# Testing of the effects concerning the number of medically diagnosed illnesses and voluntary abortions, with hypotheses 2, 7, 9, 13, 17, and 21

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#### 2 Introduction

#### 2.1 Direct effects hypotheses

In this paper, we will examine several hypotheses testing direct and indirect effects. Four hypotheses concern direct effects. They are hypotheses 2, 7, with respect to both men and women, and for females hypotheses 9 and 13.

#### 2.1.1 Hypothesis 2

Hypothesis 2 suggests that radiation dose directly explains medically diagnosed illness count. For this hypothesis we test both males and females.

#### 2.1.2 Hypothesis 7

Hypothesis 7 suggests that perceived risk directly explains medically diagnosed illness count, and we test this hypothesis with men and women.

#### 2.1.3 Hypothesis 9

Hypothesis 9 suggests that radiation dose directly explains voluntary abortions, which we use only our female sample for testing.

#### 2.1.4 Hypothesis 13

hypothesis 13 submits that perceived risk directly explains voluntary abortions. We use only our female sample to test this hypothesis.

#### 2.2 Indirect effects hypotheses

We use the female sample to test two hypotheses relating to indirect effects.

#### 2.2.1 Hypothesis 17

This hypothesis postulates that radiation dose indirectly predicts voluntary abortions mediated by the number of medically diagnosed illnesses.

#### 2.2.2 Hypothesis 21

This hypothesis postulates that perceived risk indirectly effects voluntary abortions while being mediated by the number of medically diagnosed illnesses. Altogether, we are testing 6 hypotheses in this paper.

#### 2.3 Summary tables

At the end of this paper we have summary tables indicating the nature of the hypothesis and the test results. Readers merely wanting a quick review should examine the tables at the end of this paper.

#### 3 Statistical Methods

#### 3.1 AutoMetrics automatic modeling

To test these hypotheses we use two different methods, both of which involve linear regression analysis. We use AutoMetrics for variable selection and automatic regression model building. AutoMetrics uses a general-to-specific multi-path search to find the path down which the least number of regression assumptions are violated. The researcher selects all of the variables to be included in the model. This is called the "General Unrestricted Model (GUM)." He or she then sets a probability level to determine the threshold at which variables will be removed from the model. Variables of borderline significance determined by the chosen level may be incorrectly retained or dropped from time to time. In general there are  $2^p$  paths where k= the number of variables in the GUM. The final GUM, is the union of all of the terminal models, at which point encompassing tests for non-nested models and for parsimonious encomopassing are applied to be sure that there is optimal fit among the competing models, which maximizes the  $adjusted\ R^2$  or minimizes the Schwartz criteria of the competing models.

By multi-path search, we mean that it examines every possible combination of explanatory variables to arrive at an optimal model. If a combination of explanatory variables violates a misspecification test, the path is terminated and an alternative path is tested. If a path is terminal, it reaches a combination of variables that arrives at a solution.

Candidate terminal models are compared lack of fit and ties are broken by the Schwartz criteria for the competing models. In this manner, the best model is selected.

The objective is to minimized the possibility of specification error, which might bias the magnitude and significance of our regression parameter estimates. This program can be used to test direct effects in a regression model. It furthermore evaluates the regression model assumptions to show how much we can trust the model for statistical conclusion validity. It examines the assumptions of residual normality, residual homogeneity, and functional form and provides indication whether these assumptions have been fulfilled or violated.

In the event of violation of the assumptions and as an alternative method, we use structural equation robust path modeling to further test the same hypotheses, in addition to testing the indirect hypothesis effects.

#### 3.2 Structural equation path models

Structural equation nonrecursive path models recursive path models are by definition identified. The nonrecursive models are presumed to be just-identified or over-identified, lest they be inestimable by full-information maximum likelihood. Furthermore, we test them for stability before accepting them as valid and show that they are stable. They presume unidirectional causality and preclude reverse causality. They assume a closed system, so that all important variables are deemed to be resident within the model. Such path models are not unique; there may be several alternative models using the same variables and the same data.

#### 3.3 Relative advantages of the two methods

AutoMetrics is preferred for variable selection and model-building of regression models from a large number of variables, especially when the number of variables exceeds the number of cases, and therefore at reducing the probability of specification error, but structural path models are better at revealing mediating effects in the same model, even though they are not unique. Both protocols can complement one another.

#### 3.4 Direct effects

Our hypotheses have been formulated in a path analytical framework, where we distinguish between direct and mediating effects. In order to clarify whether an effect is mediated or direct helps demystify much of the potential causal association among variables. However, this is not something which has been AutoMetrics has been designed to do. It emphasizes variable selection and model building in a manner that is theoretically defensible and has a way around the multiple testing problem. When we do our data mining with AutoMetrics, we nonetheless use a conservative approach by specifying a minute probability as the level of variable selection (p = 0.00001), which is consistent with a Sidak constraint on the experimentwise error rate, even though AutoMetrics has a 1-cut technique for sorting the t values and making 1 cut to circumvent the need for a multiple testing probability correction.

AutoMetrics is ideal for variable selection and regression model-building because of its multi-path approach to testing all different combinations of variables to determine which combinations do not violate the regression assumptions. The path is terminated in the event of a failure of a regression mis-specification test.

Direct effects can be tested with an ordinary least squares regression analysis. Researchers may attempt to test for such effects in vacuo at first. We will attempt to place them in a more realistic setting amidst multiple possible confounders and to partial out potentially confounding effects so that we can identify the specific effects we intend to test and then to test them within such a setting. We find the optimal model with an AutoMetrics general-to-specific modeling approach and control for all other potential confounders. Because AutoMetrics performs this variable selection and regression model building better than a path model can perform variable selection, we may use that procedure when variable selection and potential confounder identification is warranted.

We employ the general-to-specific modeling approach applied in AutoMetrics, including all of the principal potentially confounding variables as explanatory variables, and by the theory of reduction, reducing the model to the optimal one with respect to fulfillment of the regression assumptions.

#### 3.4.1 Exclusion criteria

When the endogenous variable in our hypothesis happens to be the number of medically diagnosed illnesses, we have to eliminate the subjective counts of illnesses as explanatory variables because their inclusion would be tantamount to testing an approximate tautology, which would add little value to our testing protocol. Nor would it help us understand what properly estimates of the count of medically diagnosed illnesses. It would merely cause other highly explanatory variables to be excluded from the analysis on account of collinearity. We have to eliminate basis functions from the candidate variable pool that are transformations of the dependent variable as well for the same reasons. Finally, all variables that are collinear must be examined for inclusion-exclusion on the basis of their contribution to the explanation and their contribution to fulfillment of the regression assumptions.

