DOI 10.25336/csp29397(suppl)
Canadian Studies in Population 45
no. 3–4 (2018), p. 179–87

Supplement to review essay on Rex B. Kline's *Principles and Practice of Structural Equation Modeling*

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This supplement provides contextualizing discussions for several sections of the review essay, and ends by listing editorial corrections to Kline's text.[1]

Section 1: Causal structures versus regression equations

Finding causal action: Does real "isolation" (p. 27) require real causal isolation? Is "independence of residuals and predictors" (p. 27) founded in real causal separateness? Kline says regression equations make "less sense" if they misrepresent the causal world (p. 27) but why only "less sense" rather than SEM-nonsense? Are regression predictions likely to hold if some regression-predictors happen to be effects rather than causes? What is the nature of explanation and explained variance (p. 29) if the supposed-explanatory variables are not causes? What if not causal actions account for why regression estimates change with introduction of control variables (p. 32)? Kline describes the "statistical and conceptual assumptions of regression" (p. 33) as including that "*there are no causal effects among the predictors* (*i.e., there is a single equation*)" (p. 34; emphasis in the original). This is false. Regression does not *assume* there are no causal effects among the predictors, even though it does not estimate potential effects among the predictors in one equation. (A regression equation paralleling Equation 5 would acknowledge and adjust for the covariance between the two causes, but would leave unspecified whether the covariance arose from either of the causes influencing the other.) What produces bias in regression estimates (p. 35) if not misrepresentation of worldly effects? Isn't a "serious specification error" (p. 35) a causal error? Kline downplays the causal basis of several matters by repeatedly presenting how the numbers and equations work out, rather than attempting to explicate how *proper* representations and *mis*-representations of the world's causal structuring lead to the statistics working out as they do. The overlap in Kline's Venn diagram (p. 40) could be usefully connected to the covariance term in essay Equation 7 above, but his couching this in a discussion of part- and partial correlations and regression R^2 values (p. 39–41) disconnects it from structural equation models. (A similar comment applies to Equations 2.13 and 2.14.)

Kline's timid differentiation between SEM's causal-focus and regression's causal-indifference is sprinkled throughout the text and appears in many guises. For instance, Kline's example of left-out error variables (p. 36) would be easier to understand if the substantial correlations were characterized as originating in overlooked causal connections, and if "relevant predictors" (p. 36) had their relevance grounded in causal actions, rather than leaving the foundations of relevance unspecified—or, worse yet, permitting readers to incorrectly presume that higher correlation constitutes or justifies the relevance of a "predictor." For another example, "Suppression" (p. 36–37) naturally and easily meshes with the causal understanding that some effects may counteract other effects. Isn't "suppression" just another name for counteracting causal actions? Kline could not entirely avoid appealing to "indirect effects" as a foundation of suppression—though he does manage to delay it until the section's second-last sentence (p. 37).

1. Cited references are listed at the end of the Review essay.

Additional connections appear in the discussion of indicators in factor analysis, where Kline says "Common variance is shared among the indicators and is a basis for observed covariances among them" (p. 190). Actually, a latent factor functions like the common cause in Figure 1c, and gives each indicator its own variance (via essay Equation 3), and produces covariance among each pair of indicators though causal actions (via essay Equation 10). There is no sharing of variance—each indicator has its own. And consider Chapter 13 on Confirmatory Factor Analysis Models (CFA models). According to Mulaik, in doing CFA "the researcher begins with a conception of a set of latent exogenous ***causal*** variables having specified ***effects*** on a set of endogenous manifest 'indicator variables'" (2009: 219; emphasis added). In factor analysis, the effects of latents on their indicators are historically called loadings or pattern coefficients, and Kline says, "Pattern coefficients are interpreted as regression coefficients" (p. 301)—where regression's causal indifference is clearly inconsistent with Mulaik's emphasis on cause! And consider "reification": it would indeed be an error to claim an underlying latent factor "*must* correspond" (p. 300; emphasis in the original) to some real thing because a model can be wrong; but it is not an error, and actually is SEM-positive, to strive to make SEM latent variables correspond to real worldly entities. It is not just that models are "most meaningful" (p. 306) when they are consistent with the data provided by the causal world—they lose meaning if the model's structure fails to correspond to the world's structure. And causal actions explain why different model coefficients can have nearly identical modification indices (p. 312)—this happens when two currently omitted coefficients have nearly identical causal implications for the covariances between the observed variables. And notice that Chapter 10 considers "Structural Regression Models," as if the latent-level structures are regression, not causal, structures. See page 232, where the effect in a path model striving for causal correctness is supposed to be "interpreted exactly as a regression coefficient," despite regression not striving for causal properness.

