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Chernobyl sick child syndrome persisting through life: testing the infancy inflammation hypothesis conditioned by socio-economic origin

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Abstract: Previous research has shown that irradiation in-utero or during early childhood may cause longlasting damage. Studies on the Chernobyl accident have found that all birth groups which received high doses of radiation in early life may have severe persisting disadvantage in health. However, we do not know which of the groups irradiated in early life was the most vulnerable. We also do not know whether the accident directly left permanent scars on the health of those who were irradiated with low doses. Most importantly, we do not know whether radiation affected the health of these individuals indirectly, through the socio-economic conditions in early life. The recent wave of the Ukrainian Longitudinal Monitoring Survey of 2007 with individual-level data, and regional-level data on radiation doses from the National report of Ukraine on the Chernobyl accident of 2006 contain all the information needed to establish mechanisms between early-life irradiation and health status in adulthood. The study design distinguishes between different birth groups with varying exposure, such as exposed in-utero, exposed during infancy, exposed between the ages one and five, and conceived after the end of exposure period. The study covers two exposure periods, which lasted three months and eight months after the accident. Parental educational status is used as a socio-economic variable. Important controls include migration and place of birth. We find no direct effect from irradiation at low doses in early life on self-perceived health in young adulthood. However, we find that radiation strongly affected adult health through the socio-economic conditions in early life, especially for those who were exposed during infancy. This negative effect is found to be ameliorated to almost negligible levels by family resources. Individuals irradiated during infancy have the probability of 0.5 to have bad health in young adulthood if they had lived in poor families, and of 0.1 if they had lived in wealthy families. Much lower vulnerability and its amelioration is indicated for the group exposed while in-utero and between the ages one and five. However, only the effect for those irradiated during infancy is robust to different specifications. This effect is found also for those who received low doses of radiation.

Key words: critical period approach, infancy inflammation hypothesis, socio-economic origin, Chernobyl accident, Ukraine, self-reported health

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List of tables

List of figures

List of appendices

1. Introduction

1.1 Research problem

In discussing nutrition and infections as possible attributes of a drastic decline in mortality and morbidity in the world during the last 300 years, Floud et al. (2011) provide some findings on the main causes of infant deaths. The authors exemplify that until the early twentieth century, before the advancements in food storage were introduced, one of the main causes of high urban infant mortality rate among low-socioeconomic classes was diarrhea, inflicted by contaminated milk which was supplied from rural farms - the only source of cheap milk for poor families (p.327). While the role of nutrition is also not undermined by authors throughout the essay, this particular example implicitly points to an interplay between different factors which might ameliorate damage from the infectious environment. Thus, in a potentially severe environment, family access to resources becomes the key factor in providing better health for children. In the authors' discussion, these advantages in health and wealth in childhood largely determine wellbeing over the life-time. This argumentation constitutes the framework of our study.

In the current literature, the pathways from early life to future health are conceptualized within a life course approach. The idea that diseases in childhood leave permanent damage is not new, the heart sequelae (White, 1931) or cancer (Cherry, 1924) caused by preceding events in childhood were recognized in studies long ago. However, it was until the thalidomide episode in 1950s-60s when the public and scientists became aware that environmental susceptibility might start in-utero. Thalidomide was first synthesized in Germany in 1954, and after extensive trials was prescribed to pregnant women throughout the world to treat morning sickness until 1961, when medical scientists established its association with gross limb and other deformities in new born babies (Joseph, 1962). Since then the notion of early scholars, that exposure during a particular period in early life may have lasting or lifelong effects on the anatomic and immune systems that are not modified in any dramatic way by later life experiences, has gained a new prominence.

Scholars have limited the timing of critical periods to early life. In a study on the mortality decline in England, Wales, Scotland and Sweden, Kermack et al (1934) identified that the environment up to the age of five years is the important factor of life-long health, and that improved conditions at later ages have little direct effect. With regard to underlying biological links, current researchers further proposed that the very first years of development or life are especially important for future health. The *fetal origin* hypothesis suggests that intrauterine environment can program the fetus to have particular metabolic responses and affect growth, which cause future diseases, thereby emphasizing in-utero as a critical period (Barker, 1994). The *infancy inflammation* hypothesis postulates that exposure to infections and injures during infancy can be associated with adult morbidity and mortality (Bengtsson, 1997). It has been explained that infectious environment at first year of life causes inflammatory responses which permanently damage immune and anatomic structure, and lead to diseases in adulthood (Liuba, 2003, Palinski & Napoli, 2002). While emphasizing infancy as the crucial critical period, inflammation studies argue that both fetuses and children after the first year of life may also be vulnerable to inflammogens (Finch & Crimmins, 2004).

Epidemiological and historical literature has produced some evidence on the critical period hypotheses. The bulk of the literature provided strong support on correlations between early events and future diseases. Several historical studies on the basis of macro and regional data have showed that factors in early life can account for trends in mortality rates and morbidities (Preston & van de Walle, 1978, Fridlizius, 1989, Finch & Crimmins, 2004). Cohort and case medical studies suggested that infectious diseases during first years of life (Lauer et al, 1978, Kaplan & Salonen, 1990, Willerson & Ridker, 2004) and low birth weight (Barker, 1994, Painter et al, 2008) may be associated with diseases of ageing. Based on longitudinal data for individuals, contemporary studies have supported the causality between early events and future outcomes. The strand of literature with individual historical data has identified that exposure to infectious diseases during infancy leads to adult mortality (Bengtsson & Lindström, 2000, 2003, Bengtsson & Alter, 2008). Some findings from individual data have indicated that intrauterine environment is an important determinant of future diseases (Stein et al, 2006, Almond et al, 2010). The individual-based studies have shown that socio-economic conditions in early life may also explain later health outcomes (Palloni et al, 2009) as well as they are able to mediate the damage from exposure to infectious diseases after birth (Bengtsson & Helgertz, 2012).

