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Competing Models

This document describes tests of competing models for the social phobia example in Chapter 11. I assume you are familiar with Mplus programming, instrumental variables (Chapter 6), and strategies for testing competing models (Chapter 7). The primary model for the social phobia example has two causal paths among the mediators, PSKILLS2 → NEGAPP2 and from PSKILLS2 → EXTERN2. The competing model adds reverse causal paths from NEGAPP2 → PSKILLS2 and from EXTERN2 → PSKILLS2 to the original model. I first consider competing model tests using FISEM. I then offer comments for competing model tests using LISEM.

FISEM TESTS OF COMPETING MODELS

As discussed in Chapter 6, to test for reciprocal causality between two variables, X and Y, one typically needs an instrumental variable for X and an instrumental variable for Y; otherwise, the model can be under-identified. For the causal relationship between PSKILLS2 and NEGAPP2 and the causal relationship between PSKILLS2 and EXTERN2, both variable pairs meet this requirement. The instrumental variable for PSKILLS2 is the baseline measure of perceived social skills, the instrumental variable for NEGAPP2 is the baseline measure of negative cognitive appraisals, and the instrumental variable for EXTERN2 is the baseline measure of external locus of control. Given this, I can add the two reciprocal causal paths to the original model without creating estimation problems. Using the syntax line from Table 11.1 from Chapter 11 that reads

```
PSKILLS2 ON TREAT HYPER SEX PSKILLS1 (p2 b4-b6) ;
```

I add the predictors NEGAPP2 and EXTERN2 to the line as follows:

```
PSKILLS2 ON TREAT HYPER SEX PSKILLS1 NEGAPP2 EXTERN2 (p2 b4-b6 p2a p2b) ;
```

which introduces the reciprocal causal designations. Note that I added two labels to make the number of predictors referenced after the ON keyword equal to the number of labels provided. If I had not used labels in the original statement (because they are optional), I would not have to add the new labels.

For the original model, the chi square statistic for model fit was 50.341 with df = 57 and a scaling correction factor of 1.0094. For the competing model, the chi square statistic for model fit was 47.954 with df = 55 and a scaling correction factor of 1.0101. If I perform a chi square difference test between the two models per Chapter 7 (using the correction factor in the program *Scaled chi sqr difference test* on my website), the chi square difference was 2.40, df = 2, p < 0.31, which is statistically non-significant; I cannot confidently conclude that the model with reciprocal causation fits better than the model without reciprocal causation.

If the traditional model chi square difference test is statistically non-significant, it follows that the MacCallum, Browne and Cai (2006) close fit difference test for the models as discussed in Chapter 7 also is statistically non-significant, so application of it is moot. When I applied the CFI test described in Chapter 7, the improvement in fit by the reciprocal causality model compared to the original model was zero, taking into account model parsimony. All signs at the global level point away from a model with reciprocal causation.

However, as noted in Chapter 7, I prefer not to rely exclusively on global, omnibus tests such as these, but also to dig deeper and examine the scenario at the level of path coefficients. Given there were only two additional, complementary paths, I examined the path coefficients for the outcome PSKILLS2 when both of new paths were included in the model. Here is the Mplus output:

	Estimate	S.E.	Est./S.E.	Two-Tailed P-Value
PSKILLS2 ON				
TREAT	1.331	0.134	9.936	0.000
HYPER	0.009	0.069	0.128	0.898
SEX	0.056	0.053	1.040	0.298
PSKILLS1	0.554	0.073	7.635	0.000
NEGAPP2	0.145	0.103	1.419	0.156
EXTERN2	-0.016	0.102	-0.162	0.872

Both of the reverse causality paths were statistically non-significant, which is consistent with a decision to exclude them. Note also that the direct effect of TREAT on PSKILLS2 remains strong even with their inclusion; in the original analysis, this path was 1.17 whereas in the reciprocal causality model, the path is 1.33. To be sure, the standard error of the TREAT to PSKILLS2 coefficient and its critical ratio is much lower than in the original analysis; in the original analysis the estimated standard error for TREAT was 0.05 and the critical ratio was 23.64. However, this result is primarily a consequence of making use of instrumental variables in conjunction with reverse causality. As I discussed in Chapter 7, doing so or using instrumental variables in conjunction with correlated disturbances can be sample size

demanding and tends to inflate standard errors. We only want to use the strategy in the way I am doing here if there is strong theoretical justification for doing so.

