σ_{ab} should be considered whenever decomposing the effects of a lower level random-effects mediational model. If σ_{ab} is the same sign as *ab* and is not considered, the amount of mediation would be underestimated.

It might also be the case that σ_{ab} is the opposite

sign of *ab*. Consider the case where *ab* is positive, and σ_{ab} is negative. For example, suppose that the effect of instruction quality (the degree to which the lesson is taught well) on a student's learning is mediated by student motivation to learn. Overall, high quality instruction leads to more motivation to learn, and more motivation to learn leads to more learning. However, it might be the case that some students are extrinsically motivated to learn and so are greatly affected by instruction quality (i.e., their *a* parameter is large), whereas other students are intrinsically motivated and are not as affected by instruction quality (i.e., their *a* parameter is relatively small). This variation would be captured by σ_a^2, which is the amount of variation in the *a* parameter due to Level 2 unit, or in this case, participant. It may also be the case that the students who are extrinsically motivated to learn—have a large *a* parameter—have not developed good study skills. Consequently, an increase in their motivation to learn has little impact on learning (i.e., their *b* parameter is small). Conversely, students who are intrinsically motivated to learn—have a small *a* parameter—have developed their study skills, and consequently, an increase in their motivation has a great impact on learning (i.e., their *b* parameter is large). In this case, σ_{ab} is negative because small *a*s are paired with large *b*s, and large *a*s are paired with small *b*s.

As the prior example illustrates, it is possible for *ab* to be positive and σ_{ab} to be negative. However, a change in scale of *M* might artificially create this state of affairs. Imagine that *M* was standardized by dividing each M_{ij} by the standard deviation of *M* for upper level unit *j,* s_{Mj}. Even if it were true that both σ_a^2 and σ_b^2 were zero before such a standardizing, the standardizing within each upper level unit would result in a spurious negative σ_{ab} because the new a_j path would now equal $s_{Mj}a_j$, and the new b_j path would now equal b_j/s_{Mj}. Because one path is multiplied and the other divided by the same value, the result would be a negative correlation between *a* and *b*. For instance, if s_M were large, then the new *a* path would be relatively large and the *b* path relatively small. But if s_M were small, then the new *a* path would be small and the *b* path would be large. Thus, the presence of a negative σ_{ab} might be due to an artifact of scale transformation, therefore, such transformations (e.g., standardizing within Level 2 units) should be avoided.

We suspect, but do not know, that typically σ_{ab} and *ab* will have the same sign. Regardless if σ_{ab} and *ab* have the same or opposite sign, it is critical to measure and interpret σ_{ab} in multilevel mediation.

Level 2 Variables

Although the major focus of the present article is lower level or Level 1 mediational analyses, typically in a multilevel model there are Level 2 variables that can be used to explain the coefficients of the Level 1 equations. For instance, in cases where the Level 2 unit is group, group-level variables such as classroom might explain the coefficients of the Level 1 equations. In cases where the Level 2 unit is person, person variables such as attitudes, aptitudes, or personality traits might explain the coefficients in the Level 1 equations. Therefore, it is important to consider Level 2 variables in this type of model. To illustrate, consider again the stressor–coping–mood example. Bolger and Zuckerman (1995) examined how individual differences in the personality variable of neuroticism were related to individual differences in stress reactivity (the total stressor-to-mood link). They then examined the extent to which these reactivity differences could be explained in terms of individual differences in coping choice (the stressor-to-coping link) and coping effectiveness (the coping-to-mood link). In this section, we consider the incorporation of such Level 2 variables within our approach. We shall see that such variables can be treated as potential moderators of the mediational process.

Consider a variable *Q* that is measured for each upper level unit. The Level 2 equations would be as follows:

$$a_j = a + dQ + u_{1j}, \tag{13}$$

$$b_j = b + eQ + u_{2j}, \tag{14}$$

and

$$c_j' = c' + fQ + u_{3j}. \tag{15}$$

The terms *a, b*, and c' would be the overall effects when *Q* is zero. If zero for *Q* were not meaningful, then *a, b*, and c' would be uninterpretable. Thus, it is important that zero is a meaningful value for *Q*, and if it is not, then *Q* should be centered. The Level 2 variables may also explain the intercepts, but we do not consider this because our focus is on the mediation of effects. We can view *Q* as a moderator variable in that it would explain some of the Level 2 variation in the effect of *X* on *Y*.