#### 3.4.2 Inclusion Criteria

Those variables that add to the partial  $R^2$  and enhance fulfillment of the basic regression assumptions necessary for statistical congruency could be included. The regression assumptions for cross-sectional data include independence of vectors, (weak) exogeneity of the explanatory variables, homogeneity of variance of the residuals, normality of the residuals, lack of collinearity among the explanatory variables, and linear and additive functional form. Weak exogeneity means that all of the useful information can be gleaned from the conditional relationship between the endogenous and exogenous variables through the parameter estimate and that the explanatory variables are pre-determined. Strong exogeneity is needed for forecasting, such that there is no correlation of the errors with variables in the models, through which feedback and bias may result [12, 128-137], [5, 174].

#### 3.4.3 Misspecification tests for cross-sectional models

Independence of observations is generally assumed and tested with tests for serial correlation. Residual normality is tested with a Jarque-Bera test. Heteroskedasticity is tested with two forms of White's general specification test. Functional form is generally tested with the Ramsey Reset test.

#### 3.5 Mediated or indirect effects

Some of the effects are discussed in a path analytic framework. When these hypotheses include indirect effects and mediators, it is clear that these effects cannot be estimated by a single regression analysis alone. For hypotheses 17 and 21, the count of medically diagnosed illnesses is identified as a mediating effect. These hypotheses have to be tested with some sort of path analytic framework. Our model is a longitudinal one that traverses several waves of time. For this reason, we have to use a robust path analysis to accommodate the panel-specific hetereoskedasticity and the inter-panel serial correlation, for which reason we ultimately rely on robust panel models for our hypothesis tests.

#### 4 Male model direct effects on number of medically diagnosed illnesses

#### 4.1 Male AutoMetrics regression model

## 4.1.1 Hypothesis 2: Direct effects of radiation dose on medically diagnosed male illness count

When we examine Table 2, we find no evidence of direct effects from cumulative dose extending to the count of the medically diagnosed illnesses in any wave for the males. Hypotheses 2 is not supported by data from our male subsample.

## 4.1.2 Hypothesis 7: Direct effects of perceived risk of exposure on the number of medically diagnosed illness

Nor do we find that perceived risk to be selected as a predictor of the count of diagnosed illnesses by doctors. from the male regression analysis in Table 2. Strictly speaking, hypothesis 7 is inconsistent with the data from our male subsample.

A reader might argue that there are similar variables indicating perception of risk that are selected. Among them are the proportion of the radioactively contaminated area and the proportion of pollution due to Chornobyl. However, if we test the alpha between crhrw1, crhrw2, crhrw3, radw1, radw2, radchw1, and radchw2, we only obtain an alpha of 0.5714, which is generally not high enough for a scale, so we would not make too much out of the similarity among these items.

Table 1: Variable list for male regression model

Variable name	type	format	Variable label
pillw1	byte	%8.0g	number of pills for pain per week in 1976-1986
medcow3	byte	%8.0g	number of medical visits for a medical condition per year 1997-now
age	byte	%8.0g	* Respondent's age
emplw14	byte	%8.0g	emplw1==3. voluntary
occ5w1	byte	%15.0g	factory laborer machinist transp cleaner in 1986
movew3	byte	%8.0g	Total number of moves experienced in time period 1996-NOW
shhlw2	byte	%8.0g	Percentage of strains and hassles related to health in 1996
shhousw3	byte	%8.0g	Percentage of strains and hassles
contw1	byte	%15.0g	related to housing NOW use of any contraception method in 1976-1986
radw1	byte	%8.0g	believed % of the radioactively
radw2	byte	%8.0g	contaminated area in 1986 believed % of the radioactively
radchw1	byte	%8.0g	contaminated area in 1996 believed % of polution related to
radchw2	byte	%8.0g	chornobyl in 1986 believed % of polution related to
dafter	int	%8.0g	chornobyl in 1996  * how many days lapsed after  Chornobyl accident before you  heard about the acciden
dauthw2	byte	%8.0g	level of danger by authorities
medw3	byte	%8.0g	(in percent) in 1996 level of danger by general media
neiw1	byte	%8.0g	(in percent) NOW level of danger by neighbors (in
carcin	byte	%8.0g	percent) in 1986  * a person exposed to carcinogen is likely to get cancer (% of
WHPel	double	%9.0g	agreement) Wtd Health Profile Pt 1 Energy
HP2probsoc	byte	%9.0g	Level Subscale Hlth profile Pt2: Hlth causing probs with social life
bf14			bf14= $max(0, radw2 - 10) * bf12$
bf11			bf11= max(0, 20 - sufamw1)
bf12			bf12= radw2 if radw2 !~ .
<b>-</b>			

Table 2: H2 and H7: Male model for count of medically diagnosed illnesses

EQ(10) Modelling icdxcnt by OLS-CS
The dataset is: /Users/robertyaffee/Documents/data/research/chwk/
phase3/data/ox/chwide10sep2012mold.dta
The estimation sample is: 2 - 338
Dropped 10 observation(s) with missing values from the sample

