A robust path analysis of depression among Ukrainian residents of Kiev and Zhitomyr Oblasts after Chornobyl

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Contents

1 Acknowledgements

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2 Introduction

In this analysis we examine some plausible causal etiological paths of Depression, Anxiety, and PTSD among residents of Zhitomyr and Kiev Oblasts in the years since Chornobyl. We will focus on omnibus measures of fit, as well as statistically significant paths, broken down into direct, indirect, and total effects. We employ path analysis to allow us to find out which variables are mediating ones and which have direct effects. The path analysis permits us to decompose total into direct, indirect, and spurious effects. After a short introduction to path analysis,

we begin with an analysis of our depression models for men and women. We guard against selection bias by a random generation of phone numbers from a computer and the attachment of those phone numbers to the area codes provided by the telephone company.

3 Path analysis

3.1 Nomenclature

Although we may refer to these models as causal, they are really only models of association. Causality requires an invariable space-time relationship between two phenomena that may be likened to a logical and probabilistic chain of events, where an effect temporally follows a cause, when the two these phenomena are spatially contiguous to one another, conditional upon specified conditions affecting these phenomena. For the time being, we exclude matters of quantum entanglement as being beyond the scope of this analysis.

To determine that the relationship between these two phenomena may be causal, we would have to be able to conduct a controlled experiment to demonstrate that the cause is proximate, facilitating, necessary and/or sufficient for the effect to occur, given specific circumstances. Without such circumstances, we cannot know whether models are causal[3, 56-78]. In a sense, we are statistically analyzing what David Hume in his Enquiery Concerning Human Understanding (1748) referred to as an association and the models which we develop are to be construed only as reflections of a possible causal path.

3.2 Path effect specification

In path analysis, we endeavor to model reflections of a possible causal paths among variables. The coefficients in such a system are called path coefficients. Although some practitioners standardize these coefficients, we do not, lest we lose the sense of scale and mean location of the metric being used when interpreting the effects of different equations.

When all effects are in a regression model, the regression coefficients are called direct path coefficients. When a variable y intervenes between w and z, the indirect effect is computed by the product of the regression coefficients in each of the component paths from w to y and from y to z. The sum of all of the indirect paths plus the direct effect is called the total effect.

The *spurious or unmeasured effect* is that difference between the total effect and the zero-order effect (correlation if standardization is employed or regression coefficient if variables are not standardized) between the exogenous and the endogenous variable, where the zero-order correlation is the bivariate correlation between the exogenous and endogenous variable with 0 controls for other variables to hold them constant to partial out other effects [1, 359-360].

We use a robust path model, by controlling for the serial correlation across the waves by applying a cluster control of id across the waves of the study.

4 Model structure

Because we make the working assumption that variables are fixed effects, we rely on the submodel structural equation formulation of Joreskog and Sorbom for observed variables, except that we adopt Sorbom's formulation of mean structures.

If and only there are no feedback loops, our models will be simplified to

$$y = \alpha + \gamma x + \zeta \tag{1}$$

with ϕ = covariance matrix among observed variables [2, 9,136-137], [4, 210].

However, in the event that our model is nonrecursive, we rely on their formulation of it as

$$y = \alpha + \beta y + \gamma x + \zeta \tag{2}$$

where α is a px1 vector of constants, β is an pxp matrix of parameter estimates for those endogenous observed variables, ξ is a nxq matrix of exogenous observed variables, and ζ= px1 vector of equation errors, with n= number of observations.

The mean of the vector is

$$y = (I - \beta)^{-1}(\alpha + \gamma\kappa) \tag{3}$$

The mean of vector ξ is denoted by vector, κ, which has an order of nx1.

5 Assumptions

Because the building blocks of path analysis consist of covariance structure analysis and regression analysis, the assumptions of linear structural equation modeling are are essential to assure statistical conclusion validity. The uncorrelated errors assumption ($E(\xi\zeta) = 0$) is an essential assumption. According to this principle, the errors of the equations are uncorrelated with the explanatory variables in the model. Otherwise, the equation errors could be driving both the explanatory and endogenous variable, rendering the explanatory variable endogenous rather than exogenous and rendering the model spurious.

What is not modeled is in the error term and if there are important omitted variables correlated with the explanatory variables, the errors will be correlated with the explanatory variables, allowing for omitted variable bias or specification error that can engender the same spurious result.

For these reasons, the optimal model building strategy of choice is one of a general-to-specific nature. There is no other way to minimize the probability of omitted variable bias assumption violations.

We make a working assumption of linearity of functional form. We have used basis functions to linearize nonlinear function forms and assume that these transformations will capture delayed effects or threshold effects sufficiently, even though this may never totally accomplished.

Any model that is to be estimated must be identified. Without adequate identification the model cannot be estimated with unique solutions for its variables. If the model is non-recursive, it contains feedback loops or cyclical effects. There must be enough variables from outside the loop to allow that loop to be estimated. The rank condition which is necessary and sufficient for this condition to hold should be tested for a model to be proposed.

Hidden in the assumption of the feedback loop is the assumption of a dynamic equilibrium is a condition that also must exist. The dynamic equilibrium is otherwise known as covariance stationarity is necessary if the model is to be estimated by non-Bayesian methods. Covariance stationarity requires stability of the mean, the variance, and the autocovariance. From the stability of the variance derives the requirement of residual homoskedasticity. For this condition of stability of the mean to obtain, level shifts in the middle of a dataset being estimated by a model of the equations are not to be tolerated without proper modeling of those effects. If feedback loops obtain within the model, we assume that the moduli (absolute value) of the eigenvalues are all within the unit circle so that the system is stable in the long run. Without such stability, variancescould not be properly estimated. Also, without such just or over-identification, the variables in the system would not be estimable.