Key parameters in the mediational model are the variances of effects (e.g., σ_a^2) and the covariances of effects (e.g., σ_{ab}). However, if there are Level 2 variables in the model, these variances and covariances are partial variances with the variance due to the

Level 2 variable removed. So the variances and covariances refer to the residual values of u_{1j}, u_{2j}, and u_{3j} (see Equations 13, 14, and 15).

It is generally advisable to center Q or at least to make sure that Q is initially scaled or rescaled so that a zero value on Q is meaningful. One should avoid "group centering" (Kreft, DeLeeuw, & Aiken, 1995). The scaling of Level 2 variables is critical to the interpretation of the estimates of a, b, and c'.

It is possible for a Level 2 effect to be mediated by a Level 1 variable. The estimation and testing of such a mediational process is described by Krull and MacKinnon (1999) and Raudenbush and Sampson (1999). Recall that the focus of this article is the analysis of mediation of a Level 1 effect.

Estimation

We have described a complication that arises in lower level mediational analysis in a fully random-effects model using a multilevel framework. Here we discuss a general procedure for testing lower level mediation in multilevel models and suggest an interim procedure for addressing this complication. (Our approach using HLM5 is described in great detail at http://users.rcn.com/dakenny/mlm-med-hlm5.doc)

The first step in lower level mediational analysis is determining if the model is a random-effects model. Of particular interest is whether both of the effects in the indirect path are random or vary at Level 2. To determine the nature of these effects, researchers should inspect the variance components of the random effects when they regress the mediator on the putative causal variable and the outcome variable on the mediator. Assuming that there is sufficient power, researchers can determine if these effects are indeed random by using statistical tests of whether the amount of variance is greater than zero. Variance components statistically greater than zero indicate that the effects of the variables specified in the model vary by Level 2 unit and, consequently, indicate that the model is a random-effects model.

If at least one of the two effects in the indirect path is nonrandom (i.e., fixed), then ordinary mediational analysis procedures that have been used to date can be used to estimate and to test the mediated effects. If, however, both a and b are random, then the covariance between a and b might be nonzero. One would then test the covariance to determine if it is statistically different from zero. However, even if the covariance is not statistically different from zero, we think it best not to fix it to zero. In conducting tests of statistical significance of random effects, researchers should consider the possibility that in their particular study there is low power to detect effects. In such cases, it may be worthwhile to allow the statistically nonsignificant random effects to be estimated and to estimate σ_{ab}.

Multilevel modeling techniques can be used to estimate the effects and the variances, but it is unclear how the covariance σ_{ab} can be estimated. It would not seem possible to do so with the computer programs that allow for only a single outcome variable because in the model considered in this article there are two outcome variables, M and Y. With these programs, it is possible to estimate the covariance between two effects, but the outcome variable must be the same variable. It is inadvisable to use the empirical Bayes estimates to estimate σ_{ab} because their variance is shrunken; therefore, it would likely underestimate the absolute value of σ_{ab}.

One straightforward way to estimate σ_{ab} would be to compute the covariance[1] of the OLS estimates of a_j and b_j. To do this, one correlates the estimates of a_j and b_j and then multiplies that correlation by the product of the standard deviations of the sample estimates of a_j and b_j. We believe that this is an unbiased estimate of σ_{ab}. To test the null hypothesis that σ_{ab} equals zero, we use the usual test of a correlation coefficient. This correlation is between the estimates of a and b.

However, the correlation between estimated a and b does not tell us how correlated a and b are because sampling error is not controlled. To avoid this problem, we suggest using the disattenuated correlation between population a and b. To determine the disattenuated correlation between population a and b, we divide the estimated σ_{ab} by the square root of the product of the estimates of $\sigma_a{}^2$ and $\sigma_b{}^2$, assuming of course the two variances are nonnegative. This disattenuated correlation of a and b will almost always be greater than the correlation between estimated a and b. There is no guarantee that the correlation will be in bounds, that is, between 1 and −1. We think that even if the correlation is out of bounds, one should still use the estimate of σ_{ab}.

