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# Is Chernobyl radiation the main cause of cancer deaths?
## The research on cancer mortality before and after 1986

**Abstract**

**T**he Chernobyl catastrophe is still referred to as the biggest radioactivity fallout problem in the history of the world. The cloud that was released from the broken reactor traveled across larger parts of the European continent and the after-effects of the radiation are said to be felt and faced throughout the impacted regions up to this day – and that this process is far from over.

This thesis aims at examining the size as well as the significance of the effects of cancer-related mortality on people after the catastrophe in 1986 compared to the cancer deaths that occurred before the Chernobyl accident. The time period that is used in this paper spans from 1966 up to 2005, which equals to twenty years before and twenty years after the reactor explosion. Within this period two countries are examined. Poland is used as the treatment group as that country is located very close to the danger zone, whereas Spain is used as a control group because it is located outside of the impacted area. Two cancer types are examined within both of these countries. Lung cancer is examined as the one caused by radiation fallout, while stomach cancer is used as the one with the development not impacted by the radioactive particles. Moreover, a number of other factors, besides radiation, is used to statistically establish whether or not the radiation is the sole and significant determinant of cancer. These are: smoking habits, obesity and air pollution.

The results show that the Chernobyl's radiation still affects lung cancer mortality of people in Poland, but to a much lesser degree than the polluted air or even heavy smoking. Stomach cancer in Poland is caused by a combination of all of the modeled variables. Spanish lung cancer mortality is also affected by heavy smoking and air pollution, whereas stomach cancer deaths in this country are caused mainly by excessive nicotine intake.

Keywords: Cancer; Chernobyl disaster; mortality; lung; stomach; radioactivity; fallout.

# Contents

# **1.** Introduction

**T**he catastrophe in Chernobyl came to be a known and popular topic in the media, politics and various discussion panels. Whenever a topic of radiation is concerned, the accident in the Ukrainian nuclear power plant is brought up as the biggest accident in terms of fallout and as the one with voluminous long-term consequences and after-effects. The radiation impacts the health and lives of flora and fauna as well as human beings in the contaminated regions.

As the radioactive cloud spread over a large part of Europe, the soil in those regions was impacted by the particles. In turn, many plants, including fruits and vegetables, were deemed inedible and new methods of growing and cleaning had to be established. In many regions it is of continuous danger to pick up berries and mushrooms. A fact that is worth mentioning is that the Ukrainian Worm Wood Forest, which was the name of the pine forest surrounding the area of the power plant and the nearby city of Pripyat, had to have its name changed to Red Forest as the trees turned their color to a dead brownish red after the catastrophe. The forest is located in the alienation zone, which is the area of 30-kilometer radius around the Chernobyl Nuclear Power Plant (CNPP). This zone is referred to as the area where the doses of radiation are still the biggest, most dangerous and quite active. Animals were also the victims of the radiation. It is still said that the radiation particles are present in the earth of many regions where the cloud flew over. Because of the contamination a number of fish and birds have been severely mutated or their development has been stunted. Many species of boar and deer may still be under the radioactive influence on many impacted territories. Similar methods, as in the case of plants, had to be created in order to make animal products safe for the market and people again. Many farms in the contaminated areas still, after all those years, have restrictions put on them in terms of which kinds of livestock (especially cows and sheep) that can be sent out onto the market.

Human beings were impacted by radiation as well. Many deaths, cancer types and chromosomal changes were registered in the years following the catastrophe in the reactor number four. The most prevalent types of cancers that are connected to the radiation fallout are thyroid and leukemia. Other common types include lung cancer as well as prostate, rectum and breast ones. Apart from cancers, a big number of other non-cancer diseases and problems were also introduced as being connected to the fallout influence. Among these, issues with the respiratory and digestive systems are worth mentioning. In addition, radiated people were often diagnosed with lymphatic illnesses and even abnormal aging. According to the Greenpeace report (2006), people living in the contaminated areas (especially in Belarus and Ukraine) can have their biological age exceeding the natural calendar one by up to nine years. There was also a magnitude of Down syndrome cases among children born in Poland, Germany and Austria between 1987 and 1989. "A statistically significant increase in frequency was apparent in January 1987, corresponding to children conceived during the period of maximal Chernobyl fallout." (Greenpeace 2006, p. 131).

## ***1.1.*** *The story of the accident*

**T**he Chernobyl disaster occurred at the Chernobyl Nuclear Power Plant in Ukraine. "The plant was located approximately 100 km north of the Ukrainian capital Kiev, near the border with Belarus." (Rosen 2006, p. 1). On 26$^{th}$ April 1986, Saturday, around 1:23 at night, reactor number four suffered a dangerous power increase, which led to the explosion in the reactor's core. When the reactor's graphite moderator was exposed to the atmosphere it went aflame.

The resulting smoke turned into a nuclear cloud. At the same time the reactor itself emitted large quantities of fuel and core materials into the air. Since the reactor was not encased in any type of protection, the pollution was able to spread quite easily. It is a known fact that the whole accident took place, ironically, during a scheduled test of the reactor's emergency core cooling unit.

The contamination spread over large parts of the European continent in a short matter of time. It is said that the cloud traveled westward as far as Austria and Switzerland, northward it reached Scandinavia, while in the south it spread to Italy and Greece. Clark (1986) wrote that the deposition of radionuclides took place in two ways during the passage of the cloud: dry and wet; the latter one is connected with rainfall. "Radioactive fallouts from the Chernobyl clouds touched many territories, where more than three billion people live." (Greenpeace 2006, p. 8). Then it is added in that report that more than half of the territories in thirteen nations were contaminated by the radiation from the power plant and about one-third was in eight additional nations. Most of Eastern European countries experienced several days of very clear and sunny weather during the several days following that 26th April. Only later, it became known to people in these nations that such cloudless sky was caused by the fact that the radiation cloud was passing by.

**Figure 1.1.1** Map of the area majorly contaminated by the Chernobyl cloud
Source: http://www.our-energy.com/chernobyl_disaster.html (accessed: 04.05.2011)


There was no information and no media coverage on the subject from the Soviet Union's side until the third day after the catastrophe, so the effects of the fallout were already taking place way before any sort of preventive methods were put into action. Soviet Union tried to hush this event up and was succeeding until the alarms of high radiation levels were set off in the Swedish power plant located in Forsmark (with the distance of over one thousand kilometers from Chernobyl). Only then Soviet authorities began to spread the news about the accident along with the hints that by the 29th April emissions were far from over. The preventive actions in all of the impacted nations were mostly about giving people doses of iodine, so as to protect their bodies and organisms from absorbing the contaminated iodine from the radiation. The most common version of this iodine is called the Lugol's solution, which is a mixture elemental iodine and potassium iodide in water. Polish people were advised by their

government, contrary to what the Soviet authorities were ordering, to drink that solution for protection against the radioactive iodine in the first days after the catastrophe[1].

The catastrophe had the biggest effect, of course, on those that were in close proximity to the broken reactor. Workers who were present in the plant during the explosion died instantly. Many of the firefighters brought in to extinguish the flames that were injured during this action died while being hospitalized. Some of them died from large and acute doses of radiation. The inhabitants of Pripyat were ordered to evacuate the city as late as the second day after the disaster. About 100000 people had to be relocated behind the borders of the danger zone. There is also a known case of the "liquidators", the circa 300 000 up to 600 000 clean-up workers that were recruited from the Soviet and Baltic nations to dispose of the debris and to clean the overall alienation area. A lot of workers were experiencing major health problems after the work was done, which was the result of the radiation doses received by their bodies despite wearing the protective gear. This was especially true for those that entered in close contact with the power plant. In some cases, even the offspring of these people had some physical mutations. In summary, there were about 31 immediate casualties: 28 from maximum exposure to radiation, two from the explosion and one from a heart attack. In addition, 238 were diagnosed with injuries caused by acute radiation. No one can tell for sure, though, how many additional radiation victims were and still are out there on the contaminated territories. "In addition to the lack of reliable information provided to the people affected in the first few years after the accident, there was widespread mistrust of official information and the false attribution of most health problems to radiation exposure from Chernobyl." (World Health Organization 2006, Fact sheet N$^{o}$ 303).

Nonetheless, the effects of the catastrophe on human health, because of the traveling cloud, are said to be visible up to the present times and in a quite big area. It also seems very plausible to imply that the after-effects of radiation will continue to influence us in the upcoming future. "(…) it is reasonable to conclude that the Chernobyl accident has caused, and will continue to cause, a significant amount of morbidity and mortality across Europe (…) and beyond." (Greenpeace 2006, p. 135). The aim of this thesis is to examine how big and significant were and are the effects of cancer mortality on people after the catastrophe compared to the cancer deaths that occurred before the Chernobyl accident.

### ***1.2.*** *Problems and discrepancies*

**M**y initial idea was to write about and research thyroid and leukemia cancers since those are the most prominent types caused by radiation. The data for leukemia was complete in terms of observations, but had to be put aside as there are no major and known deterministic factors other than radiation that are causing this cancer type. On the other hand, the data for thyroid cancer mortality in Poland was quite incomplete. This resulted in more than twenty years worth of yearly observations that were missing from my dataset.

I started to pursue this problem wanting to find the missing thyroid observation and as the result I found out that it might have been a combined effect of the government's cover ups and strikes in the medical industry that stood for the greater part of missing observations. The strikes of the medical staff took place in the late 1990s. Cover ups about the death toll, radiation spread and its consequences were done during the Soviet times and especially in the

[1] This fact is given here courtesy of my parents.

post-Chernobyl days. These resulted in missing or misleading information. This is why one may get varying estimates of the mortality figures depending on the sources. This is quite apparent in the above paragraph in which I mention the Pripyat evacuation, “liquidators” and figures on deaths – one can never be completely sure about these numbers.

There are two sides to the nuclear power plant argument. This also adds to the confusion in terms of data and information. Scientists are apt to say that even though the effects of Chernobyl were big and grave at the time, it is mostly in the past by now. They may also add that nuclear energy is still in big demand. Environmental agencies, on the contrary, will state that the health consequences are very far from being over and that all of the power plants should be shut down and closed. This is another aim of my thesis: to see which of the two sides of this argument is more correct in its assumptions about the after-effects of the Chernobyl disaster.

## **2.** Data

**T**he time period that is examined in this paper spans between 1966 and 2005, which makes up to twenty years before and twenty years after the catastrophe. Since the accident in Chernobyl occurred quite early in the first half of the year 1986, so for simplicity reasons this year is placed in the "after" period. I am using a dummy variable for indication of these two separate periods. Poland is researched as the treatment group as it is located in the contaminated area as well as quite close to the power plant (CNPP was positioned about 450 kilometers from the eastern Polish border). Spain, on the other hand, is used a control group as this country was not impacted by the radiation from the Chernobyl cloud.

In both nations two genders are examined along with mortality because of two types of cancer per each gender. These chosen cancer types are stomach cancer and lung cancer. Lung cancer is researched as it is a type that is clearly caused by radiation. Stomach cancer can also be caused by radiation, but to a much lesser degree; it is, thus, used for comparison purposes. "The total predicted number of cases possibly attributable to Chernobyl in Europe (whose population were more 570 million people in 1986) up to 2065 is large in absolute terms, about 23 000 for all cancers excluding leukemia, thyroid cancer and nonmelanoma skin cancer (…)." (Cardis et al. 2006, p. 1230). In order to determine whether or not the radiation is a sole and major factor contributing to cancer mortality, additional variables are furthermore used for both countries. Overweight people are more probable to be the victims of the stomach cancer – obesity can be, thus, treated as a deterministic factor of that cancer type. People living in the areas with high air pollution are running a higher risk of being diagnosed with lung cancer – air pollution can, as such, be treated as the deterministic factor of that type of cancer. In addition, both chosen cancer types are said to be caused by excessive smoking and nicotine intake[2]. It has to be noted that general mortality numbers for both genders in both countries are also, of course, incorporated into the analysis.

In addition, age-specific mortality rates for each cancer type per each gender in each of the two nations are included. This is done in order to get a better and more specific outlook on the differences between the two periods. There are only three age groups that are used: 0-29 years, 30-59 years and 60-85+. The latter group may seem to be the broadest, but as the age progresses the number of observations gets rapidly smaller. For people at the age of 90+, mortality in both countries decreases to very small single-digit figures. Why only three age groups? It is done so for the purpose of using chi-square cross-tabulations testing method; more about this is explained in the Methods section.

### ***2.1.*** *Dataset sources*

**G**eneral mortality rates for Poland and Spain are taken from website that belongs to Human Mortality Database (HMD). All of the data for cancer mortality along with age-specific observations is taken from the International Agency for Research on Cancer (IARC) website. IARC is a part of the World Health Organization (WHO). Data on the cancer-determining variables consists of: number of smoked cigarettes in million pieces per year (proxy for

[2] Information about the deterministic causes of cancers is taken from the online articles located on the MedicineNet website (http://www.medicinenet.com) as well as from the book entitled "Onkologi" (2008) written by Dalianis, Henriksson and Ringborg. For further information, please check the "Sources" section at the end of this paper.

smoking habits), available fat per person per day in grams (proxy for obesity) as well as per capita emissions of sulfur dioxide, $SO_2$, in kilograms per year (proxy for air pollution). This whole part of the dataset also comes from the WHO.[3]

There are certain drawbacks with the dataset:
a) It needs to be noted that there are missing observations for Poland for both cancer types as well as for smoking-related deaths in 1997 and 1998. This is because of the aforementioned worker strikes among the medical staff.
b) The part of the dataset that is describing the smoking habits does not cover the whole examined period of time – it covers the years between 1970 and 2000 (for both countries).
c) The part of the dataset that is describing the obesity does not cover the whole examined period of time – it covers the period between 1970 and 2005 (for both countries).
d) The part of the dataset that is describing the air pollution does not cover the whole examined period of time – it covers the time between 1970 and 2000 (for both countries).
e) The problem with all of the deterministic variables is that they are not gender-specific.

[3] For the information on all of the used variables along with their names and their descriptions, please refer to tables A.2.1 and A.2.2, both of which are located in the Appendix section of this thesis.

## **3.** Methods

**I**n order to examine the data two kinds of tests are performed. The first uses the cross-tabulation method with Pearson Chi-square tests. The second one is done with the help of the time series analysis methods. The reason for using two tests instead of one is to get a better and deeper look at the differences between the two time periods – before and after the Chernobyl catastrophe in terms of cancer mortality. The first method is performed by using the aforementioned age-specific cancer mortality figures. It is done with the help of the statistical software called SPSS. The second test is done by creating statistical models with the help of the general cancer mortality numbers and the bespoken deterministic variables. Program entitled STATA is used in this part. Below follows information, rules and restrictions of those two chosen test types as well as author's theory-to-practice approaches.

### ***3.1.*** *Cross-tabulations*

**A**nalysis of charts created by cross-tabulations is done by using the Pearson Chi-square ($\chi^2$) method. This method is based on comparing the observed frequencies in a table with their expected counterpart values. The formula for this test is located below (formula (1)). In this equation O stands for observed values, E is for estimated frequencies, while subscripts r and c stand for rows and respectively columns.

$$\chi^2 = \sum_{r=1}^{\infty}\sum_{c=1}^{\infty}\frac{(O_{rc} - E_{rc})^2}{E_{rc}} \tag{1}$$

"It was proposed in 1900 by Karl Pearson, the British statistician (…)." (Agresti 2007, p. 35). The test is done by creating a difference between the observed and awaited values in each cell of the contingency table. This difference is then taken to its square power and divided by the estimated frequencies. The test statistic, as such, is the sum of all these ratios. It takes the value of zero only when $O_{rc} = E_{rc}$. The $\chi^2$-test can never attain negative value as the difference is squared. Bigger differences between the observed and expected values create greater values of chi-square. This, in turn, means a stronger support for the alternative hypothesis.

The null hypothesis is rejected if the calculated sum is bigger than the critical value for a given significance level. The amount of the degrees of freedom (df) is calculated by using the formula (2):

$$df = (r-1)\times(c-1) \tag{2}$$

Under $H_0$ there are $r-1$ unique row proabilites and $c-1$ sole column probabilties. This means that there are $(r-1)+(c-1)$ parameters. Since the probability cannot exceed one, so these parameters can be equal, at the maximum, to $r\times c-1$. The whole derivation is placed in formula (3) below:

$$\begin{aligned} df &= (r\times c-1)-[(r-1)+(c-1)] = r\times c-1-r+1-c+1 = r\times c+1-r-c = \\ &= (r-1)\times(c-1) \end{aligned} \tag{3}$$

It is the null hypothesis that decides the structure and distribution of the observations. The main idea is that there is independence between the time periods and each of the tested variables. This means that it is assumed that the number of observations for each variable is equal in both time periods.

The significance of the p-values is defined in a standard statistical manner: one-star $10\% \leq P < 5\%$, two-star $1\% < P \leq 5\%$ and three-star $P \leq 1\%$. The latter one is when the significance is the strongest and when the null hypothesis is most likely to be rejected. In this thesis a significance level of $\alpha = 5\%$ is used.

For the test to work all table cells need to have expected values that are bigger than one. In addition only 20% of all the cells can have estimations below the value of five. Agresti (2007) points out that this test, like any other significance test, has limitations. It indicates whether there is an association between the variables, but it does not give any information about its power or direction. "Rather than relying solely on these tests, study the nature of the association. It is sensible to study residuals (…)." (Agresti 2007, p. 40).

Standardized residuals are, thus, being used in the contingency tables as they are providing a deeper and more detailed look into the nature of the relationship. According to Agresti (2007) there is a lack of fit of the null hypothesis in the table cell when the residual in that very cell exceeds the value of two (when there are few cells in the table) and of three (when there are many cells). It has to be noted that in the table with equal amount of columns and rows the variable residuals will have the same absolute values (one will be positive, the other one negative) between the periods. "This is because the observed counts and the estimated expected frequencies have the same row and column totals." (Agresti 2007, p. 39).

All in all, Pearson chi-square cross-tabulations are usually used as a basic background test that gives a bit more scientific representation of the data. This testing method is used in order to show the differences between the two examined time periods. In addition, it allows one to observe how much the results differ from their expected values. Adding residuals to the contingency tables helps in realizing how the results are placed in comparison to the accepted levels for the null hypothesis of equal amounts of observations in both time periods.