Although we construct our summary measures of Chornobyl related health threat from factor scores, in waves one though three, with alpha reliability coefficients in excess of 0.78, we make a simplifying working assumption in our exploratory mode that these variables are fixed effects without measurement error. This permits us to eschew use of the measurement equations of the structural equation modeling system and to rely on the submodel of Joreskog and Sorbom, plus Sorbom's formulation of mean structures [2, 9,136-137].

Regression models presume a causal direction from the exogenous to the endogenous variable variable and then from one to another endogenous variable. We furthermore assume that multicollinearity is not a problem in controlling for the effects of other variables. We assume that our cluster control of serial correlation is robust enough to attend to issues that otherwise may have derived from serial correlation of our residuals and deviations from homoskedasticity. Finally, we assume that all models are stationary, lest we be unable to rely on the consistency of our statistical analysis.

Linear structural equation models in general assume independence of observations and multivariate normality of the observed and latent variables. Sometimes joint normality is too restrictive and conditional normality or general symmetry may suffice. If too many of the variables appear to be ski jumps without clear modes or maxima, the models may not converge at all. However, there are estimation algorithms that such as asymptotic distribution free (ADF) or quasi-maximum likelihood (QML) which relax this assumption. When we request ADF, we obtain a kind of weighted least square which can correct for heteroskedasticity. WHen we request cluster robust estimates, the estimation method becomes QML, which relaxes the independence of observations by allowing clustering (correlation among id) across the waves, while requiring independence of the clustered observations [4, 57].

6 Dose-Depression response path models

6.1 male model

We begin examining the relationship between the initial dose of radiation to which a respondent was exposed and the links to self-reported depressive symptoms in waves one and two, and depression as defined by the Brief symptom inventory in wave three. In Figure 1, we display a path diagram of our findings then a list of the output to illustrate the presentation. In Table 2 we present the output of our analysis. For us to understand the either the table or the figure, we must be familiar with the variable names, which we present in Table 1.

Table 1

variable name	variable label
avgcumdosew1	Cumulative external dose in mGy for wave 1
radhlw1	how much believed personal health is affected by radiation in 1986
radhlw2	how much believed personal health is affected by radiation in 1996
radchw1	believed % of pollution related to Chornobyl in 1986
radchw2	believed % of pollution related to Chornobyl in 1996
depagw1	Self-reported depressive symptoms aggregated to wave 1 in 1986
depagw2	Self-reported depression symptoms to wave 2:1987 thru 1996
BSIdep	Brief symptom inventory depression subscale score

What can we learn from these results. First, we see that the model, not using the cluster robust variance estimates fits the data nicely from the likelihood ratio test provided at the bottom of Table 2. Second, we note that all of the paths are statistically significant. The nonsignificant paths, with the exception of one constant, have been trimmed from the model to support parsimony.

In the path diagram below, the reader will find numbers on the right hand side of the boxes that represent observed variables. The upper right hand number is the mean and the lower right hand number is the variance when the variables are exogenous. When the variables are endogenous, the numbers represent the constant in the regression model. The reader will also note that the errors are represented by circles and the number attached to the circle represents the error variance of the equation. The numbers along side the arrows represent the path coefficient of that path.