We know of no current computer program that will estimate this entire model in a straightforward fash-

[1] If there were Level 2 variables, one would need to compute the partial covariance, controlling for the Level 2 variables.

ion. One might think that HLM5 would accomplish this purpose. Although it allows for the parameter *b* to be a random variable, parameter *a* cannot be a random variable with this program. Because *a* is not random, it follows that σ_{ab} is zero. Various multilevel modeling programs (e.g., MLwiN; Rasbash et al., 2000) allow for multivariate outcomes, and some structural equation modeling programs (e.g., LISREL 8; Jöreskog and Sörbom, 1996) have options that allow for multilevel data, but so far as we know, none of these programs allow for paths from one outcome to another.

In the absence of a general program, we recommend the following piecemeal and interim strategy. We first estimate the effect of *X* on *Y* to obtain σ_c^2 and *c*. We then estimate the effect of *X* on *M* to obtain σ_a^2 and *a*. Next, we estimate the effect of *X* and *M* on *Y* to obtain estimates of σ_b^2, $\sigma_{c'}^2$, *b*, and *c*′. To estimate σ_{ab}, we compute the OLS estimates of the slopes and then compute their covariance across Level 2 units or partial covariance if there are Level 2 variables. We can use the same procedure to estimate $\sigma_{ac'}$ and $\sigma_{bc'}$. With all of these estimates we can decompose effects using the formulas that we have provided.

The estimation method that we have developed requires the assumption that *a*, *b*, and *c*′ have a joint multivariate normal distribution. Although this assumption is standard in multilevel modeling, the violation of the assumption would be more serious here. Normality is assumed here to identify a model. That is, Equations 9 and 11 were derived by assuming normal distribution. Without normality, the equations would not hold. Alternatively, we need not make the assumption of normality, and we could compute *c* and σ_c^2 directly. So for instance, we could estimate *c* and then subtract estimated *c*′ and *ab*. By this method, the remainder would reflect not only σ_{ab} but also the effect of nonnormality. By using the estimation procedure that we have developed, we can directly estimate σ_{ab}. More details about this estimation procedure are provided in the two examples that follow.

Examples

We present two rather detailed examples of the piecemeal strategy to compute σ_{ab} that we outlined above. We first present an example using a simulated data set. We used a simulated data set for two reasons. First, we want to show that our method, and not the usual method, correctly decomposes effects and determines their variance. Second, with simulated data, we know that the assumption of multivariate normality is exactly met in the population.

Simulated Data

Using a QBasic computer program, we generated a simulated multilevel data set based on the population parameters displayed in Table 1. The distributions for all random variables were normal. So, for instance, *a*, *b*, and *c*′ were generated as random normal variables. The data set consisted of 200 upper level units each with 10 lower level observations per variable, therefore, 2,000 observations per variable. We realize that this data set is much larger and balanced than the typical multilevel data set, but we wanted to reduce the effects of sampling error on the solution. In choosing parameter values in the simulation, we selected values that created sizable mediation effects that varied considerably.

The specified model is a fully random-effects model. Although we did correlate *a* and *b,* we did not correlate *a* or *b* with *c*′. The sample data (available at http://users.rcn.com/dakenny/mul-lev-sim.txt) were analyzed by HLM5 (Raudenbush et al., 2000). Additionally, we used MLwiN (Rasbash et al., 2000) and SAS's PROC MIXED, and their estimates were virtually identical to those of HLM5. We note that the

Table 1
Simulation Model and Sample Statistics Estimated by HLM5 (Raudenbush et al., 2000)