The course of industrialization offered researchers an additional source of environmental hazard to investigate – pollutants, the long-term influence of which is underexplored in the field. Epidemiologists launched research on the effects of pollutants at a time with other life course scholars, and likewise supported the importance of early life factors (Douglas & Waller, 1966, Mann et al, 1992). The findings of early scholars that the long term biological mechanism of pollutants is purely inflammatory (Ophuls, 1921), have been revitalized by current researchers (Finch & Crimmins, 2006). Cohort and case epidemiological studies showed that the series of responses caused by pollution inflammogens are dependent on the dose of exposure, but at any dose they can be stored in bodies for a long time and persistently affect health (Pellmar & Ledney, 2005, Lukyanova et al, 2005). Recently, historians have begun to investigate the long term effects of early life exposure to toxins with individual level data. The studies, often based on natural experiments of exposure, indicate the negative effects for those exposed either in-utero (Almond et al, 2009, Sanders, 2011, Black et al, 2013) or during early childhood (Nilsson, 2009, Yemeluanau & Amialchuk, 2012). Taken together, the micro level studies on pollutants are scarce, but they all point to the considerable differences among pollutants in their longlasting effect on health.

Among noninfectious inflammogens, radiation is the most dangerous for long-life health status. This seems to stem from the scientific fact that radionuclides, in addition to generating common inflammatory effects, are the only inflammogens capable of causing direct cell death and even affecting genetic codes (Hall & Giaccia, 2011). Additional danger is that human bodies accumulate radioisotopes as part of normal metabolic processes (Lukyanova, 2003). Researchers have been documenting health outcomes of early life exposure to radiation since the Hiroshima and Nagasaki atoming bombings in 1945. Diseases such as cataracts, leukemia and other cancers, malformed offspring, premature ageing, and neurological problems were detected among those adults, exposed at high levels of bombings radiation in their childhood (Nakane & Ohta, 1986, Saaki et al, 2002, Yamada et al, 2004). However, much higher risks and scope of these diseases appeared after the Chernobyl accident in former Soviet Union in 1986, the most severe radiation accident in the history of industrial world (WHO, 2007). The

Chernobyl fallout spread over Belarus, Ukraine, Russia, and at lesser levels over European countries. According to available data (UNSCEAR, 2008a), the whole amount of released radioisotopes due to the disaster was approximately one hundred times of that received by the atmosphere from the atom bombs in Japan. Given the scale of the accident, the long term health consequences for those exposed to Chernobyl radiation during early life are considered to be severe by current epidemiologists (Yablokov et al, 2009). The *sick child syndrome* is regarded, albeit metaphorically, as a characteristic of the population of children affected by the Chernobyl radiation during their first years of life (Stepanova, 2006). Throughout their development from childhood to older ages, these individuals have systematically indicated higher susceptibility to infections, mental disorders, dysfunction of metabolic systems, and diseases of the sense organs in comparison to non-exposed cohorts.

The Chernobyl radioactive fallout, which irradiated the entire Ukrainian population, has been chosen in this study as a quasi-experiment in early life exposure to radiation. Based on individual-level cross-sectional data coming from a recent wave of national surveys, we are able to match the varying irradiation in early life among different birth cohorts to health outcomes in their young adulthood. The richness of data allows us to control for potential confounders, such as migration and place of birth, as well as to identify the socio-economic status of the individual at the exposure period in early life. In this study, we will also incorporate regional level information on radiation doses received due to the accident, and then estimate the effects for birth cohorts irradiated at low dose levels. An exposure to radionuclides released after the Chernobyl accident had long since decayed to negligible levels. Among radioisotopes, which covered the entire Ukraine, iodine isotopes decomposed in a three-month period, whereas cesium isotopes were available in the topsoil during eight months, and in some regions will continue to irradiate for decades to come (see Appendix A). To account for this, we will first establish exposure period at a physical life of iodine radionuclides, and then lengthen it to eight months to consider an exposure to cesium radionuclides. The use of two exposure periods will enable us to identify the potential downward bias in our results related to this peculiarity of exposure.

This study is not only seeking to explore biological pathway from exposure to Chernobyl radiation. Instead, following the historical literature, we argue that, if any, health responses from this exogenous insult might have been conditioned by the socio-economic status of an exposed individual at the time of accident. In other words, to the extent that individuals at particular periods of their early life were environmentally vulnerable, early life events could be mediated by socio-economic environment at a household level. Conceptually, there are several reasons why this mediation is possible. On the one hand, parents can distribute more resources to their children in order to compensate for negative outcomes of exposure. On the other hand, individuals from more affluent families may originally experience a weaker effect from negative insults due to high initial level of available resources, and they may potentially be subject to less and smaller early shocks than children from poor families. Given this conceptuality, we will principally examine socio-biological pathways from irradiation during early life to health in later life.

1.2 Research question

By what mechanisms is damage from exposure to radiation in early life transferred to health status in adulthood? More specifically, does exposure to radiation in early life cause long-lasting damage directly, through biological processes, and do socio-economic conditions in early life have a direct effect on health in adulthood? Can exposure to radiation in early life be mediated by socio-economic conditions?

This study is quasi-experimental, and therefore we expect to indicate the causality between environmental factors in early life and future outcomes. With individual-based data covering birth cohorts exposed to the Chernobyl accident in early life, we are able to establish exogenous shock to their health and identify its consequences for future health. Later health outcomes are observed in young adulthood. Since any health responses are likely to be mediated by socio-economic environment in early life, we incorporate early-life socio-economic status into the analysis. Socio-economic conditions in early life may strengthen or weaken the impact from radiation exposure as an additional determinant, and may indicate possibility to compensate for negative health consequences at a family level. This will allow to propose some policy implications.

The study is structured as follows. Section 1 discusses previous research and states the theoretical approach linking early life exposure and child socio-economic status to adult health outcomes. Based on theoretical model, Section 1 formulates empirically testable hypotheses. Section 2 motivates exposure period, and describes the data and methodology. In Section 3 the results are presented and discussed. The study ends with some conclusions.

2. Theory

2.1 Previous research

The idea that early life conditions and experiences affect adult health through accumulation of risk factors was developed in research in the first half of the twentieth century. The early origins of the notion can be traced back to Hellstenius who in the 1870s emphasized the importance of proper nutrition and treatment in childhood for good health in adult life. The author indicated the close association between the health status of Swedish cohorts mustering to the army and harvests during their fetal stage (Hellstenius, 1871 cited in Bengtsson & Mineau, 2009, p.1561). The idea of the importance of early life factors began to dominate the discussion of health scholars and public authorities much later, in the 1930s, in line with attempts to explain the dramatic decline in mortality in Europe observed since 1850s. By using macro data, demographers supported their arguments with findings on age-specific mortality for Sweden, England, Wales and Scotland (Kermack et al, 1934, Derrick, 1927). Derrick (1927) first clearly indicated stronger regularities in birth cohort changes in adult mortality rates relative to period variations. Kermack et al (1934) argued that 'each generation after the age of five years seems to carry along with it the same relative mortality throughout adult life, and even into extreme old age' (p.679). They also concluded that environment in early life is a major determinant of reductions in the death rates of the various age groups, and improved conditions at later ages, including the particular year under consideration, have little direct effect.