### *3.2. Time series analysis*

**T**ime series is the name for the variables that have their values spread across as well as being dependent on time. The most common model in time series is called AR(1) or an autoregressive process of order one; this model is used in this part of the paper to describe time series. This is the most basic type of the AR(p) models. AR(1) can be written as shown in formula (1) below:

$$y_t = \rho \times y_{t-1} + u_t \tag{1}$$

In that equation t is the time index (1,…,T), $u_t$ is the so-called white noise with a zero-mean and a variance that is equal to a constant $\sigma^2$. The parameter $\rho$ is called as an autoregressive coefficient and it has an absolute value equal to less than one. In this equation the dependent variable $y_t$ is determined by its own past effect ($y_{t-1}$, also known as the first lag), with a variable impact that is equal to $\rho$. It is also determined by a random shock occurring at time t, the aforementioned white noise factor. This equation can be extended by adding certain

components, e.g. a constant and a trend. In addition, more difficult models can be made by using autoregressive processes of higher orders than 1 (up to AR(p)), as well as by adding moving average processes of various orders (MA(q)) to the equation. It needs to be mentioned that all of the information and methods mentioned in this section can be also applied to more difficult models and multivariate cases.

Time series can be stationary or non-stationary (unit roots). The difference is dependent on whether or not certain assumptions are violated. In stationary series the observations are fluctuating around the constant mean. Stationary series, moreover, need to have a constant variance. The covariance between two random observations within these series should not time-dependent. This means that for stationarity all of the below equations need to be true (formula (2)):

$$\begin{aligned} E(y_t) &= \mu \\ Var(y_t) &= \sigma^2 \\ Cov(y_t, y_{t+s}) &= Cov(y_t, y_{t-s}) = \gamma_s \end{aligned} \tag{2}$$

It is quite important to differentiate between those two types of series as "(…) there is a danger of obtaining apparently significant regression results from unrelated data when non-stationary series are used in regression analysis." (Hill et al. 2008, p. 333). This is called a spurious regression. One has to use the Augmented Dickey-Fuller test (ADF) to test the type of the series with a regard to the inclusion or exclusion of constant and a trend. Dickey-Fuller tests have their own critical values that are called τ-statistics (tau). They tend to be more negative than standard t-statistics; therefore the null hypothesis of unit root is less likely to be rejected. In addition, these critical values change depending on the chosen deterministic components of the equation. The number of lags is decided upon by examining an ACF chart (autocorrelation function). The null hypothesis, which stands here for the presence of unit roots in the series, is rejected when the calculated tau is less than the critical one.

To make a unit root series into a stationary one it has to be differenced. There is a general rule that a series integrated of order d (I(d)) needs to be differenced d-times before it becomes stationary. The method can be followed in formula (3), shown here for I(1) series.

$$y_t - y_{t-1} = y_{t-1} + u_t - y_{t-1} \Rightarrow \Delta y_t = u_t \tag{3}$$

Since $u_t$ is stationary, so is the difference of $y_t$. "Stationary series are said to be integrated of order zero, I(0)." (Hill et al. 2008, p. 338).

Finally, one has to test whether the series are cointegrated. It implies that two processes, both of I(1) order, have similar stochastic trends and they never drift too far apart. Moreover, a linear difference of those processes is stationary. If the processes, thus, are $y_t$ and $x_t$, while their difference is $e_t$, so that difference must be of order I(0). This aforementioned difference can be pictured with the help of the formula (4).

$$e_t = y_t - \beta_1 - \beta_2 \times x_t \tag{4}$$

Testing for cointegration is done by the means of testing for stationarity of the least squares residuals with Dickey-Fuller. The critical values here are different from the aforementioned

ADF test. That is because "(…) we are basing this test upon estimated values of the residuals (…)" (Hill et al. 2008, p. 339). It is also of big importance here to use the critical values in accordance to the used set of deterministic components. If the residuals are stationary, then the series are cointegrated. The null hypothesis, which stands here for no cointegration as well as the presence of unit root in the residuals, is rejected when the calculated $\tau$ is smaller than the tabulated one.

All of the above steps and tests need to be done in order to create a proper time series model for data testing. The choice of the correct model is based on whether the cointegration occurs in or it is absent from the series. If the cointegration is present between the time series, then one ought to use the VEC (vector error correction) model. VAR (vector autoregressive) model is to be used in the situation when the null hypothesis of the lack of cointegration was not rejected. It has to be noted here that the "(…) VEC model is a special form of the VAR for I(1) variables that are cointegrated." (Hill et al. 2008, p. 348). These two models in their equation forms are shown below in formulas (7) and (8):

VAR model

$$\begin{aligned} y_t &= \beta_{10} + \beta_{11} \times y_{t-1} + \beta_{12} \times x_{t-1} + \upsilon_t^y \\ x_t &= \beta_{20} + \beta_{21} \times y_{t-1} + \beta_{22} \times x_{t-1} + \upsilon_t^x \end{aligned} \tag{5}$$

VEC model

$$\begin{aligned} y_t &= \alpha_{10} + (\alpha_{11} + 1) \times y_{t-1} - \alpha_{11} \times \beta_0 - \alpha_{11} \times \beta_1 \times x_{t-1} + \upsilon_t^y \\ x_t &= \alpha_{20} + \alpha_{21} \times y_{t-1} - \alpha_{21} \times \beta_0 - (\alpha_{21} \times \beta_1 - 1) \times x_{t-1} + \upsilon_t^x \end{aligned} \tag{6}$$

It is quite clear that in the VAR model case the first variable is dependent on the past value of the other value as well as its own first lag. The second variable is, of course, a function of the lag of the other variable from the system and the first lag of itself. If the variables are non-stationary then they need to be exchanged with their corresponding first differences in the VAR framework.

In the VEC model, the α-coefficients (with subscripts '$_{11}$' and '$_{21}$') are named as error correction coefficients as they illustrate the extent to which differences of the tested variables react to the cointegrating error ($e_{t-1}$). For example, if $\alpha_{21}$ is negative then $\Delta x_t$ decreases. As the counter-effect, to correct the error, a positive $\alpha_{11}$ guarantees that $\Delta y_t$ rises. "Having the error correction coefficients less than one in absolute value ensures that the system is not explosive." (Hill et al. 2008, p. 348).

### *3.3. Time series analysis – working with the data*

**S**ince working with and adjusting the data within time series analysis is somewhat harder and more time-consuming than it is when one performs cross-tabulation tests, so a separate section devoted to showing the reader the process of getting the results and models seems like a sound solution here.

The first step is to create a number of additional variables representing the cancer mortality ratios. This means that each cancer mortality variable for each gender in each country is divided by its respective general mortality counterpart. This choice is based on the fact that only such ratios capture the annual changes in both cancer mortality and general mortality. These ratios are also a way to register the fluctuations of yearly cancer deaths as a proportion of overall deaths per each year. In addition, it is only by the means of these ratios that both

cancer and all-cause mortality can be included in the models. Thus, twelve new variables are added to the basic dataset. It is of importance to note here that it is those ratios, in their stationary forms, that will henceforth function as dependent variables in this thesis[4].

As a result, one can begin the time series testing. First, all of the variables need to be tested for unit root. This is done with the help of the Augmented Dickey Fuller test. This test is easily carried out in STATA; one just needs to consider the amount of necessary lags as well as the inclusion/exclusion of certain components.

In terms of the variables from the Polish part of the dataset, it is only the ratios for female and male lung cancer mortality that are non-stationary. In addition, the same can be said about the proxy variable for air pollution. All three turn out to be of order I(1). In terms of the Spanish data, the deterministic proxy variables for obesity and air pollution turn out to be of order I(1). Female and male lung cancer mortality ratios are unit root of order I(2) and in turn their sum (variable 'ltspr') is I(1). The summary is presented below in Figure 3.3.1.

**Figure 3.3.1.** Summary of the stationarity testing

| Country | Variable | Lags | Test value | Critical value | Components |
|---|---|---|---|---|---|
| Poland | Lung male | 0/3 | -3,282/-5,928 | -3,41/-3,41 | T&C/T&C |
| | Lung female | 0/1 | 5,500/-2,115 | -1,94/-1,94 | No/No |
| | Lung total | 2 | -4,232 | -3,41 | T&C |
| | Stomach male | 1 | -5,991 | -1,94 | No |
| | Stomach female | 1 | -5,223 | -1,94 | No |
| | Stomach total | 3 | -4,306 | -1,94 | No |
| | Smoking habits | 0 | -3,097 | -2,86 | C |
| | Obesity | 0 | -3,872 | -2,86 | C |
| | Air pollution | 1/0 | -1,493/-2,406 | -1,94/-1,94 | No/No |
| Spain | Lung male | 3/2/1 | 0,415/-2,941/-11,371 | -1,94/-3,41/-1,94 | No/T&C/No |
| | Lung female | 1/2/2 | 3,709/-1,246/-7,100 | -1,94/-1,94/-1,94 | No/No/No |
| | Lung total | 3/0 | 0,840/-7,669 | -1,94/-2,86 | No/Constant |
| | Stomach male | 0 | -5,564 | -3,41 | T&C |
| | Stomach female | 1 | -5,485 | -1,94 | No |
| | Stomach total | 0 | -5,107 | -3,41 | T&C |
| | Smoking habits | 0 | -6,009 | -3,41 | T&C |
| | Obesity | 0/0 | 2,641/-4,994 | -1,94/-1,94 | No/No |
| | Air pollution | 0/0 | -2,736/-5,331 | -3,41/-1,94 | T&C/No |

Note: In all columns - Symbol / is used to indicate the result differences between the non-differenced and differenced series. In the Component column - T&C stands for "trend + constant", C signifies "just constant", whereas No is used for "no components".
The level of significance is 5%.

In the next step tests for cointegration are performed. A set of additional interaction variables is created to aid in this part of the time series analysis procedure. These variables are created by multiplying each stationary cancer mortality ratio with the Chernobyl dummy variable. The purpose of these variables is to account for the impact of the Chernobyl fallout on human cancer mortality. Testing for cointegration is also carried out in STATA, but the whole procedure is a bit more difficult than the unit root testing. Firstly, the regression is done where

[4] For the list of all of the additionally created variables outside of the main dataset please refer to the Appendix (Table A.3.1).

each cancer mortality ratio is used a dependent variable. The list of the included independent variables for each respective country is as follows: necessary determining factor variables, the aforementioned interaction variable as well as the standalone Chernobyl dummy. This means that, e.g. for male lung cancer mortality in Poland the proper mortality ratio is used as the dependent variable, whereas smoking habits and air pollution are used as the determining factor variables. Moreover, the Chernobyl dummy variable is included along with the correct interaction term. On the other hand, when e.g Spanish female stomach mortality model is considered then smoking habits and obesity are used as the determining independent variables. In addition, the Chernobyl dummy variable and the proper interaction term are also included as the explanatory variables, while a proper mortality ratio is used as the dependent variable. These regressions are, as such, performed for twelve different models: two cancer types in two countries for two genders with the addition of their summed total. It has to be mentioned that all of the variables that are used here have to be in their stationary forms. The next step is to perform a Dickey-Fuller test on the residuals saved from each separate regression with the respect to the number of lags and included components.

The results of cointegration testing are quite straightforward – cointegration occurs when lung cancer mortality figures are used as independent variables. It holds true for both Poland and Spain as well as for both genders and their total sum. There is no cointegration for stomach cancer deaths variables, irrespective of the country and gender. The summary of the results is located in the table below (Figure 3.3.2).

**Figure 3.3.2.** Summary of the cointegration testing

| Country | Variable | Test value | Critical value | Components | Cointegration |
|---|---|---|---|---|---|
| Poland | Lung male | -8,741 | -2,76 | No | Yes |
| | Lung female | -9,244 | -2,76 | No | Yes |
| | Lung total | -3,776 | -2,76 | No | Yes |
| | Stomach male | -2,712 | -2,76 | No | No |
| | Stomach female | -2,739 | -2,76 | No | No |
| | Stomach total | -2,687 | -2,76 | No | No |
| Spain | Lung male | -8,051 | -2,76 | No | Yes |
| | Lung female | -9,499 | -2,76 | No | Yes |
| | Lung total | -11,668 | -2,76 | No | Yes |
| | Stomach male | -2,455 | -2,76 | No | No |
| | Stomach female | -2,598 | -2,76 | No | No |
| | Stomach total | -2,438 | -2,76 | No | No |

Note: In the Components column - T&C stands for “trend + constant”, C signifies “just constant”, whereas No is used for “no components”.
The level of significance is 5%.

It is, as such, quite clear that all of the lung cancer deaths series need to be tested with the VEC model as the cointegration exists there between them and their relevant independent variables. In turn, all of the stomach cancer mortality series will be included in the VAR model because of the lack of cointegration between them and their independent variables. This means that in each case six models need to be created, tested and interpreted.

### *3.4. VAR models*

**I**n this part of the paper Vector Autoregressive models for the part of the dataset that is not cointegrated will be estimated and tested. This means that the stomach cancer mortality models are examined in this part of the paper. In terms of estimation, the modeling process is quite easy as one only needs to find out the proper amount of lags that have to be included in the model. This is done with the help of Information Criteria along with a Likelihood Ratio test. Testing the models can be a bit harder as it focuses on model stability as well as normality in the residuals. This latter part is of big importance as the normality in the residuals implies a proper estimation of the model in question. "Recall that hypothesis tests and interval estimates for the coefficients rely on the assumption that the errors, and hence the dependent variable y, are normally distributed." (Hill et al. 2008, p. 89). This is tested with the Jarque-Bera (J-B) test for normality in the residuals, which has a null hypothesis of normal distribution in the errors. It needs to be noted here that the necessary information about model estimation and its testing (for each single model) is presented in this section, whereas the models themselves are placed in the Results section.

All of the VAR models need fours lags in their specifications in accordance to the Selection-Order Criteria. The tests for stability of these models return positive results as all of the eigenvalues for each model are located within the unit circle. As such, all six VAR models are stable. When the normal distribution of the standard residuals is considered, only the model representing Spanish female deaths appears to not fulfill this requirement. In all of the other models the residuals appear to be normally distributed as the values of the Jarque-Bera tests prevent one from rejecting the null hypothesis. For the outputs of the J-B, please refer to the figures placed in the Appendix (Figure A.3.2 – A.3.7). For the summary of all of the necessary test specifications and test results, please refer to the chart presented in Figure 3.4.1 below. It is not very surprising to see that all of the models require the same amount of lags and that all of them turn out to be stable and have their eigenvalues located in the unit circle, since all of these models are quite similar to one another.

**Figure 3.4.1.** Summary of VAR modeling specifications

| Country | Model | Lags | Model stability | Normal residuals |
|---|---|---|---|---|
| Poland | Stomach male | 4 | Yes | Yes |
| | Stomach female | 4 | Yes | Yes |
| | Stomach total | 4 | Yes | Yes |
| Spain | Stomach male | 4 | Yes | Yes |
| | Stomach female | 4 | Yes | No |
| | Stomach total | 4 | Yes | Yes |

### *3.5. VEC models*

**T**his section of the paper is dedicated to testing for VEC (Vector Error Correction) models. While all of the necessary information about model estimation and its testing (for each single model) is presented in this section, the models themselves can be found in the Results section. VEC-modeling requires the knowledge of the number of test ranks. These ranks can also be interpreted as the amount of underlying cointegration relationships that take place in the model. To calculate the number of these relationships one should use the Johansen test for cointegration. In addition, the number of lags is decided by using, as in the VAR cases, the Information Criteria test. VEC models, similarly as VAR models, need also to be tested for

residual normality; this is done, accordingly, with the help of the Jarque-Bera test for normality in the error terms.

For the summary of model specifications and test results, please refer to the chart placed in the Figure 3.5.1 below. In all of the models four lags are used; similarly as with the VAR models. On the other hand, the estimated models differ from one another in terms of the amount of test ranks (cointegration relationships). Polish male lung cancer mortality ratio along with the Spanish female one are tested for up to three relationships. In both countries total mortality is only tested with one test rank. In addition, all of the models appear to have normally distributed residuals as the results of the Jarque-Bera tests are too big to allow the rejection of the null hypothesis. For the outputs of the J-B test for each of the VEC models, please see the Appendix section (Figure A.3.8 – A.3.13).

**Figure 3.5.1.** Summary of VEC modeling specifications

| Country | Variable | Rank | Lags | Normal residuals |
|---|---|---|---|---|
| Poland | Lung male | 3 | 4 | Yes |
| | Lung female | 2 | 4 | Yes |
| | Lung total | 1 | 4 | Yes |
| Spain | Lung male | 2 | 4 | Yes |
| | Lung female | 3 | 4 | Yes |
| | Lung total | 1 | 4 | Yes |

# **4.** Results

**W**hy is mortality so important? First answer that one may stumble upon is that it is a part of a natural human life-cycle and that everybody sooner or later faces death. Mortality can also be defined as "(…) the process by which the members of the population are reduced by death (…)." (Hinde 1998, p. 2). Why should demographers be concerned with it? It is a part of the balancing equation: $P_{t+1} = P_t + B_t - D_t + I_t - E_t$. In this equation deaths are represented by $D_t$. In connection with births/fertility ($B_t$), mortality creates a part of this equation that is referred to as the natural increase. This increase can be positive or negative depending on which of the two factors is greater than the other. In addition it is a well-known issue that mortality varies with "(…) age, sex, occupation, marital status and so on." (Hinde 1998, p. 19). The author then adds that it is one of the main tasks within demography to try to measure and understand those differences.

As mentioned before, the research question of this paper is to see whether Chernobyl radiation fallout really does have a significantly big effect on human cancer mortality. The main focus is on examining cancer-related deaths in Poland, while Spain is used as a control group. Lung cancer is used as the one that is clearly caused by radiation, whereas its stomach counterpart is used for comparison purposes as its development is not caused by radionuclides. In addition, proxies for smoking habits, obesity and air pollution are used as the determining factors. The gathered data is examined in two ways: 1) age-specific cancer mortality figures are tested with Pearson chi-square methods; 2) general cancer mortality figures are examined with the help of times series methods and VAR and VEC models (deterministic factor variables are used in this part of the analysis).