Similarly, in Chapter 14 we read: "path coefficients are interpreted for SR models as ***regression coefficients*** between factors. Total ***effects*** between factors can be decomposed into direct and total indirect ***effects***, just as in path analysis" (p. 340; emphasis added). Are regression coefficients really effects? Kline stumbles repeatedly over whether or not the latent levels of structural equation models strive for causal standing.

Chapter 17 on interaction and multilevel modeling begins with regression and yet causal effects soon appear in the text and section titles (p. 427, 431). By page 432 we are told that "just as a mediational model is a causal model, so too is a model of moderation"—but the location of the transition from regression to causal action was left a mystery. Kline's artificial data illustrating this chapter's introductory "regressions" (p. 424–29) actually has a known causal foundation—one that is not reported, and is sloughed over. Then, consider moderated mediation, where the "interactive *effect* is represented in the figure by the *regression* of *M* on *X*, *W*, and *XW*" (p. 434; emphasis added). Does the estimate really constitute an effect if the equation is not causal and merely a regression? The delay in introducing required causal action robs Kline of yet another opportunity to instruct his readers on the necessity of attending to causes *before* specifying interactions or multilevel models, and robs him of the opportunity to instruct readers to check causal specifications both before and after running models that contain interactions or multilevel effects.

Section 2: Reciprocal effects

Here is a difficulty produced by Kline's omission of reciprocal effects. We are told that "within-time associations in panel models are typically specified as unanalyzed" (p. 139), and Figure 6.8b complies with the absence of within-time causal arrows. Unfortunately, this results in a seemingly unnoticed logical difficulty. Consider just the *X* row and *M* row of Figure 6.8b (p. 140), and see

the absence of an effect of X_{12} on M_{12} and the presence of an indirect effect *ec'* connecting X_{11} to M_{13}. The model precludes *X* from influencing *M* at any specific time, yet permits *X* to effect *M* by skipping from some earlier time to some later time. It is logically awkward to claim that *X* does not cause *M* at any specific time while simultaneously claiming *X*'s causal actions somehow jump to causing *M* at a later time. The past is influencing the future without ever going through the present? How small (but not infinitely small) time frames for "the-present" connect "the-past" to "the-future" is a matter well worth considering, especially since SEmodels need ***not*** be confined to modelling causal actions occurring instantaneously at the indicator-observation times (Hayduk 1985, 1996).

Section 3: Separating observed from latent variable

Here are some "unusual" features of Chapters 6 and 7, originating in their focus on observed-variable path models, despite almost all measured variables containing some measurement error. The rarity of error-free variables probably explains why the only real example in Chapters 6 and 7 is a seven-line discussion on page 159, and even this example ignores likely measurement errors in the relevant modelled variables. The points in these chapters were obviously made with fictional examples, and could have been made as easily, and more appropriately, using fictional latent-variable examples. The artificiality of purely "Observed-Variable (Path) Models" will be obvious to anyone noticing that Chapter 7 begins by reporting "two general…requirements" for such models, the second requirement being that "Every ***latent*** variable…must be assigned a scale" (p. 145; emphasis added). Inserting required features for latent variables in observed-variable models is probably a consequence of Kline's chapter reorganization, but at least this moves latents toward where they should have been all along.

Section 4: Some nit-picking concerns connected to Chapter 8

To my knowledge, there has been no demonstration of an appropriate way to address the multiple testings of independencies (and manufacturing of multiple dependencies by controlling for colliders) derived from DAG investigations (e.g., p. 172). Another feature for which I know of no current resolution concerns the "basis set" of non-redundant conditional independencies (p. 173). Independencies beyond the minimal set are redundant if the independencies hold, but if one or more do not hold, that raises the possibility of dredging diagnostic assistance from the initially declared-redundant set. DAGs also await ways of appropriately assessing fixed model coefficient values, and differentiating between properly and improperly fixed/specified coefficients. Kline cannot be held responsible for these loose ends in the DAG literature, but a caution should at least report that various extensions remain to be pursued. What Kline can be faulted for is his failure to integrate the new DAG testing precision with his discussions of testing in Chapters 11 and 12.