	Coefficient	Std.Error	HACSE	t-HACSE	t-prob	Part.R^2
pillw1	0.186596	0.04793	0.03236	5.77	0.0000	0.0980
medcow3	0.126010	0.02280	0.03274	3.85	0.0001	0.0462
age	0.0280895	0.004911	0.003946	7.12	0.0000	0.1421
emplw14	-1.85601	1.332	0.4230	-4.39	0.0000	0.0592
occ5w1	-1.10699	0.3823	0.3449	-3.21	0.0015	0.0326
movew3	0.665057	0.2217	0.2756	2.41	0.0164	0.0187
shhlw2	0.00777531	0.002345	0.002228	3.49	0.0006	0.0383
shhousw3	0.00492730	0.002415	0.002660	1.85	0.0649	0.0111
contw1	-0.342896	0.1129	0.1049	-3.27	0.0012	0.0338
radw1	-0.00743013	0.003314	0.003594	-2.07	0.0395	0.0138
radw2	0.0202849	0.006974	0.007029	2.89	0.0042	0.0265
radchw1	0.00467527	0.003481	0.004018	1.16	0.2455	0.0044
radchw2	-0.00697845	0.003332	0.003610	-1.93	0.0541	0.0121
dafter	0.00439510	0.001575	0.0005668	7.75	0.0000	0.1642
dauthw2	0.00766027	0.003209	0.003601	2.13	0.0342	0.0146
medw3	-0.00528337	0.002951	0.003545	-1.49	0.1371	0.0072
neiw1	-0.00764893	0.002508	0.002720	-2.81	0.0052	0.0252
carcin	0.00831244	0.002684	0.002773	3.00	0.0029	0.0285
WHPel	0.0110772	0.002791	0.002823	3.92	0.0001	0.0479
HP2probsoc	-0.348068	0.2661	0.2914	-1.19	0.2331	0.0046
bf14	-0.000161419	7.398e-05	6.608e-05	-2.44	0.0151	0.0191
sigma	1.27035	RSS	49	93.815878		
log-likelihood	-531.388					
no. of observati	ons 327	no. of par	ameters	21		
mean(icdxcnt)	2.13456	se(icdxcnt	:)	1.70535		
When the log-lik	elihood consta	nt is NOT i	ncluded:			
AIC	0.540643	SC		0.784035		
HQ	0.637760	FPE		1.71741		
When the log-lik	elihood consta	nt is inclu	ded:			
AIC	3.37852	SC		3.62191		
HQ	3.47564	FPE		29.3325		
Normality test:	$Chi^2(2) =$	9.0841 [0	.0107]*			
Hetero test:	F(37,288) =	1.6733 [0	.0112]*			
RESET23 test:	F(2,304) =	4.1100 [0	.0173]*			

#### 4.2 Male Path Model

Another test of hypotheses 2 and 7 for males can be performed with a path analytical model, as shown in Figure 1. The color-coded model exhibits the radiation dose variables as rose colored boxes with red direct effects. The perceived risk variables are represented as orange boxes with dark orange direct effects. Fear of consuming contaminated food variables are colored light blue-gray

medium blue direct effects, whereas the count of medical diagnoses is colored a lighter grey with green arrows extending form it to two of the Nottingham health scales—energy level in cyan and physical ability in Kakhi. Sleep is golden with purple arrows protruding from it.

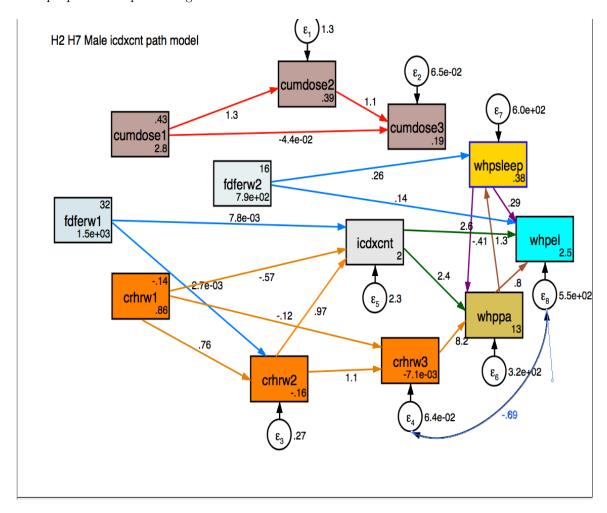


Figure 1: Male icdxcnt path model

#### 4.2.1 Hypothesis 2: Male path model test results

From our analysis of the male path model, we find no evidence supporting a direct effect of cumulative radiation dose on the number of medically diagnosed diseases, as shown in Figure 1.

#### 4.2.2 Hypothesis 7: Male path model test results

From our analysis of the male path model, we find evidence to support the hypothesis that perceived risk of exposure directly predicts the number of medically diagnosed illnesses in waves 1 and 2. Therefore, we have to say that there is partial evidence in support of this hypothesis. The evidence is partial insofar as 2 out of 3 waves exhibit this evidentiary support.

#### 5 Female model direct effects on number of medically diagnosed illnesses

#### 5.1 Female AutoMetrics regression model

The regression analysis to test hypotheses 2 and 7 among the female subsample follows. The variable selection process for identifying the optimal model did not trim down the model as much as we might have preferred even though we used a minute variable selection probability (p=0.0001) for both the male and the female. We had to prune out some nonsignificant paths from the female to obtain this much of a reduction. Moreover, 99 observations were dropped owing to the missing values on some of the variables selected for the optimal regression model.

## 5.1.1 Hypothesis 2: Direct dose effects on medically diagnosed illness count

To test the hypotheses 2, and 7, we have to ask whether for a direct effect were any of the cumulative dose or perceived risk variables selected to directly predict the count of medical diagnoses. From waves 1 and 2, the cumulative external dose variables were identified as directly predicting the number of medical diagnoses.

Table 3: Hypothesis 2 female test

Table 3

Hypothesis 2 female test results								
Variable	Coefficient	Std.Error	HACSE	t-HACSE t-prob	Part.R^2			
cumdose1	1.98759	0.6911	0.6521	3.05 0.0026	0.0412			
cumdose2	-0.759593	0.2981	0.2730	-2.78 0.0059	0.0346			

Although cumdose3 was not selected, we can say that we have partial evidence in female support of hypothesis 2 from Table 3.

### 5.1.2 Hypothesis 7 Direct perceived risk effects on medically diagnosed illness count

Hypothesis 7 stipulates that perceived risk of exposure directly explains or predicts the number of medically diagnosed illnesses. But the crhrw variables are not to be found among those selected for the optimal regression model to predict female icd9 count. According to a strict interpretation of the meaning of the variables, we cannot therefore say that we have evidence from our female model to support hypothesis 7.

#### 5.2 Female path model

Therefore, we examine the female path model in Figure 2 and the direct effects in the clustered robust Table 6.

#### 5.2.1 Hypothesis 2

We find no evidence to support hypothesis 2 in any wave.

#### 5.2.2 Hypothesis 7

Nor do we find any evidence in this path model to support hypothesis 7 in any wave.