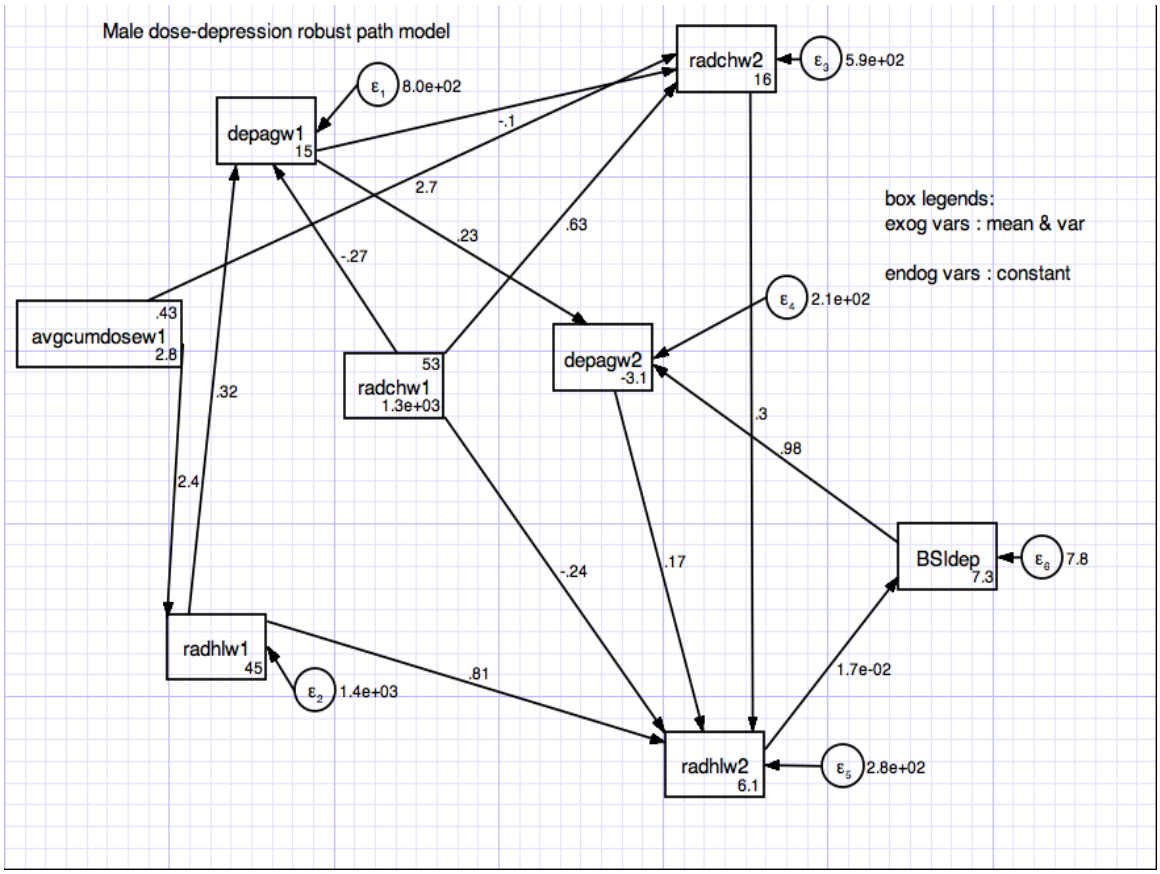

Figure 1: Dose-depression robust path diagram for males

Table 2 Dose - Depression path model for males

Endogenous variables

Observed: radhlw1 radchw2 depagw1 depagw2 radhlw2 BSIdep

Exogenous variables

Observed: avgcumdosew1 radchw1

Structural equation model Number of obs = 339
Estimation method = ml
Log pseudolikelihood= -10885.385

(Std. Err. adjusted for 339 clusters in id)

	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
Structural						
radhlw1 <-						
avgcumdos~1	2.41282	.6121466	3.94	0.000	1.213035	3.612606
_cons	44.52567	2.057047	21.65	0.000	40.49394	48.55741
radchw2 <-						
depagw1	-.1015498	.0423147	-2.40	0.016	-.184485	-.0186145
avgcumdos~1	2.680468	.5755574	4.66	0.000	1.552396	3.80854
radchw1	.6250525	.0472639	13.22	0.000	.5324169	.7176882
_cons	15.88568	3.505764	4.53	0.000	9.01451	22.75685
depagw1 <-						
radhlw1	.3222346	.0481044	6.70	0.000	.2279517	.4165174
radchw1	-.2704084	.0482875	-5.60	0.000	-.3650502	-.1757665
_cons	15.14743	3.003698	5.04	0.000	9.260287	21.03457
depagw2 <-						
depagw1	.2311921	.037071	6.24	0.000	.1585343	.3038499
BSIdep	.9811087	.2931539	3.35	0.001	.4065376	1.55568
_cons	-3.084811	2.241914	-1.38	0.169	-7.478882	1.309261
radhlw2 <-						
radhlw1	.8126957	.0266565	30.49	0.000	.7604498	.8649415
radchw2	.300364	.0601198	5.00	0.000	.1825312	.4181967
depagw2	.1656212	.0573098	2.89	0.004	.0532961	.2779463
radchw1	-.2355238	.0600088	-3.92	0.000	-.3531388	-.1179087
_cons	6.136548	2.035603	3.01	0.003	2.146839	10.12626
BSIdep <-						
radhlw2	.0172557	.0042348	4.07	0.000	.0089556	.0255558
_cons	7.315476	.2382199	30.71	0.000	6.848574	7.782379
Variance						
e.radhlw1	1368.577	59.50623			1256.778	1490.321
e.radchw2	592.9015	66.29016			476.2256	738.163
e.depagw1	797.2865	82.03237			651.6806	975.4255
e.depagw2	213.1001	36.88705			151.7899	299.1743
e.radhlw2	279.1774	44.9786			203.5834	382.841
e.BSIdep	7.791174	1.366722			5.524408	10.98804

LR test of model vs. saturated: chi2(14) = 19.53, Prob > chi2 = 0.1456 †

† The above LR test is for the non-robust version of this model.

There is no direct dose-depression response effect for males regardless of which depression measure we use.According to the output, the sum of the three indirect path effects from cumulative external dose to depression as measured by the Brief Symptom inventory is sum of indirect effects (b=0.0479, z=3.04, p=0.002), which is also the total effect of dose on male respondent depression measured by the Brief Symptom inventory. But if we accept the self-reports of depression as an adequate measure of it, the wave 1 dose-depression response impact is 0.777 and in wave 2 it is .23, both larger than the impact according to the Brief symptom inventory.

6.2 female model

The female dose-depression model is a little more complex than that of the males. First, there is a statistically significant direct effect from dose to depression for female respondents (b = 1.00 z=2.74, p=.006). However, the sum of the indirect effects on depression, measured by the BSI depression score, is also statistically significant (b= 0.415 z=2.72 p=0.007). The total effect (b=1.415 z=3.37 p=.001) is also statistically significant.

If we accept the self-reported depression in waves one and two as measures, we find no direct effect in either waves one or two, but the sum of the seven indirect paths are (b=13.933 z=2.97 p=0.007) in wave one and in wave two (b=5.024 z=3.04 p=0.002) are substantial. They are a much larger impacts than that measured by the Brief symptom inventory. They comprise evidence that the mediating or indirect effects are significant and in some cases more substantial than conventionally measured direct effects in cases of cumulative external dose on depression for Ukrainian female residents of Zhitomyr and Kiev Oblasts.

6.3 Comparison of male to female dose-depression responses

Although the males experienced no direct dose-depression impact, the females did. For the males, the total impact of dose was through one of several indirect paths. However, for the females, this direct impact was statistically significant and positive, with higher exposures leading to more depression. For the males, the only effects experienced were indirect ones through perceived threat to belief in the proportion of pollution due to Chornobyl. However, both for males and females, the total dose effects were to enhance the level of the depression. As for the self-reports of waves one and two, they indicated greater and declining impacts for both males and females than the Brief symptom inventory depression subscale did. Indeed, regardless of what wave we consider the Brief symptom inventory depression subscale scores were lower than the self-reports on the part of males and females.