Parameter	Population	Sample (*SE*)
Paths		
c	.672	.673 (.044)
c (inferred)	.672[a]	.704[a]
a	.600	.585 (.032)
b	.600	.646 (.036)
c′	.200	.201 (.027)
Variances and covariances		
σ_c^2	.274	.290
σ_c^2 (inferred)	.274[b]	.312[b]
$\sigma_{(ab)}^2$	.235[c]	.254[c]
σ_a^2	.160	.135
σ_b^2	.160	.184
$\sigma_{c'}^2$	.040	.058
σ_{ab}	.113	.125

Note. The parameters $\sigma_{(ab)}^2$ as well as the inferred values of *c* and σ_c^2 are not free parameters but rather equal a function of the other parameters in the model. See the text for the formulas. Also, $\sigma_{ac'}$ and $\sigma_{bc'}$ are set to zero. Note that σ_{ab} refers to the covariance of *a* and *b,* whereas $\sigma_{(ab)}^2$ refers to variance of the product *ab*.
[a] Calculated using Equation 9.
[b] Calculated using Equation 12.
[c] Calculated using Equation 11.

method that we propose can use virtually any multilevel estimation computer program. Table 1 presents the population values of the parameters.

A random-effects mediational model was estimated using the Baron and Kenny (1986) steps. The resulting parameter estimates from the simulation data set are displayed in the right column of Table 1. We now use HLM5 notation, not the notation that we have used so far in the article, to describe the estimation steps. First, using HLM5, we estimated the unmediated effect of X on Y or path c. In this model the Level 1 equation is $Y = \beta_0 + \beta_1(X) + r$. The Level 2 equations are $\beta_0 = \gamma_{00} + u_0$ and $\beta_1 = \gamma_{10} + u_1$.

Second, the effect of X on the mediator M, which is path a in Figure 1, was estimated. The Level 1 equation in this model using HLM5 notation is $M = \beta_0 + \beta_1(X) + r$. The Level 2 equations for this model are the same as those for the model of the unmediated effect, that is, $\beta_0 = \gamma_{00} + u_0$ and $\beta_1 = \gamma_{10} + u_1$. While estimating the effect of X on M, we created a file containing the OLS estimates of the Level 1 path a coefficients[2] (i.e., the a coefficients for each individual), which are necessary to estimate σ_{ab}.

Next, the effects of M and X on Y—paths b and c' displayed in Figure 1, respectively—were estimated. In this model the Level 1 equation is $Y = \beta_0 + \beta_1(X) + \beta_2(M) + r$, and the Level 2 equations are $\beta_0 = \gamma_{00} + u_0$, $\beta_1 = \gamma_{10} + u_1$, and $\beta_2 = \gamma_{20} + u_2$. As was done with path a coefficients, a file containing the path b coefficients was created.

Finally, the OLS estimates of the b coefficients and OLS estimates of the a coefficients (contained in the first residual file created) were then copied into a single file and the covariance between a and b or σ_{ab} was estimated. Lower level path a and path b coefficients were correlated, $r = .472$, $p < .01$, with a covariance of .125, as displayed in Table 1. The disattenuated correlation between a and b (as opposed to the correlation between estimated a and b) is .793. Table 1 displays a summary of population or theoretical and sample or estimated model parameters. We used HLM5 to estimate the model parameters.

We can perform the decomposition of the total effect both for the theoretical and empirical values. The population total effect or c is equal to .672. In the standard single level model formulation (see Equation 8), the total effect should equal the direct plus the indirect effect or 0.20 + (0.60)(0.60) or 0.56. Clearly, the standard single-level model formulation underestimates the total effect. The population total effect 0.672 is underestimated by 0.113, which exactly equals the covariance between a and b. The population total effect inferred using the suggested formula for random-effects models (Equation 9), which considers σ_{ab}, estimates the population total effect exactly. The population Level 2 variance of estimated ab using the suggested formula for random-effects models (Equation 11) equals 0.235. This population variance is underestimated at 0.141 when the standard single level model approach, Equation 10, is used.