However, if we permit a broader construction of perceived risk of exposure, we might find some support in the regression analysis in Table 5.2.2. Among them are found forms of fear of exposure in waves 1 and 3, including airw1, radw1, radfmw3, radhlw3, radtlw3, radtlw3, goferw3, and fdferw3. These refer respectively to fear of air and water pollution due to Chornobyl, the percent of the pollution due to Chornobyl, percent belief that the health of one's family has been affected by Chornobyl, the percent belief that one's own health has been affected by Chornobyl, fear of long-term or lifetime exposure in wave 3, a fear of going outdoors in wave 3, and a fear of consuming contaminated food in wave 3. All these are forms of fear of exposure. In a broad sense, we could say that these forms of fear of exposure directly predict the count of medically

diagnosed diseases in waves 1 and 3. In these respects, we have a penumbra of support for prediction of the number of medically diagnosed illnesses in waves 1 and 3 among females.

Table 4: Direct effects on count of medical diagnoses among females

EQ(40) Modelling icdxcnt by OLS-CS
The dataset is: /Users/robertyaffee/Documents/data/research/
chwk/phase3/data/ox/chwide10sep2012fold.dta
The estimation sample is: 2 - 362
Dropped 99 observation(s) with missing values from the sample

	Coefficient	Std.Error	HACSE	t-HACSE	t-nroh	Part.R^2
age	0.0303113	0.009709	0.01002	3.02	0.0028	0.0406
edu2	-0.945011	0.4160	0.3901	-2.42	0.0162	0.0265
marrw13	-0.343753	0.2589	0.2567	-1.34	0.1820	0.0200
marrw21	1.06130	0.3432	0.3254	3.26	0.0013	0.0469
marrw23	0.703971	0.2866	0.2494	2.82	0.0052	0.0356
marrw31	-0.875476	0.3574	0.3119	-2.81	0.0055	0.0352
emplw31	-0.984046	0.2025	0.2288	-4.30	0.0000	0.0382
occ3w1	-0.396961	0.2792	0.2705	-1.47	0.1437	0.0099
occ6w3	-1.02105	0.6429	0.3448	-2.96	0.0034	0.0390
inc1w2	-0.459441	0.2142	0.1790	-2.57	0.0109	0.0296
dvcew1	3.60276	0.8846	1.408	2.56	0.0112	0.0294
sepaw1	-4.16134	1.159	1.492	-2.79	0.0058	0.0348
accdw3	0.773016	0.2460	0.2434	3.18	0.0017	0.0446
shrelaw1	0.00932040	0.002417	0.002762	3.37	0.0009	0.0501
suprtw3	-0.00937121	0.002117	0.002771	-4.13	0.0001	0.0731
sufamw2	0.00651415	0.002322	0.002271	2.87	0.0045	0.0368
contw1	0.833298	0.2182	0.2544	3.28	0.0012	0.0473
contw2	-0.496042	0.2180	0.2285	-2.17	0.0311	0.0213
ncontw2	-0.528386	0.1855	0.1914	-2.76	0.0063	0.0341
beerw2	-0.0331456	0.02358	0.01277	-2.59	0.0101	0.0302
liqw1	0.126191	0.06953	0.07014	1.80	0.0734	0.0148
hospw2	-0.00454063	0.006422	0.004734	-0.959	0.3386	0.0042
mhoutw1	-1.20100	0.3831	0.4093	-2.93	0.0037	0.0383
mhoutw2	1.26193	0.3903	0.4420	2.86	0.0047	0.0364
goferw3	-0.0158404	0.006980	0.005756	-2.75	0.0064	0.0339
fdferw3	0.0147139	0.004684	0.003201	4.60	0.0000	0.0891
trrepw2	0.00812814	0.002751	0.002663	3.05	0.0026	0.0414
evacselfr	-1.05776	0.3268	0.3379	-3.13	0.0020	0.0434
airw1	0.00671090	0.002608	0.002338	2.87	0.0045	0.0367
radw1	-0.0142683	0.002840	0.002743	-5.20	0.0000	0.1113
radtlw3	-0.0357084	0.002971	0.003232	-11.0	0.0000	0.3611
radhlw3	-0.0190519	0.005414	0.006575	-2.90	0.0041	0.0374
radfmw3	0.0102532	0.005407	0.006743	1.52	0.1298	0.0106
source	0.174867	0.05289	0.05719	3.06	0.0025	0.0415
dafter	-0.00245213	0.01767	0.01201	-0.204	0.8385	0.0002
medw2	0.00866203	0.003004	0.003056	2.83	0.0050	0.0359
cloud	-0.00735021	0.002581	0.002768	-2.66	0.0085	0.0316
chsize	-0.00969579	0.003040	0.003293	-2.94	0.0036	0.0386
HP2probsoc	-0.702894	0.2902	0.2807	-2.50	0.0130	0.0282
HP2inthob	0.427081	0.2689	0.2272	1.88	0.0615	0.0161
BSIsoma	0.154072	0.02037	0.02671	5.77	0.0000	0.1335
BSIhos	-0.0898308	0.03126	0.03985	-2.25	0.0252	0.0230
bf12	0.0108460	0.003499	0.003406	3.18	0.0017	0.0448
bf22	0.000638930	2.965e-05	3.473e-05	18.4	0.0000	0.6105
cumdose1	1.98759	0.6911	0.6521	3.05	0.0026	0.0412
cumdose2	-0.759593	0.2981	0.2730	-2.78	0.0059	0.0346

#### Omnibus model fit statistics

sigma	1.18433	RSS	302.969341
log-likelihood	-390.795		
no. of observations	262	no. of parameters	46
mean(icdxcnt)	3.19466	se(icdxcnt)	2.46436
When the log-likeli	hood consta	nt is NOT included:	
AIC	0.496432	SC	1.12294
HQ	0.748237	FPE	1.64890
When the log-likeli	hood consta	nt is included:	
AIC	3.33431	SC	3.96081
HQ	3.58611	FPE	28.1623
Normality test: C	hi^2(2) =	16.293 [0.0003]**	
Hetero test: F	(75,186) =	1.4124 [0.0322]*	
RESET23 test: F	(2,214) =	6.9797 [0.0012]**	

We should mention that in both cross-sectional regression analyses, all assumptions necessary for statistical congruency with the theory were violated. Therefore, we would do better to be careful about placing too much faith in all the details of these models.

#### 6 Female model direct effects on abortions

#### 6.1 Female path model

## 6.1.1 Hypothesis 9: Radiation dose directly explains number of medically diagnosed illnesses

This hypothesis pertains to the female model insofar as it deals with voluntary abortions. To address this hypothesis and the next one, we use a structural equation path model, along with a clustered-robust structural path model, the path diagram for which is displayed in Figure 2.