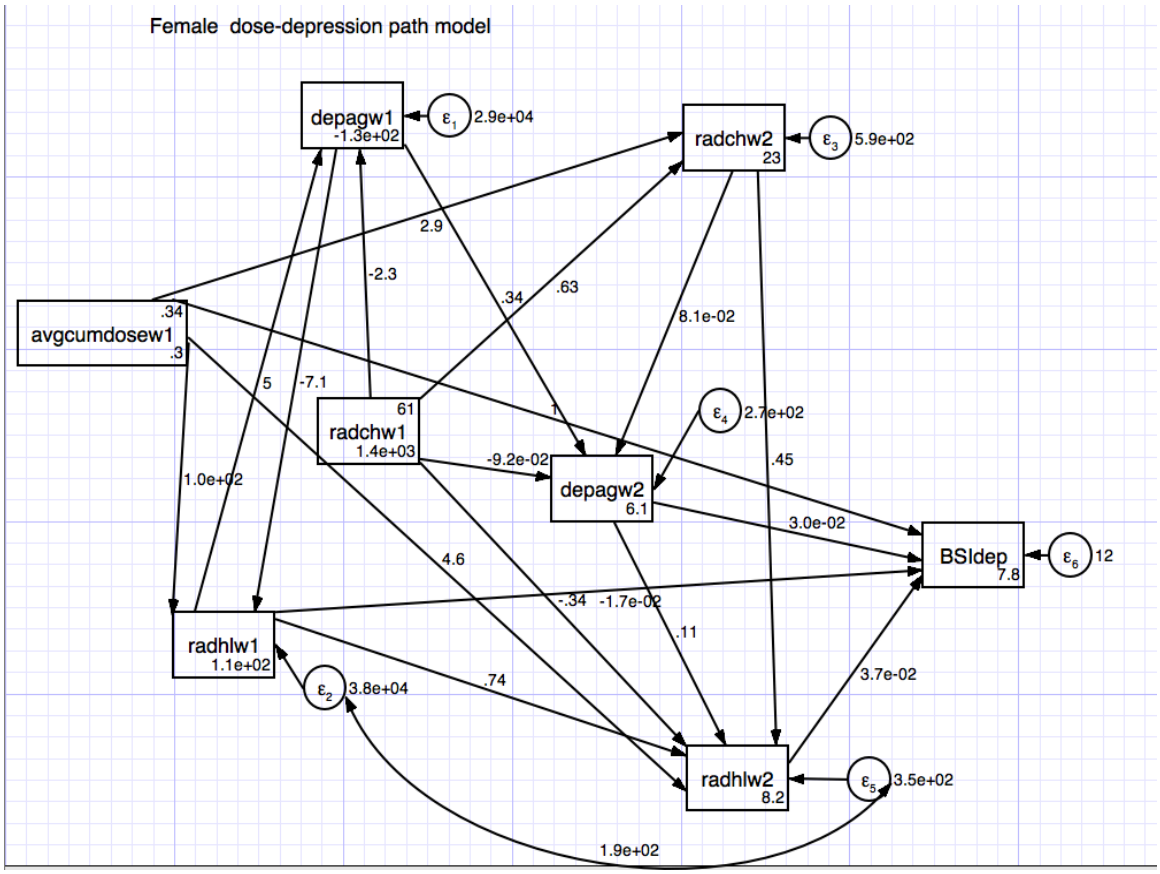

Figure 2: Dose-depression robust path diagram for females

Table 3 Female dose depression model

Endogenous variables

Observed: radhlw1 radchw2 radhlw2 BSIdep depagw1 depagw2

Exogenous variables

Observed: avgcumdosew1 radchw1

Structural equation model Number of obs = 362
Estimation method = ml
Log pseudolikelihood= -11345.165

(Std. Err. adjusted for 362 clusters in id)

	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
Structural						
radhlw1 <-						
depagw1	-7.079177	4.037319	-1.75	0.080	-14.99218	.8338219
avgcumdos~1	101.3973	62.0894	1.63	0.102	-20.2957	223.0903
_cons	112.0013	33.90253	3.30	0.001	45.55355	178.449
radchw2 <-						
avgcumdos~1	2.873174	2.184521	1.32	0.188	-1.408409	7.154757
radchw1	.6309132	.0417481	15.11	0.000	.5490884	.712738
_cons	22.79068	3.362662	6.78	0.000	16.19998	29.38137
radhlw2 <-						
radhlw1	.7370445	.034112	21.61	0.000	.6701862	.8039028
radchw2	.4526271	.058448	7.74	0.000	.3380712	.567183
depagw2	.1144146	.0703453	1.63	0.104	-.0234598	.2522889
avgcumdos~1	4.552042	3.290513	1.38	0.167	-1.897245	11.00133
radchw1	-.3397368	.0579504	-5.86	0.000	-.4533175	-.2261561
_cons	8.176407	2.403323	3.40	0.001	3.465981	12.88683
BSIdep <-						
radhlw1	-.0169352	.0090695	-1.87	0.062	-.034711	.0008407
radhlw2	.0368707	.0098539	3.74	0.000	.0175574	.0561839
depagw2	.0297888	.0088121	3.38	0.001	.0125173	.0470602
avgcumdos~1	1.003901	.3661598	2.74	0.006	.2862408	1.721561
_cons	7.780477	.3698462	21.04	0.000	7.055592	8.505362
depagw1 <-						
radhlw1	5.044272	3.33268	1.51	0.130	-1.487661	11.57621
radchw1	-2.326271	1.535252	-1.52	0.130	-5.335308	.6827672
_cons	-130.9285	97.56776	-1.34	0.180	-322.1578	60.30077
depagw2 <-						
radchw2	.0814283	.0376128	2.16	0.030	.0077085	.155148
depagw1	.3438149	.0575026	5.98	0.000	.2311118	.4565179
radchw1	-.0924591	.0378049	-2.45	0.014	-.1665554	-.0183629
_cons	6.09155	1.760725	3.46	0.001	2.640593	9.542507

Continued on the next page ...