## ***4.1.*** *Cross-tabulations – results*

**P**earson $\chi^2$-tests are performed with the help of statistical software called SPSS. In addition, checking for Pearson p-values and abnormally big residuals is being carried out. The null hypothesis here states that each variable has an equal amount of observations in both of the tested periods; this means that the reactor radiation is said to have no effect on cancer mortality in the whole "after"-period (if all other deterministic factors are held constant).

*Group 0-29 years*

The output for this group is presented in Figure 4.1.1 below. One can see the comparison between the countries in regard to the period, gender and, in turn, to the cancer type. In the case of every cancer as well as gender, the amount of deaths is smaller in the second examined time-period. The biggest value is in the first period of male lung cancer mortality (466). The biggest difference between the two periods is for male lung cancer mortality in Spain (544).

It becomes also quite clear that the residuals in the second periods are not exceeding the limit of two and therefore we cannot reject the null hypothesis of equal figures in both periods. This is confirmed by the p-values, which are equal to 0,878 for male lung cancer mortality and 0,244 for the stomach counterpart. For female cancer deaths the p-values are smaller: 0,064 for lung and 0,020 for stomach. It is worth mentioning that the biggest residuals in the second period are attained by women, with 1,4 for female stomach cancer mortality in Spain as the

biggest and 0,0 for lung cancer Spanish men. The correlation between lower p-values, smaller differences and higher standard residual values is quite clear here.

**Figure 4.1.1.** Contingency table for age-group 0-29 years

| Cancer | Gender | | | | Country: Poland | Country: Spain | Total |
|---|---|---|---|---|---|---|---|
| Lung | Men | Period | 1966-1985 | Count | 466 | 544 | 1010 |
| | | | | Expected Count | 467,4 | 542,6 | 1010,0 |
| | | | | Std. Residual | ,0 | ,1 | |
| | | | 1986-2005 | Count | 217 | 249 | 466 |
| | | | | Expected Count | 215,6 | 250,4 | 466,0 |
| | | | | Std. Residual | ,1 | ,0 | |
| | | Total | | Count | 683 | 793 | 1476 |
| | | | | Expected Count | 683,0 | 793,0 | 1476,0 |
| | Women | Period | 1966-1985 | Count | 214 | 249 | 463 |
| | | | | Expected Count | 224,8 | 238,2 | 463,0 |
| | | | | Std. Residual | -,7 | ,7 | |
| | | | 1986-2005 | Count | 105 | 89 | 194 |
| | | | | Expected Count | 94,2 | 99,8 | 194,0 |
| | | | | Std. Residual | 1,1 | -1,1 | |
| | | Total | | Count | 319 | 338 | 657 |
| | | | | Expected Count | 319,0 | 338,0 | 657,0 |
| Stomach | Men | Period | 1966-1985 | Count | 464 | 371 | 835 |
| | | | | Expected Count | 454,5 | 380,5 | 835,0 |
| | | | | Std. Residual | ,4 | -,5 | |
| | | | 1986-2005 | Count | 181 | 169 | 350 |
| | | | | Expected Count | 190,5 | 159,5 | 350,0 |
| | | | | Std. Residual | -,7 | ,8 | |
| | | Total | | Count | 645 | 540 | 1185 |
| | | | | Expected Count | 645,0 | 540,0 | 1185,0 |
| | Women | Period | 1966-1985 | Count | 366 | 250 | 616 |
| | | | | Expected Count | 349,0 | 267,0 | 616,0 |
| | | | | Std. Residual | ,9 | -1,0 | |
| | | | 1986-2005 | Count | 175 | 164 | 339 |
| | | | | Expected Count | 192,0 | 147,0 | 339,0 |
| | | | | Std. Residual | -1,2 | 1,4 | |
| | | Total | | Count | 541 | 414 | 955 |
| | | | | Expected Count | 541,0 | 414,0 | 955,0 |

*Group 30-59 years*

The output for this group is presented in Figure 4.1.2 below. Once again one can see the comparison between the countries in regard to the period, gender and, last but not least, to the cancer type. The numbers are bigger than their respective counterparts in the younger age-group. In this group one can see that it is not always the case that the amount of deaths decreases between the periods. It actually holds true that mortality figures are greater in the case of the lung cancer. The biggest value (84 688) is for the second period of male lung cancer mortality. The biggest difference, in absolute terms, is for lung cancer among Spanish men (|24713|).

The residuals are clearly exceeding the limit of two for all lung cancer death cases. In the second period, the biggest residual value is for Spanish males who died of lung cancer (12,3). It is worth noting that for all stomach cancer women the residuals attain the value of zero. The p-values are as follows: men 0,000 for lung cancer and 0,053 for stomach one, women 0,000 for lung and 0,926 for stomach cancer. The correlation between the tested factors is yet again very clear. The null hypothesis of equal amount of observations in both time periods for all variables is rejected for lung cancer deaths among both genders. On the other hand, it cannot be rejected for stomach ones.

**Figure 4.1.2.** Cross-tabulation for age-group 30-59 years

| Cancer | Gender | | | | Country: Poland | Country: Spain | Total |
|---|---|---|---|---|---|---|---|
| Lung | Men | Period | 1966-1985 | Count | 61643 | 35270 | 96913 |
| | | | | Expected Count | 58701,6 | 38211,4 | 96913,0 |
| | | | | Std. Residual | 12,1 | -15,0 | |
| | | | 1986-2005 | Count | 84688 | 59983 | 144671 |
| | | | | Expected Count | 87629,4 | 57041,6 | 144671,0 |
| | | | | Std. Residual | -9,9 | 12,3 | |
| | | Total | | Count | 146331 | 95253 | 241584 |
| | | | | Expected Count | 146331,0 | 95253,0 | 241584,0 |
| | Women | Period | 1966-1985 | Count | 8999 | 5768 | 14767 |
| | | | | Expected Count | 10025,0 | 4742,0 | 14767,0 |
| | | | | Std. Residual | -10,2 | 14,9 | |
| | | | 1986-2005 | Count | 19674 | 7795 | 27469 |
| | | | | Expected Count | 18648,0 | 8821,0 | 27469,0 |
| | | | | Std. Residual | 7,5 | -10,9 | |
| | | Total | | Count | 28673 | 13563 | 42236 |
| | | | | Expected Count | 28673,0 | 13563,0 | 42236,0 |
| Stomach | Men | Period | 1966-1985 | Count | 32779 | 22503 | 55282 |
| | | | | Expected Count | 32915,2 | 22366,8 | 55282,0 |
| | | | | Std. Residual | -,8 | ,9 | |
| | | | 1986-2005 | Count | 19631 | 13111 | 32742 |
| | | | | Expected Count | 19494,8 | 13247,2 | 32742,0 |
| | | | | Std. Residual | 1,0 | -1,2 | |
| | | Total | | Count | 52410 | 35614 | 88024 |
| | | | | Expected Count | 52410,0 | 35614,0 | 88024,0 |
| | Women | Period | 1966-1985 | Count | 14400 | 11004 | 25404 |
| | | | | Expected Count | 14404,3 | 10999,7 | 25404,0 |
| | | | | Std. Residual | ,0 | ,0 | |
| | | | 1986-2005 | Count | 7731 | 5896 | 13627 |
| | | | | Expected Count | 7726,7 | 5900,3 | 13627,0 |
| | | | | Std. Residual | ,0 | ,0 | |
| | | Total | | Count | 22131 | 16900 | 39031 |
| | | | | Expected Count | 22131,0 | 16900,0 | 39031,0 |

*Group 60-85+ years*

The output for this group is presented in Figure 4.1.3 below. One can, again, see the comparison between the countries in regard to the period, with gender and cancer type put as the layers. Numbers here are mostly greater than in the two aforementioned age-groups. Figures for the second examined period in this group are bigger again only for the lung cancer mortality. The biggest value is for the second period male lung cancer victims in Spain (216 518), while the biggest difference is also for the lung cancer mortality among men in Spain. In absolute terms its value reaches |11 7384|.

All of the residuals in this group are greater than two. The biggest post-Chernobyl residual value is attained by Spanish male lung cancer deaths (value of 14,5). When the smallest residual in the "after"-period is regarded, then it has to be noted that it occurs for stomach cancer victims among women in Spain (value of 4,9). The p-values are equal to 0,000 for all of the tested groups in this case. It seems only natural as the standard residuals do exceed the limit value of two, sometimes by a big difference. The relationship between lower p-values, smaller differences and higher standard residual values is again very visible. Since all of the p-values are equal to maximum three-star significance, then the null hypothesis of equal figures in both periods of time has to be rejected for all variables in this age-group.

**Figure 4.1.3.** Pearson chi-square table for age-group 60-85+ years

| | | | | | Country | | |
|---|---|---|---|---|---|---|---|
| Cancer | Gender | | | | Poland | Spain | Total |
| Lung | Men | Period | 1966-1985 | Count | 114313 | 99134 | 213447 |
| | | | | Expected Count | 107671,5 | 105775,5 | 213447,0 |
| | | | | Std. Residual | 20,2 | -20,4 | |
| | | | 1986-2005 | Count | 206997 | 216518 | 423515 |
| | | | | Expected Count | 213638,5 | 209876,5 | 423515,0 |
| | | | | Std. Residual | -14,4 | 14,5 | |
| | | Total | | Count | 321310 | 315652 | 636962 |
| | | | | Expected Count | 321310,0 | 315652,0 | 636962,0 |
| | Women | Period | 1966-1985 | Count | 21851 | 16289 | 38140 |
| | | | | Expected Count | 23963,3 | 14176,7 | 38140,0 |
| | | | | Std. Residual | -13,6 | 17,7 | |
| | | | 1986-2005 | Count | 47165 | 24541 | 71706 |
| | | | | Expected Count | 45052,7 | 26653,3 | 71706,0 |
| | | | | Std. Residual | 10,0 | -12,9 | |
| | | Total | | Count | 69016 | 40830 | 109846 |
| | | | | Expected Count | 69016,0 | 40830,0 | 109846,0 |
| Stomach | Men | Period | 1966-1985 | Count | 89037 | 77210 | 166247 |
| | | | | Expected Count | 86469,6 | 79777,4 | 166247,0 |
| | | | | Std. Residual | 8,7 | -9,1 | |
| | | | 1986-2005 | Count | 60292 | 60562 | 120854 |
| | | | | Expected Count | 62859,4 | 57994,6 | 120854,0 |
| | | | | Std. Residual | -10,2 | 10,7 | |
| | | Total | | Count | 149329 | 137772 | 287101 |
| | | | | Expected Count | 149329,0 | 137772,0 | 287101,0 |
| | Women | Period | 1966-1985 | Count | 57932 | 62115 | 120047 |
| | | | | Expected Count | 56941,3 | 63105,7 | 120047,0 |
| | | | | Std. Residual | 4,2 | -3,9 | |
| | | | 1986-2005 | Count | 36073 | 42067 | 78140 |
| | | | | Expected Count | 37063,7 | 41076,3 | 78140,0 |
| | | | | Std. Residual | -5,1 | 4,9 | |
| | | Total | | Count | 94005 | 104182 | 198187 |
| | | | | Expected Count | 94005,0 | 104182,0 | 198187,0 |

*Total*

The output for the sums of all age-groups is presented in the table that is located below (Figure 4.1.4.). This table represents the differences between the countries in regard to the period, gender and, in turn, to the cancer type. The numbers are bigger in the second period in comparison to the first one only when lung cancer is considered. They decrease in the period after the Chernobyl reactor meltdown in terms of stomach cancer. The biggest number is for post-Chernobyl Polish lung cancer deaths among men (value of 291 902). When one looks at the differences, the biggest difference, in absolute terms, is for male lung cancer mortality in Spain (|141 802|).

It is quite obvious that all of the residuals in this group should be greater than two. The biggest residual value in the second period is attained by the male lung cancer deaths in Spain (value of 20,6). It needs to be noted that the smallest value of the residuals occurs for stomach cancer victims among women in Spain (value of 5,3). The p-values are three-star significant for all of the variables in this case. The relationship that occurs between lower p-values, smaller differences and higher standard residual values is again quite clear. When the null hypothesis of equal figures in both periods of time is regarded, it has to be rejected for all variables in this age-group.

**Figure 4.1.4.** Cross-tabulation for the sums of the respective age-groups

| Cancer | Gender | | | | Country: Poland | Country: Spain | Total |
|---|---|---|---|---|---|---|---|
| Lung | Men | Period | 1966-1985 | Count | 176422 | 134948 | 311370 |
| | | | | Expected Count | 165702,7 | 145667,3 | 311370,0 |
| | | | | Std. Residual | 26,3 | -28,1 | |
| | | | 1986-2005 | Count | 291902 | 276750 | 568652 |
| | | | | Expected Count | 302621,3 | 266030,7 | 568652,0 |
| | | | | Std. Residual | -19,5 | 20,8 | |
| | | Total | | Count | 468324 | 411698 | 880022 |
| | | | | Expected Count | 468324,0 | 411698,0 | 880022,0 |
| | Women | Period | 1966-1985 | Count | 31064 | 22306 | 53370 |
| | | | | Expected Count | 34245,9 | 19124,1 | 53370,0 |
| | | | | Std. Residual | -17,2 | 23,0 | |
| | | | 1986-2005 | Count | 66944 | 32425 | 99369 |
| | | | | Expected Count | 63762,1 | 35606,9 | 99369,0 |
| | | | | Std. Residual | 12,6 | -16,9 | |
| | | Total | | Count | 98008 | 54731 | 152739 |
| | | | | Expected Count | 98008,0 | 54731,0 | 152739,0 |
| Stomach | Men | Period | 1966-1985 | Count | 122280 | 100084 | 222364 |
| | | | | Expected Count | 119590,0 | 102774,0 | 222364,0 |
| | | | | Std. Residual | 7,8 | -8,4 | |
| | | | 1986-2005 | Count | 80104 | 73842 | 153946 |
| | | | | Expected Count | 82794,0 | 71152,0 | 153946,0 |
| | | | | Std. Residual | -9,3 | 10,1 | |
| | | Total | | Count | 202384 | 173926 | 376310 |
| | | | | Expected Count | 202384,0 | 173926,0 | 376310,0 |
| | Women | Period | 1966-1985 | Count | 72698 | 73369 | 146067 |
| | | | | Expected Count | 71555,8 | 74511,2 | 146067,0 |
| | | | | Std. Residual | 4,3 | -4,2 | |
| | | | 1986-2005 | Count | 43979 | 48127 | 92106 |
| | | | | Expected Count | 45121,2 | 46984,8 | 92106,0 |
| | | | | Std. Residual | -5,4 | 5,3 | |
| | | Total | | Count | 116677 | 121496 | 238173 |
| | | | | Expected Count | 116677,0 | 121496,0 | 238173,0 |

To sum up, the differences between the two time periods are significantly big for the age-group 60-85+ in both countries and for both types of cancers. For the group 30-59 the null hypothesis cannot be rejected for stomach cancer deaths in both Poland and Sweden, whereas the differences between two examined time periods seem to be statistically significant when lung cancer is regarded. In the youngest group (0-29) the null hypothesis of having equal amount of observations in each period cannot be rejected at all, regardless of the country, gender or the cancer type. In the case of the 'total', the calculated residuals are big enough to provide evidence against the null hypothesis. For the overview of the results of the Pearson chi-square cross-tabulations tests, please check the table below (Figure 4.1.5). It is quite clear, but needs to be nonetheless noted, that whenever the difference results in a negative value, it means that the amount of observations is greater in the second period (the difference is calculated in the following manner: "period before" – "period after").

**Figure 4.1.5.** Results of the cross-tabulation tests

| Country | Cancer type | Gender | Age-group | Residuals | $H_0$ rejected? | Difference |
|---|---|---|---|---|---|---|
| Poland | Lung | Male | 0-29 | 0,1 | No | 249 |
| | | | 30-59 | -9,9 | Yes | -23045 |
| | | | 60-85+ | -14,4 | Yes | -92684 |
| | | | *Total* | -19,5 | Yes | -115480 |
| | | Female | 0-29 | 1,1 | No | 109 |
| | | | 30-59 | 7,5 | Yes | -10675 |
| | | | 60-85+ | 10,0 | Yes | -25314 |
| | | | *Total* | 12,6 | Yes | -35880 |
| | Stomach | Male | 0-29 | -0,7 | No | 283 |
| | | | 30-59 | 1,0 | No | 13148 |
| | | | 60-85+ | -10,2 | Yes | 28745 |
| | | | *Total* | -9,3 | Yes | 42179 |
| | | Female | 0-29 | -1,2 | No | 191 |
| | | | 30-59 | 0,0 | No | 6669 |
| | | | 60-85+ | -5,1 | Yes | 20859 |
| | | | *Total* | -5,4 | Yes | 28719 |
| Spain | Lung | Male | 0-29 | 0,0 | No | 295 |
| | | | 30-59 | 12,3 | Yes | -24713 |
| | | | 60-85+ | 14,5 | Yes | -117384 |
| | | | *Total* | 20,8 | Yes | -141802 |
| | | Female | 0-29 | -1,1 | No | 160 |
| | | | 30-59 | -10,9 | Yes | -2027 |
| | | | 60-85+ | -12,9 | Yes | -8252 |
| | | | *Total* | -16,9 | Yes | -10119 |
| | Stomach | Male | 0-29 | 0,8 | No | 202 |
| | | | 30-59 | -1,2 | No | 9392 |
| | | | 60-85+ | 10,7 | Yes | 16648 |
| | | | *Total* | 10,1 | Yes | 26242 |
| | | Female | 0-29 | 1,4 | No | 86 |
| | | | 30-59 | 0,0 | No | 5108 |
| | | | 60-85+ | 4,9 | Yes | 20048 |
| | | | *Total* | 5,3 | Yes | 25242 |

Note: Null hypothesis is rejected when the residuals exceed the absolute value of 2

It is interesting to see that the null hypothesis is rejected more often in the cases of lung cancer mortality. What can be the reason of this? It is also interesting to see that the pattern of rejecting/accepting the null hypothesis is clearly the same in both countries - one country being affected by the Chernobyl radioactive fallout, while the other one is not. Are there any other deterministic factors at play besides radiation (e.g. lifestyle and environment) that can cause cancer mortality? In the next section, the data will be tested with the help of time series analysis and additional deterministic factors that can cause cancer to find the answers to these questions and doubts.