A critic might further argue that a different competing model I should consider is one that reverses the paths between PSKILLS2 → NEGAPP2 and PSKILLS2 → EXTERN2 without introducing reciprocal causality, i.e., the causal direction only goes the opposite way to what I originally thought. I would reject this suggestion because it is not consistent with past research or theory. When considering competing models, you are not at the whims of a semi-informed critic who, to quote the classic adage, "knows not of what they speak."

Implementation of the strategies discussed above for Bayesian FISEM use the same basic approach but you do not have access to the chi square difference test or the test of close difference. However, you have available the CFI test as well as the examination of the local path coefficients. As well, there are specialized model comparison methods you can use that make use of information theory via the Deviance Information Criterion (Ando, 2010). I discuss these in Chapter X of the main text.

LISEM TESTS OF COMPETING MODELS

Tests of competing models also can be pursued in LISEM frameworks, although the ease of and ability to do so often depends on the form of the competing model and the statistical method being used. For example, in Bollen's MIIV-SEM framework, it is as simple as changing the R syntax line in Table 11.11 from

```
PSKILLS2 ~ TREAT+PSKILLS1+HYPER+SEX
```

to

```
PSKILLS2 ~ TREAT+PSKILLS1+HYPER+SEX+NEGAPP2+EXTERN2
```

For OLS-based LISEM, the somewhat dated approach was to use two stage least squares but current recommendations generally favor a more complex maximum likelihood method. It turns out that this method is captured in Mplus through standard SEM programming, so one would simply isolate the piece of the full model one wants to focus on and conduct the analysis in Mplus in an LISEM spirit. Here is the syntax:

```
TITLE: EXAMPLE CHAPTER 11 FISEM ;
  DATA: FILE IS c:\mplus\ret\newchap11\chap11M.txt ;
   VARIABLE:
      NAMES ARE ID CR1 SPAI1 SPIN1 CR3 SPAI3 SPIN3
        NEGAPP2 PSKILLS2 EXTERN2 NEGAPP1 PSKILLS1 EXTERN1
        HYPER SEX TREAT ;
      USEVARIABLES ARE NEGAPP2 PSKILLS2 EXTERN2 NEGAPP1 PSKILLS1
```

```
      EXTERN1 HYPER SEX TREAT ;
  MISSING ARE ALL (-9999) ;
ANALYSIS:
  ESTIMATOR = MLR ;  !Robust maximum likelihood
MODEL:
PSKILLS2 ON TREAT HYPER SEX PSKILLS1 NEGAPP2 EXTERN2 ;
EXTERN2 ON TREAT HYPER SEX EXTERN1 PSKILLS2  ;
NEGAPP2 ON TREAT HYPER SEX NEGAPP1 PSKILLS2  ;
OUTPUT:
  SAMP STAND(STDYX) MOD(ALL 4) RESIDUAL CINTERVAL TECH4 ;
```

and here is the output from the analysis:

	Estimate	S.E.	Est./S.E.	Two-Tailed P-Value
PSKILLS2 ON				
TREAT	1.331	0.134	9.937	0.000
HYPER	0.009	0.069	0.128	0.898
SEX	0.056	0.053	1.040	0.298
PSKILLS1	0.554	0.073	7.635	0.000
NEGAPP2	0.145	0.103	1.419	0.156
EXTERN2	-0.016	0.102	-0.161	0.872

which are quite close to the results in the FISEM analysis. One would only want to move to this type of LISEM if the sample size was such that it precluded pursuing the full model in FISEM or if there were parts of the broader FISEM analysis that were problematic and that undermined its application.

For approaches to address reciprocal causality in quantile regression, see Muller (2019).