	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
Variance						
e.radhlw1	37555.87	41182.72			4377.982	322167.5
e.radchw2	587.2238	61.23885			478.6696	720.3964
e.radhlw2	348.7998	39.91475			278.7207	436.4988
e.BSIdep	12.3783	1.466825			9.812825	15.61451
e.depagw1	28821.15	37161.41			2302.443	360772.7
e.depagw2	272.5456	43.332			199.5753	372.196
Covariance						
e.radhlw1						
e.radhlw2	185.2487	318.3273	0.58	0.561	-438.6614	809.1588

LR test of model vs. saturated: chi2(9) = 11.91, Prob > chi2 = 0.2182 †

† = Model fit of non-robust version of otherwise same model

7 Path Models for Perceived Chornobyl related health threat and Depression

Another one of our concerns was distinguishing between the physiological and the psychological. In these models we focus on the psychological impact of perceived threat on one's health while trying to distinguish it from the reconstructed external dose sustained at the time of the Chornobyl catastrophe. From the factor scores of three variables, we constructed summary perceived Chornobyl health threat scales. The variables used for this scale construction asked the percent to which your health was affected by Chornobyl, the percent to which your family's health was affected by Chornobyl, and a percent belief that most of the cancer cases in Zhitomyr and Kiev are due to the effects of Chornobyl. We designated these summary scores crhtw1, crhtw2, and crhtw3 according to the wave in which the Choronobyl related health threat was perceived, after finding that their alpha reliabilities were respectively as 0.796, 0.832, and 8.833.

In these cases we wish to differentiate between the reconstructed external dose and the perceived Chornobyl related health threat. We begin with the male model. Instead of using the perceived threat to oneself (radhlw1, radhlw2, and radhlw3), we employ the perceived Chornobyl related health threat factor scores (crhtw1 crhtw2 and crhtw3) to see how dose and the perceived risk impact depression on the part of the respondent.

7.1 male model

From the bottom of Table 4, we see that the nonrobust version of this same model appears to be consistent with the data. However, when we add our perceived Chornobyl health risk summary scores for waves one through three to the equation, a different dynamic emerges. No longer is there any statistically significant impact of dose on depression for males. The total effect appears to have dissipated into a statistically nonsignificant relationship (b = -0.0022, z=-0.19, and p=0.851). Moreover, there was no longer a significant dose impact on wave one or wave two self-reported depressive symptoms.

What impacts explain or predict the BSI depression subscale? The variables that have a direct impact on the BSI depression subscale are self-reported depressive symptoms in wave 2 (depagw2 b=0.0318 z=3.50 p=0.000) and our perceived Chornobyl health risk in wave two (crthw2 b=0.700 z=4.20 p=0.000). If we want to compare the direct impact of these two coefficients, we can compare their standardized versions (crthw2 stdzd b = 0.2267) and (depagw2 stdzd b=0.187) in order to compare the relative size of their impacts regardless of their different metrics.

From an examination of Figure 3, we can see that when we consider indirect paths, we find only two indirect paths begin with cumulative external dose (avgcumdosew1), whereas three indirect paths that impact BSI depression begin with the summary perceived Chornobyl health risk score (crhtw1) in wave 1. Moreover, we find that all indirect paths but one, have a summary perceived Chornobyl health risk score (crhtw2) as an intervening variable.

The total effect, which add the sum of the indirect effects to the direct effect, we can identify those which appear to have the most impact. In terms of magnitude of the total effect path coefficients, perceived health risk from Chornobyl in wave 2 (crhtw2 b = 0.701 z=4.20 p=0.000) seems to impact BSI depression the most and that for wave 1 has the second largest coefficient (b = 0.685 z=5.07 p=0.000). The proportion of pollution due to Chornobyl in wave 2 is most significant(b=.007 z=10.11, p=0.000), but the magnitude of the impact may be smaller.

Current depression may well be predicted by self-reported depressive symptoms at previous waves as well as our new summary perceived health risk score at the previous wave. Wave 2 self-reported depressive symptoms stem from mostly from Chornobyl related perceived risk summary score, secondarily from the self-reported depressive symptoms at wave 1 and the thirdly from the proportion of the pollution believed due to Chornobyl in wave 1. How does this model compare with a comparable one for the females?

7.2 female model

When we model the relationship between dose, perceived Chornobyl health risk summary score for the above models, and depression for the females, we obtain a path diagram displayed in Figure 4 and the model output shown in Table 5. Not all of these paths are statistically significant at the 0.05 level. Two of them are of

borderline statistical significance, with p=0.55. Another path coefficient has a p = 0.65. However, a little collinearity in the model could reduce the statistical significance or the removal of it might enhance the statistical significance of some of the variables. For this reason, if the significance of the parameters is borderline, we do not necessarily prune those variables from the model, as their inclusion may have heuristic value for us in the future. The model is nonetheless a stable one that is consistent with the data when we estimate a non-robust version of it, which the likelihood ratio test at the bottom of table 5 indicates.

According to this model, the direct effect of cumulative external dose on the BSI depression subscale are not quite statistically significant at the 0.05 level. Nor are the direct effects of perceived Chornobyl related health risk from wave 1 which has approximately the same significance level. However, given a slight change in conditions, these variables may or may not have a direct impact. When these magnitude of these effects are standardized, the greatest direct impact in order of their absolute value comes from the perceived Chornobyl health threat at wave 2 (crhtw2 beta = .349), the second largest is that of age (beta = .207), the third comes from perceived Chornobyl health risk at wave 1 (crhtw1 beta = -.152) , and the fourth largest comes from cumulative external dose at wave 1 (avgcumdosew1 beta = 0.119).