Section 5: A passel of technical teasers connected to deficient testing

Regarding page 60, paragraph 2, second line: is the null hypothesis of the *model* test really a hypothesis that is "false by the degree indicated" by some index? I vote no, it is not.

Regarding page 239, point 2: the error rate might be high (for some unspecified test, for some unspecified conditions, and without reference), but what kind of error rate would be typical for reasonable conditions?

Regarding page 239, fourth-last line: if the global test indicates model misspecification, the coefficient estimates are likely to be biased. Would you want to pay more attention to the biased estimates?

What constitutes the "population" in model testing? In χ^2 testing the Σ matrix, namely the model-and-estimate implied covariance matrix is employed as the population. This may or may not correspond to the "worldly population" that provided the data, and it is not dependent upon the data coming from a random sample of any "worldly population." Several of Kline's wordings about these distinct population are sufficiently ambiguous as to invite misinterpretation (p. 235, 236, 265), as well as confusion about the nature of the "multivariate normality" and the hypothetical "random sample" (p. 270) relevant for maximum likelihood estimation and χ^2.

The χ^2-difference test is justified only if the "more complex of the two models compared" (p. 281) actually fits the data, and not merely that it should "fit the data reasonably well" (p. 281, and see p. 306).

"Given two models with similar fit to the data, the simpler model is preferred, assuming that the simpler model is theoretically plausible" (p. 128)—so the model is "preferred" even if both models are similarly highly **in**consistent with the data!

A perfectly fitting model may not be perfect because seriously misspecified models can fit perfectly (Hayduk 2014a). And there is no known way to calibrate the amount of ill fit to the seriousness of the corresponding model causal misspecification—not even with a noncentrality parameter. So what is the nature of the "perfection," and what makes some amount of ill-fit "acceptable" when Kline says, "what is considered 'acceptable' departure from perfection is related to the value of the noncentrality parameter for the χ^2" (p. 60)? The size of a claimed-acceptable noncentrality is actually an index of the degree of shamefulness in the researcher's disregard for evidence. Zero-noncentrality with zero-shamefulness is as easy as using the ordinary χ^2.

Kline cites references that discuss ways SE model-testing differs from coefficient-testing (Hayduk 1996, 2014a; Hayduk and Glaser 2000), so it is surprising that his discussion of "Cognitive errors in significance testing" (p. 55–56) fails to distinguish between model tests and coefficient tests. I would have hoped that Kline would see that the testing differences also make it incorrect to treat confidence intervals around fit indices the same as confidence intervals around coefficient estimates. The difference between model adequacy and model fit means that if model "respecification is driven entirely by empirical criteria such as statistical significance, the researcher should worry—a lot, actually—about" *model misspecification* and not just "capitalization on chance" (p. 283). The issue that killed "automatic modification" was not capitalization on chance (p. 283); it was that ill-fit is prone to being inappropriately reduced by inclusion of additional *mis*specified coefficients matching *real* (not merely chance) residual covariances.

Overall, Kline could have avoided multiple imprecisions by acknowledging SEM's commitment to seeking causal-theory, and emphasizing attention to evidence signaling the causal solidity of some models and causal dubiousness of others.

Rex Kline was one of the authors of a recent American Psychological Association publication which provided new reporting standards for structural equation models (see Table 7 in Appelbaum et al. 2018). I provided the following two replacements for sections of these standards to the SEMNET listserv (26 January 2018). Interested readers might see SEMNET for Kline's response.

> Replace the first sentence in the **Abstract** section with:
>
> *Report a test of whether or not the model is consistent with the data, and the implications of informative localized-ill-fit.*
>
> Change the title from Model fit to **Model Testing** and replace the first bullet point in this section with:
>
> *Report the most powerful model test—usually chi-square (possibly adjusted) with its degrees of freedom and probability. All fit indices (even with values commonly reported as acceptable) are deficient at detecting model misspecification and hence cannot replace or displace the evidence provided by model testing.*

Section 6: Improving Kline's Figure 17c example

The model-implied consequences of an intervention at X can be investigated by observing how the terms on the right of essay Equation 19 change in response to the postulated intervention. The model claims the effect of an X intervention on Y will be the difference between the Y value provided by this equation before the intervention and the Y value after the postulated intervention. Any right-hand terms left unchanged by, held constant by, or precluded by the imagined/postulated intervention will contribute equally to Y-before and Y-after intervention, and hence will not contribute to changing Y. The terms that are changed as a result of a postulated intervention document the components of the causal system contributing to changing Y's value.