The boxes in the path diagram are color coded to help interpret the diagram. The boxes representing the variables have "w" suffixes referring to the wave to which they refer. Wave 1 is 1986, wave 2 is the decade from 1987-1996, inclusive. Wave 3 is the years from 1997 to the time of the interview. Any variable without a wave reference refers to any and all waves. Cumulative dose variables are represented by rose colored boxes, with red direct effects. Perceived risk of exposure to Chornobyl radiation exposure are depicted by orange colored boxes whose direct effects are symbolized by dark orange arrows. Fear of consuming contaminated food and fluids are cyan boxes with medium blue arrows indicated their direct effects. The light gray boxes represent voluntary abortions with purple direct effects. The gold box is the number of medically diagnosed illnesses with forest green direct effects. The gold box is a Nottingham sleep box with purple direct effects, the Nottingham energy level variable is represented by a yellow box with olive direct effects, whereas the Nottingham physical ability scale is indicated by a mint colored box with mint colored arrows.

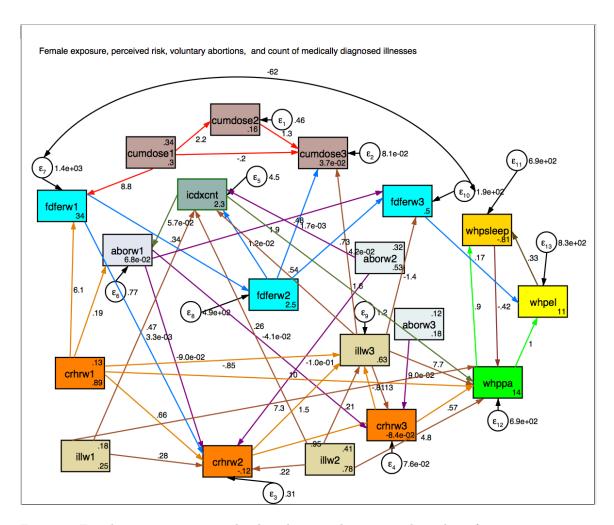


Figure 2: Female exposure, perceived risk, voluntary abortions and number of medically diagnosed illnesses

Table 5: Female Path model parameter estimates

The model output supporting this path diagram is included in the Table 5. The clustered robust direct effects for hypothesis testing follows in Table 6. If we examine

(1 observations with missing values excluded;
specify option 'method(mlmv)' to use all observations)

Endogenous variables

Observed: cumdose2 cumdose3 fdferw1 crhrw2 crhrw3 aborw1 illw3 icdxcnt

fdferw3 fdferw2

Exogenous variables

Observed: cumdose1 crhrw1 aborw2 aborw3 illw1 illw2

Structural equation model Number of obs = 362

Estimation method = ml

Log likelihood = -9550.3837

		OIM				
	Coef.	Std. Err.	z	P> z	[95% Conf.	Interval]
Structural cumdo~2 <-						
cumdose1	2.188894	.0649526	33.70	0.000	2.061589	2.316199
_cons	.1613576	.0418234	3.86	0.000	.0793853	.2433299
cumdo~3 <-						
cumdose2	1.314717	.0221427	59.37	0.000	1.271318	1.358116
illw3	.0419176	.0130489	3.21	0.001	.0163424	.0674929
fdferw2	.0016627	.0005875	2.83	0.005	.0005112	.0028143
cumdose1	201453	.0554729	-3.63	0.000	3101778	0927282
_cons	.0366882	.020967	1.75	0.080	0044063	.0777827
fdferw1 <-						
cumdose1	8.753535	3.523999	2.48	0.013	1.846624	15.66045
crhrw1	6.059022	2.054354	2.95	0.003	2.032563	10.08548
_cons	33.6814	2.286827	14.73	0.000	29.1993	38.1635
crhrw2 <-						
fdferw1	.0034748	.0007769	4.47	0.000	.0019522	.0049974
aborw1	1068016	.0329075	-3.25	0.001	1712992	042304
crhrw1	.6594244	.0322813	20.43	0.000	.5961542	.7226945
aborw2	1003075	.0393379	-2.55	0.011	1774084	0232066
illw1	.2582233	.0603799	4.28	0.000	.1398808	.3765657
illw2	.2110993	.0338218	6.24	0.000	.1448097	.2773889
_cons	1179535	.0454289	-2.60	0.009	2069924	0289145
crhrw3 <-						
crhrw2	1.095005	.051542	21.24	0.000	.9939849	1.196026
illw3	.0706981	.0142521	4.96	0.000	.0427646	.0986316
crhrw1	1190519	.0379068	-3.14	0.002	1933479	0447559
aborw3	.0930538	.0341644	2.72	0.006	.0260927	.1600149
_cons	0619315	.0187248	-3.31	0.001	0986315	0252315

Table 5 of Female Model of Number of Medical diagnoses- continued:

		OIM				
	Coef.	Std. Err.	z	P> z	[95% Conf.	<pre>Interval]</pre>
aborw1 <-						
icdxcnt	.0599619	.0201282	2.98	0.003	.0205113	.0994124
crhrw1	.1937856	.0491656	3.94	0.000	.0974228	.2901483
_cons	.0573689	.0796464	0.72	0.471	0987352	.2134731
illw3 <-						
crhrw2	.6326686	.0885617	7.14	0.000	.4590909	.8062462
crhrw1	7649751	.0799265	-9.57	0.000	9216281	6083221
illw2	. 1856439	.0637737	2.91	0.004	.0606498	.310638
_cons	.6285453	.0585738	10.73	0.000	.5137428	.7433478
icdxcnt <-						
illw3	.5447237	.102339	5.32	0.000	.3441429	.7453044
fdferw2	.0118556	.0044208	2.68	0.007	.003191	.0205202
aborw2	.4824779	.1549793	3.11	0.002	.1787241	.7862317
illw1	.4690125	.2310932	2.03	0.042	.0160781	.9219468
illw2	.2521321	.1344499	1.88	0.061	0113847	.515649
_cons	2.262121	.1607007	14.08	0.000	1.947153	2.577089
fdferw3 <-						
aborw1	1.886615	.8010501	2.36	0.019	.3165854	3.456644
illw3	-1.447259	.621503	-2.33	0.020	-2.665382	2291355
fdferw2	.7301559	.0324097	22.53	0.000	.6666342	.7936777
_cons	.5025388	1.008382	0.50	0.618	-1.473853	2.47893
fdferw2 <-						
fdferw1	.3398738	.0307564	11.05	0.000	.2795923	.4001552
_cons	2.493675	1.636532	1.52	0.128	7138674	5.701218
Variance						
e.cumdose2	.4605615	.0342333			.398124	.5327911
e.cumdose3	.0809005	.0060133			.0699329	.093588
e.fdferw1	1370.578	101.8784			1184.764	1585.534
e.crhrw2	.3111668	.0231746			.2689049	.3600707
e.crhrw3	.0774229	.0068501			.0650967	.0920832
e.aborw1	.7684275	.0571281			.6642339	.8889651
e.illw3	.9925255	.0737764			.8579662	1.148188
e.icdxcnt	4.529445	.3366919			3.915361	5.239842
e.fdferw3	188.6199	14.09157			162.9278	218.3634
e.fdferw2	489.7427	36.40229			423.3492	566.5488
Covariance						
e.fdferw1						
e.fdferw3	-62.16211	31.18092	-1.99	0.046	-123.2756	-1.048643
e.crhrw2						
e.crhrw3	0375643	.0178112	-2.11	0.035	0724736	002655