The variables that do have statistically significant direct effects are self-reported depressive symptoms at wave 2 and perceived Chornobyl health risk in decade after Chornobyl.

Most of the variables appear to have had a significant indirect effect on BSI depression. Among those variables who have a significant sum of indirect effects, however, are age of the respondent (b = .018, z=4.99 p=0.000), the external cumulative dose at wave 1 (b = .473 z=3.57 p=0.000), both previous self-reports of depressive symptoms (depagw1 b = .011 z=6.02 p=0.000 and depagw2 b= 012 z=6.32 p=0.000), the perceived Chornobyl health risk at wave 1 (crhtw1 b=.972 z=15.53 p=0.000), as well as the percent of the wave 2 pollution believed to be due to Chornobyl (radchw2 b = .0218 z=10.02, p=0.000). The latter was of borderline significant impact for wave 1(with radchw1 b=-0.00850 z=-1.93 p=.053).

The variables that explain self-reported depressive symptoms in wave one include age and cumulative external dose. The variables that explain such symptoms at wave two include the self-reported depressive indications at wave one and the perceived Chornobyl health threat in the previous wave.

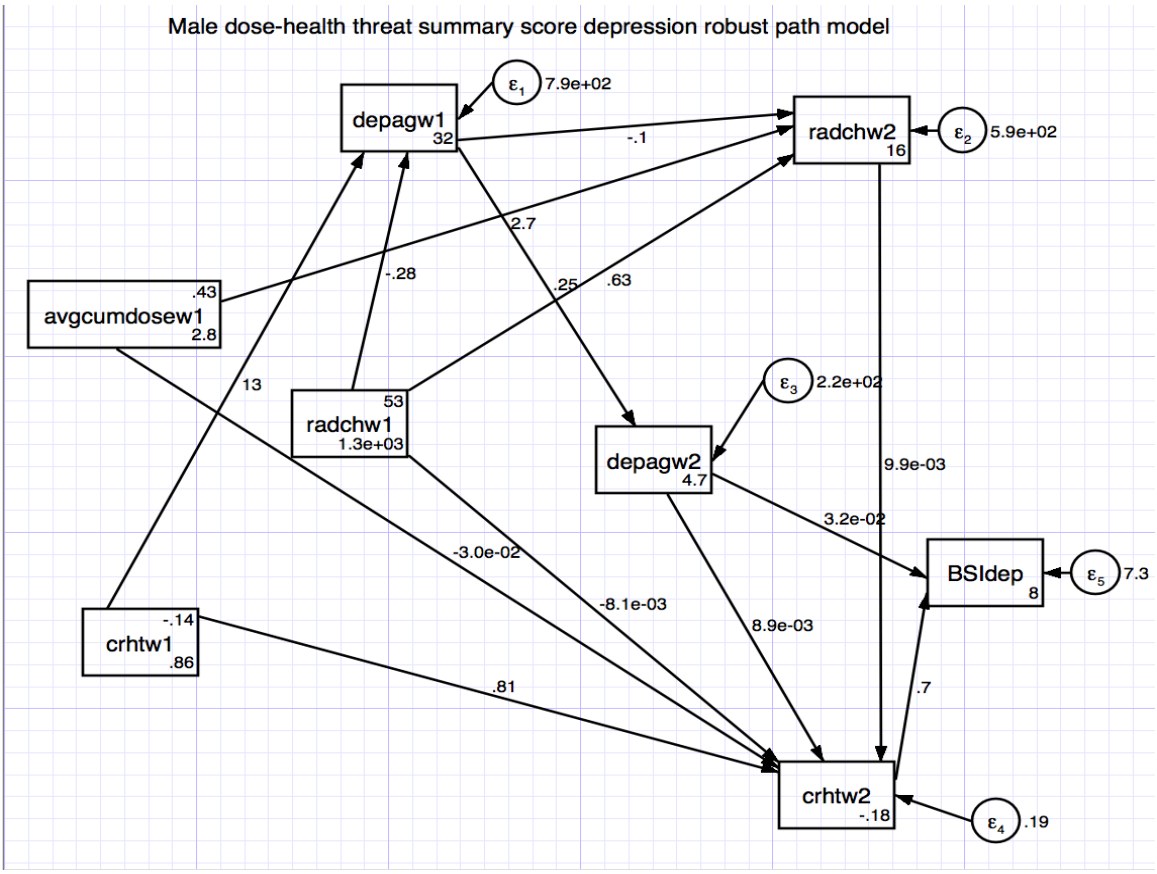

Figure 3: Dose-Perceived risk-depression path diagram for males

Table 4 Male cumulative external dose, perceived risk and the pathways to depression

Endogenous variables

Observed: radchw2 crhtw2 depagw1 depagw2 BSIdep

Exogenous variables

Observed: avgcumdosew1 radchw1 crhtw1

Structural equation model Number of obs = 339
Estimation method = ml
Log pseudolikelihood= -8393.8297

(Std. Err. adjusted for 339 clusters in id)