In the 1920s-30s, medical scientists also began to apply the concept of the influential developmental periods in early life. The importance of early development was studied for different birth cohorts, and more specifically, for the in-utero (e.g. Stockard, 1921) and infancy stages (e.g. Watson, 1928). From animal experiments, Stockard (1927) concluded that there are critical periods in the development of organs when they may be damaged acutely by severe environmental conditions, and there are passive periods when organs suffer only slightly. This finding, which echoes many others published at that time, reflects the underlying assumption that later on formed the so-called life course approach: individual's development is cumulative, and the effects of unfavorable conditions and injuries in early childhood may be irreversible. In explaining biological mechanisms of better health, the research of the 1930s concentrated their interest not only on nutrition, but especially on the infectious disease load which, as proposed, might be carried from early childhood to health status later in life. By analyzing case data, epidemiological studies suggested a link between different infections and adult health, namely between rheumatic fever and heart sequela (White, 1931), tuberculosis and cancer in later life (Cherry, 1924), different infections, such as influenza, pneumonia, and diarrhea among others, and the development of arteriosclerosis (Ophuls, 1921). In explaining these links, researchers also suggested that infectious diseases generate inflammatory processes that may influence future health (Ophuls, 1921). Taken together, all these historical and epidemiological studies predicted the dramatic fall in mortality for the middle-aged generation of the interwar period. In line with explanations of mortality trends for the previous decades, reductions in death rates were supposed to occur due to dramatic improvements in the cohort's early life conditions. When these predictions were not confirmed, theories on early origins were strongly criticized, and as a result, the concept stagnated until the 1970s.

Due to the difficulties in explaining the long-life health trajectories with other concepts, early-origins theories regained interest within both medical and historical studies in the 1970s-80s. Longitudinal research on chronic disease launched by medical scientists ascribed adult diseases to illnesses in early life (Kiernan et al, 1976, Cole & MacMahon, 1969, Lauer et al, 1978). These studies suggested that factors such as malnutrition and infections might directly cause early life diseases, and then, through health behavior in adulthood, indirectly increase risks of cardiovascular and respiratory diseases in old ages. Revising the methodology of Kermack and his collegues, demographers have also launched a bulk of studies that use macro data on mortality. Linking adult and infant death rates for Norway, Forsdahl (1978) argued that poor living conditions in early childhood might expedite aging and increase mortality. The author attributed the causes of adult diseases to poor diet in early childhood, and concluded that surviving individuals would carry with them a life-long vulnerability. A decade later, Floud et al (1990) made similar findings when investigating the effects of nutritional status in the UK by using data on height and mortality in historical populations. By applying the vast European and American statistics on health and nutrition, Fogel (1993) further suggested that decrease in net-nutrition, either due to the lack of nutrients or increased demands because of disease during fetal stage or early childhood may lead to chronic diseases in later life.

Other historians attributed adult morbidity not only to nutrition, but also to infectious diseases in early life. Having compiled mortality and health statistics for the three largest urban areas of France, Preston and van de Walle (1978) concluded that long term physical development is as closely associated with disease and death rates from infectious disease in early childhood as with nutritional indicators. Other historians strongly advocated exposure to infectious diseases in early life as a main contributor to adult risk factors and suggested the causal mechanism alternative to nourishment. Fridlizius (1989) extended Kermack et al's analysis on mortality trends for Sweden by identifying four different causes, such as tuberculosis, alcohol, improvement in social conditions and other factors. The author concluded that the development of disease and death in adulthood seemed to be primarily related to infectious diseases, such as smallpox, in the first five years of life, which later in life contributed to adult risk factors. Fridlizius supposed that exposure to infectious illnesses in early childhood may reduce immunity to other diseases in later life. Lunn (1991) has made similar findings in the study on mortality trends for developing countries. The researcher argued that infectious disease, such as measles, by itself frequently causes more severe growth faltering or impairment of immune status than nutrition alone. In all, the historical and medical research of the 1970s-80s, which emphasized either the role of malnutrition or exposure to infections in early life, strongly supported that risk factors are accumulated over the individual's life.

In line with the development of early origins literature, studies have recognized that socio-economic status in childhood can adversely affect health in later life. Among early findings, Engels could first showed distinct mortality patterns related to all age groups, and to upper and lower classes in Manchester in 1844 (Engels, 1845). During the 1920s-30s, morbidity and death trends have been attributed to socio-economic conditions in early life, both by public officers and scholars. During the Great Depression of the 1930s, national activist Wall Hannington claimed that 'unemployment has existed for so long … that many of the youths who are to-day leaving school where probably handicapped from the moment of their birth…' (cited in Kuh et al, 2004c, p.9). Medical researchers and demographers also implicitly

discussed poor socio-economic conditions within their studies on the nutritional or infectious origins of later life morbidity. In the 1970s, the attention to socio-economic environment has gained renewed prominence in the field, although the studies, which directly investigated the impact of children's socio-economic background, were relatively scarce among other early origins literature. Ravelli et al (1976) examined the relationship between prenatal and early postnatal exposure to famine during the Dutch Hunger Winter 1944-45 and the health status of the recruits to the army, who were originated from families with either manual or non-manual workers. Although early life socio-economic status was discussed in the study as a potential confounding effect, researchers have found much higher obesity rates for those exposed in-utero, and these health outcomes were accentuated with social origin. In a review of early origins studies of the 1980s, Elo and Preston (1992) noted that only a few of them investigate the links between household characteristics in childhood and adult mortality. In the population-based studies, Finnish researchers found greater relative risks of adult mortality and morbidity from coronary heart disease for individuals that originated from lowest socio-economic class (Notkola et al, 1985, Kaplan & Salonen, 1990). However, controlling for other factors, such as socio-economic status in adulthood or adult health behaviour, makes the effects almost negligible. In the longitudinal macro-data study for the US, Mare (1986) explored the association between different early life factors and adult mortality rates. They found that cohorts born in labour families demonstrate much higher death rates than those who were born in the families of professionals, but that this socio-economic gradient disappears when own education of individuals is taken into account. Like other early origins literature, studies linking parental characteristics and adult morbidity pointed to the interplay of different factors throughout life, which accumulate the damage from early life exposures and can partially or wholly modify them in later ages. Until recently, this proposition has been questioned in the life-course studies.