Kline postulates an intervention in which X=0 constitutes a control condition and X=1 denotes provision of a treatment (p. 435). (Readers familiar with Pearl (2000) will recognize the parallel to Pearl's $do(x)$.) Providing the treatment (namely, shifting X's value from 0 to 1) in a world structured like essay Figure 4 would have two basic ways of influencing Y—namely, directly (with whatever complications moderation/interaction implies) and indirectly through M (also with whatever complications moderation/interaction implies). To minimize space, we will focus on understanding/interpreting the *indirect* effect on Y of an intervention changing X from 0 to1. The focus on indirectness requires retaining terms containing both X and X's β_1 effect on M, but we must consider all the terms in Equation 19, which we duplicate here for convenience.

$$Y = \theta_0 + \boldsymbol{\theta_1 X} + \{\theta_2\beta_0 + \boldsymbol{\theta_2\beta_1 X} + \theta_2 e_M\} + \{\boldsymbol{\theta_3 X \beta_0} + \boldsymbol{\theta_3 X \beta_1 X} + \boldsymbol{\theta_3 X e_M}\} + e_Y \qquad (19)$$

Now consider each right-hand term.

θ_0 is the intercept corresponding to the net impact of variables not currently in the Y equation, which our "postulated intervention" did not address. This term forces clarification of our postulated intervention by demanding we add the assumption/presumption that the excluded causes of Y remain constant and hence do not contribute to changing Y as the intervention changes X from 0 to 1.

$\theta_1 X$ is the direct effect of X on Y but we are seeking effects connected to, or functioning through variable/mechanism M, so we must clearly assert that our postulated intervention specifies this effect has somehow been rendered inoperative.

$\theta_2 \beta_0$ neither the θ_2 effect nor the β_0 intercept are altered by the postulated change in X from 0 to 1, so this term remains constant, and hence does not contribute to a change in Y by the intervention of interest.

$\boldsymbol{\theta_2 \beta_1 X}$ involves the indirect pathway due to the presence of both β_1 and θ_2, and this term's contribution to Y will change from $\theta_2 \beta_1(0)$ to $\theta_2 \beta_1(1)$ as the treatment changes X from 0 to 1, so this term contributes to changing Y by the intervention of interest.

$\theta_2 e_M$ our postulated X intervention and the causal action working through M did not change either θ_2 (the basic effectiveness of M) or any unknown sources of the mediator-moderator M (namely, e_M), so this term remains constant. This term instructs us that it would be possible to trace the consequences of an intervention changing the causal effectiveness of one variable at influencing another (like changing θ_2) rather than seeking the consequences of changing the value of a variable (like X), but our particular intervention did not introduce such a change.

$\theta_3 X \beta_0$ This term will prove to be contentious, and I will return to it momentarily.

$\boldsymbol{\theta_3 X \beta_1 X}$ involves an indirect effect of X through M due to β_1, and this term changes due to our postulated change of X from 0 to 1.

$\theta_3 X e_M$ This term is also contentious, and I will return to it momentarily.

e_Y is rendered constant by again increasing the precision of our postulation, namely by adding the assumption/presumption that the intervention of interest does not alter any of the unknown causes of Y.

Now we return to Kline and notice the third Equation in 17.8 (p. 436) reports what Kline calls the Natural Indirect Effect (NIE) of an $X=0$ to $X=1$ intervention in this model as

$$NIE = (\theta_2 + \theta_3)\,\beta_1 \tag{S1}$$

Combining the two terms containing β_1 highlighted above, and writing this as an effect (namely as an effect appearing in front of the X variable) results in something similar but not quite identical to Kline's NIE equation.