	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
Structural						
radchw2 <-						
depagw1	-.1015498	.0423147	-2.40	0.016	-.184485	-.0186145
avgcumdos~1	2.680468	.5755574	4.66	0.000	1.552396	3.80854
radchw1	.6250525	.0472639	13.22	0.000	.5324169	.7176882
_cons	15.88568	3.505764	4.53	0.000	9.01451	22.75685
crhtw2 <-						
radchw2	.0098726	.0015787	6.25	0.000	.0067783	.0129668
depagw2	.0089195	.0013667	6.53	0.000	.0062409	.0115982
avgcumdos~1	-.0295446	.0099938	-2.96	0.003	-.0491321	-.0099572
radchw1	-.0081225	.0015666	-5.18	0.000	-.011193	-.0050519
crhtw1	.8109274	.0264141	30.70	0.000	.7591567	.8626981
_cons	-.1807124	.0531546	-3.40	0.001	-.2848936	-.0765313
depagw1 <-						
radchw1	-.2796966	.0481504	-5.81	0.000	-.3740697	-.1853235
crhtw1	13.44711	1.897476	7.09	0.000	9.728125	17.16609
_cons	32.23328	3.551726	9.08	0.000	25.27202	39.19453
depagw2 <-						
depagw1	.2458306	.0365588	6.72	0.000	.1741768	.3174845
_cons	4.656015	.6972411	6.68	0.000	3.289448	6.022583
BSIdep <-						
crhtw2	.7005313	.1745123	4.01	0.000	.3584935	1.042569
depagw2	.0318522	.0123239	2.58	0.010	.0076978	.0560067
_cons	7.980278	.1737475	45.93	0.000	7.639739	8.320816
Variance						
e.radchw2	592.9015	66.29016			476.2256	738.163
e.crhtw2	.1932635	.0278806			.1456645	.2564165
e.depagw1	785.3806	79.30772			644.3559	957.27
e.depagw2	221.9766	36.65741			160.5976	306.8142
e.BSIdep	7.300579	1.276708			5.182045	10.28522

LR test of model vs. saturated: chi2(12) = 9.10, Prob > chi2 = 0.6946 †

† represents the fit of the non-robust version of the same model.

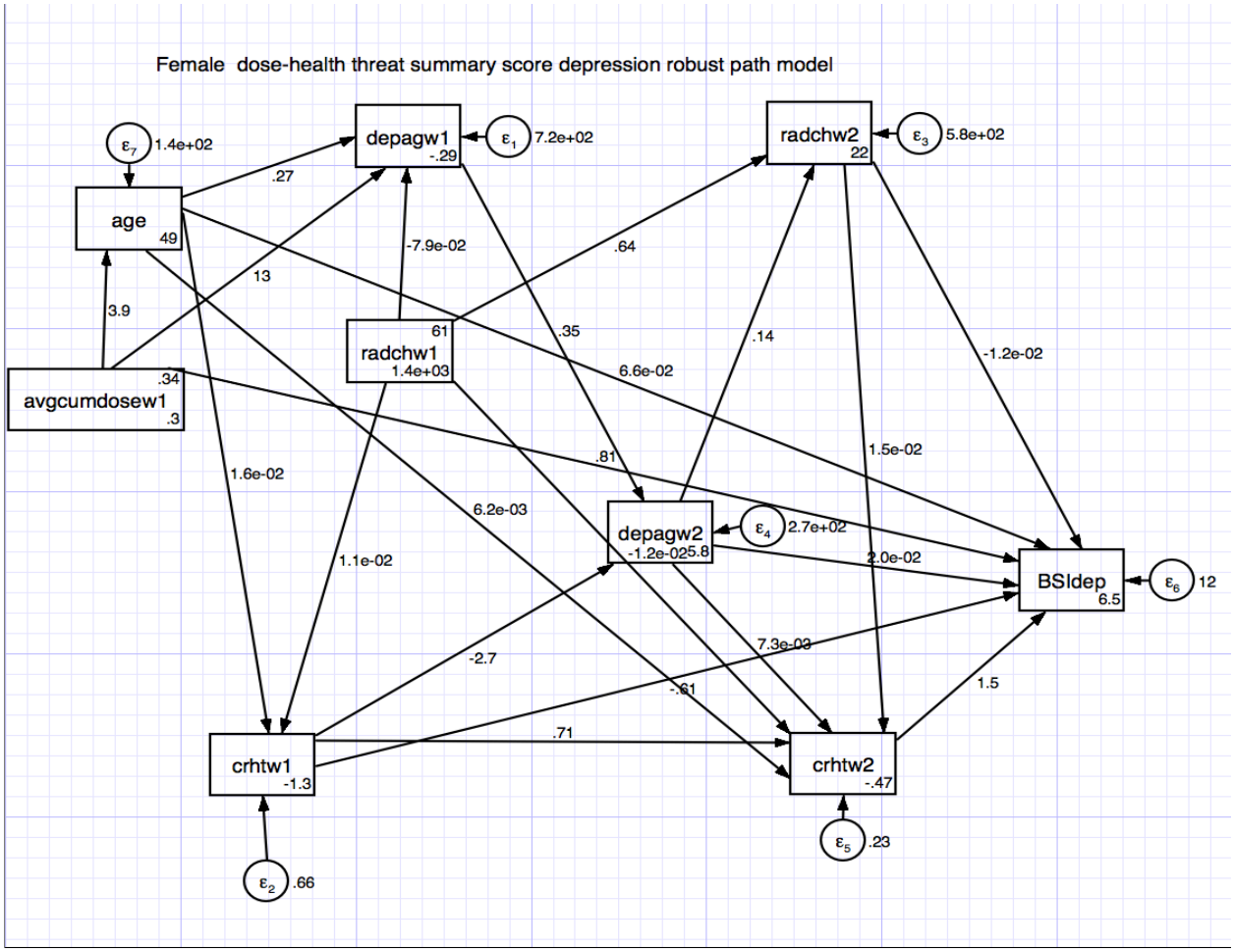

Figure 4: Dose-Perceived risk-depression path diagram for females

Table 5 Female Perceived Chornobyl health risk and its impact on Depression

Endogenous variables

Observed: depagw1 BSIdep age depagw2 crhtw1 radchw2 crhtw2

Exogenous variables

Observed: avgcumdosew1 radchw1

Structural equation model Number of obs = 362
Estimation method = ml
Log pseudolikelihood= -10064.647