### *4.2. Time series analysis – results*

**I**n this section the results of the time series models, Vector Autoregressive and Vector Error Correction ones, are presented and interpreted. In addition to standard outputs of the tested models, Granger causality is examined for VAR models and cointegrating equations are inspected for VEC models. Each cancer type in each country is presented firstly in terms of the total mortality and then in respect to each gender.

VAR models are presented and interpreted below:

*VAR model for total stomach cancer mortality in Poland*

It is quite clear from the output (Figure 4.2.1) that the tested variables do not have an impact on the cancer mortality in this model as most of the coefficients are not significant. However, mortality ratio is affected by its own fourth lag. The p-value in this case indicates three-star significance (0,000). In addition, the coefficient value is clearly positive. This means that the current mortality ratio increases by circa 0,84 if the past fourth unit value goes up by one.

**Figure 4.2.1.** VAR model for total stomach cancer mortality in Poland

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| stplr | | | | | | |
| stplr L4. | .8427704 | .0729823 | 11.55 | 0.000 | .6997277 | .9858131 |
| stplrd L4. | -.2880964 | .2056831 | -1.40 | 0.161 | -.6912279 | .1150352 |
| smokepl L4. | -4.85e-08 | 4.76e-08 | -1.02 | 0.308 | -1.42e-07 | 4.47e-08 |
| fatpl L4. | -.0000506 | .0000475 | -1.07 | 0.286 | -.0001437 | .0000424 |
| chernobyl L4. | .0060919 | .004094 | 1.49 | 0.137 | -.0019323 | .014116 |
| _cons | .0108015 | .0056466 | 1.91 | 0.056 | -.0002657 | .0218687 |

Granger causality Wald tests

| Equation | Excluded | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| stplr | stplrd | 1.9619 | 1 | 0.161 |
| stplr | smokepl | 1.0394 | 1 | 0.308 |
| stplr | fatpl | 1.1373 | 1 | 0.286 |
| stplr | chernobyl | 2.2141 | 1 | 0.137 |
| stplr | ALL | 12.359 | 4 | 0.015 |

In terms of Granger-causality, the results also turn out to be insignificant because their p-values are greater than the critical value for the 5%-level. This is not true when the variable 'All' is regarded as its p-value is equal to 0,015. Apparently, even though single variables do not Granger-cause changes in total stomach cancer mortality in Poland, a combination of them does that.

*VAR model for male stomach cancer mortality in Poland*

In the male stomach cancer case, the situation is quite similar to the one presented above. The fourth lag of the male mortality ratio for this cancer type is the only variable that appears to have an impact on the variable itself. The p-value is three-star statistically significant in this case and the coefficient has a positive direction. The interpretation is a follows: If the mortality ratio increases by one unit within the fourth lag, then it means an increase by around 0,89 for the current ratio value. In addition, the constant appears to be significant with p-value equal to 0,014. Output is shown in Figure 4.2.2 below.

**Figure 4.2.2.** VAR model for male stomach cancer mortality in Poland

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| smplr | | | | | | |
| smplr L4. | .889619 | .0679757 | 13.09 | 0.000 | .756389 | 1.022849 |
| smplrd L4. | -.2427517 | .1736471 | -1.40 | 0.162 | -.5830938 | .0975904 |
| smokepl L4. | -8.25e-08 | 5.58e-08 | -1.48 | 0.139 | -1.92e-07 | 2.68e-08 |
| fatpl L4. | -.0000818 | .0000576 | -1.42 | 0.155 | -.0001946 | .000031 |
| chernobyl L4. | .0077183 | .0048333 | 1.60 | 0.110 | -.0017548 | .0171914 |
| _cons | .0160517 | .006549 | 2.45 | 0.014 | .003216 | .0288874 |

Granger causality Wald tests

| Equation | Excluded | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| smplr | smplrd | 1.9543 | 1 | 0.162 |
| smplr | smokepl | 2.1876 | 1 | 0.139 |
| smplr | fatpl | 2.0197 | 1 | 0.155 |
| smplr | chernobyl | 2.5501 | 1 | 0.110 |
| smplr | ALL | 20.027 | 4 | 0.000 |

The results of the Granger causality test are not significant, except for the case when all of the variables are combined into one (variable 'All'). The p-value is equal to 0,000. This means that male mortality is affected by a combination of all of the included variables and not by independently single variables as all other p-values are insignificant.

*VAR model for female stomach cancer mortality in Poland*

The results of the model representing stomach cancer deaths among Polish women closely follow the results of the previous two models. In the output (Figure 4.2.3), the coefficients are insignificant and, thus, their variables are not impacting the mortality ratio. This does not hold true, however, for the past value of the ratio itself. There is three-star significance in that case. This means that the present ratio goes up by almost 0,77 if the ratio four years back had increased in value by one unit.

**Figure 4.2.3.** VAR model for female stomach cancer mortality in Poland

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| sfplr | | | | | | |
| sfplr L4. | .766943 | .093451 | 8.21 | 0.000 | .5837825 | .9501035 |
| sfplrd L4. | -.3833829 | .3001001 | -1.28 | 0.201 | -.9715683 | .2048025 |
| smokepl L4. | -3.35e-08 | 4.69e-08 | -0.71 | 0.475 | -1.26e-07 | 5.85e-08 |
| fatpl L4. | -.0000201 | .0000467 | -0.43 | 0.667 | -.0001116 | .0000714 |
| chernobyl L4. | .00489 | .0039129 | 1.25 | 0.211 | -.0027792 | .0125592 |
| _cons | .0070279 | .0056095 | 1.25 | 0.210 | -.0039665 | .0180223 |

Granger causality Wald tests

| Equation | Excluded | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| sfplr | sfplrd | 1.632 | 1 | 0.201 |
| sfplr | smokepl | .50996 | 1 | 0.475 |
| sfplr | fatpl | .18476 | 1 | 0.667 |
| sfplr | chernobyl | 1.5618 | 1 | 0.211 |
| sfplr | ALL | 5.4924 | 4 | 0.240 |

All of the results are statistically insignificant, even for the variable 'All', when it comes to testing for Granger causality as all p-values in the output above are bigger than the critival value. As such, the cancer mortality ratio here is not Granger-caused by any of the variables included in this VAR model.

*VAR model for total stomach cancer mortality in Spain*

From the output presented below (Figure 4.2.4) it can be gathered that only the past fourth lag of the current cancer mortality ratio value is significant. It has a p-value equal to 0,000. In addition, the coefficient here has a positive direction. The interpretation of this coefficient is as follows: With an increase in value by one unit of the fourth lag of the mortality ratio itself, the current value increases by about 0,82. This means that the rest of the included variables do not affect the dependent variable.

**Figure 4.2.4.** VAR model for total stomach cancer mortality in Spain

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| stspr | | | | | | |
| stspr L4. | .8166114 | .0880013 | 9.28 | 0.000 | .6441319 | .9890908 |
| stsprd L4. | -.1839749 | .1811601 | -1.02 | 0.310 | -.5390423 | .1710924 |
| smokesp L4. | -1.88e-08 | 1.19e-08 | -1.59 | 0.113 | -4.21e-08 | 4.44e-09 |
| dfatsp L4. | .0000434 | .0000498 | 0.87 | 0.383 | -.0000542 | .000141 |
| chernobyl L4. | .0028686 | .0039848 | 0.72 | 0.472 | -.0049416 | .0106787 |
| _cons | .0041293 | .0028227 | 1.46 | 0.144 | -.0014031 | .0096617 |

Granger causality Wald tests

| Equation | Excluded | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| stspr | stsprd | 1.0313 | 1 | 0.310 |
| stspr | smokesp | 2.5155 | 1 | 0.113 |
| stspr | dfatsp | .75969 | 1 | 0.383 |
| stspr | chernobyl | .51822 | 1 | 0.472 |
| stspr | ALL | 5.4163 | 4 | 0.247 |

In terms of Granger causality, all of the results appear to be non-significant as their p-values are bigger than the critical value for the 5%-level. Therefore, it can be stated that the Spanish total stomach cancer mortality ratio is not Granger-caused by any other variable included in this particular VAR model.

*VAR model for male stomach cancer mortality in Spain*

In the Spanish male case, the results are quite similar to those for the models above. For the output, please refer to Figure 4.2.5. The mortality ratio in this VAR is positively affected by the fourth its own lag value. There is three-star significance in this case. With a rise in value by one unit in the fourth lag ratio, the present ratio goes up by circa 0,85. Nevertheless, a significant coefficient also occurs when smoking habits are regarded (p-value of 0,036). The value of this coefficient is, however, negative and very low.

**Figure 4.2.5.** VAR model for male stomach cancer mortality in Spain

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| smspr | | | | | | |
| smspr L4. | .8524019 | .1103906 | 7.72 | 0.000 | .6360402 | 1.068763 |
| smsprd L4. | -.2200977 | .2171339 | -1.01 | 0.311 | -.6456723 | .2054768 |
| smokesp L4. | -2.98e-08 | 1.42e-08 | -2.10 | 0.036 | -5.75e-08 | -2.02e-09 |
| dfatsp L4. | .0000462 | .0000617 | 0.75 | 0.454 | -.0000747 | .000167 |
| chernobyl L4. | .0049114 | .0060498 | 0.81 | 0.417 | -.0069459 | .0167687 |
| _cons | .0047936 | .0041035 | 1.17 | 0.243 | -.0032492 | .0128363 |

Granger causality Wald tests

| Equation | Excluded | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| smspr | smsprd | 1.0275 | 1 | 0.311 |
| smspr | smokesp | 4.4201 | 1 | 0.036 |
| smspr | dfatsp | .55991 | 1 | 0.454 |
| smspr | chernobyl | .65907 | 1 | 0.417 |
| smspr | ALL | 6.33 | 4 | 0.176 |

From Granger causality test, it can be also gathered that smoking habits are indeed impacting male cancer stomach mortality in Spain. The p-value here is equal, again, to 0,036. None of the other included variables seem to have an impact on the model's dependent variable as their p-values are insignificant.

*VAR model for female stomach cancer mortality in Spain*

The final VAR model also returns insignificant results (Figure 4.2.6). This is not true only for the past value of the dependent variable itself. The p-value is equal to 0,000 and according to the positive coefficient, an increase by one unit for the lag four ratio equal to a increase by circa 0,75 unit for the present ratio.

**Figure 4.2.6.** VAR model for female stomach cancer mortality in Spain

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| sfspr | | | | | | |
| sfspr L4. | .75172 | .0825999 | 9.10 | 0.000 | .5898271 | .9136128 |
| sfsprd L4. | -.1037082 | .1792392 | -0.58 | 0.563 | -.4550105 | .2475942 |
| smokesp L4. | -1.17e-08 | 1.17e-08 | -1.00 | 0.317 | -3.47e-08 | 1.12e-08 |
| dfatsp L4. | .0000472 | .0000475 | 1.00 | 0.320 | -.0000458 | .0001403 |
| chernobyl L4. | .0004377 | .0030109 | 0.15 | 0.884 | -.0054636 | .006339 |
| _cons | .004105 | .0022715 | 1.81 | 0.071 | -.0003471 | .0085571 |

Granger causality Wald tests

| Equation | Excluded | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| sfspr | sfsprd | .33478 | 1 | 0.563 |
| sfspr | smokesp | 1.0003 | 1 | 0.317 |
| sfspr | dfatsp | .99023 | 1 | 0.320 |
| sfspr | chernobyl | .02113 | 1 | 0.884 |
| sfspr | ALL | 6.0393 | 4 | 0.196 |

In addition, testing for causality in the model also brings insignificant results. None of the included variables appear to be Granger-causing the mortality ratio in this VAR model as their p-values are not significant.

In the next part of this section VEC models are presented and interpreted:

*VEC model for total lung cancer mortality in Poland*

From the output of this model (Figure 4.2.7) it can be seen that none of the three past lags of each of the included variables have a short-run impact on the mortality ratio as all of the respective p-values are insignificant. Only the constant in the VEC appears to be significant with a p-value of 0,036.

**Figure 4.2.7.** VEC model for total lung cancer mortality in Poland

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| D_ltplr | | | | | | |
| _ce1 | | | | | | |
| L1. | -.1217914 | .1466161 | -0.83 | 0.406 | -.4091537 | .165571 |
| ltplr | | | | | | |
| LD. | -.4619894 | .4800048 | -0.96 | 0.336 | -1.402782 | .4788028 |
| L2D. | -.0952707 | .4282467 | -0.22 | 0.824 | -.9346187 | .7440774 |
| L3D. | .097487 | .4453953 | 0.22 | 0.827 | -.7754717 | .9704458 |
| ltplrd | | | | | | |
| LD. | -.6825652 | 1.094638 | -0.62 | 0.533 | -2.828016 | 1.462885 |
| L2D. | .5031675 | .959691 | 0.52 | 0.600 | -1.377792 | 2.384127 |
| L3D. | .7331543 | .8608652 | 0.85 | 0.394 | -.9541104 | 2.420419 |
| smokepl | | | | | | |
| LD. | -1.16e-07 | 1.42e-07 | -0.82 | 0.414 | -3.93e-07 | 1.62e-07 |
| L2D. | -7.16e-08 | 1.11e-07 | -0.64 | 0.520 | -2.90e-07 | 1.47e-07 |
| L3D. | -3.37e-08 | 8.10e-08 | -0.42 | 0.677 | -1.92e-07 | 1.25e-07 |
| dso2pl | | | | | | |
| LD. | .0005291 | .0004645 | 1.14 | 0.255 | -.0003814 | .0014396 |
| L2D. | .0004165 | .0003516 | 1.18 | 0.236 | -.0002727 | .0011057 |
| L3D. | .0002624 | .0001923 | 1.36 | 0.172 | -.0001146 | .0006394 |
| chernobyl | | | | | | |
| LD. | .0332515 | .0457694 | 0.73 | 0.468 | -.0564547 | .1229578 |
| L2D. | -.013527 | .034314 | -0.39 | 0.693 | -.0807812 | .0537273 |
| L3D. | -.0256919 | .031905 | -0.81 | 0.421 | -.0882246 | .0368408 |
| _cons | .0016481 | .0007858 | 2.10 | 0.036 | .0001081 | .0031882 |

| beta | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| _ce1 | | | | | | |
| ltplr | 1 | . | . | . | . | . |
| ltplrd | -3.014721 | .2722611 | -11.07 | 0.000 | -3.548343 | -2.481099 |
| smokepl | -9.92e-07 | 9.43e-08 | -10.52 | 0.000 | -1.18e-06 | -8.07e-07 |
| dso2pl | .0040752 | .0004787 | 8.51 | 0.000 | .003137 | .0050134 |
| chernobyl | .1628478 | .0171834 | 9.48 | 0.000 | .129169 | .1965265 |
| _cons | .0545222 | . | . | . | . | . |

The results for the long-run equilibrium (when the cointegrating equation is equal to zero) look a bit more promising. The constant in that case is not significant, but the rest of the variables return three-star significant results. The first case here is $dltplr_t = 3{,}015 * ltplrd_t$. It can be, as such, constituted that there is a one unit rise in the interaction term which is equal to 3,015 unit increase of the mortality ratio itself. Next, $dltplr_t = 9{,}92e^{-07} * smokepl_t$ - this means that as the amount of smoked cigarettes goes up by one unit, then the mortality ratio itself goes up by a small amount as well. In addition, from this equation $dltplr_t = -0{,}004 * dso2pl_t$ it can be gathered that if the emissions of the $SO_2$ increase by one, then the mortality ratio decreases by about 0,0041.

In the case of the broken long-term equilibrium, which takes place when the cointegrating equation is different from zero, the error correction for the mortality ratio is negative and insignificant (for ce1 it is equal to about -0,12 with a p-value of 0,406). In turn, only the proxy for air pollution returns a significant result (coefficient: -816,7; p-value: 0,005). The coefficient for smoking habits is greatly positive and equal 499 754,7, but the p-value is insignificant (0,406). The coefficient for the interaction terms is positive (0,81), but its p-

value is insignificant (0,472). It means that only the variable for $SO_2$ emissions is reacting to the cointegration error and adjusting if the equilibrium is broken. The other output mentioned in this paragraph is placed in the Appendix (Figure A.4.1).

*VEC model for male lung cancer mortality in Poland*
The situation in this VEC model is quite similar to the previous one. It is only the constant that turns out to be significant (p-value equal to 0,038), whereas none of the past values of any of the tested variables appear to have a short-run impact on the dependent variable. The output is presented in Figure 4.2.8 below.