Originating from early literature, it was until 1990s when researchers claimed that damage from environmental events during particular periods in early life may be completely irreversible. Previous medical and historical studies allowed Barker and his team to suggest and demonstrate with empirical data that events in early life might be linked to various diseases which manifest themselves only in middle ages or later. In his early works, Barker and his colleagues examined mortality statistics for England and Wales to compare adult death rates from specific causes with causes of infant deaths in the tradition of the previous macro studies (Barker & Osmond, 1986, Barker, 1991). The author argued that exposure to infectious diseases in prenatal and early postnatal life strongly influences risk of coronary heart disease in older ages. In a series of later historical cohort studies for the UK, Barker obtained and analyzed health data from hospital records based on a large number of cases (1994, 1995). Various markers of fetal and infant growth were found to be adversely associated with coronary heart disease, stroke, diabetes, and respiratory disease and their associated risk factors. Most importantly, the associations between birth weights categories and coronary risk factors remained unchanged when other potential adult factors have been considered. In these later studies, the researcher drew on findings of the 1970s' research on the nutrition and growth, and thereby emphasized the role of proper nourishment especially in-utero and during infancy. As a result, the idea of critical periods was formulated, which has been attributed to periods in early life when any damages cannot be modified to any extent by factors in later life. In addition, Barker (1990) suggested a new biological mechanism between early life exposure and adult diseases. The intrauterine environment – and nutrition in

particular – was argued to program the fetus to have particular metabolic characteristics which can cause disease in later life. Barker's studies in the current literature are related to a so-called fetal origin or fetal programming hypothesis which points to the in-utero as the most critical period and improper nutrition as a main cause of future diseases.

In parallel, in the 1990s, epidemiologists and historians revitalized the idea that infectious illnesses, which take place during early life, may be even more important. In case studies medical scientists related exposure to Chlamydia pneumonia or Helicobacter pylori in early life to cardiovascular disease in adult life (Thom et al, 1992, Patel et al, 1995). Infliction from stomach infection associated with poor housing conditions and overcrowding, has been considered as a marker of low socio-economic status in childhood (Murray et al, 1995). Epidemiologists suggested biological mechanisms rather than nutritional in creating the relationship between infectious diseases and adult morbidity, so called the inflammatory responses. It has been argued that infections generate inflammation markers, such as increase in fibrinogen and total leukocyte count (Murray et al, 1995) or C-reactive protein (Mendall et al, 1996), which persist during life course and cause ischemic disease and cancer in adult life. However, as in the case of case studies, prospective historical studies have failed systematically to demonstrate close correlations between infections in early childhood and adult diseases like did case studies (e.g. Danesh, 1998). Only a decade later, with cohort-specific mortality data for Sweden, Finch and Crimmins (2004) demonstrated that exposure to infections during infancy causes future diseases, such as vascular diseases and many other morbidities of ageing. Scholars have also argued that, among all childhood periods, the vulnerability to inflammogens is most pronounced during first year of life, and also likely to emerge in-utero (Finch & Crimmins, 2006). Moreover, they suggested that slowed infant growth and later deficiencies in the fetal origin hypothesis may be due to exposure to infections that causes inflammatory responses and impair nutrient absorption (Finch & Crimmins, 2004). In this explanation, the authors exploited findings obtained almost a century ago. While vindicating these ideas, current studies refer to the so-called inflammation hypothesis, or the nfancy inflammation hypothesis. All macro and regional studies are questionable in their results, since they enable to produce only correlations. Current studies based on individual data provides better possibility to justify the critical period hypotheses.

Contemporary studies based on longitudinal individual-level data allow to examine the life course hypotheses more soundly. Bengtsson and his colleagues appear to be the first to apply vast historical data on individuals in early origins studies (Bengtsson & Lindström, 2000). The methodological approaches implemented by these scholars differ considerably from previous historical and epidemiological studies which used aggregated data on mortality and were essentially correlational. Studies on the basis of individual level data enabled inclusion of different context factors, and also to test causal models. However, the validity of causal relationship strongly depends on the type and time span of data (Bengtsson & Mineau, 2009). Those prospective studies on early life factors which were able to provide testability of causal models with longitudinal data had large age-spans. This data also made it possible to incorporate environmental macro factors such as prevalence of infectious diseases or national incomes in the models, and thereby test different early origins hypotheses. Moreover, different pathways from exposures in early life and later in life can be examined with interaction terms. The essays of Bengtsson and Lindström (2000, 2003) appear then to be demonstrative of using this approach. By using longitudinal data for individuals for 1766-

1894 from the rural population in southern Sweden, the researchers analyzed the relationship between both measures of nutrition and exposure to infectious diseases experienced by cohorts, either in-utero or during infancy, and mortality rates for the survivors of these cohorts between the ages of 55 and 80. Strong evidence was found for the hypothesis that individuals exposed to infectious during first year of life experienced higher death rates, especially from infectious diseases. Similar results have been obtained in many other studies based on the approach of Bengtsson and Lindström. The most recent attempts include studies on historical populations of Quebec (Gagnon & Mazan, 2009) and Ardennes (Bengtsson & Alter, 2008).

Early origins ideas are also examined in retrospective studies, but here some methodological problems arise (Bengtsson & Mineau, 2009). Such data usually lack in details on the socio-economic and other factors related to early life of the individuals. Additionally, selection processes for cohorts are possible since such data do not contain information on migration and deaths before the observed events. For instance, with retrospective data from the recent US censuses, Case and Paxson (2009) examined the reductions in childhood mortality from typhoid, malaria, measles, influenza, and diarrhea, and found lowered cognitive ability in old ages for those who were exposed during infancy. However, this study solely produces correlations. In order to apply causal models, current demographical studies on the life course use methodologies which may account for possible confounding effects or rely on natural experiments. By applying an instrumental-variables identification strategy to the US historical data, Barreca (2010) found considerably lower levels of educational attainment for those who were exposed to malaria in-utero and during postnatal stage. In a recent paper, Bengtsson and Helgertz (2012), adopting a quasi-experiment of the exposure before age five to the 1918 Influenza pandemic in Sweden, estimate that children exposed during infancy are likely to transfer their poor early life socio-economic status to later life. The many other contemporary studies on the effects of exposure to infections in early life did not consider different birth cohorts and focused on one group of children. Bleakley (2007) examines the impact of hookworm eradication among school-aged children on both short and long term outcomes. He has found substantial increases in school enrolment, attendance and literacy after the decreases in hookworm infection, and substantial income gain for the follow-up of the affected cohorts. Compiled data from several censuses, Chay et al. (2009) indicate that reduced exposure to pneumonia and diarrhea in early childhood, measured with post neonatal mortality rates, largely improved school performance among Blacks in comparison to Whites. Taken together, contemporary individual-level studies indicate that infectious environment in early life is an important determinant of health and socio-economic outcomes in later life.