$$[\text{indirect effect of}]\ X = [(\theta_2 + \theta_3 X)\beta_1]X \tag{S2}$$

$$[\text{indirect effect}] = (\theta_2 + \theta_3 X)\beta_1 \tag{S3}$$

The difference is the "extra" X inside the parentheses. If X moves from 0 to 1, both Kline's Natural Indirect Effect coefficient (Equation S1) and our indirect effect coefficient (Equation S3) report exactly the same change in Y's value, because both provide zero contribution when $X = 0$ and contribute $(\theta_1 + \theta_3)\,\beta_1$ when $X = 1$, because the θ_3 is left unchanged by being multiplied by the "extra" X of 1 in Equations S2 and S3. A difference would arise if we had been considering a progressive X treatment, where a subject might receive partial treatment or multiple doses of the treatment, so X might take values like 0, 0.5, 1, or 1.5. If this is possible, our indirect effect calculation differs from Kline's because the "extra" X in our formula alters the θ_3 portion of the effect (Equation S2 or S3). This cautions that Kline's calculation of NIE applies only to dichotomous X variables scaled 0–1, not to X variables having non-dichotomous scalings, and not even to dichotomous interventions scored 1–2 rather than 0–1.

Now consider the terms skipped above. The value of the $\theta_3 X e_M$ term, and hence Y, would change as X switches from 0 to 1 due to the postulated intervention if both θ_3 and e_M are non-zero. The disturbances or errors in equations are routinely presumed to average zero, but this equation contains each particular individual's precise error/disturbance value not the average of multiple cases' errors or disturbances (just as X and Y in the equation refer to specific, not averaged, values). This has two consequences. First Kline should have reported either that his NIE calculation (p. 436) presumes or assumes he is seeking the indirect effect for a case having precisely a zero disturbance/error, or that his calculation acknowledges the indirect effect will differ between cases and he is seeking only the average of those cases' indirect effects. Second, Kline could have reported the possibility of assessing the extent to which variations in X's indirect effect on Y originates in variations in M's disturbance/error variable. The variance of M's disturbance/error is routinely estimated in structural equation models and hence the standard deviation of the disturbance/error is available. Calculating the magnitude of $\theta_3 X e_M$ for error values one or two standard deviations above and below zero error would report the fluctuations in the postulated indirect effect likely to arise from the 0–1 change in X combining with modest or nearly-extreme disturbance/error values.

The consequences of the other skipped term, namely $\theta_3 X \beta_0$, are more awkward but intriguing. Here too, if X changes from 0 to 1, and if both θ_3 and β_0 are non-zero, this term alters Y's value in a way which involves M because β_0 is part of M's equation. Above it was relatively straight forward to presume $e_M = 0$, or equals zero upon averaging, to eliminate the term, but a corresponding presumption of $\beta_0 = 0$ would be dubious. The β_0 intercept is not an average. It possesses only a single value that is included/operative for each and every case, and like all intercepts its value depends on the scale and effects of all the variables included in the equation and even the scales and effects of excluded variables.

In what sense are the $\theta_3 X \beta_0$ and $\theta_3 X e_M$ terms "effects"? Neither term contains an "effect" if by effect we mean a coefficient depicting a regression-style slope, but both terms originate in M acting as a moderator variable that influences, changes, or adjusts the causal effect of variable X as it proceeds to impact Y. The term containing e_M describes effect-variations explicitly produced though the actions of unobserved causes that differ between individuals. The term containing β_0 describes effect-variations produced through features that the model does not permit to vary between individuals, and that are partially controlled by both the zero-point and scale-units of the M variable (which are likely arbitrary).

And in what sense are the $\theta_3 X \beta_0$ and $\theta_3 X e_M$ terms "indirect" or "direct"? Tracing these terms back to Equation 19 finds that these originated in the multiplicative term connecting X and M, namely $\theta_3 X M$, and that the changes in these terms result from M responding to the postulated 0–1 change in X. Thus these terms clearly implicate the mediator/moderator variable, and can lay claim to being indirect consequences of an X intervention working though the mediator/moderator variable M. The $\theta_3 X M$ term does not indicate whether M is modifying X's effect, or if X is modifying M's effect (namely whether the curvature in Kline's Figure 17.2 should be considered as changing slopes paralleling the X axis, or the other axis). Viewing this term as X conditioning/altering M's effect makes the term seem like an indirect effect working through M. Viewing the term as M conditioning/altering X's effect makes the term seem more like a direct effect. Kline, following Valeri and VanderWeele (2013), includes the term as part of what they call the "natural direct effect" NDE (p. 435–436, Equation 17.7) but that placement is debatable, and is better viewed as open to the researcher's preference for how this term would be most usefully considered in their specific context.