In the sample, the estimated total effect c, 0.673, is virtually identical to the population value of 0.672. We can also decompose the sample total effect using the standard single level model approach to decomposition or Equation 8. This approach underestimates the total effect as 0.579. The correct equation for random-effects models, Equation 9, is closer to the total effect, somewhat overestimating it as 0.704. Using the suggested formula for multilevel random-effects models (Equation 11), we estimate $\sigma_{(ab)}^2$ as 0.254, which is not that far from the population value, .234, and much closer than using the standard single level model approach (Equation 10), which results in a value of 0.144.

Finally, we examined how the introduction of the mediator affects the variation in the effect of X on Y. In the population, σ_c^2 is 0.274 and $\sigma_{c'}^2$ is 0.040. Thus, the mediator explains 85% of the variation of the effect of X on Y. Using sample estimates, σ_c^2 is estimated as 0.29 and $\sigma_{c'}^2$ as 0.058; therefore, the mediator explains 80% of the variation of the effect of X on Y.

Example Using an Existing Data Set

We next apply our estimation method for random-effects models to an actual data set. Korchmaros and Kenny (2001) examined the mediation of genetic relatedness on willingness to help by emotional closeness. That is, the decision to help kin is mediated by feelings of closeness. Korchmaros and Kenny (2002) followed up this study with an investigation of the mediation of the effect of genetic relatedness on emotional closeness. It seemed very plausible that perceived similarity was a possible mediator. Korchmaros and Kenny (2002) asked persons to list their

[2] Actually the residual file created by HLM5 (Raudenbush et al., 2000) contains residual coefficients, the coefficients minus the "average" coefficient. Because we seek to compute a covariance between two sets of coefficients and covariances subtract off the mean, this is not a problem.

family members by name and to report each family member's relationship to them (e.g., mother, grandfather, or stepsister). Degree of genetic relatedness or proportion of genes identical by descent was inferred from this information (e.g., .50 for parents, .25 for grandparents, and .00 for stepsiblings). Ten family members were selected from each person's list of family members. Persons then rated each of their 10 family members on emotional closeness and similarity on 7-point scales. The data set consisted of 72 upper level units (i.e., persons) each with 10 lower level observations (i.e., family members) per variable. Thus, there were 2,160 observations in total, 720 per variable.

In this data set the putative causal variable is genetic relatedness, the mediator is perceived similarity, and the outcome variable is emotional closeness. Possibly, the effect of genetic relatedness on perceived similarity varies by person. Some people may spend more time with their kin than others spend with their kin, and as a consequence, these people become more similar to them, variation that is captured by σ_a^2. One could also imagine that the effect of similarity on emotional closeness might vary by person. Some persons may be more inclined to be attracted to similar others, whereas others may be less attracted because they feel they are similar because of undesirable factors. This variation would be captured by σ_b^2. For persons who have a large b (i.e., similarity lies in desirable traits; therefore, similarity leads to emotional closeness), it seems reasonable to expect that they would spend more time with their kin and so their a parameter would also be large. By the same token, for those whose b path was small, we would expect that their a path would also be small. Thus, it seems plausible that σ_{ab} would be positive.

A random effects mediational model was estimated using HLM5. The results are presented in Table 2. First, the unmediated effect of genetic relatedness on emotional closeness—path c—was estimated and equaled 5.923, $t(71) = 19.199$, $p < .001$. On average, emotional closeness increased 5.923 points on a 7-point scale for every 1-unit change in genetic relatedness (i.e., a change from no genetic relationship to perfect genetic relationship as is found in monozygotic [identical] twins). The variance in this path is 1.386, $\chi^2(71, N = 720) = 95.388$, $p = .028$, which indicates that the effect of genetic relatedness on emotional closeness varied by Level 2 unit or participant, a random effect.

Second, the effect of genetic relatedness on perceived similarity—path a—was estimated, and it equaled 3.643, $t(71) = 9.405$, $p < .001$. On average, perceived similarity increased 3.643 points on a 7-point scale for every 1-unit change in genetic relatedness. There was variation in the a paths of 2.919, $\chi^2(71, N = 720) = 100.430$, $p = .012$, indicating a random effect. As in the previous example, while estimating the effect of the putative causal variable on the mediator (in this case, genetic relatedness and perceived similarity, respectively), we used HLM5 to create a file containing the OLS estimates of the Level 1 path a coefficients for each individual).