Recent studies also applied individual level data for testing fetal origin hypothesis. Unlike for the infectious studies, research on fetal nutrition has obtained mixed results, and has some methodological problems. There is an abundant contemporary literature on close associations between low birth weights and future diseases (see for reviews Doblhammer, 2004; Currie, 2009; Almond & Currie, 2011a). The findings suggested that poor nutrition in-utero is positively associated with such diseases as cancer, diseases of respiratory, nervous and mental systems, infectious diseases, and mortality. The studies on fetal programming predominantly rely on natural experiments. Almond and his team appear to have become strong supporters of the nutrition hypothesis, and tested it in different historical cases with the US data. Adhering to the nutritional explanation of the exposure to infectious diseases, the scholars apply a natural experiment to the 1918 Influenza Pandemic. They found that children of infected

mothers were much likely to be disabled and less educated in adulthood (Almond & Mazumder, 2005, Almond, 2006). These results have recently been questioned by Brown (2011) who showed that Almond's findings are caused by change in the socio-economic composition of the examined cohorts. A number of recent studies evaluated the impact of famines on the health and socio-economic outcomes of the survivors. Stein et al (2006) revised the study on the exposure to Dutch Hunger Winter with individual-level data, and found significant increase in adiposity later in life for those who were exposed to food reductions in-utero. Several studies used China's Great famine of 1959-1961 as a quasi-experiment in exposure to malnutrition in-utero and early childhood. Findings from these studies, different in the cohorts' compositions, appear to be inconclusive on the importance of fetal nutrition. Meng and Qian (2006) found that those who were at the age from one to three at the onset of the famine have much larger negative health and educational outcomes in adulthood, but no effects for in-utero cohort was found. Chen and Zhou (2007) showed significant height reductions for children exposed to nutritional loss up to four years old, with most pronounced effect for the 1959 birth cohort. Recently, Almond et al (2010) have used the case of China Famine to examine fetal origins with data collected from different censuses. They found that individuals who were exposed in-utero were likely to be illiterate, notworking and not-married in older ages, but no effects on health were indicated. Mixed results from studies on the China Famine for the in-utero cohort may potentially point to possible selection processes in the used data. Unlike other natural experiments on famines, exposure to nutritional loss in China lasted three years, and was likely to strongly affect family planning decisions. Additionally, due to the exposure of such duration it tends to be difficult to distinguish between effects for the solely in-utero cohort and the cohort exposed after birth.

Other researchers consider economic shocks for testing nutrition hypothesis in individual-level studies. With data on military recruits of 20 years old, Banerjee et al (2010) investigated the effect of the nineteenth-century blight to French vineyards from the phylloxera insect. Men affected during the year of birth were found to be shorter, but no morbidities caused with the nutrition crisis was shown. Cutler and Norton (2007) examined the long-term outcomes of economic upheaval during the depression Dust Bowl era in the US while focusing exclusively on survivors who were exposed in-utero. They did not find any significant effects on future health for this treatment cohort. By compiling individual-level self-reported data with macroeconomic variations in grain prices in the Britain in 1780-1850, Baten et al (2007) relate ages of war and economic downturn for individuals affected in-utero with their numeracy and occupational status later in life. They have indicated that the affected individuals were likely to be less numerate and to occupy the positions with limited intellectual requirements. Since height estimated on the basis of grain prices has been found to have strong and stable effect on numeracy, the researchers argue on the importance of both genetic and nutritional factors in early life. Van der Berg et al (2006) explore the relationships between adult survival rates and exposure to economic shocks in early childhood by using historical individual-level and macroeconomic data for individuals born in Netherlands in 1812-1912, and followed up to the year 2000. The empirical analysis demonstrates that those who were born at the years of economic downturn are likely to live a fewer years that those born in a boom. The same approach has been applied to the Danish Twin Registry covering births in 1873–1906 in the study of van der Berg et al (2009). The study indicates a significant negative causal effect of macroeconomic conditions early in life on individual mortality rates

at higher ages for the groups exposed during infancy, but not during earlier or later periods. In all, like studies on long-term famines, studies on economic shocks suffer from the methodological problems arising from long duration of exposure and subsequent self-selection biases, and provide mixed results for the importance of fetal nutrition.

Relatively a few recent studies have examined the impact of socio-economic status in childhood on adult health outcomes. In the historical literature, two traditions in measuring socio-economic status have been established, either with occupation, like in the UK studies, or with educational measures, like in the studies on Europe and America (Kuh et al, 2004b). If both measures are included in the models, current researchers normally consider occupation as an approximation of social class, while education is related to the stock of human capital. Due to the obvious difficulties of exploiting natural experiments in early life exposure to poor socio-economic conditions *per se*, the majority of studies on socio-economic status are correlational. Notwithstanding, they consistently support the view that poor socio-economic conditions in childhood may affect future health and wealth either directly or indirectly, through educational and socio-economic status at different stages of life. One way to estimate this effect is to gradually extend the model with different measures or recompose a sample. With this estimation strategy, studies of Smith and his colleagues provide perhaps the most convincing evidence that socio-economic conditions affect later life morbidity through accumulation of risks over the life course. In a series of early studies, on the basis of the US censuses, the researchers showed that adverse socio-economic conditions in childhood affect age-related diseases (Smith et al 1998) and mortality (Blane et al, 1996) independently of socio-economic status in adulthood. In a later study, using a randomized study sample which was followed 1937-1998, scholars found that exposure to health-damaging environments during adulthood related to socio-economic conditions may accumulate health disadvantage obtained from childhood (Holland et al, 2000). Recently, Smith et al. (2009) examine possible links between poor health and low socio-economic conditions in childhood and different outcomes of adult socio-economic status with the Income Survey data on siblings. Both indirect and direct effects of morbidity and social environment in childhood were established. Many others have come up with similar results (e.g. Harding, 1999; Power et al, 1999; Landenberg et al, 2003). By gradually extending the regression models with socio-economic and educational variables, Palloni et al. (2009) used the 1958 British Cohort to show persistent influence of socio-economic background on adult health and socio-economic status.