LR test of model vs. saturated: chi2(72) = 88.19, Prob > chi2 = 0.0944

Stability analysis of simultaneous equation systems

Eigenvalue stability condition stability index = .2167462
All the eigenvalues lie inside the unit circle.

Table 6: Female Path model clustered robust direct effects

Before proceeding to a discussion of the direct effects and the hypotheses they test, we should mention that the model fits the data well. The LR test of model vs. saturated:  $\chi^2(72) = 88.19$ ,  $Prob > \chi^2 = 0.0944$ . Moreover, the model is a stable on. The stability index = .2167462 so all moduli reside within the unit circle, satisfying the condition for model stability.

Direct effects

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

		JG)	a. Err.	adjusted 10	or 362 clusters in id)
		Robust			
	Coef.	Std. Err.	z	P> z	Std. Coef.
Structural					
cumdo~2 <-					
cumdose1	2.188894	.0836046	26.18	0.000	.8708001
cumdo~3 <-					
cumdose2	1.314717	.1775052	7.41	0.000	1.038299
fdferw1	0	(no path)			0
crhrw2	0	(no path)			0
aborw1	0	(no path)			0
illw3	.0419176	.0271581	1.54	0.123	.0278226
icdxcnt	0	(no path)			0
fdferw2	.0016627	.0007575	2.20	0.028	.0243433
cumdose1	201453	.3961209	-0.51	0.611	0632933
crhrw1	0	(no path)			0
aborw2	0	(no path)			0
illw1	0	(no path)			0
illw2	0	(no path)			0
fdferw1 <-					
cumdose1	8.753535	3.105798	2.82	0.005	.1271528
crhrw1	6.059022	2.116446	2.86	0.004	.1511972
crhrw2 <-					
fdferw1	.0034748	.0007847	4.43	0.000	.1515927
crhrw2	0	(no path)			0
aborw1	1068016	.0262047	-4.08	0.000	1111255
illw3	0	(no path)			0
icdxcnt	0	(no path)			0
fdferw2	0	(no path)			0
cumdose1	0	(no path)			0
crhrw1	.6594244	.0368569	17.89	0.000	.7178849
aborw2	1003075	.0391308	-2.56	0.010	0840425
illw1	. 2582233	.0592224	4.36	0.000	.1480249
illw2	.2110993	.0600579	3.51	0.000	.2157106

 ${\tt Table\ 6\ of\ Female\ direct\ effects\ on\ Number\ of\ Medical\ diagnoses-\ continued:}$ 

		Robust			
	Coef.	Std. Err.	z	P> z	Std. Coef.
crhrw3 <-					
fdferw1	0	(no path)			0
crhrw2	1.095005	.0639497	17.12	0.000	1.071873
aborw1	0	(no path)			0
illw3	.0706981	.0178725	3.96	0.000	.0926496
icdxcnt	0	(no path)			0
fdferw2	0	(no path)			0
cumdose1	0	(no path)			0
crhrw1	1190519	.0464989	-2.56	0.010	1268684
aborw2	0	(no path)			0
aborw3	.0930538	.0557219	1.67	0.095	.0439917
illw1	0	(no path)			0
illw2	0	(no path)			0
aborw1 <-					
fdferw1	0	(no path)			0
crhrw2	0	(no path)			0
aborw1	0	(no path)			0
illw3	0	(no path)			0
icdxcnt	.0599619	.0231791	2.59	0.010	.1538714
fdferw2	0	(no path)			0
cumdose1	0	(no path)			0
crhrw1	.1937856	.0554688	3.49	0.000	.2027567
aborw2	0	(no path)			0
illw1	0	(no path)			0
illw2	0	(no path)			0
illw3 <-					
fdferw1	0	(no path)			0
crhrw2	.6326686	.1148443	5.51	0.000	.4725716
aborw1	0	(no path)	0.01	0.000	0
illw3	0	(no path)			0
icdxcnt	0	(no path)			0
fdferw2	0	(no path)			0
cumdose1	0	(no path)			0
crhrw1	7649751	.1182343	-6.47	0.000	6220546
aborw2	0	(no path)	0.11	0.000	.0220340
illw1	0	(no path)			0
illw2	.1856439	.0936275	1.98	0.047	.1416958
	1000409	.0000210	1.50	0.01	.1410350

Table 6 of Female Model of Number of Medical diagnoses- continued:

				0	
		Robust			
	Coef.	Std. Err.	Z	P> z	Std. Coef.
icdxcnt <-					
fdferw1	0	(no path)			0
crhrw2	0	(no path)			0
aborw1	0	(no path)			0
illw3	.5447237	.0694747	7.84	0.000	.2731281
icdxcnt	0	(no path)			0
fdferw2	.0118556	.0051406	2.31	0.021	.13112
cumdose1	0	(no path)			0
crhrw1	0	(no path)			0
aborw2	.4824779	.1846312	2.61	0.009	.1513996
illw1	.4690125	.1626082	2.88	0.004	.1006944
illw2	.2521321	.1373089	1.84	0.066	.0964927
fdferw3 <-					
fdferw1	0	(no path)			0
crhrw2	0	(no path)			0
aborw1	1.886615	1.43568	1.31	0.189	.074969
illw3	-1.447259	.5740939	-2.52	0.012	0739975
icdxcnt	0	(no path)			0
fdferw2	.7301559	.0550973	13.25	0.000	.8234611
cumdose1	0	(no path)			0
crhrw1	0	(no path)			0
aborw2	0	(no path)			0
illw1	0	(no path)			0
illw2	0	(no path)			0
fdferw2 <-					
fdferw1	.3398738	.0395434	8.59	0.000	.5021123
cumdose1	0	(no path)			0
crhrw1	0	(no path)			0

In the medical diagnosis count panel of Table 6, we observe no direct effects from either cumulative external dose (hypothesis 2) or perceived risk (hypothesis 7).