**Figure 4.2.8.** VEC model for male lung cancer mortality in Poland

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| D_dlmplr | | | | | | |
| _ce1 | | | | | | |
| L1. | -2.012947 | 1.271742 | -1.58 | 0.113 | -4.505515 | .479621 |
| _ce2 | | | | | | |
| L1. | -.2448959 | 3.088844 | -0.08 | 0.937 | -6.298918 | 5.809127 |
| _ce3 | | | | | | |
| L1. | -1.87e-08 | 1.12e-07 | -0.17 | 0.868 | -2.39e-07 | 2.02e-07 |
| dlmplr | | | | | | |
| LD. | .5984492 | 1.243201 | 0.48 | 0.630 | -1.83818 | 3.035078 |
| L2D. | .8219199 | 1.052293 | 0.78 | 0.435 | -1.240536 | 2.884376 |
| L3D. | .6324757 | .5122979 | 1.23 | 0.217 | -.3716098 | 1.636561 |
| dlmplrd | | | | | | |
| LD. | -.1177626 | 2.339188 | -0.05 | 0.960 | -4.702487 | 4.466962 |
| L2D. | -.4240046 | 1.717261 | -0.25 | 0.805 | -3.789775 | 2.941766 |
| L3D. | -.0689201 | .7463345 | -0.09 | 0.926 | -1.531709 | 1.393869 |
| smokepl | | | | | | |
| LD. | 3.04e-08 | 1.76e-07 | 0.17 | 0.863 | -3.14e-07 | 3.75e-07 |
| L2D. | -7.82e-09 | 1.10e-07 | -0.07 | 0.943 | -2.24e-07 | 2.08e-07 |
| L3D. | -5.23e-08 | 1.15e-07 | -0.46 | 0.649 | -2.77e-07 | 1.73e-07 |
| dso2pl | | | | | | |
| LD. | .0000759 | .0003431 | 0.22 | 0.825 | -.0005966 | .0007484 |
| L2D. | .000012 | .0002312 | 0.05 | 0.959 | -.0004412 | .0004652 |
| L3D. | .0000565 | .0002443 | 0.23 | 0.817 | -.0004222 | .0005353 |
| chernobyl | | | | | | |
| LD. | .0025523 | .003877 | 0.66 | 0.510 | -.0050466 | .0101512 |
| L2D. | .0032057 | .0032004 | 1.00 | 0.317 | -.003067 | .0094784 |
| L3D. | .0005976 | .0054432 | 0.11 | 0.913 | -.0100709 | .0112662 |
| _cons | .0055071 | .002656 | 2.07 | 0.038 | .0003015 | .0107128 |

| beta | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| _ce1 | | | | | | |
| dlmplr | 1 | . | . | . | . | . |
| dlmplrd | 4.44e-16 | . | . | . | . | . |
| smokepl | (omitted) | | | | | |
| dso2pl | -.0005777 | .0002304 | -2.51 | 0.012 | -.0010292 | -.0001262 |
| chernobyl | -.0025901 | .001442 | -1.80 | 0.072 | -.0054163 | .0002361 |
| _cons | -.0001663 | . | . | . | . | . |

The constant is insignificant in the cointegrating equations, while the proxy for air pollution turns out to be significant. The p-value here is equal to 0,012 and the proper equation look for when the equilibrium's cointegration is equal to zero is $dlmplr_t = 0{,}0006 * dso2pl_t$. This constitutes that a rise by about 0,0006 unit in the Polish male lung cancer deaths ratio is equal to the increase in air pollution by one unit. The p-value for the Chernobyl dummy variable is not significant as can be gathered from the above output.

When the cointegrating equations are not zero (broken equilibrium situation), then it can be seen that the error correction for the mortality ratio in the VEC model is insignificant (p-value of 0,113) with a negative coefficient. The error is not adjusted for by the air pollution variable as, even though its coefficient is positive (3376,8), the p-value is insignificant (0,155). The output mentioned here is located in Figure A.4.2 in the Appendix.

*VEC model for female lung cancer mortality in Poland*

In the case of this VEC model, the results may come as a bit of surprising as quite some coefficients appear to be significant in their short-term forms (output is located in Figure 2.4.9 below). The first coefficient is, of course, the constant with a p-value equal to 0,019. The second lag of the mortality ratio variable is also significant, but barely, with a p-value of 0,049 and a negative coefficient, which is equal to circa -2,55. This means that if the second lag increases by one unit, then the dependent variable in the present time decreases by -2,55 units. All three lags of the interaction term are significant. First lag has a p-value of 0,002 and a positive coefficient of 10,9; second lag's p-value is 0,005, while the positive coefficient is equal to 6,76; for the third lag the p-value is a bit higher and equal to 0,19 and the coefficient is lower, but still positive, and equal to 2,7. This constitutes that if the Chernobyl effect in each of its lags is increased by one unit, then the mortality ratio increases by about 10,9 and 6,76 as well 2,7, respectively. In addition, second lag of smoking habits is significant (0,47) and has a very small, but still positive, coefficient. This means that an increase in the number of smoked cigarettes has a small, albeit significant, impact of lung cancer mortality among women in Poland. All three lags of $SO_2$ are also greatly significant, with p-values that are equal to 0,001, 0,002 and 0,005 for each respective lag. The coefficients of all three lags are negative and their interpretation is as follows: If the emissions are increased by one in each of the past values, then this results in a mortality ratio decrease by 0,0005 for the first lag, 0,0004 for the second one and 0,0002 for the third lag.

**Figure 4.2.9.** VEC model for female lung cancer mortality in Poland

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| D_dlfplr | | | | | | |
| _ce1 | | | | | | |
| L1. | 2.369195 | 2.045879 | 1.16 | 0.247 | -1.640655 | 6.379045 |
| _ce2 | | | | | | |
| L1. | -11.37235 | 3.640102 | -3.12 | 0.002 | -18.50682 | -4.237883 |
| dlfplr | | | | | | |
| LD. | -3.066207 | 1.738655 | -1.76 | 0.078 | -6.473908 | .3414946 |
| L2D. | -2.547646 | 1.296519 | -1.96 | 0.049 | -5.088777 | -.0065146 |
| L3D. | -1.170436 | .724139 | -1.62 | 0.106 | -2.589722 | .2488503 |
| dlfplrd | | | | | | |
| LD. | 10.88814 | 3.556777 | 3.06 | 0.002 | 3.916987 | 17.8593 |
| L2D. | 6.764908 | 2.424674 | 2.79 | 0.005 | 2.012634 | 11.51718 |
| L3D. | 2.732338 | 1.162379 | 2.35 | 0.019 | .4541167 | 5.01056 |
| smokepl | | | | | | |
| LD. | 4.04e-08 | 5.72e-08 | 0.71 | 0.479 | -7.16e-08 | 1.53e-07 |
| L2D. | 8.96e-08 | 4.51e-08 | 1.98 | 0.047 | 1.11e-09 | 1.78e-07 |
| L3D. | 8.50e-08 | 5.27e-08 | 1.61 | 0.107 | -1.84e-08 | 1.88e-07 |
| dso2pl | | | | | | |
| LD. | -.0005203 | .0001554 | -3.35 | 0.001 | -.0008249 | -.0002157 |
| L2D. | -.0003699 | .0001209 | -3.06 | 0.002 | -.0006068 | -.000133 |
| L3D. | -.0002317 | .0000829 | -2.80 | 0.005 | -.0003941 | -.0000693 |
| chernobyl | | | | | | |
| LD. | -.0094062 | .0028692 | -3.28 | 0.001 | -.0150298 | -.0037826 |
| L2D. | -.0067414 | .0022126 | -3.05 | 0.002 | -.0110779 | -.0024048 |
| L3D. | -.0052142 | .001578 | -3.30 | 0.001 | -.008307 | -.0021213 |
| _cons | .0049729 | .0021152 | 2.35 | 0.019 | .0008272 | .0091187 |

| beta | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| _ce1 | | | | | | |
| dlfplr | 1 | . | . | . | . | . |
| dlfplrd | -1.11e-16 | . | . | . | . | . |
| smokepl | 1.49e-08 | 9.29e-09 | 1.60 | 0.110 | -3.37e-09 | 3.31e-08 |
| dso2pl | -.0000618 | .0000286 | -2.16 | 0.030 | -.0001178 | -5.83e-06 |
| chernobyl | -.0006162 | .0001768 | -3.49 | 0.000 | -.0009626 | -.0002698 |
| _cons | (omitted) | | | | | |

By examining the cointegrating equations for long-term equilibrium (when equations are equal to zero) we see that air pollution, again, has a significant effect on the dependent variable. The equation is equal to $dlfplr_t = 0{,}0006 * dso2pl_t$, which is a similar result to the one in the male case. As such, the interpretation of the equation in that aforementioned model clearly applies here as well: A rise by about 0,0006 unit in the Polish female lung cancer deaths ratio is equal to the increase in air pollution by one unit. The constant is omitted in this model, while the p-value for smoking habits is insignificant.

When the equilibrium is broken and the equations are different from zero, then it is quite clear that the mortality ratio does not adjust for the error as the coefficient of about 2,4 is insignificant (p-value is equal to 0,247). Similar can be said about the air pollution variable, which has a negative coefficient (-4 377,216), but is also insignificant (0,392). For the other output, please see Figure A.4.3 in the Appendix.

*VEC model for total lung cancer mortality in Spain*
In the short-term equilibrium, all of the results of this VEC model are insignificant. This is even true for the constant. This means that there is no effect of the past values on the dependent variable. Output is located in the figure below (Figure 2.4.10).

**Figure 4.2.10.** VEC model for total lung cancer mortality in Spain

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| D_dltspr | | | | | | |
| _ce1 | | | | | | |
| L1. | -1.83269 | 1.307766 | -1.40 | 0.161 | -4.395865 | .7304845 |
| dltspr | | | | | | |
| LD. | .4129629 | 1.284074 | 0.32 | 0.748 | -2.103776 | 2.929702 |
| L2D. | .5288486 | 1.07322 | 0.49 | 0.622 | -1.574624 | 2.632321 |
| L3D. | .4906922 | .5786286 | 0.85 | 0.396 | -.643399 | 1.624783 |
| dltsprd | | | | | | |
| LD. | -1.05301 | 1.381525 | -0.76 | 0.446 | -3.760748 | 1.654729 |
| L2D. | -1.182896 | 1.255419 | -0.94 | 0.346 | -3.643472 | 1.277679 |
| L3D. | -.5361388 | .8133578 | -0.66 | 0.510 | -2.130291 | 1.058013 |
| smokesp | | | | | | |
| LD. | -7.39e-08 | 6.88e-08 | -1.07 | 0.282 | -2.09e-07 | 6.09e-08 |
| L2D. | -9.55e-08 | 6.35e-08 | -1.50 | 0.133 | -2.20e-07 | 2.90e-08 |
| L3D. | -1.41e-08 | 2.61e-08 | -0.54 | 0.589 | -6.53e-08 | 3.70e-08 |
| dso2sp | | | | | | |
| LD. | -.0003472 | .0002453 | -1.42 | 0.157 | -.0008279 | .0001335 |
| L2D. | -.0002214 | .0001772 | -1.25 | 0.211 | -.0005686 | .0001259 |
| L3D. | -.0000524 | .0001113 | -0.47 | 0.638 | -.0002706 | .0001658 |
| chernobyl | | | | | | |
| LD. | .0053593 | .00556 | 0.96 | 0.335 | -.0055381 | .0162567 |
| L2D. | .0008469 | .006063 | 0.14 | 0.889 | -.0110364 | .0127303 |
| L3D. | .0069693 | .0061955 | 1.12 | 0.261 | -.0051738 | .0191123 |
| _cons | .0001553 | .0003077 | 0.50 | 0.614 | -.0004478 | .0007585 |

| beta | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| _ce1 | | | | | | |
| dltspr | 1 | . | . | . | . | . |
| dltsprd | -.8930342 | .068221 | -13.09 | 0.000 | -1.026745 | -.7593235 |
| smokesp | -5.29e-08 | 6.51e-09 | -8.14 | 0.000 | -6.57e-08 | -4.02e-08 |
| dso2sp | -.0002168 | .0000344 | -6.31 | 0.000 | -.0002842 | -.0001495 |
| chernobyl | .0020499 | .0001033 | 19.85 | 0.000 | .0018475 | .0022523 |
| _cons | .0020588 | . | . | . | . | . |

In terms of the long-run equilibrium (when all equations are equal to zero), however, all of the included variables are significant. For the interaction variable the equation is $dltspr_t = 0{,}893 * dltsprd_t$. This means that a when the value of the interaction terms goes up by one unit, then the mortality ratio itself increases by about 0,893. When smoking habits are regarded the equation is $dltspr_t = 0{,}529e^{-08} * smokesp_t$. According to this an increase by one

unit in the smoking habits leads to quite small, but still significant, increase in cancer mortality. Finally, when one looks at the air pollution emissions $dltspr_t = 0{,}0002 * dso2sp_t$; this means that the mortality ratio increases by circa 0,0002 unit when the $SO_2$ emissions are increased by one unit. The constant, however, is not significant.

When the long-term error equilibrium is broken, then it can be seen that the mortality ratio is not correcting for the error as its p-value is insignificant (0,161). The coefficient here is equal to circa -1,83. However, the interaction ratio appears to be significant with a p-value of 0,034 and a coefficient -1,86. In addition, the proxy for smoking habits is also significant with a p-value of 0,05 and positive coefficient equal to $3{,}49e^{07}$. The proxy for air pollution returns insignificant results (p-value equals to 0,845), even though the coefficient here is greatly positive and equal to about 839,9. This means that, even though the mortality ratio in this model is not adjusting for the error, but other variables included in that particular model (except for the air pollution proxy variable) are doing that correction. Please see Figure A.4.4 in the Appendix section for the additional output.

*VEC model for male lung cancer mortality in Spain*

From the output below (Figure 4.2.11) it can be seen that while most of the variables are insignificant in the short-run equilibrium, the significant results are for all three lags of the air pollution proxy. The constant is, yet again, also significant (0,000). In terms of all three lags of the 'dso2sp' variable, the coefficients are -0,0014, -0,00096 and -0,00052, respectively. The p-values are, in turn, equal to 0,001, 0,002 and 0,015, for the respective lags. This means that if the $SO_2$ emission increase by one unit, the mortality ratio in this VEC model goes down by 0,0014 in the first lag, by 0,00096 in the second and by 0,00052 in the third lag.

**Figure 4.2.11.** VEC model for male lung cancer mortality in Spain

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| D_d2lmspr | | | | | | |
| _ce1 | | | | | | |
| L1. | -5.228882 | 1.973477 | -2.65 | 0.008 | -9.096827 | -1.360937 |
| _ce2 | | | | | | |
| L1. | -.3505407 | 1.998403 | -0.18 | 0.861 | -4.267339 | 3.566257 |
| d2lmspr | | | | | | |
| LD. | 2.484859 | 1.70679 | 1.46 | 0.145 | -.8603878 | 5.830106 |
| L2D. | .8754615 | 1.106302 | 0.79 | 0.429 | -1.29285 | 3.043773 |
| L3D. | .1594942 | .4427809 | 0.36 | 0.719 | -.7083405 | 1.027329 |
| d2lmsprd | | | | | | |
| LD. | .6661518 | 1.750855 | 0.38 | 0.704 | -2.765462 | 4.097765 |
| L2D. | .7909947 | 1.194954 | 0.66 | 0.508 | -1.551072 | 3.133062 |
| L3D. | .6401386 | .5566815 | 1.15 | 0.250 | -.4509372 | 1.731214 |
| smokesp | | | | | | |
| LD. | 4.93e-09 | 9.76e-08 | 0.05 | 0.960 | -1.86e-07 | 1.96e-07 |
| L2D. | -1.29e-07 | 8.18e-08 | -1.57 | 0.116 | -2.89e-07 | 3.16e-08 |
| L3D. | 8.14e-09 | 5.02e-08 | 0.16 | 0.871 | -9.02e-08 | 1.06e-07 |
| dso2sp | | | | | | |
| LD. | -.001373 | .0004166 | -3.30 | 0.001 | -.0021894 | -.0005566 |
| L2D. | -.0009611 | .0003042 | -3.16 | 0.002 | -.0015573 | -.000365 |
| L3D. | -.0005246 | .0002155 | -2.43 | 0.015 | -.000947 | -.0001021 |
| chernobyl | | | | | | |
| LD. | .0164269 | .0085159 | 1.93 | 0.054 | -.0002639 | .0331178 |
| L2D. | -.0003932 | .0116098 | -0.03 | 0.973 | -.0231479 | .0223616 |
| L3D. | .0207606 | .0094916 | 2.19 | 0.029 | .0021574 | .0393638 |
| _cons | .0216232 | .0051455 | 4.20 | 0.000 | .0115382 | .0317083 |

| beta | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| _ce1 | | | | | | |
| d2lmspr | 1 | . | . | . | . | . |
| d2lmsprd | (omitted) | | | | | |
| smokesp | -2.51e-08 | 2.13e-08 | -1.18 | 0.238 | -6.69e-08 | 1.66e-08 |
| dso2sp | -.0003518 | .0001017 | -3.46 | 0.001 | -.0005511 | -.0001524 |
| chernobyl | .0005238 | .0002853 | 1.84 | 0.066 | -.0000354 | .001083 |
| _cons | .0053243 | . | . | . | . | . |

In the long-run equilibrium (when all of the cointegrating equations are equal to zero), the significant results is delivered by the air pollution variable. The p-value is 0,001, while the coefficient is equal to circa -0,00035. The equation, as such, is $d2lmspr_t = 0{,}00035 * dso2sp$. This constitutes that an increase by one unit in the sulfur dioxide emissions increases the Spanish male lung cancer mortality ratio by 0,00035 unit. The constant is not significant and so are the other p-values that are mentioned in the above output.

When the equation is not equal to zero as it is in the broken equilibrium case, the mortality ratio is surely significant (0,008) and has a negative coefficient of -5,23. However, the error is not picked up and corrected by the air pollution variable as, even though its coefficient is positive (about 3493), but its p-value is insignificant and equal to 0,277. This means that the mortality ratio variable acts up as the adjusting indicator in this model, whereas the $SO_2$ emissions one does not. For the other output connected to this model, please see the Figure A.4.5 in the Appendix.

*VEC model for female lung cancer mortality in Spain*

The final VEC model is for Spanish female mortality with lung cancer as its cause. In the short-run equilibrium none of the past lags of the included variables turn out to be significant; it is even true for the constant. This means that the independent variables included in the model have no effect on the dependent variable. The output is located in the table below (Figure 4.2.12).