These observations warn against becoming attached to specific definitions of features like NIE and NDE (natural indirect and direct effects; p. 435, Equation 17.8) because these may not correspond to the causal actions a researcher wishes to investigate in their particular model. Indeed, Kline's definitions for these terms apply only when the model is structured exactly as in Figure 4, and only when specific features of the postulated intervention are assumed (remember the demanded 0–1 coding of X, the possibility of non-dichotomous X values, the arbitrary scale for β_0, and the required 0 for e_M). Introducing additional model variables, and/or additional interactions or nonlinearities will change the equation for Y and/or the equations for the variables "replaced" in Y's equation (as M's equation replaced M in Y's equation to obtain Equation 19 above). Such changes stymie any routine definition of entities such as NIE because they introduce terms not addressed by NIE.

Mean-centering M and Y (by subtracting the means from the appropriate data values) would set the intercept terms to zero, and hence eliminate some terms and alter the appropriate definitions, but would require countervailing un-centering if the interpretations were to be applied to variables having their original scales. Mean-centering an intervention like X would likely introduce confusion, because a 0 would no longer correspond to absence of the treatment, or 1 to presence of the treatment.

Editorial Corrections and Other Small Improvements

1) Page xiii: the Chapter 6 and 7 titles should both contain or both omit the hyphen between Observed and Variables.
2) I fail to understand why Chapter 2 begins, opposite to usual traditions, by using upper-case B for unstandardized effects and lower-case b for standardized effects. This might be helpful if LISREL notation was to be used routinely, but that seems not to be the case.
3) The phrase "controlling for their intercorrelation" two lines above the equation on the middle of page 30 is inaccurate—with R^2 there is no controlling.
4) Equation 2.12 is not a regression equation. The equation should include variables on the right side for it to be a regression equation.
5) The right sides of equations 2.12 and 2.13 are identical and cannot correspond to the different kinds of entities on the left of these equations.
6) Page 80, second line: should read "final 1–7" not 0–7.
7) Table 4.2 would be easier to read if the rescaled variances were presented in italics.
8) In both Equations 4.8 and 4.9, the term "$r_{XX} \times r_{XX}$" should read "$r_{XX} \times r_{YY}$".
9) Page 133, first sentence: should be reworded to claim *only* no direct causal connection between X and Y because there is in fact some other causal connection between the variables.
10) Page 133, footnote 5: should end with "but this practice is not consistent with SEM".
11) The discussion of causal loops would benefit from references clarifying how loops function (Hayduk 1987: Chapter 8) and how loops alter effects that touch any variable in a loop (Hayduk 1996: Chapter 3).
12) Page 151: The reference to "in the next chapter" at the end of the middle paragraph would be more helpful if it pointed specifically to "Instrumental Variables" (p. 180).
13) Page 161, last line of 1st paragraph: "excluded variables" should be "excluded effects", and some additional headings could clarify the implicit segmentation of Appendix 7.A.
14) Page 167, last line: Instead of "a back-door path that starts" it would be more accurate to say, "a back-door path between X and Y that starts".
15) Page 168, first line: Instead of "A back-door path may convey a spurious association between variables at either end, but never causation", it would more accurate to say, "A back-door path may convey a spurious association between the variables at the ends of the path, but not a direct or indirect effect between the variables at the ends of the path."
16) Page 168, second-last line of the 1st paragraph: The statement that the models "are equivalent" should instead clarify that the models are NOT causally equivalent (they contain contrasting causal effects) even though they imply equivalent-conditional-independencies.
17) Page 170, end of 1st paragraph: The claim that "multiple regression assumes no causal effects among the predictors" is incorrect. Multiple regression does not estimate/report effects among the predictors, but it does not assume these do not exist.
18) Page 170, paragraph 2, third-last line: Replace "where X, is specified to directly cause Y" with "where X_1 is specified to directly cause Y_1".
19) Page 175, end of second-last sentence in the 2nd paragraph: refers to X and Y but should refer to X_1 and Y_1.
20) Page 177, 3rd paragraph, last line: should end "(see Appendix 8.A)".
21) Page 180: The section on "Instrumental Variables" should clarify that the rules locate what the model claims as instrumental variables, but does not guarantee the corresponding worldly variables actually are instrumental variables (because the model may be misspecified). Farther on (page 182, paragraph 2), it is stated: "Exogenous variables make ideal instruments because by definition they are unrelated to all disturbances in the model." The world is not controlled by this definition, and hence a researcher's exogenous variables may or may not be unrelated to the modeled disturbances, and hence may or may not be acceptable instruments.
22) Page 189: "factor indeterminancy" should be "factor score indeterminacy".
23) Page 197, 3rd paragraph, first line: "affect" should be "effect".
24) Page 199, first line: Add the names of the Greek characters so this reads "(λ, lambda)" and "(φ, phi)".
25) Page 211: The term "matrix" which appears twice in the first two lines could be more helpfully described as a "list" or "vector".