Another empirical strategy is to include interaction terms between environmental variables of socio-economic status and exposed birth cohorts. This allows for measuring both direct and indirect effects at the same time. As previously mentioned, Bengtsson first applied this technique in the historical studies, and later on used the same strategy for socio-economic factors. On the basis of the same data from four Swedish parishes, Bengtsson & Broström (2009) extended previous studies (Bengtsson & Lindström, 2000, 2003) by including measures of indirect effects of exposure to malnutrition and infectious diseases at birth, and supposed their influence through socio-economic status in childhood and adult life. They found that although socio-economic disadvantage persists through adult life, it has no effect on mortality at older ages. The aforementioned study of Bengtsson and Helgertz (2012) showed that poor child socio-economic status accentuates the biological damage from exposure to the Spanish flu in its effects on social class in adulthood for those who were exposed after birth and during early childhood. This points to the idea that adverse effects are

potentially modifiable by socio-economic class during early childhood at the time of exposure, but not later in life. To our knowledge, no other study has used this approach in order to investigate the effects of socio-economic conditions.

The piece of literature examined the long term impacts of pollutants on fetal and child health. Early research revealed that exposure to pollutants in childhood can be modified through the life course. With the UK 1946 birth cohort, who were exposed in childhood to high levels of smoke and sulphur dioxide pollution in urban areas, studies detected chest diseases among adolescents (Douglas & Waller, 1966), but relatively small symptoms for the same cohort in young adulthood (Colley et al, 1973). For the same data, the later study showed that long term effects for those exposed before the age of two are accumulated at middle ages through adult socio-economic status and smoking, and also strongly depends on parental and grandparental respiratory disease, and parental smoking (Mann et al, 1992). Recent studies indicate significant associations between low birth weight and exposure to vehicle pollution. By using data from birth certificates in New Jersey, Currie et al (2009) establish that the variations in carbon monoxide substantially reduce birth weight and gestation. Similar study on Pennsylvania shows the same links, and identifies that the effect is also strong if to follow mothers over time or consider mothers fixed effects model (Currie & Walker, 2009). These studies also demonstrated that influence of pollutants on child health may have numerous confounding factors, such as migration or socio-economic status. For this reason, the majority studies on early life exposure to toxins have focused on natural experiments.

The contemporary findings from quasi-experimental data support that damage from toxaemia may have long-lasting effects for exposure during early life. A bulk of studies considered a natural experiment in the implementation of the Clean Air Act of 1970s and the recession of the early 1980s for identification of effects from early life exposure. In two essays, on the basis of US county-level data, Chay and Greenstone (2003a, b) found that a decline in particulates led to fewer infant deaths and incidents of low birth weight. Reyes (2007) used the same natural experiment to estimate the long term effects of banning leaded gasoline on crime within the cohort exposed before the age of five. Assuming association between early life exposure to lead and aggressive and criminal behavior in adulthood, the researcher found significant and large effects for those exposed before the age of six, but the effects were not robust to state-specific time trends. To the same experiment, Sanders (2011) applied the data on individuals born in Texas. Findings suggested that high school test scores improved for the cohort exposed to lower levels of ambient particulates in-utero. In order to identify educational and socio-economic outcomes of exposure in early life, Nilsson (2009) also correlated reductions in ambient lead levels in 1970s with individual-level data for Sweden. He found that the reduced lead exposure before the age of two improves school performance, cognitive ability, and labor market outcomes among young adults, and the effect is larger for exposed children from low-income families. Importantly, the study showed a nonlinear relationship between local air lead levels in early childhood and adult outcomes at relatively low levels of exposure, thereby indicating the existence of a threshold in the long-term impact of lead. In another study, the researcher used a Swedish alcohol policy experiment conducted in the 1960s to estimate long term impact of prenatal exposure based on individual-level data (Nilsson, 2008). The findings showed that the cohort exposed in-utero during the experiment had fewer years of schooling, lower future earnings, and higher welfare dependence rates at adulthood compared to related cohorts.

The experiments on exposure to radiation in early life allowed epidemiologists and historians to test the causal effects. Since the beginning of testing and production of nuclear power in the 1940s, numerous accidents may potentially serve as quasi experiments. However, due to the lack of available data, only few of them have been used in the research. The Chernobyl accident of April 1986 appears to be the incident most exploited by medical scientists and demographers. Five years after the accident, epidemiologists began to follow children exposed to radioisotopes in the most contaminated areas of Belarus, Ukraine, and Russia, and found much higher risks of different diseases. A number of studies have showed that the risk of thyroid cancer and hypothyroidism in adulthood is much higher for children exposed before the age of five, with the largest effect for those exposed during infancy (e.g. Stsjazhko et al, 1995, Davis et a., 2004, Ostroumova et al, 2009). The literature has documented no significant increases in the development of thyroid cancer for those exposed in-utero at the time of the accident (Parshkov, 1999, Hatch et al, 2010). Other cancers, such as leukemia, have been found to be related to radiation pollution for individuals exposed in-utero and during early childhood (Ivanov et al, 1998, Davis et al, 2006, Noshchenko, 2010). A number of other illnesses, such as lung diseases, digestive and blood disorders, immune deficiencies, neurological and mental disorders, and susceptibility to cardiac and infectious diseases have been identified for the cohorts exposed during childhood in the numerous epidemiological case-control and cohort studies (e.g. Romanenko et al, 2009, Loganovsky et al, 2008, Svendsen et al, 2010). Similar results have been obtained for other countries that received much lower doses of Chernobyl radiation, such as Greece (Petridou et al, 1996), Israel (Kordysh et al, 1995), Romania (Davidescu et al, 2004), and other European countries (Parkin et al, 1996).