**Figure 4.2.12.** VEC model for female lung cancer mortality in Spain

| | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| D_d2lfspr | | | | | | |
| _ce1 | | | | | | |
| L1. | -3.370944 | 3.384387 | -1.00 | 0.319 | -10.00422 | 3.262332 |
| _ce2 | | | | | | |
| L1. | -1.865661 | 3.704286 | -0.50 | 0.615 | -9.125928 | 5.394607 |
| _ce3 | | | | | | |
| L1. | 5.10e-09 | 2.58e-08 | 0.20 | 0.843 | -4.55e-08 | 5.57e-08 |
| d2lfspr | | | | | | |
| LD. | .869349 | 2.83162 | 0.31 | 0.759 | -4.680523 | 6.419221 |
| L2D. | -.1494463 | 1.679147 | -0.09 | 0.929 | -3.440515 | 3.141622 |
| L3D. | -.1230462 | .5394288 | -0.23 | 0.820 | -1.180307 | .9342148 |
| d2lfsprd | | | | | | |
| LD. | 2.157624 | 3.162026 | 0.68 | 0.495 | -4.039834 | 8.355081 |
| L2D. | 1.734374 | 2.068389 | 0.84 | 0.402 | -2.319595 | 5.788342 |
| L3D. | .4195596 | .9437557 | 0.44 | 0.657 | -1.430167 | 2.269287 |
| smokesp | | | | | | |
| LD. | -2.93e-08 | 2.48e-08 | -1.18 | 0.238 | -7.78e-08 | 1.93e-08 |
| L2D. | -1.58e-09 | 2.63e-08 | -0.06 | 0.952 | -5.32e-08 | 5.00e-08 |
| L3D. | -4.49e-09 | 9.75e-09 | -0.46 | 0.645 | -2.36e-08 | 1.46e-08 |
| dso2sp | | | | | | |
| LD. | -.0000903 | .0001491 | -0.61 | 0.545 | -.0003826 | .0002019 |
| L2D. | -.0000552 | .0001243 | -0.44 | 0.657 | -.0002989 | .0001885 |
| L3D. | .0000161 | .0000863 | 0.19 | 0.852 | -.0001531 | .0001852 |
| chernobyl | | | | | | |
| LD. | -.0007647 | .0022498 | -0.34 | 0.734 | -.0051742 | .0036449 |
| L2D. | .0009686 | .002001 | 0.48 | 0.628 | -.0029534 | .0048905 |
| L3D. | -.0009502 | .0024764 | -0.38 | 0.701 | -.0058038 | .0039035 |
| _cons | -.0002874 | .0002263 | -1.27 | 0.204 | -.0007309 | .0001561 |

| beta | Coef. | Std. Err. | z | P>\|z\| | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| _ce1 | | | | | | |
| d2lfspr | 1 | . | . | . | . | . |
| d2lfsprd | (omitted) | | | | | |
| smokesp | (omitted) | | | | | |
| dso2sp | -.0000106 | .0000186 | -0.57 | 0.567 | -.0000471 | .0000258 |
| chernobyl | -.000056 | .0000426 | -1.31 | 0.189 | -.0001395 | .0000275 |
| _cons | -.0000561 | . | . | . | . | . |

Similar can be said about the long-run equilibrium. None of the variables appear to have an effect on the mortality ratio in question as all of the p-values that are visible in the output are insignificant. This may be caused by some problems with the model and with the variable structure; this is confirmed by the fact that only this model lacks normally distributed residuals.

# **5.** Discussion

**T**he accident that took place on the 26th April, 1986, in Chernobyl, Ukraine, has changed the world and its view on nuclear energy. The consequences of the core meltdown in the reactor number four are still estimated as the biggest and most dangerous for human health, especially in terms of the delayed effects of the radiation fallout. Up to the recent times, the accident in the Nuclear Power Plant in Chernobyl was assessed as the most threatening. Only the current accident in Japan in the nuclear power plant located in Fukushima was evaluated to be of the same danger level to humanity as Chernobyl. Does this mean that all of the nuclear power plants should be shut down? The answer to that question is not easy and definitely not straightforward. As mentioned before, there are two sides to the nuclear energy argument. By asking both sides for help in answering that question, one will surely receive very mixed messages. However, at the present Poland is creating projects for its own nuclear power plant in the northern part of the country[5]. It is estimated to be completed by the year 2020.

Radiation causes many health problems for people who are exposed to it. Sometimes the effects on health are immediate, whereas at times they can take up to several years to develop. This is based on the doses as well as types of radiation. Apart from various diseases and mutations because of the exposure, human beings are also apt to be the victims of different types of cancer. The most prevalent types that are connected to contact with radionuclides are thyroid and leukemia, but a plethora of other types follows. Among these types lung cancer is often mentioned in the literature on radiation. The aim of this thesis, as mentioned before, is to test whether the Chernobyl radiation really has an effect on the development of lung cancer among people.

From the cross-tabulation part of the analysis, it can be gathered that there is no difference between the examined countries when it comes to rejecting the null hypothesis. More elderly die of those two cancer types in the period after the catastrophe. The middle age-group also seems to be more prone to die but only when the lung cancer is considered. On the whole, it is clear from these tests that lung cancer mortality is greater in both countries than the stomach cancer one as there are more negative results in the lung cancer part of "Difference" column of the summarizing chart (as shown in Figure 4.1.5). However, according to the calculated differences between the first and the second time period, it is Spain that has bigger mortality because of both cancer types. The results can be striking as a bit uncanny in the light of the fact that Spain was not within the reach of the Chernobyl's radioactive cloud. In addition, it is also true that there are other factors that can have a delayed effect on health and mortality, besides smaller and non-acute doses of radiation. Chain-smoking, living in the areas with heavily polluted air or unhealthy eating do not have immediate effects on health or mortality. They may take years to actually impact human health and well-being; cancer on its own is also known to take time to develop. One may start smoking by the age of 18 only to be diagnosed with lung cancer when he or she turns 60. That is why these deterministic factors are used in the time series testing.

All of the VAR models, which are in fact all models with stomach cancer mortality as the dependent variable, support the hypothesis that Chernobyl is not the cause of increased mortality in the time period after the accident. The fourth lag of the interaction term in all

[5] This fact is given here courtesy of my parents.

models is insignificant. In addition, all other lagged variables, except the past value of the dependent variables themselves, return insignificant results as well. This means that the hypothesis that stomach cancer is caused by excessive smoking or excessive eating is not supported here. When one, on the other hand, looks at Granger causality it becomes clear that in the male model for Poland the mortality ratio is impacted (Granger-caused) by a combination of all of the included variables. The reason for this might be that since the researched time period is quite short, then the effect of single factors on mortality cannot be registered by the time series analysis. What is registered, however, is the combined effect of the included variables. Similar can be said about the total model in Poland. Smoking habits appear to Granger-cause cancer mortality among men in Spain, whereas other variables do not. People in Southern Europe are known for their heavy smoking habits and men, in general and not only that in particular region, tend to have greater nicotine intakes than women. As such, they may be more apt to be diagnosed with cancer.

VEC models are created for lung cancer mortality data. In the short-run equilibrium only the Polish female model and Spanish male model return a number of significant results. Polish female mortality appears to be negatively affected by the Chernobyl radiation and by, to a smaller degree, smoking habits. The $SO_2$ emissions appear to be, in fact, decreasing the mortality ratio for Polish females. The effect of the latter factor is similar when Spanish males are considered – the rise in the amount of the emissions seems to be helping with the decrease of the mortality ratio. In the long-run equilibrium, however, the effect of air pollution clearly becomes negative in these two models. As such, the increasing amount of air pollutants results in increased lung cancer mortality. Moreover, the same can be said about the models representing Polish males and Spanish total. The reason for this is, as mentioned before, that there exists a delayed effect on health and that only the long-run analysis registers this as a negative impact. This means that both genders in Poland along with Spanish men are prone to be affected by air pollution. The explanation for Poland is quite simple – as the country became independent, the number of purchased and driven cars as well as the number of big factories increased rapidly over a short period of time. This obviously resulted in an increase in air pollution. Chernobyl has a negative long-run effect on mortality in total models for both countries, but the effect is much greater in Poland; 3,015 as opposed to Spanish 0,893. The reason for this result is that even though Spain was not affected by the radioactive cloud, the effects of the contamination might have also reached out into this country, via e.g. travelers, exported goods, etc. Poland's effect is bigger as this country is the neighboring nation of Ukraine. The reason for only the totals being impacted is that in those two models there are the biggest amounts of annual observations. It is, hence, easier for the time series analysis to register the causality factor. Similar can be said about the smoking habits. The effect of this habit on mortality is also only picked up in the total models for both Spain and Poland. The effect value for Poland is bigger than then one for Spain, although both of them are causing an increase in the respective mortality ratios, for Poland it is $9{,}92e^{-07}$ as opposed to the Spanish $0{,}529e^{-08}$. These numbers in their decimal forms are roughly equal to 0,009 and 0,0002, respectively. This means that in the long-run Polish people are more affected by heavy smoking and tend to die of lung cancer more often than Spanish people.

There are also certain issues with this paper that need to be discussed. The development of the lung cancer is, of course, much less common among people who were exposed to the fallout in comparison to the thyroid one. That is why the literature on the health effects of radiation usually deals with the thyroid cancer - more data, easier detection of causality and better results are just a few reasons. The fact that lung cancer is less common among radiation

victims might also be the explanation for the fact that results of the Chernobyl radiation effect are only conclusive and significant in the total models in this paper.

On the other hand, some problems with the scarcity of the significant results may be due to the fact that all twelve models researched in this thesis are quite simple. In addition, the choice and the structure of the included variables might also be impacting the results. It is especially pertinent when the deterministic variables are regarded. None of these variables is direct as all of them are used as proxies. One may then argue about whether or not they are good proxies and even their relevance to the research may be questioned. The results may also be impacted by a clear lack of the variable that describes the yearly radiation doses. The latter issue cannot be resolved, however, due to the lack of open availability of such data.

It is furthermore true that in time series analysis one should use much longer periods than 40 years. The more annual observations are used, the better and easier the causality can be picked up and registered by the models. This is especially important when dealing with non-stationary variables that need to be differenced. Taking differences decreases the amount of yearly observations in the variables. This is especially an apparent issue for all of the deterministic factor variables as none of them covered the whole researched time period to begin with. Nevertheless, there were also hindrances from extending the dataset for both time periods. Mortality by each of the two cancers as the cause covers the period 1951-2005 for Spain and 1959-2005 for Poland. General mortality, in turn, spans the period 1908-2006 for Spain and 1958-2009 for Poland. I believe that I have managed to find a middle-ground for the examined time periods as well as for making these periods relevant to the topic of this paper and to my research question.

# **6.** Conclusions

The accident in the Chernobyl's Nuclear Power Plant altered our view on the nuclear power It needs to be added that the hazards that this accident brought into the world have actually changed us. Exposure to radionuclides can be harmful to human health and can result in immediate or delayed diseases, malfunctions or mutations. One of the most common consequences of being exposed to nuclear fallout is the development of cancer. A number of books and articles state that it is the thyroid and leukemia cancers that are the most common results of exposure to radioactivity. Many authors analyze just these two cancer types when writing about the consequences of the Chernobyl's reactor meltdown. Nonetheless, there are also other types of cancers that are caused by radionuclides and lung cancer is one them. In this paper the main aim is to test the hypothesis whether the Chernobyl radiation is still causing the development of lung cancer. The main part of the analysis uses the time series methods to test the significance of that hypothesis by creating VEC and VAR models.

To sum up the VEC model results, air pollution affects the mortality of Polish and Spanish males as well as Polish females in terms of lung cancer deaths. In addition, it is both the Chernobyl effect, which is conveyed by the interaction terms, as well as the heavy smoking habits that are resulting in an increased mortality in the total models in both countries. To sum up the VAR model results, total and male stomach cancer mortality ratios in Poland are impacted by a combination of all variables. In the case of the Spanish stomach cancer male mortality ratio it is mainly the excessive smoking that is causing more cancer deaths.

The results show that the radioactive contamination coming from the Chernobyl's accident is still affecting lung cancer mortality of people in Poland. However, it is the polluted air and even heavy smoking that pose much larger threats to human health nowadays. One cannot deny the influence of the radioactive fallout on the frequency of lung cancer diagnosis among Polish citizens, but it seems that it is the living in our modern and stressful times that is really taking its toll on lung cancer mortality. For the future research on this topic, one can only wish for longer time periods, different cancer types. Moreover, future papers on this or similar topic can really make use of more reliable and differently structured data. It should also be mentioned, that researchers should gain easier access to data on annual national radiation doses, which would really help and simplify their data analysis and modeling.

## **7.** Sources

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**4.** Fat intake – http://data.euro.who.int/hfadb/
*(available fat per person per day in grams)* (accessed: 08.05.2011)

**5.** $SO_2$ emissions – http://data.euro.who.int/hfadb/
*(sulfur dioxide emissions per capita per year in kilograms)* (accessed: 08.05.2011)

# **8.** Appendix

**Table A.2.1.** List of used variables and their explanations – for POLAND

| Variable name | Description |
|---|---|
| year | Time period (1966-2005) |
| mpl, fpl, tpl | General mortality for males, females and resp. total in Poland |
| lmpl, lfpl, ltpl | Lung cancer mortality for males, females and resp. total in Poland |
| smpl, sfpl, stpl | Stomach cancer mortality for males, females and resp. total in Poland |
| smokepl | Millions of consumed cigarettes per year in Poland (proxy of smoking habits) |
| fatpl | Available fat per person per day in grams in Poland (proxy of obesity) |
| so2pl | $SO_2$ emission in Poland per capita per year in kilograms (proxy of air pollution) |
| ch | Dummy variable for "before" and "after" periods |

**Table A.2.2.** List of used variables and their explanations – for SPAIN

| Variable name | Description |
|---|---|
| year | Time period (1966-2005) |
| msp, fsp, tsp | General mortality for males, females and resp. total in Spain |
| lmsp, lfsp, ltsp | Lung cancer mortality for males, females and resp. total in Spain |
| smsp, sfsp, stsp | Stomach cancer mortality for males, females and resp. total in Spain |
| smokesp | Millions of consumed cigarettes per year in Spain (proxy of smoking habits) |
| fatsp | Available fat per person per day in grams in Spain (proxy of obesity) |
| so2sp | $SO_2$ emission in Spain per capita per year in kilograms (proxy of air pollution) |
| ch | Dummy variable for "before" and "after" periods |

**Table A.3.1.** List of additionally created variables in the time series analysis section

| Variable name | Description |
|---|---|
| lmspr, lfspr, ltspr | Ratios of lung cancer mortality in Spain for men, women and resp. total. |
| lmplr, lfplr, ltplr | Ratios of lung cancer mortality in Poland for men, women and resp. total. |
| smspr, sfspr, stspr | Ratios of stomach cancer mortality in Spain for men, women and resp. total. |
| smplr, sfplr, stplr | Ratios of stomach cancer mortality in Poland for men, women and resp. total. |
| dlmplr, dlfplr | First difference of the male and resp. female lung cancer mortality ratios in Poland |
| dso2pl, dso2sp | First difference of the Polish and resp. Spanish air pollution proxies |
| d2lmspr, d2lfspr | Second difference of the male and resp. female stomach cancer mortality ratios in Spain |
| dfatsp | First difference of the Spanish obesity proxy |
| dlmplrd, dlfplrd, ltplrd, smplrd, sfplrd, stplrd, d2lmsprd, d2lfsprd, dltsprd, smsprd, sfsprd, stsprd | Interaction terms between each of the stationary cancer mortality ratios and the Chernobyl dummy |

**Figure A.3.2.** Jarque-Bera test in VAR modeling for male stomach cancer mortality in Poland

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| smplr | 2.620 | 2 | 0.26986 |
| smplrd | 0.950 | 2 | 0.62197 |
| smokepl | 1.926 | 2 | 0.38181 |
| fatpl | 5.661 | 2 | 0.05897 |
| chernobyl | 1.601 | 2 | 0.44916 |
| ALL | 12.757 | 10 | 0.23756 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| smplr | -.75952 | 2.596 | 1 | 0.10714 |
| smplrd | -.03048 | 0.004 | 1 | 0.94844 |
| smokepl | -.3916 | 0.690 | 1 | 0.40614 |
| fatpl | -.84102 | 3.183 | 1 | 0.07441 |
| chernobyl | -.31471 | 0.446 | 1 | 0.50439 |
| ALL | | 6.919 | 5 | 0.22675 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| smplr | 3.1454 | 0.024 | 1 | 0.87740 |
| smplrd | 3.9168 | 0.946 | 1 | 0.33086 |
| smokepl | 4.048 | 1.236 | 1 | 0.26632 |
| fatpl | 4.4843 | 2.478 | 1 | 0.11542 |
| chernobyl | 4.0133 | 1.155 | 1 | 0.28249 |
| ALL | | 5.838 | 5 | 0.32226 |

**Figure A.3.3.** Jarque-Bera test in VAR modeling for female stomach cancer mortality in Poland

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| sfplr | 0.141 | 2 | 0.93200 |
| sfplrd | 8.796 | 2 | 0.01230 |
| smokepl | 5.836 | 2 | 0.05405 |
| fatpl | 0.919 | 2 | 0.63153 |
| chernobyl | 3.309 | 2 | 0.19121 |
| ALL | 19.001 | 10 | 0.04026 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| sfplr | .08621 | 0.033 | 1 | 0.85490 |
| sfplrd | -.1339 | 0.081 | 1 | 0.77638 |
| smokepl | -.77048 | 2.671 | 1 | 0.10217 |
| fatpl | -.16503 | 0.123 | 1 | 0.72628 |
| chernobyl | .78896 | 2.801 | 1 | 0.09420 |
| ALL | | 5.709 | 5 | 0.33556 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| sfplr | 3.309 | 0.107 | 1 | 0.74313 |
| sfplrd | 5.7833 | 8.715 | 1 | 0.00316 |
| smokepl | 4.6771 | 3.164 | 1 | 0.07526 |
| fatpl | 3.8415 | 0.797 | 1 | 0.37209 |
| chernobyl | 3.6718 | 0.508 | 1 | 0.47614 |
| ALL | | 13.291 | 5 | 0.02080 |