#### 6.1.2 Hypothesis 9

Hypothesis 9 suggests that there are dose direct effects on abortions. Therefore, we have to look in the direct effects table under the abortions panel on page 20 for evidence of a path from cumulative dose. We find none and therefore infer that this hypothesis is inconsistent with the data.

## 6.1.3 Hypothesis 13: Perceived risk directly explains female voluntary abortions

Yet a review of Table 6.1.1 on page 13 shows no paths from any wave of cumulative external dose to female voluntary abortions. For example, in the voluntary abortions panel in wave 1 (aborw1 panel) on about page 18, we find a direct

Table 7: Female clustered robust Indirect effects

effect from crhrw1 crhrw1 stdized  $\beta = -0.127$ , p = 0.010).. Therefore, we conclude that Hypothesis 13 is partially consistent with our data because only in wave 1 do we find such a path.

#### 7 Female Model Indirect effects

To review the female indirect effects we examine the table of indirect effects from two vantage points: From the point of the target endogenous variable, abortions, and from the panel of the hypothesized mediator—count of medical diagnosed ailments.

## 7.0.4 Hypothesis 17: medical illness count mediates an external dose abortion relationship

This hypothesis postulates that radiation dose indirectly predicts voluntary abortions mediated by the number of medically diagnosed illnesses.

## 7.0.5 Hypothesis 21: medical illness count mediates the perceived risk abortion relationship

This hypothesis postulates that perceived risk indirectly effects voluntary abortions while being mediated by the number of medically diagnosed illnesses.

		(St	d. Err.	adjusted	for 362 clusters in id)
	Coef.	Robust Std. Err.	z	P> z	Std. Coef.
Structural					
cumdo~2 <-					
cumdose1	0	(no path)			0
cumdo~3 <-					
cumdose2	0	(no path)			0
fdferw1	.0006564	.0000708	9.27	0.000	.0141971
crhrw2	.0264616	.0048034	5.51	0.000	.0131192
aborw1	0028261	.0006934	-4.08	0.000	0014579
illw3	0000923	.0000118	-7.84	0.000	0000613
icdxcnt	0001695	.0000655	-2.59	0.010	0002243
fdferw2	-2.01e-06	8.71e-07	-2.31	0.021	0000294
cumdose1	2.883522	.4074403	7.08	0.000	.905956
crhrw1	0111166	.0091448	-1.22	0.224	006
aborw2	0027361	.0021867	-1.25	0.211	0011365
illw1	.0067535	.004901	1.38	0.168	.0019194
illw2	.0133079	.0099226	1.34	0.180	.006742

cumdose1	0 (no	path)	0
crhrw1	0 (no	path)	0

Table 7 Female Indirect effects---continued:

		Robust			
	Coef.	Std. Err.	z	P> z	Std. Coef
crhrw2 <-					
fdferw1	0000334	3.60e-06	-9.27	0.000	001457
crhrw2	0022022	.0003997	-5.51	0.000	002202
aborw1	.0002352	.0000577	4.08	0.000	.000244
illw3	0034807	.0004439	-7.84	0.000	004659
icdxcnt	0063899	.0024701	-2.59	0.010	017061
fdferw2	0000758	.0000328	-2.31	0.021	002237
cumdose1	.0301244	.0129844	2.32	0.020	.019090
crhrw1	.001411	.0117009	0.12	0.904	.001536
aborw2	0028621	.0020782	-1.38	0.168	00239
illw1	0035656	.0022471	-1.59	0.113	00204
illw2	0027222	.0017641	-1.54	0.123	002781
crhrw3 <-					
fdferw1	.0039223	.000892	4.40	0.000	.167499
crhrw2	.0422186	.0076637	5.51	0.000	.041326
aborw1	1214574	.0298006	-4.08	0.000	123704
illw3	0039671	.000506	-7.84	0.000	005198
icdxcnt	0072828	.0028153	-2.59	0.010	019034
fdferw2	0000863	.0000374	-2.31	0.021	002495
cumdose1	.0343338	.0149175	2.30	0.021	.021298
crhrw1	.6990942	.0607879	11.50	0.000	.744993
aborw2	1175859	.0439989	-2.67	0.008	09643
aborw3	0	(no path)			
illw1	.290242	.0614378	4.72	0.000	.162864
illw2	.2506192	.0695552	3.60	0.000	.250683
aborw1 <-					
fdferw1	.0003127	.0000337	9.27	0.000	.013112
crhrw2	.0206191	.0037429	5.51	0.000	.019816
aborw1	0022022	.0005403	-4.08	0.000	002202
illw3	.0325907	.0041567	7.84	0.000	.04193
icdxcnt	000132	.000051	-2.59	0.010	000338
fdferw2	.0007093	.0003076	2.31	0.021	.020131
cumdose1	.0027375	.0015074	1.82	0.069	.001667
crhrw1	0098663	.0051401	-1.92	0.055	01032
aborw2	.0267983	.0170058	1.58	0.115	.021579
illw1	.0333853	.017801	1.88	0.061	.018393
illw2	.025488	.0144102	1.77	0.077	.025031

Table 7 Female Indirect effects---continued:

		Robust			
	Coef.	Std. Err.	z	P> z	Std. Coef
illw3 <-					
fdferw1	.0021773	.0004952	4.40	0.000	.070949
crhrw2	0013932	.0002529	-5.51	0.000	001040
aborw1	0674212	.0165424	-4.08	0.000	052399
illw3	0022022	.0002809	-7.84	0.000	002202
icdxcnt	0040427	.0015628	-2.59	0.010	008062
fdferw2	0000479	.0000208	-2.31	0.021	001057
cumdose1	.0190588	.0087984	2.17	0.030	.009021
crhrw1	.4180898	.0815523	5.13	0.000	.339977
aborw2	0652722	.0273323	-2.39	0.017	040849
illw1	.1611139	.0508709	3.17	0.002	.068986
illw2	.1318337	.0396335	3.33	0.001	.100624
icdxcnt <-					
fdferw1	.0052154	.0005628	9.27	0.000	.085215
crhrw2	.3438706	.0624207	5.51	0.000	.128788
aborw1	0367259	.009011	-4.08	0.000	014311
illw3	0011996	.000153	-7.84	0.000	000601
icdxcnt	0022022	.0008513	-2.59	0.010	002202
fdferw2	0000261	.0000113	-2.31	0.021	000288
cumdose1	.0456532	.0214625	2.13	0.033	.010835
crhrw1	1645425	.0494464	-3.33	0.001	067088
aborw2	0355553	.0162838	-2.18	0.029	011157
illw1	.0877626	.0316495	2.77	0.006	.018842
illw2	.1729375	.059496	2.91	0.004	.066184
fdferw3 <-					
fdferw1	. 2455998	.0288682	8.51	0.000	.409202
crhrw2	8747185	.1587821	-5.51	0.000	033406
aborw1	.0934213	.0229217	4.08	0.000	.003712
illw3	.0646733	.0082485	7.84	0.000	.003306
icdxcnt	.1187267	.0458954	2.59	0.010	.012106
fdferw2	.0014076	.0006103	2.31	0.021	.001587
cumdose1	2.149866	.819284	2.62	0.009	.052031
crhrw1	2.35263	.6698573	3.51	0.000	.09781
aborw2	.1450239	.0713757	2.03	0.042	.004640
illw1	1701884	.1326033	-1.28	0.199	003725
illw2	4113862	. 2384233	-1.73	0.084	016054
fdferw2 <-					
fdferw1	0	(no path)			
cumdose1	2.975097	1.08866	2.73	0.006	.06384
crhrw1	2.059303	.7588785	2.71	0.007	.07591

Table 8: Tabular summary of Direct effects hypothesis test results

Hyp #	Endog.var	Exog var	Interp	Gender	Confirmation	model
2	icdxcnt	dose	strict	male	none	regression
2	icdxcnt	dose	strict	female	partial	regression
7	icdxcnt	perceived risk	strict	male	partial	regression
7	icdxcnt	perceived risk	strict	female	partial	regression
2	icdxcnt	dose	strict	male	none	path
7	icdxcnt	perceived risk	strict	male	partial	path
2	icdxcnt	dose	strict	female	none	path
7	icdxcnt	perceived risk	strict	female	none	path
9	abortions	dose	strict	female	none	path
13	abortions	perceived risk	strict	female	partial	path

## 8 Recapitulation of Direct effect Hypothesis test results

# 9 Recapitulation of Indirect effect hypothesis test results

The only abortion panel in the female indirect effects table on about page 23. We find that there are indirect paths originating with cumulative external dose in wave 1 and in perceived risk in waves 1 and 2. Therefore, there is a basis for examining the indirect path further. If the number of medical diagnosed illnesses mediates the source terms and voluntary abortions, there will be indirect paths from those sources found within the number of diagnosed illnesses (icdxcnt) panel on page 24.

## 9.1 Hypothesis 17: medical illness count mediates an external dose abortion relationship

In the icdxcnt panel, we find significant indirect effects originating with cumulative external dose in wave 1 (cumdose1 stdized  $\beta=0.011,\ p=0.033$ ). Therefore we have evidence of a significant indirect path from cumulative external dose to the count of medically diagnosed illnesses. From the abortion panel on the previous page, we have significant path originating in the number of medically diagnosed illnesses extending to voluntary abortions icdxcnt stdized  $\beta=-0.0003,\ p=0.010$ ). Therefore, we can conclude that there is partial support for hypothesis 17 in the female data. This is partial because we only have evidence of it originating in wave 1.

## 9.2 Hypothesis 21 medical illness count mediates the perceived risk abortion relationship

In order to find evidence for hypothesis 21, we have to follow the same procedure, first examining the abortion panel and looking for evidence of an indirect path. Then we have to return to the icdxcnt panel and search for evidence of significant paths from perceived risk.

In the female indirect effects abortion panel for wave 1, we indeed find evidence of a significant paths originating the hypothesized mediator - the count of medically diagnosed illnesses (icdxcnt stdized  $\beta = -.0003$ , p = 0.010)..

In the female indirect effects icdxcnt panel on the next page, we find evidence of statistically significant perceived risk indirect paths originating in waves 1 and 2. crhrw1 stdized  $\beta=-0.067$ , p=.001) and (crhrw2 stdized  $\beta=-0.001$ , p=0.000). Therefore, there appears to be evidence in partial support of Hypothesis 21 stemming from two of the three waves. Hypothesis 21 is not inconsistent with all of our female data. We have partial evidentiary support for Hypothesis 21.

Table 9: Tabular summary of Indirect effects hypothesis test results

	Hyp #	Endog.var	Exog var	Interp	Gender	confirmation	effects confirmed
ſ	17	abortions	dose	strict	female	partial	path
	19	abortions	perceived risk	none	female	partial	path model

Table 10: Files on which this analysis is based

File type	Filename		Gender
PDF report	H2H7icdxcnt.pdf	this report	
dataset	chwide16sep2012.dta	latest data	both
path diagram	icdxcntMaleV2.pdf	pdf	male
path diagram	icdxcntFemV2.pdf	pdf	female
AutoMet Output	H2H7AutoMetrics.out	OLS regression	
AutoMet output	abortions	dose	female
stata output	abortions	perceived risk	female
stata output	H2H7H9H13icd9cntAbortns.pdf	dose and pcvd risk	female
stata output	H2H7H9H13icd9cntAbortns.smcl	dose and pcvd risk	female
stata output	H2H7H9H13icd9cntAbortns.pdf	dose and pcvd risk	female
stata output	maleIcdxcntWPv2.pdf	output	male path model
sembuilder	icdxcntMaleV2.stsem	male	structural equation path mode
sembuilder	aborICDcntFemV2.stsem	female	structural equation path mode

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