Historical studies, which used individual-level experimental data on radiation in order to identify the effects of exposure in early life, are scarce. Almond et al (2009) examines the effect of Chernobyl radiation on the Swedish cohort that was in-utero at the time of the accident. The findings showed that those affected by pollution were more likely not to qualify for high school and had lower grades in comparison to surrounding cohorts. The impact on cognitive abilities from irradiation in-utero was dose-dependent, and captured even at low doses. This effect was robust to restriction to siblings' comparisons. Almond also found that the cognitive damage had been particularly accentuated for children from low-income families. In another study, Yemeluanau and Amialchuk (2012) estimated the effect from radiation after the Chernobyl accident with Belarusian individual-level data. They found significant large effects on health, wages, and possibility to be unemployed at young adulthood for those who were exposed to radiation before the age of three and resided at most contaminated areas. The probability of self-reported bad health for the cohort residing in areas with low radiation doses was also much higher in comparison to the older cohort. Recently, Black et al (2013) used nuclear power testing in 1950s-60s in Norway to examine the effects of fetal inflammation. The findings showed that exposure to radiation in-utero led to lower high school attainment and completion, and wages in adulthood, but that child socio-economic status did not strengthen the impact. The evidence from contemporary historical studies seems clear. It points to long-lasting health damage from the exposure in early life to radiation even at low doses that are considered to be almost harmless for health by the general public and academia.

2.2 Theoretical model

In the current literature, a socio-economic status in early life is hypothesized to influence later health through several mechanisms, both directly and indirectly. A schematic simplified model of these relations, that is implicitly used in all life course studies, is presented in Figure 1. Primarily, it is suggested that a socio-economic origin provides constraints to socio-economic status at different stages of an individual's life through access to economic resources and medical treatment (path a), and specifically, limits educational opportunities (path b). Consequently, socio-economic status in younger adulthood and middle-ages affects the disease and mortality risk in older ages. Another important mechanism is acting through the influence of family's socio-economic conditions on the child health capital. The child's health, produced with the family resources, directly determines health in adulthood. The important periods of health production are limited to early life, so called the critical periods. These critical periods open a window to the influence from the exogenous hazards. Damaging environmental factors affect health in early life directly, but due to the influence of the socio-economic conditions in a family, are additively strengthened or mediated (path c). Therefore, the pathway from child socio-economic status is socio-biological. Since environmental hazards affect both cognitive and non-cognitive abilities of a child, they also influence his or her educational experiences. As a result, through a child's health capital and education, environmental shocks may indirectly influence socio-economic status in adulthood. The last potential pathway includes the effect through the health behavior of children. The health habits develop in early life under the direct influence of the parents' health behavior, and later on are affected by educational experiences (path d). A health behavior acquired during childhood and adolescence contributes to the disease risks, which gradually cumulate and thereby affect health in older ages. Even such a simplified model highlights a complex interrelationship between social and biological mechanisms in long-lasting effects on health, and provides a sound basis for epidemiological and historical research within a life course approach.

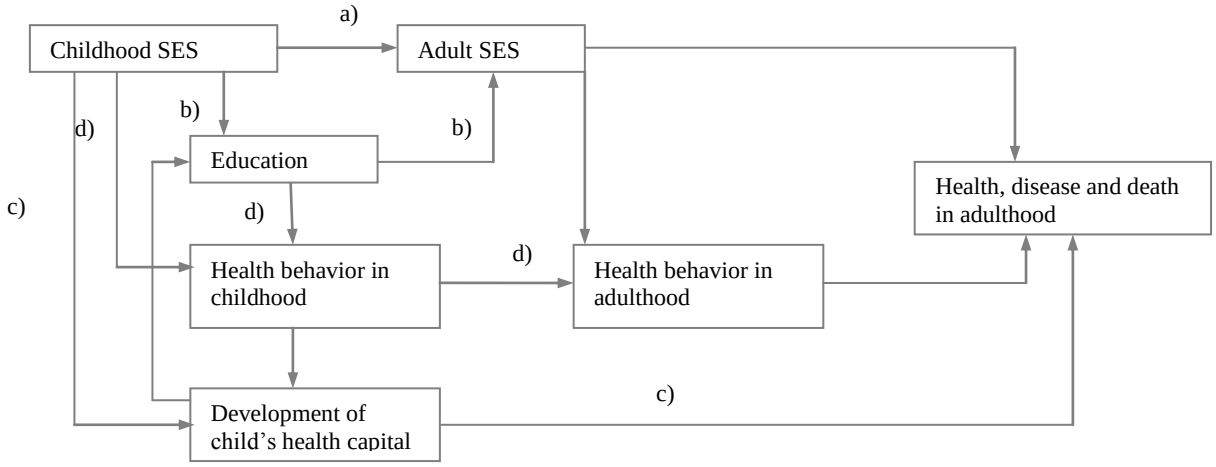


Figure 1 – Pathways between childhood socio-economic status and adult health

Source: adapted from Ben-Schlomo & Kuh (2002), Kuh et al. (2004c)

A life course approach distinguishes between different types of models. The researchers consider a life course approach for conceptualizing a critical period model or a model with accumulation of risk (Ben-Schlomo & Kuh, 2002). The critical period model assumes that an exposure during particular period has long-lasting impact on health, or does not manifest itself until adult ages, and this impact is partially or wholly irreversible. As suggested, exposure in later life can also influence health, but in comparison to a critical period this effect is negligible and modifiable. To date, two critical periods have been suggested by scholars, namely in-utero (fetal programming hypothesis) and infancy (infancy inflammation hypothesis). In suggesting that other periods in early childhood may also be potentially environmentally vulnerable, scholars normally distinguish between critical and sensitive periods. In a critical period, development of cells and organs occurs in such a rapid way that any exogenous insult can easily modify it in ether a positive or negative direction. In this way, several environmental factors may either damage health additively, or eliminate the effects of each other. Favorable socio-economic conditions may eliminate damage from exposure to inflammation or nutrition insults (Almond & Currie, 2011a). However, this mediation is possible only within a critical period. A sensitive period is a time period when an exposure has a stronger impact on health than it would have at other times, and is partially or wholly modifiable by later life experiences (Kuh et al, 2004d). Critical period may not taper simultaneously after birth or at the age of one, and the susceptibility of an individual to favorable or unfavorable environmental conditions will be gradually decreasing when it ends. Models of risk accumulation postulate that exogenous factors during a life course are cumulative in their impact on health, although there are some developmental periods when the damage is more apparent than at any other period (Kuh et al, 2004c). Exogenous factors may affect health separately or create clusters of risks, but they have influence throughout the whole life course. The health behavior is assumed to be an important factor or modifier of the environmental insults. Socio-economic status at different stages of life is then potentially interrelated with health behavior of an individual and partially determined by the biological effect from exogenous events. Therefore, when the importance of later life risk factors and modifiers can be recognized in the critical period models, they only postulate that an exposure in an early life critical period results in permanent and irreversible disease or damage in a life course. Only under this assumption, the role of development of health capital and its remediation with socio-economic status in childhood become crucial for later outcomes.