**Figure A.3.4.** Jarque-Bera test in VAR modeling for total stomach cancer mortality in Poland

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| stplr | 0.364 | 2 | 0.83352 |
| stplrd | 8.626 | 2 | 0.01339 |
| smokepl | 2.880 | 2 | 0.23692 |
| fatpl | 2.138 | 2 | 0.34330 |
| chernobyl | 0.325 | 2 | 0.84982 |
| ALL | 14.334 | 10 | 0.15829 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| stplr | -.28234 | 0.359 | 1 | 0.54921 |
| stplrd | -.0803 | 0.029 | 1 | 0.86474 |
| smokepl | -.51332 | 1.186 | 1 | 0.27619 |
| fatpl | -.57008 | 1.462 | 1 | 0.22654 |
| chernobyl | -.08712 | 0.034 | 1 | 0.85337 |
| ALL | | 3.070 | 5 | 0.68917 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| stplr | 2.9303 | 0.005 | 1 | 0.94111 |
| stplrd | 5.7644 | 8.597 | 1 | 0.00337 |
| smokepl | 4.2272 | 1.694 | 1 | 0.19304 |
| fatpl | 3.7751 | 0.676 | 1 | 0.41103 |
| chernobyl | 3.5089 | 0.291 | 1 | 0.58938 |
| ALL | | 11.264 | 5 | 0.04639 |

**Figure A.3.5.** Jarque-Bera test in VAR modeling for male stomach cancer mortality in Spain

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| smspr | 0.788 | 2 | 0.67430 |
| smsprd | 8.283 | 2 | 0.01590 |
| smokesp | 12.972 | 2 | 0.00152 |
| dfatsp | 0.388 | 2 | 0.82363 |
| chernobyl | 0.679 | 2 | 0.71222 |
| ALL | 23.110 | 10 | 0.01035 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| smspr | .02209 | 0.002 | 1 | 0.96332 |
| smsprd | -.46065 | 0.920 | 1 | 0.33760 |
| smokesp | -.97223 | 4.096 | 1 | 0.04299 |
| dfatsp | -.29849 | 0.386 | 1 | 0.53436 |
| chernobyl | .27828 | 0.336 | 1 | 0.56239 |
| ALL | | 5.739 | 5 | 0.33242 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| smspr | 2.1482 | 0.786 | 1 | 0.37530 |
| smsprd | 5.6071 | 7.363 | 1 | 0.00666 |
| smokesp | 5.8624 | 8.876 | 1 | 0.00289 |
| dfatsp | 2.9573 | 0.002 | 1 | 0.96457 |
| chernobyl | 2.4372 | 0.343 | 1 | 0.55802 |
| ALL | | 17.371 | 5 | 0.00385 |

**Figure A.3.6.** Jarque-Bera test in VAR modeling for female stomach cancer mortality in Spain

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| sfspr | 52.330 | 2 | 0.00000 |
| sfsprd | 0.733 | 2 | 0.69321 |
| smokesp | 22.403 | 2 | 0.00001 |
| dfatsp | 0.057 | 2 | 0.97174 |
| chernobyl | 2.893 | 2 | 0.23545 |
| ALL | 78.416 | 10 | 0.00000 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| sfspr | 2.0143 | 17.583 | 1 | 0.00003 |
| sfsprd | -.10642 | 0.049 | 1 | 0.82467 |
| smokesp | -1.3412 | 7.795 | 1 | 0.00524 |
| dfatsp | .07849 | 0.027 | 1 | 0.87022 |
| chernobyl | .72802 | 2.297 | 1 | 0.12965 |
| ALL | | 27.750 | 5 | 0.00004 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| sfspr | 8.6635 | 34.748 | 1 | 0.00000 |
| sfsprd | 3.7945 | 0.684 | 1 | 0.40829 |
| smokesp | 6.6722 | 14.609 | 1 | 0.00013 |
| dfatsp | 2.8318 | 0.031 | 1 | 0.86104 |
| chernobyl | 3.7416 | 0.596 | 1 | 0.44019 |
| ALL | | 50.666 | 5 | 0.00000 |

**Figure A.3.7.** Jarque-Bera test in VAR modeling for total stomach cancer mortality in Spain

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| stspr | 3.718 | 2 | 0.15580 |
| stsprd | 3.836 | 2 | 0.14688 |
| smokesp | 15.981 | 2 | 0.00034 |
| dfatsp | 0.178 | 2 | 0.91486 |
| chernobyl | 2.188 | 2 | 0.33481 |
| ALL | 25.902 | 10 | 0.00387 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| stspr | .78898 | 2.697 | 1 | 0.10051 |
| stsprd | -.24627 | 0.263 | 1 | 0.60819 |
| smokesp | -1.1842 | 6.077 | 1 | 0.01369 |
| dfatsp | -.1609 | 0.112 | 1 | 0.73767 |
| chernobyl | .70355 | 2.145 | 1 | 0.14304 |
| ALL | | 11.295 | 5 | 0.04584 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| stspr | 3.9708 | 1.021 | 1 | 0.31230 |
| stsprd | 4.8162 | 3.574 | 1 | 0.05871 |
| smokesp | 6.0235 | 9.904 | 1 | 0.00165 |
| dfatsp | 2.7536 | 0.066 | 1 | 0.79758 |
| chernobyl | 3.2003 | 0.043 | 1 | 0.83485 |
| ALL | | 14.607 | 5 | 0.01218 |

**Figure A.3.8.** Jarque-Bera test in VEC modeling for male lung cancer mortality in Poland

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| D_dlmplr | 0.063 | 2 | 0.96880 |
| D_dlmplrd | 0.549 | 2 | 0.76009 |
| D_smokepl | 2.254 | 2 | 0.32399 |
| D_dso2pl | 8.956 | 2 | 0.01136 |
| D_chernobyl | 0.103 | 2 | 0.94979 |
| ALL | 11.925 | 10 | 0.29009 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_dlmplr | .05241 | 0.012 | 1 | 0.91312 |
| D_dlmplrd | .00514 | 0.000 | 1 | 0.99146 |
| D_smokepl | .37634 | 0.614 | 1 | 0.43338 |
| D_dso2pl | -1.048 | 4.759 | 1 | 0.02914 |
| D_chernobyl | -.15337 | 0.102 | 1 | 0.74952 |
| ALL | | 5.487 | 5 | 0.35937 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_dlmplr | 2.782 | 0.051 | 1 | 0.82047 |
| D_dlmplrd | 2.2884 | 0.549 | 1 | 0.45892 |
| D_smokepl | 4.2305 | 1.640 | 1 | 0.20028 |
| D_dso2pl | 4.9682 | 4.197 | 1 | 0.04050 |
| D_chernobyl | 2.9683 | 0.001 | 1 | 0.97372 |
| ALL | | 6.438 | 5 | 0.26588 |

**Figure A.3.9.** Jarque-Bera test in VEC modeling for female lung cancer mortality in Poland

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| D_dlmplr | 0.102 | 2 | 0.95019 |
| D_dlmplrd | 0.628 | 2 | 0.73045 |
| D_smokepl | 0.347 | 2 | 0.84062 |
| D_dso2pl | 1.673 | 2 | 0.43320 |
| D_chernobyl | 0.192 | 2 | 0.90854 |
| ALL | 2.943 | 10 | 0.98274 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_dlmplr | .09076 | 0.036 | 1 | 0.85015 |
| D_dlmplrd | .14864 | 0.096 | 1 | 0.75700 |
| D_smokepl | .06574 | 0.019 | 1 | 0.89115 |
| D_dso2pl | -.55987 | 1.358 | 1 | 0.24383 |
| D_chernobyl | -.21029 | 0.192 | 1 | 0.66156 |
| ALL | | 1.700 | 5 | 0.88889 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_dlmplr | 2.7523 | 0.066 | 1 | 0.79651 |
| D_dlmplrd | 2.2989 | 0.532 | 1 | 0.46558 |
| D_smokepl | 3.5507 | 0.329 | 1 | 0.56653 |
| D_dso2pl | 3.5391 | 0.315 | 1 | 0.57473 |
| D_chernobyl | 2.9864 | 0.000 | 1 | 0.98872 |
| ALL | | 1.242 | 5 | 0.94074 |

**Figure A.3.10.** Jarque-Bera test in VEC modeling for total lung cancer mortality in Poland

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| D_ltplr | 1.799 | 2 | 0.40682 |
| D_ltplrd | 58.083 | 2 | 0.00000 |
| D_smokepl | 0.312 | 2 | 0.85575 |
| D_dso2pl | 1.323 | 2 | 0.51607 |
| D_chernobyl | 0.558 | 2 | 0.75651 |
| ALL | 62.075 | 10 | 0.00000 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_ltplr | .55353 | 1.328 | 1 | 0.24922 |
| D_ltplrd | 2.1344 | 19.741 | 1 | 0.00001 |
| D_smokepl | .15341 | 0.102 | 1 | 0.74946 |
| D_dso2pl | .53831 | 1.256 | 1 | 0.26247 |
| D_chernobyl | .00383 | 0.000 | 1 | 0.99364 |
| ALL | | 22.426 | 5 | 0.00043 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_ltplr | 3.6594 | 0.471 | 1 | 0.49249 |
| D_ltplrd | 8.9492 | 38.343 | 1 | 0.00000 |
| D_smokepl | 3.4398 | 0.210 | 1 | 0.64711 |
| D_dso2pl | 2.7507 | 0.067 | 1 | 0.79526 |
| D_chernobyl | 2.2823 | 0.558 | 1 | 0.45506 |
| ALL | | 39.649 | 5 | 0.00000 |

**Figure A.3.11.** Jarque-Bera test in VEC modeling for male lung cancer mortality in Spain

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| D_d2lmspr | 0.366 | 2 | 0.83267 |
| D_d2lmsprd | 0.417 | 2 | 0.81163 |
| D_smokesp | 1.290 | 2 | 0.52464 |
| D_dso2sp | 1.216 | 2 | 0.54447 |
| D_chernobyl | 0.389 | 2 | 0.82308 |
| ALL | 3.679 | 10 | 0.96067 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_d2lmspr | .0518 | 0.012 | 1 | 0.91414 |
| D_d2lmsprd | -.18705 | 0.152 | 1 | 0.69699 |
| D_smokesp | -.54556 | 1.290 | 1 | 0.25610 |
| D_dso2sp | .42105 | 0.768 | 1 | 0.38076 |
| D_chernobyl | .28332 | 0.348 | 1 | 0.55534 |
| ALL | | 2.569 | 5 | 0.76606 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_d2lmspr | 2.4279 | 0.355 | 1 | 0.55152 |
| D_d2lmsprd | 2.5047 | 0.266 | 1 | 0.60615 |
| D_smokesp | 3.018 | 0.000 | 1 | 0.98502 |
| D_dso2sp | 3.6428 | 0.448 | 1 | 0.50345 |
| D_chernobyl | 2.8042 | 0.042 | 1 | 0.83848 |
| ALL | | 1.110 | 5 | 0.95322 |

**Figure A.3.12.** Jarque-Bera test in VEC modeling for female lung cancer mortality in Spain

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| D_d2lfspr | 0.502 | 2 | 0.77795 |
| D_d2lfsprd | 0.749 | 2 | 0.68774 |
| D_smokesp | 0.250 | 2 | 0.88257 |
| D_dso2sp | 0.250 | 2 | 0.88259 |
| D_chernobyl | 0.147 | 2 | 0.92907 |
| ALL | 1.898 | 10 | 0.99707 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_d2lfspr | .03608 | 0.006 | 1 | 0.94014 |
| D_d2lfsprd | -.3404 | 0.502 | 1 | 0.47857 |
| D_smokesp | -.22949 | 0.228 | 1 | 0.63284 |
| D_dso2sp | .22084 | 0.211 | 1 | 0.64572 |
| D_chernobyl | -.12917 | 0.072 | 1 | 0.78801 |
| ALL | | 1.020 | 5 | 0.96097 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_d2lfspr | 2.323 | 0.497 | 1 | 0.48102 |
| D_d2lfsprd | 3.4771 | 0.247 | 1 | 0.61950 |
| D_smokesp | 3.1412 | 0.022 | 1 | 0.88314 |
| D_dso2sp | 2.8116 | 0.038 | 1 | 0.84452 |
| D_chernobyl | 2.7372 | 0.075 | 1 | 0.78442 |
| ALL | | 0.878 | 5 | 0.97179 |

**Figure A.3.13.** Jarque-Bera test in VEC modeling for female lung cancer mortality in Spain

Jarque-Bera test

| Equation | chi2 | df | Prob > chi2 |
|---|---|---|---|
| D_dltspr | 0.057 | 2 | 0.97180 |
| D_dltsprd | 0.093 | 2 | 0.95480 |
| D_smokesp | 1.235 | 2 | 0.53919 |
| D_dso2sp | 1.207 | 2 | 0.54685 |
| D_chernobyl | 0.333 | 2 | 0.84654 |
| ALL | 2.925 | 10 | 0.98312 |

Skewness test

| Equation | Skewness | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_dltspr | -.11172 | 0.054 | 1 | 0.81609 |
| D_dltsprd | -.12632 | 0.069 | 1 | 0.79258 |
| D_smokesp | -.40132 | 0.698 | 1 | 0.40348 |
| D_dso2sp | .5115 | 1.134 | 1 | 0.28698 |
| D_chernobyl | .24001 | 0.250 | 1 | 0.61734 |
| ALL | | 2.205 | 5 | 0.82018 |

Kurtosis test

| Equation | Kurtosis | chi2 | df | Prob > chi2 |
|---|---|---|---|---|
| D_dltspr | 2.9464 | 0.003 | 1 | 0.95553 |
| D_dltsprd | 2.8531 | 0.023 | 1 | 0.87851 |
| D_smokesp | 2.2956 | 0.537 | 1 | 0.46349 |
| D_dso2sp | 3.2603 | 0.073 | 1 | 0.78641 |
| D_chernobyl | 2.7222 | 0.084 | 1 | 0.77251 |
| ALL | | 0.721 | 5 | 0.98180 |

**Table A.4.1.** Additional output for VEC model for total lung cancer Polish mortality

| | | | | | | |
|---|---|---|---|---|---|---|
| D_ltplrd | | | | | | |
| _ce1 | | | | | | |
| L1. | .8112038 | 1.127633 | 0.72 | 0.472 | -1.398916 | 3.021324 |
| ltplr | | | | | | |
| LD. | -6.733777 | 3.691744 | -1.82 | 0.068 | -13.96946 | .5019075 |
| L2D. | -2.31024 | 3.293669 | -0.70 | 0.483 | -8.765712 | 4.145232 |
| L3D. | 2.094885 | 3.42556 | 0.61 | 0.541 | -4.619089 | 8.808859 |
| ltplrd | | | | | | |
| LD. | 6.873984 | 8.418919 | 0.82 | 0.414 | -9.626794 | 23.37476 |
| L2D. | -2.367318 | 7.381037 | -0.32 | 0.748 | -16.83388 | 12.09925 |
| L3D. | -8.09239 | 6.620962 | -1.22 | 0.222 | -21.06924 | 4.884456 |
| smokepl | | | | | | |
| LD. | 8.75e-07 | 1.09e-06 | 0.80 | 0.422 | -1.26e-06 | 3.01e-06 |
| L2D. | 7.84e-07 | 8.57e-07 | 0.91 | 0.360 | -8.95e-07 | 2.46e-06 |
| L3D. | 4.65e-07 | 6.23e-07 | 0.75 | 0.455 | -7.56e-07 | 1.69e-06 |
| dso2pl | | | | | | |
| LD. | -.003101 | .0035728 | -0.87 | 0.385 | -.0101036 | .0039016 |
| L2D. | -.0018456 | .0027043 | -0.68 | 0.495 | -.007146 | .0034548 |
| L3D. | -.0012666 | .0014793 | -0.86 | 0.392 | -.0041661 | .0016328 |
| chernobyl | | | | | | |
| LD. | -.3132561 | .3520147 | -0.89 | 0.374 | -1.003192 | .37668 |
| L2D. | .0668705 | .2639112 | 0.25 | 0.800 | -.4503859 | .584127 |
| L3D. | .2984761 | .2453832 | 1.22 | 0.224 | -.1824662 | .7794183 |
| _cons | .0042599 | .0060433 | 0.70 | 0.481 | -.0075847 | .0161045 |
| D_smokepl | | | | | | |
| _ce1 | | | | | | |
| L1. | 499754.7 | 601885 | 0.83 | 0.406 | -679918.3 | 1679428 |
| ltplr | | | | | | |
| LD. | -3027257 | 1970504 | -1.54 | 0.124 | -6889374 | 834860.3 |
| L2D. | 118062.7 | 1758028 | 0.07 | 0.946 | -3327608 | 3563734 |
| L3D. | 1815712 | 1828426 | 0.99 | 0.321 | -1767936 | 5399361 |
| ltplrd | | | | | | |
| LD. | 3981890 | 4493680 | 0.89 | 0.376 | -4825561 | 1.28e+07 |
| L2D. | -2182506 | 3939700 | -0.55 | 0.580 | -9904177 | 5539164 |
| L3D. | -6119824 | 3534003 | -1.73 | 0.083 | -1.30e+07 | 806693.7 |
| smokepl | | | | | | |
| LD. | .0219901 | .5817318 | 0.04 | 0.970 | -1.118183 | 1.162163 |
| L2D. | .2130379 | .4572172 | 0.47 | 0.641 | -.6830914 | 1.109167 |
| L3D. | -.0095935 | .3323759 | -0.03 | 0.977 | -.6610382 | .6418513 |
| dso2pl | | | | | | |
| LD. | -1883.611 | 1907.029 | -0.99 | 0.323 | -5621.318 | 1854.096 |
| L2D. | -425.7407 | 1443.461 | -0.29 | 0.768 | -3254.873 | 2403.392 |
| L3D. | -627.5766 | 789.6119 | -0.79 | 0.427 | -2175.188 | 920.0343 |
| chernobyl | | | | | | |
| LD. | -172214.6 | 187891.3 | -0.92 | 0.359 | -540474.7 | 196045.5 |
| L2D. | 67447.65 | 140865.2 | 0.48 | 0.632 | -208643 | 343538.3 |
| L3D. | 220490.6 | 130975.7 | 1.68 | 0.092 | -36216.96 | 477198.3 |
| _cons | .0021903 | 3225.664 | 0.00 | 1.000 | -6322.182 | 6322.187 |
| D_dso2pl | | | | | | |
| _ce1 | | | | | | |
| L1. | -816.7028 | 287.7839 | -2.84 | 0.005 | -1380.749 | -252.6566 |
| ltplr | | | | | | |
| LD. | -27.2092 | 942.1723 | -0.03 | 0.977 | -1873.833 | 1819.415 |
| L2D. | 1136.317 | 840.5794 | 1.35 | 0.176 | -511.1878 | 2783.823 |
| L3D. | 838.9606 | 874.2393 | 0.96 | 0.337 | -874.517 | 2552.438 |
| ltplrd | | | | | | |
| LD. | 4270.572 | 2148.598 | 1.99 | 0.047 | 59.39698 | 8481.746 |
| L2D. | 256.068 | 1883.719 | 0.14 | 0.892 | -3435.954 | 3948.09 |
| L3D. | 234.3458 | 1689.74 | 0.14 | 0.890 | -3077.484 | 3546.175 |
| smokepl | | | | | | |
| LD. | -.0007201 | .0002781 | -2.59 | 0.010 | -.0012653 | -.000175 |
| L2D. | -.0002953 | .0002186 | -1.35 | 0.177 | -.0007237 | .0001332 |
| L3D. | -.0001825 | .0001589 | -1.15 | 0.251 | -.0004939 | .000129 |
| dso2pl | | | | | | |
| LD. | 1.880236 | .9118223 | 2.06 | 0.039 | .0930971 | 3.667375 |
| L2D. | 1.037622 | .6901734 | 1.50 | 0.133 | -.3150931 | 2.390337 |
| L3D. | .2175993 | .3775432 | 0.58 | 0.564 | -.5223719 | .9575704 |
| chernobyl | | | | | | |
| LD. | -138.7514 | 89.8379 | -1.54 | 0.122 | -314.8305 | 37.32762 |
| L2D. | 11.50654 | 67.35295 | 0.17 | 0.864 | -120.5028 | 143.5159 |
| L3D. | -11.76788 | 62.62441 | -0.19 | 0.851 | -134.5095 | 110.9737 |
| _cons | 1.342575 | 1.542311 | 0.87 | 0.384 | -1.680299 | 4.36545 |