Next, the effects of perceived similarity and genetic relatedness on emotional closeness—paths b and c', respectively—were estimated. While estimating these effects, we used HLM5 to create a file containing the OLS estimates of the Level 1 path b and c' coefficients. Perceived similarity had a strong effect on emotional closeness, path b, 0.491, $t(71) = 12.799$, $p < .001$. On average, for every 1-unit increase in perceived similarity on a 7-point scale, there was a 0.491 point increase in emotional closeness, which was also measured on a 7-point scale. There was variation in the b paths, 0.061, $\chi^2(71, N = 720) = 184{,}776$, $p < .001$, indicating a random effect. The direct effect

Table 2
Estimates for the Korchmaros and Kenny (2002) Example Estimated by HLM5 (Raudenbush et al., 2000)

Parameter	Sample (*SE*)
Paths	
c	5.923 (.309)
c (inferred)	6.054[a]
a	3.643 (.387)
b	0.491 (.038)
c'	3.918 (.319)
Variances and covariances	
σ_c^2	1.386
σ_c^2 (inferred)	2.099[b]
$\sigma_{(ab)}^2$	3.057[c]
σ_a^2	2.919
σ_b^2	0.061
$\sigma_{c'}^2$	3.260
σ_{ab}	0.348
$\sigma_{ac'}$	0.252
$\sigma_{bc'}$	–0.613

Note. The parameters $\sigma_{(ab)}^2$ as well as the inferred values of c and σ_c^2 are not free parameters but rather equal a function of the other parameters in the model. See the text for the formulas. Note that σ_{ab} refers to the covariance of a and b, whereas $\sigma_{(ab)}^2$ refers to variance of the product ab.
[a] Calculated using Equation 9.
[b] Calculated using Equation 12.
[c] Calculated using Equation 11.

of genetic relatedness on emotional closeness was reduced, though remained strong being equal to 3.918, $t(71) = 12.272$, $p < .001$.

The lower level path a_j and path b_j coefficients were correlated, $r = .262$, $p = .026$, with a covariance of 0.348 and a disattenuated correlation of .825. Also using the OLS paths, we estimated[3] $\sigma_{ac'}$ as 0.252 and $\sigma_{bc'}$ as −0.613, only the latter covariance being statistically significant.

Consider the decomposition of the effects of the mediational model. Using the standard single level model formula where the σ_{ab} is not considered, the total effect is 5.706, 3.918 + (3.643)(0.491), which underestimates *c,* the actual unmediated path coefficient (5.923) by 0.217. By this computation of the total effect, only 31% of the total effect, (5.706 − 3.918)/5.706, is mediated. When using the suggested random-effects formula where the σ_{ab} is considered, the total effect is 6.054, 3.918 + (3.643)(0.491) + 0.348, which somewhat overestimates the unmediated path coefficient by 0.131. By this computation of the total effect, 35% of the total effect, (6.054 − 3.918)/6.054, is mediated. These proportions of the total effect illustrate the importance of considering σ_{ab} when estimating the amount of mediation. However, note that these proportions may not characterize the population because they are calculated using sample estimates from a relatively small sample. The importance of considering σ_{ab} when estimating the amount of mediation is also illustrated in the variance of the mediated effects or $\sigma_{(ab)}^2$. This variance or $\sigma_{(ab)}^2$ equals 1.691 for the standard single-level model specification (Equation 10) and 3.057 when allowances for σ_{ab} are made (Equation 11).

Very surprisingly, introducing the mediator increases the variance in the effect of *X* on *Y*. Without having the mediator in the model, the variance of *c* is 1.386, but having the mediator in the equation leads to a variance of c' of 3.260. We suspect that the following might be happening. There is relatively little variation in the total effect of genetic relatedness and emotional closeness—for most people there is a strong relationship between genetic relatedness and emotional closeness. However, people may vary in terms of the reason why this relationship exists. For some people, the relationship between genetic relatedness and emotional closeness is completely mediated by similarity, whereas for others, other variables such as frequency of interaction might partially mediate the relationship. The negative correlation between *b* and c' is consistent with this explanation. When there is more mediation, leading to larger *b* paths, the c' path is smaller; and when there is less mediation, leading to smaller *b* paths, the c' path is larger. Additionally, the inferred variance of *c* is somewhat overestimated. This is possibly because of nonnormality (or see Footnote 3).