26) Page 212, bottom paragraph, line 6: Delete the word "indirectly" because the effects of latents on the indicators that measure those latents are direct.
27) Page 217, first line. The word "unreliability" should be replaced with "invalidity".
28) Page 217, third last line in the first full paragraph: "improve factor measurement" should be "improve latent measurement" because the latent need not be a factor.
29) Page 223: 2nd paragraph, second line: "underlying factor" should read "underlying latent".
30) Page 224: Figure 10.7 is referred to as Figure 11.7 in the website material.
31) Page 227: There seems no reason to switch the placement of the ξ and η terms away from their usual LISREL location in Appendix Equation 10.6, and this matrix equation is missing an η from the left side.
32) Page 241: The formula producing the equation on this page should be provided.
33) Page 241, 3rd paragraph, third line: should read "(see Figures 7.5 and 11.1)".
34) Exercise 1 (pages 241 and 479) should incorporate the relevance of control/uncontrolled variables and the fact that this model fails significantly because both these features alter the permissible interpretations.
35) Page 261: An easy and generally applicable way to obtain start values for effects in complicated models can be obtained by making the square of the effect's start-value multiplied by the variance of the causal variable contribute a reasonable amount of variance into the dependent variable—paralleling the structure of the terms in essay Equation 7 above. Checking that the square of an effect estimate multiplied by the variance of the cause contributes a reasonable amount of variance into the dependent variable can sometimes provide useful direction for finding where a model is empirically underidentified (p. 157).
36) Page 273 just prior to Equation 12.4: The word "limit" is statistically incorrect and should be deleted or replaced.
37) Page 275: The H_0 near mid-page is missing a decimal, and should read "≥ .10".
38) Page 276: When readers encounter $\hat{\Delta}_M$ just prior to Equation 12.7, they would probably appreciate a reminder that this was defined in Equation 12.4 (p. 273).
39) Page 307, line 2: should read "failed at $p < .01$" because the model $p = .006$ is larger than .001.
40) Page 358: Figure 14.4 is inconsistent with the website model output (two indicators are switched and the 1.0 connected to Risk is differentially placed).
41) Page 417, above the two equations: "Factor variances and sizes" should be "Factor variances and sample sizes".
42) Page 438, second-last and third-last lines: should report how the feature is denoted, for example, as "the nonproduct factors A and B are all zero *which we denote as* $\sigma^2_{AB,A}=0=\sigma^2_{AB,B}$".
43) Pages 438–39: Just as an introductory sentence indicates that the Equations in 17.11 were obtained "by taking the products of the corresponding expressions" there should have been an introductory sentence indicating how the Equations in 17.12 are obtained. The statement might indicate the first equation in the set of Equations 17.12 corresponds to the first equation in the set of equations ending column-1 in Kenny and Judd 1984:210; and might note that the remaining equations in Kline's 17.12 parallel the first equation in the set with the last/covariance term repeatedly being dropped due to the assumed independence of error variables.
44) Page 439, the first line of text should read: where the term σ^2_{AB} *represents the variance of the product and the term* $\sigma^2_{A,B}$ represents *the square of* the covariance between factors A and B.
45) Page 439: the third line of text should read "factors A and B plus *the square of* their covariance".
46) Pages 452, 453, and 497: The year of the Hoyle and Isherwood reference should be 2013.
47) Page 455, last two lines: "have positive intercorrelations" should read "have substantial intercorrelations consistent with the signs of the indicators' loadings on the latent factor".
48) Page 456 first line: Delete "or negative".
49) Page 457 in the last bullet point: "ensure your readers" should either be "assure your readers" or change the sentence to read "to ensure that it is actually".
50) Page 497: Delete one of the duplicated journal titles for the Hoyle and Isherwood reference.
51) Page 507: The volume and pages for the Valeri and VanderWeele reference should read "18(2):137–50".
52) Page 526: The entry for "Model test statistics" should NOT include "fit indexes" because the indices are not tests.