**Table A.4.2.** Additional output for VEC model for male lung cancer Polish mortality

| | | | | | | |
|---|---|---|---|---|---|---|
| D_dso2pl | | | | | | |
| _ce1 | | | | | | |
| L1. | 3376.793 | 2372.445 | 1.42 | 0.155 | -1273.113 | 8026.699 |
| _ce2 | | | | | | |
| L1. | -5559.117 | 5762.263 | -0.96 | 0.335 | -16852.95 | 5734.711 |
| _ce3 | | | | | | |
| L1. | .0001797 | .0002096 | 0.86 | 0.391 | -.0002311 | .0005906 |
| dlmplr | | | | | | |
| LD. | -3186.871 | 2319.201 | -1.37 | 0.169 | -7732.423 | 1358.68 |
| L2D. | -2296.924 | 1963.061 | -1.17 | 0.242 | -6144.452 | 1550.604 |
| L3D. | -1009.322 | 955.696 | -1.06 | 0.291 | -2882.452 | 863.8074 |
| dlmplrd | | | | | | |
| LD. | 5806.971 | 4363.775 | 1.33 | 0.183 | -2745.869 | 14359.81 |
| L2D. | 2917.362 | 3203.565 | 0.91 | 0.362 | -3361.51 | 9196.235 |
| L3D. | 1192.11 | 1392.293 | 0.86 | 0.392 | -1536.734 | 3920.954 |
| smokepl | | | | | | |
| LD. | -.0002763 | .0003279 | -0.84 | 0.399 | -.000919 | .0003664 |
| L2D. | .0003144 | .0002054 | 1.53 | 0.126 | -.0000882 | .000717 |
| L3D. | .0001968 | .0002142 | 0.92 | 0.358 | -.000223 | .0006166 |
| dso2pl | | | | | | |
| LD. | .4439383 | .6400807 | 0.69 | 0.488 | -.8105969 | 1.698473 |
| L2D. | .1869515 | .4313787 | 0.43 | 0.665 | -.6585352 | 1.032438 |
| L3D. | .459477 | .4556522 | 1.01 | 0.313 | -.4335848 | 1.352539 |
| chernobyl | | | | | | |
| LD. | .6064695 | 7.232665 | 0.08 | 0.933 | -13.56929 | 14.78223 |
| L2D. | 6.509194 | 5.970391 | 1.09 | 0.276 | -5.192557 | 18.21094 |
| L3D. | -4.836119 | 10.15438 | -0.48 | 0.634 | -24.73834 | 15.0661 |
| _cons | .0544905 | 4.954767 | 0.01 | 0.991 | -9.656674 | 9.765655 |

**Table A.4.3.** Additional output for VEC model for female lung cancer Polish mortality

| | | | | | | |
|---|---|---|---|---|---|---|
| D_dso2pl | | | | | | |
| _ce1 | | | | | | |
| L1. | -4377.216 | 5115.402 | -0.86 | 0.392 | -14403.22 | 5648.788 |
| _ce2 | | | | | | |
| L1. | 12025.99 | 9101.506 | 1.32 | 0.186 | -5812.629 | 29864.62 |
| dlfplr | | | | | | |
| LD. | 7988.436 | 4347.235 | 1.84 | 0.066 | -531.9888 | 16508.86 |
| L2D. | 869.6059 | 3241.744 | 0.27 | 0.789 | -5484.096 | 7223.308 |
| L3D. | -834.2287 | 1810.596 | -0.46 | 0.645 | -4382.932 | 2714.475 |
| dlfplrd | | | | | | |
| LD. | -4643.881 | 8893.166 | -0.52 | 0.602 | -22074.17 | 12786.4 |
| L2D. | -3261.962 | 6062.518 | -0.54 | 0.591 | -15144.28 | 8620.356 |
| L3D. | 6546.69 | 2906.348 | 2.25 | 0.024 | 850.3524 | 12243.03 |
| smokepl | | | | | | |
| LD. | -.0002302 | .000143 | -1.61 | 0.107 | -.0005104 | .00005 |
| L2D. | .0000856 | .0001129 | 0.76 | 0.448 | -.0001357 | .0003068 |
| L3D. | -.0002826 | .0001319 | -2.14 | 0.032 | -.0005411 | -.0000241 |
| dso2pl | | | | | | |
| LD. | .3039423 | .3886216 | 0.78 | 0.434 | -.4577419 | 1.065627 |
| L2D. | -.2817016 | .3022482 | -0.93 | 0.351 | -.8740972 | .310694 |
| L3D. | -.3258061 | .2071957 | -1.57 | 0.116 | -.7319022 | .0802901 |
| chernobyl | | | | | | |
| LD. | 3.10908 | 7.174056 | 0.43 | 0.665 | -10.95181 | 17.16997 |
| L2D. | 5.173901 | 5.532215 | 0.94 | 0.350 | -5.669041 | 16.01684 |
| L3D. | -11.18136 | 3.945558 | -2.83 | 0.005 | -18.91451 | -3.448205 |
| _cons | -2.016782 | 5.288755 | -0.38 | 0.703 | -12.38255 | 8.348987 |

**Table A.4.4.** Additional output for VEC model for total lung cancer Spanish mortality

| | | | | | | |
|---|---|---|---|---|---|---|
| D_dltsprd | | | | | | |
| _ce1 | | | | | | |
| L1. | -1.864358 | .8773789 | -2.12 | 0.034 | -3.583989 | -.144727 |
| dltspr | | | | | | |
| LD. | 1.692986 | .8614839 | 1.97 | 0.049 | .0045083 | 3.381463 |
| L2D. | 1.266714 | .7200223 | 1.76 | 0.079 | -.1445037 | 2.677932 |
| L3D. | .3746541 | .3882013 | 0.97 | 0.334 | -.3862065 | 1.135515 |
| dltsprd | | | | | | |
| LD. | -2.241319 | .9268634 | -2.42 | 0.016 | -4.057937 | -.4246997 |
| L2D. | -1.90139 | .8422591 | -2.26 | 0.024 | -3.552187 | -.2505922 |
| L3D. | -.414778 | .5456809 | -0.76 | 0.447 | -1.484293 | .6547369 |
| smokesp | | | | | | |
| LD. | -6.94e-08 | 4.62e-08 | -1.50 | 0.133 | -1.60e-07 | 2.11e-08 |
| L2D. | -8.94e-08 | 4.26e-08 | -2.10 | 0.036 | -1.73e-07 | -5.86e-09 |
| L3D. | -6.79e-09 | 1.75e-08 | -0.39 | 0.698 | -4.11e-08 | 2.75e-08 |
| dso2sp | | | | | | |
| LD. | -.0003742 | .0001645 | -2.27 | 0.023 | -.0006967 | -.0000517 |
| L2D. | -.0002176 | .0001189 | -1.83 | 0.067 | -.0004505 | .0000154 |
| L3D. | -.0000274 | .0000747 | -0.37 | 0.714 | -.0001738 | .000119 |
| chernobyl | | | | | | |
| LD. | .0077651 | .0037302 | 2.08 | 0.037 | .000454 | .0150761 |
| L2D. | .0002338 | .0040677 | 0.06 | 0.954 | -.0077387 | .0082063 |
| L3D. | .0070493 | .0041566 | 1.70 | 0.090 | -.0010975 | .015196 |
| _cons | .0001917 | .0002065 | 0.93 | 0.353 | -.0002129 | .0005963 |
| D_smokesp | | | | | | |
| _ce1 | | | | | | |
| L1. | 3.49e+07 | 1.78e+07 | 1.96 | 0.050 | -58081.32 | 6.98e+07 |
| dltspr | | | | | | |
| LD. | -2.64e+07 | 1.75e+07 | -1.51 | 0.132 | -6.07e+07 | 7929845 |
| L2D. | -1.92e+07 | 1.46e+07 | -1.31 | 0.190 | -4.78e+07 | 9514970 |
| L3D. | -3739290 | 7885755 | -0.47 | 0.635 | -1.92e+07 | 1.17e+07 |
| dltsprd | | | | | | |
| LD. | 2.63e+07 | 1.88e+07 | 1.40 | 0.163 | -1.06e+07 | 6.32e+07 |
| L2D. | 2.35e+07 | 1.71e+07 | 1.37 | 0.170 | -1.00e+07 | 5.70e+07 |
| L3D. | 1.21e+07 | 1.11e+07 | 1.09 | 0.276 | -9640991 | 3.38e+07 |
| smokesp | | | | | | |
| LD. | .7638294 | .9375067 | 0.81 | 0.415 | -1.07365 | 2.601309 |
| L2D. | .7238469 | .8660237 | 0.84 | 0.403 | -.9735283 | 2.421222 |
| L3D. | -.55712 | .3556192 | -1.57 | 0.117 | -1.254121 | .1398808 |
| dso2sp | | | | | | |
| LD. | 7891.254 | 3342.544 | 2.36 | 0.018 | 1339.988 | 14442.52 |
| L2D. | 6430.491 | 2414.487 | 2.66 | 0.008 | 1698.183 | 11162.8 |
| L3D. | 2222.53 | 1517.331 | 1.46 | 0.143 | -751.3842 | 5196.443 |
| chernobyl | | | | | | |
| LD. | -58306.43 | 75773.85 | -0.77 | 0.442 | -206820.4 | 90207.57 |
| L2D. | -47802.57 | 82629.3 | -0.58 | 0.563 | -209753 | 114147.9 |
| L3D. | -135658 | 84435.02 | -1.61 | 0.108 | -301147.6 | 29831.58 |
| _cons | 6.12e-06 | 4193.811 | 0.00 | 1.000 | -8219.719 | 8219.719 |
| D_dso2sp | | | | | | |
| _ce1 | | | | | | |
| L1. | 839.9558 | 4283.736 | 0.20 | 0.845 | -7556.013 | 9235.924 |
| dltspr | | | | | | |
| LD. | 339.0693 | 4206.13 | 0.08 | 0.936 | -7904.793 | 8582.932 |
| L2D. | 575.1182 | 3515.454 | 0.16 | 0.870 | -6315.045 | 7465.282 |
| L3D. | 450.7302 | 1895.363 | 0.24 | 0.812 | -3264.114 | 4165.574 |
| dltsprd | | | | | | |
| LD. | -1631.775 | 4525.34 | -0.36 | 0.718 | -10501.28 | 7237.729 |
| L2D. | -770.062 | 4112.266 | -0.19 | 0.851 | -8829.955 | 7289.831 |
| L3D. | -714.4289 | 2664.246 | -0.27 | 0.789 | -5936.254 | 4507.396 |
| smokesp | | | | | | |
| LD. | -.0000243 | .0002253 | -0.11 | 0.914 | -.0004659 | .0004173 |
| L2D. | -.0000204 | .0002082 | -0.10 | 0.922 | -.0004283 | .0003876 |
| L3D. | -.0000182 | .0000855 | -0.21 | 0.831 | -.0001857 | .0001493 |
| dso2sp | | | | | | |
| LD. | -.5210295 | .8033898 | -0.65 | 0.517 | -2.095645 | 1.053586 |
| L2D. | -.2937203 | .5803288 | -0.51 | 0.613 | -1.431144 | .8437032 |
| L3D. | -.0106107 | .3646947 | -0.03 | 0.977 | -.7253992 | .7041779 |
| chernobyl | | | | | | |
| LD. | -2.601041 | 18.21246 | -0.14 | 0.886 | -38.2968 | 33.09472 |
| L2D. | -2.163061 | 19.86018 | -0.11 | 0.913 | -41.0883 | 36.76218 |
| L3D. | 11.80746 | 20.29419 | 0.58 | 0.561 | -27.96843 | 51.58335 |
| _cons | -.2164088 | 1.007994 | -0.21 | 0.830 | -2.192041 | 1.759224 |
| D_chernobyl | | | | | | |
| _ce1 | | | | | | |
| L1. | -597.849 | 185.3881 | -3.22 | 0.001 | -961.203 | -234.4949 |
| dltspr | | | | | | |
| LD. | 457.9239 | 182.0295 | 2.52 | 0.012 | 101.1526 | 814.6953 |
| L2D. | 322.6634 | 152.139 | 2.12 | 0.034 | 24.47638 | 620.8505 |
| L3D. | 66.6961 | 82.02604 | 0.81 | 0.416 | -94.07198 | 227.4642 |
| dltsprd | | | | | | |
| LD. | -472.8373 | 195.8441 | -2.41 | 0.016 | -856.6847 | -88.98997 |
| L2D. | -403.4283 | 177.9674 | -2.27 | 0.023 | -752.238 | -54.61867 |
| L3D. | -182.0783 | 115.3011 | -1.58 | 0.114 | -408.0643 | 43.90776 |
| smokesp | | | | | | |
| LD. | -.0000163 | 9.75e-06 | -1.67 | 0.094 | -.0000354 | 2.79e-06 |
| L2D. | -.0000195 | 9.01e-06 | -2.16 | 0.031 | -.0000371 | -1.81e-06 |
| L3D. | 5.22e-06 | 3.70e-06 | 1.41 | 0.158 | -2.03e-06 | .0000125 |
| dso2sp | | | | | | |
| LD. | -.1220092 | .0347685 | -3.51 | 0.000 | -.1901542 | -.0538643 |
| L2D. | -.0973987 | .025115 | -3.88 | 0.000 | -.1466232 | -.0481742 |
| L3D. | -.0448353 | .015783 | -2.84 | 0.005 | -.0757694 | -.0139013 |
| chernobyl | | | | | | |
| LD. | 1.986782 | .7881843 | 2.52 | 0.012 | .4419689 | 3.531595 |
| L2D. | .5295833 | .8594933 | 0.62 | 0.538 | -1.154993 | 2.214159 |
| L3D. | 2.359481 | .8782761 | 2.69 | 0.007 | .6380914 | 4.08087 |

**Table A.4.5.** Additional output for VEC model for male lung cancer Spanish mortality

| | | | | | | |
|---|---|---|---|---|---|---|
| D_dso2sp | | | | | | |
| _ce1 | | | | | | |
| L1. | 3492.971 | 3214.682 | 1.09 | 0.277 | -2807.689 | 9793.631 |
| _ce2 | | | | | | |
| L1. | -5545.071 | 3255.284 | -1.70 | 0.088 | -11925.31 | 835.1684 |
| d2lmspr | | | | | | |
| LD. | -2607.87 | 2780.263 | -0.94 | 0.348 | -8057.085 | 2841.345 |
| L2D. | -1330.836 | 1802.102 | -0.74 | 0.460 | -4862.891 | 2201.219 |
| L3D. | -295.7644 | 721.2647 | -0.41 | 0.682 | -1709.417 | 1117.888 |
| d2lmsprd | | | | | | |
| LD. | 4397.375 | 2852.043 | 1.54 | 0.123 | -1192.526 | 9987.277 |
| L2D. | 2674.905 | 1946.512 | 1.37 | 0.169 | -1140.188 | 6489.997 |
| L3D. | 1252.566 | 906.8022 | 1.38 | 0.167 | -524.734 | 3029.865 |
| smokesp | | | | | | |
| LD. | -.0000886 | .0001589 | -0.56 | 0.577 | -.0004001 | .000223 |
| L2D. | -.0001036 | .0001332 | -0.78 | 0.437 | -.0003647 | .0001575 |
| L3D. | .0000202 | .0000817 | 0.25 | 0.805 | -.0001399 | .0001803 |
| dso2sp | | | | | | |
| LD. | -.3355713 | .678547 | -0.49 | 0.621 | -1.665499 | .9943564 |
| L2D. | -.091762 | .495481 | -0.19 | 0.853 | -1.062887 | .879363 |
| L3D. | -.0950523 | .3511051 | -0.27 | 0.787 | -.7832056 | .593101 |
| chernobyl | | | | | | |
| LD. | -2.288044 | 13.87189 | -0.16 | 0.869 | -29.47645 | 24.90036 |
| L2D. | 1.746658 | 18.91169 | 0.09 | 0.926 | -35.31957 | 38.81289 |
| L3D. | 26.80115 | 15.46126 | 1.73 | 0.083 | -3.502369 | 57.10468 |
| _cons | -.0226934 | 8.381792 | -0.00 | 0.998 | -16.4507 | 16.40532 |