Conclusion

Multilevel models are being increasingly used to estimate models for both repeated measures and nested data. We consider the use of multilevel modeling to estimate mediational models in which there is lower level mediation, and all terms are random. We show that the standard formulas for indirect effects and their variance must be modified for this type of model. For each, the covariance between path *a* and *b* should be considered.

None of the standard methods of estimating multilevel models can estimate such a model. We suggest an ad hoc procedure that uses conventional methods. We present an example using simulated data, and we see that our method adequately captures the model's parameters. We also present an example using an existing data set. These examples illustrate that our method provides quite different and more accurate results than using conventional methods that have been used in single level models.

This method, though adequate, is less than ideal. First, it does not exactly reproduce the total effect as is estimated by the unmediated model. There are a few reasons for this inexact estimation. One reason is that maximum-likelihood estimation weights the estimates differently when the mediator is in the model and when it is not. Some of the difference between the coefficient of the *X*-to-*Y* path in the mediated model and the corresponding coefficient in the unmediated model is due to this difference in weighting rather than to mediation (Krull & MacKinnon, 1999). Note that a similar problem can arise for standard, single level mediational models when *X*, *M,* or *Y* are defined as latent variables or when logistic regressions are run. Because of maximum-likelihood estimation, the overall *X*-to-*Y* effect can change when the mediator *M* is included in the model.

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[3] Because *b* and c' are estimated from the same equation, they contain correlated sampling error that is ignored in the OLS estimation method of the covariance. The presence of this correlated sampling error likely biases the estimate of $\sigma_{bc'}$.

A second limitation of the proposed procedure is that we use OLS estimation to calculate σ_{ab} and do not weight *a* and *b* by their statistical precision. An ideal estimator of the covariance would take the statistical precision of *a* and *b* into account. Additionally, although we do provide for a statistical test for σ_{ab} using the correlation of the estimates of *a* and *b,* we do not directly estimate its standard error.

Finally, the estimation method that we have developed requires the assumption that *a, b,* and *c′* have a joint multivariate normal distribution. Although this assumption is standard in multilevel modeling, as mentioned previously, the violation of the assumption might be more serious here. This is because normality is assumed to identify a model.

We view our method as only an interim method, and we expect that a single step estimation method will be developed to replace our piecemeal approach. Ideally, a computer program would simultaneously estimate several multilevel models (one for *M* and one for *Y*) and allow for paths between outcome variables (from *M* to *Y*). With these programs, it would be possible to simultaneously estimate the Level 1 *a* and *b* coefficients and also to estimate σ_{ab}. It would be even better if the program would have an option for nonnormal distributions of *a* and *b*. Given that structural equation modeling software is beginning to include multilevel capabilities (e.g., LISREL 8 [Jöreskog & Sörbom, 1996]; EQS [Bentler, 1995]; Mplus [Muthén & Muthén, 2002]; Mx [Neale, 2002]), and multilevel software is beginning to add structural equation modeling capabilities (HLM5), we expect that single-step estimation methods will soon be available.

There are additional limits in our approach. First, we make all of the usual assumptions of multilevel modeling. One standard assumption of that approach is that the random effects have a multivariate normal distribution. Some of the formulas are based on the assumption of multivariate normality. However, we need not make that assumption because we can compute *c* and σ_c^2 directly. We have assumed that *a* and *b* have a bivariate normal distribution. Equations 9, 11, and 12 would be much more complex if we allow the distributions of *a* and *b* to be nonnormal. Because it is already standard practice to assume normal distributions and because the formulas are already very complicated, we do not consider nonnormality in this article. A recent article by Shrout and Bolger (2002) used bootstrapping to estimate and to take account of nonnormality in the sampling distribution of *ab* in a fixed-effects mediational model. Although it would be considerably more difficult to implement, it is possible that this or some similar approach may be useful in tackling nonnormality in the random-effects case.