(Std. Err. adjusted for 362 clusters in id)

	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
Structural						
depagw1 <-						
age	.265543	.1245006	2.13	0.033	.0215263	.5095598
avgcumdos~1	13.03686	4.856171	2.68	0.007	3.51894	22.55478
radchw1	-.0786365	.0431478	-1.82	0.068	-.1632047	.0059318
_cons	-.2884023	5.082497	-0.06	0.955	-10.24991	9.673109
BSIdep <-						
age	.0657521	.0147984	4.44	0.000	.0367478	.0947564
depagw2	.0197734	.0095985	2.06	0.039	.0009607	.0385862
crhtw1	-.6106943	.3188835	-1.92	0.055	-1.235695	.0143058
radchw2	-.0117596	.0063636	-1.85	0.065	-.0242321	.0007129
crhtw2	1.482689	.358085	4.14	0.000	.7808554	2.184523
avgcumdos~1	.8141524	.4235437	1.92	0.055	-.015978	1.644283
_cons	6.451681	.7954758	8.11	0.000	4.892577	8.010784
age <-						
avgcumdos~1	3.928799	1.115207	3.52	0.000	1.743034	6.114564
_cons	48.94976	.7401591	66.13	0.000	47.49908	50.40045
depagw2 <-						
depagw1	.3469208	.0576615	6.02	0.000	.2339063	.4599354
crhtw1	-2.725531	1.025888	-2.66	0.008	-4.736236	-.7148267
_cons	5.831444	.768957	7.58	0.000	4.324316	7.338572
crhtw1 <-						
age	.0156495	.0037127	4.22	0.000	.0083727	.0229263
radchw1	.0111706	.0012103	9.23	0.000	.0087985	.0135427
_cons	-1.338271	.1914047	-6.99	0.000	-1.713417	-.9631248
radchw2 <-						
depagw2	.1381542	.0663012	2.08	0.037	.0082061	.2681022
radchw1	.6415708	.0419538	15.29	0.000	.559343	.7237987
_cons	21.74291	3.415033	6.37	0.000	15.04957	28.43625

Continued on the next page...

Table 5 continued

	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
crhtw2 <-						
age	.0061808	.0020948	2.95	0.003	.002075	.0102866
depagw2	.0072639	.0013714	5.30	0.000	.0045759	.0099518
crhtw1	.7144429	.0359837	19.85	0.000	.6439161	.7849697
radchw2	.0147294	.0013612	10.82	0.000	.0120616	.0173973
radchw1	-.0122293	.0013853	-8.83	0.000	-.0149445	-.0095141
_cons	-.4749309	.1174283	-4.04	0.000	-.7050862	-.2447756
Variance						
e.depagw1	720.3633	92.22052			560.5072	925.8101
e.BSIdep	11.55687	1.327249			9.227496	14.47426
e.age	135.0564	8.338223			119.6638	152.4289
e.depagw2	272.1402	42.39661			200.5323	369.3185
e.crhtw1	.6619999	.0414378			.5855677	.7484085
e.radchw2	582.6394	61.3243			474.0331	716.1286
e.crhtw2	.2324073	.0244519			.1891009	.2856315

LR test of model vs. saturated: chi2(14) = 19.52, Prob > chi2 = 0.1460 †

† Fit of companion non-robust version of the same model

Within the female subsample, we note a direct dose-depression response effect of quasi- statistical significance (avgcumdosew1 b=0.814 z=1.92 p=0.055). Even the perceived health risk from 1986 is of borderline statistical significance (crhtw1 b = -.611 z=1.92, p=.055). Although some may question the significane of the wave one impact, they can take note of statistically direct effects of age (b=.066 z=4.44 p=0.000) and self-reported depressive symptoms in wave two as well as perceived Chornobyl health risk in wave two (b = 1.483 z=4.14 p=0.000).

Notwithstanding the quasi-significance of its direct effects, cumulative external dose does have a significant total effect on BSI depression (b=1.287 z=3.30 p = 0.001), whereas the other two variables that had quasi-significant direct impacts fade into apparent statistical non-significance in their total impact (crhtw1 and radchw1). Whereas self-reported wave 1 depressive symptoms had no direct effect on BSI depression, its total effect was quite statistically significant (depagw1 b=0.011 z = 6.03, p=0.000). The proportion of pollution believed due to Chornobyl in wave 1 did not exhibit a direct effect on BSI depression but the total effect of this variable almost became statistically significant (radchw1 b = - 00849 z=-1.93, p=0.053). Two variables whose direct effects were statistically significant also had total effects that remained significant were self-reported depressive symptoms at wave 2 (depagw2 b = .032 z=3.19 and p = 0.001) and perceived Chornobyl health threat at wave 2 (crhtw2 b = 0.316 z=4.14 p=0.000). Again, we observe that mediating effects can account for much of the effect of one variable on another.

7.3 Comparison of the male to the female perceived health risk - depression response path models

When we compare the path diagrams of the males to the females, we see that the females have a more elaborate path model than do the men.The female model contains one more variable than the male model and fits the data slightly better than the other model. The male model contains 5 endogenous variables, while the female model contains 7 of them. Although, strictly construed, there are no statistically significant direct effects of cumulative external dose in either model, there is an effect in the female model that is of borderline statistical significance. If we liberally allow all of these paths in the diagrams of Figures 3 and 4 to be deemed significant in the models, the female model contains more indirect paths than the male model. The male model contains two indirect paths from avgcumdosew1 or and three indirect paths from perceived Chornobyl related health threat to BSI depression, whereas the female model contains 5 indirect paths from the cumulative dose and two from the source of the perceived health threat. Even if we disallow the direct links from the avgcumdosew1 or the crthw1 to the BSI depression to be considered significant, the female model still contains more indirect paths than the male model.

References

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