Second, we assume that the mediational model is correctly specified. A mediational model is a causal model. Ideally the variable *X* is a manipulated variable, and consequently, we know that if there is a statistical association, then *X* causes *M* and *Y,* and not vice versa. The variable *M* is not manipulated and so the assumption that *M* causes *Y* is more problematic. Both substantive theory and research design (e.g., measuring *M* before *Y*) should be used to justify the causal direction. One key assumption is that there is no measurement error in either *M* or *X.* If there were measurement error in either of these variables, different methods would have to be used (Raudenbush & Sampson, 1999).

A statistical mediational analysis never establishes or proves mediation. Mediation occurs when a putative causal variable causes the putative mediator, which causes an outcome. Causation is a logical, theoretical, and experimental issue. A statistical analysis by itself cannot prove causation and, consequently, cannot prove mediation. For instance, seemingly credible estimates can often be obtained if *Y* is treated as the mediator and *M* is treated as the outcome. Like other causal models, a mediational analysis can establish that the model is false; however, it cannot ever prove that it is true.

We do believe that studying mediation with a multilevel context affords a much greater understanding of the process than a single level analysis. First and foremost, we can test to see if the mediation is the same for all upper level units. If there is no variation, then we gain confidence that the process is universal. Second, if we find that the mediation varies by upper level unit, then we can see if that variation is meaningful theoretically. So, for instance, if some people (or groups in the case where groups are the Level 2 unit) show the mediation and others do not, then we can investigate why we get mediation for some people and not for others. In this way we can probe theories in greater detail. However, some methodologists might question interpreting mediation as a causal effect when that mediational effect varies randomly.

Third, we have considered only lower level mediation. However, upper level mediation is considered in other articles (Krull & MacKinnon, 1999; Raudenbush & Sampson, 1999).

Multilevel models are now becoming common in

the social and behavioral sciences. These models allow researchers to study important social processes such as how intimate relationships affect the course of a person's health and psychological well-being (e.g., Bolger, Zuckerman, & Kessler, 2000) and how social-structural variables affect the likelihood of individual level victimization (Sampson, Raudenbush, & Earls, 1997). We hope that by providing a method of assessing mediation in multilevel models, their value to researchers will be substantially increased.

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Call for Nominations

The Publications and Communications (P&C) Board has opened nominations for the editorships of ***Comparative Psychology, Experimental and Clinical Psychopharmacology, Journal of Abnormal Psychology, Journal of Counseling Psychology,*** and ***JEP: Human Perception and Performance*** for the years 2006–2011. Meredith J. West, PhD, Warren K. Bickel, PhD, Timothy B. Baker, PhD, Jo-Ida C. Hansen, PhD, and David A. Rosenbaum, PhD, respectively, are the incumbent editors.

Candidates should be members of APA and should be available to start receiving manuscripts in early 2005 to prepare for issues published in 2006. Please note that the P&C Board encourages participation by members of underrepresented groups in the publication process and would particularly welcome such nominees. Self-nominations also are encouraged.

Search chairs have been appointed as follows:

- ***Comparative Psychology,*** Joseph J. Campos, PhD
- ***Experimental and Clinical Psychopharmacology,*** Linda P. Spear, PhD
- ***Journal of Abnormal Psychology,*** Mark Appelbaum, PhD, and David C. Funder, PhD
- ***Journal of Counseling Psychology,*** Susan H. McDaniel, PhD, and William C. Howell, PhD
- ***JEP: Human Perception and Performance,*** Randi C. Martin, PhD

To nominate candidates, prepare a statement of one page or less in support of each candidate. Address all nominations to the appropriate search committee at the following address:

Karen Sellman, P&C Board Search Liaison
Room 2004
American Psychological Association
750 First Street, NE
Washington, DC 20002-4242

The first review of nominations will begin December 8, 2003. The deadline for accepting nominations is **December 15, 2003.**