The development of early life health capital can be framed conceptually by applying the current health capital literature, and more specifically, by using the extended Grossman-type models. In the Grosmann (1972) models, health is a stock that varies over one life period in response to investments and depreciation. Parental education in the model is assumed as either a productivity shifter in the health production function or as a factor of child socio-economic status (Currie, 2009). In this original version of the model, past health investments tend to vanish with a time, and then early life investments become less important in a life course. By allowing two periods prior to health investments during childhood (Zweifel et al, 2009, Almond & Currie, 2011a), the model may frame the critical period approach (see Appendix B). In this extended version, for a given depreciation rate that is higher for the critical period during childhood, health investments becomes more influential for later health at the end of this period than investment during the rest of the childhood. Heckman (2007) further developed the extended model by incorporating a more flexible health capital production technology, and showed that recognition of a child's socio-economic origin is

conceptually important. In this latter version, the model can imply either initial differentiation between social groups in their responses to exogenous damage (model with fixed investments) or compensatory behavior of parents (model with responsive investments). In case no additional distribution of resources is taking place, individuals from upper classes, with higher initial levels of health investments in childhood, experience originally weaker effects from negative insults later in life than those from poorer classes, where baseline investments are low. This socio-economic gradient in health responses also comes from the fact that children from low-income families are subject to more and larger early life shocks. In addition to initial differences, wealthier parents are more likely to respond to environmental insults by increasing child health investments. These multiple mechanisms imply that if investments, differing across socio-economic classes, are not considered in the analysis, biological effects from exogenous insults might be understated. In general, the extended version of the Grossman health capital model is capable of shaping conceptually the early origins approach, but apparently cannot address the question of the relative importance of different critical periods during early life.

Current early origins scholars base their argumentation concerning main critical periods in early life on the implications of the epidemiological literature. To date, physiologists have established two biological mechanisms with accentuated long term influence during different critical periods. Fetal programming researchers indicate that nutrition during in-uterus generates permanent changes that persist throughout life. It has been argued that when a fetus undergoes programming the anatomic and immune structures are remodeled in order to preserve development and survival, when faced with the negative effects of malnutrition (Mile et al, 2005, Gluckman et al, 2008, Calkins & Devascar, 2011). When a newborn faces a mismatch between in-utero and external conditions, it produces the high disease risks which lead to future diseases. In this interpretation, supported by the empirical literature, proper nourishment of the children during prenatal stage is a necessary prerequisite for the absence of diseases in adulthood, such as coronary artery disease, diabetes, cancer, osteoporosis, and various psychiatric diseases (e.g. Painter et al, 2008, Lahti et al, 2009). In contrast, infancy-inflammation scholars emphasize the pathway from infections to adult health, and argue that other factors, such as nutrition and living conditions, are consequent to infections (see Appendix B). As regards biological mechanisms, it is assumed that high inflammation levels during infancy promote the process of atherogenesis or permanent dysfunction of immune system that determines later life health (Finch & Crimmins, 2004). Infections generate inflammatory markers, such as elevations of blood C-reactive protein, as well as interleukin-6, tumor necrosis factor-a, and fibrinogen, which cause future diseases. For instance, atheromas generated after insults contain macrophages and other cells that secrete inflammatory proteins which can be risk factors and even pathogens for later diseases. While the core of the inflammation literature considers infants to be most vulnerable to infections (Liuba, 2003), a lifelong process of atherogenesis is also demonstrated to begin in-utero (Palinski & Napoli, 2002). The affected mothers influence gene development in fetuses, and inflammatory responses generated in the mothers' bodies may enhance susceptibility of children to postnatal arterial structure dysfunction. Recent research has supported the infancy inflammation hypothesis with evidence for chronic diseases, heart attack, stroke, cancer, and some psychiatric disorders (e.g. Dalman et al, 2008, Willerson & Ridker, 2004). As proposed in the inflammation literature (see Appendix C), not only infections but also noninfection-related inflammogens can cause inflammatory responses.

Researchers suggest that health damage from noninfectious substances is of inflammation origin, but there are some important differences in their long-life health damage. While in the past research and policy concentrated on the effects of smoke and sulphur dioxide, the more recent focus has been on the health hazards of vehicle emissions, such as lead or ozone, as well as mercury, arsenic, and radionuclides, among others (Kuh et al, 2004a). All these toxins can lead to molecular, cellular, and even tissue damage through inflammation processes, which cause different diseases in long term, common to those of infectious origin (Finch & Crimmins, 2006). Fetuses and especially infants, with bodies' cells that are actively dividing, are suggested to be most vulnerable to these influences (Simmons et al, 2005, Ho et al, 2006). Besides generating inflammation processes, the intensity of the insult caused by pollutants is dose-dependent (Kimeldorf & Hunt, 1965). There are particular dose thresholds beyond which toxins not only generate the inflammation responses, which influence is manifested in adulthood, but immediately cause severe anatomic and immune damage. For instance, radionuclides at very high doses are capable of breaking chemical bonds in genetic code strands which cause mutations and cell death in both the short and long run (Hall & Giaccia, 2011). In addition, unlike infections, pollution inflammogens may even at low doses be stored in the body due to their ability to bind to the bone structure or directly to cells. The human bodies do not recognize harmful elements and therefore accumulate them as part of normal metabolism. For instance, bodies absorb radioactive iodine as biological iodine, and cesium isotopes as potassium (Lukyanova, 2003). Although biological activity differs considerably among toxins, the notion that mobilization of skeletal lead or tissue and organ radiation stores, either maternal or infantile, can persist many years after external exposure and cause damages, is well acknowledged in the literature (e.g. Gulson et al, 2003). Prevalence of radionuclides in the placenta has also been widely documented (Lukyanova, 2003, Zadorozhnaya et al, 1993). Radionuclides cause disturbances in blood circulation and dystrophic and involutive changes in placentas. Such damage is dose-dependent (Lukyanova et al, 2005). These results show that the impact of pollutants on health appears to be systemic, and then becomes apparent from early life throughout all stages of an individual's life.

Current studies have established the links between particular radionuclides and future diseases. The bulk of the findings has been obtained by epidemiologists who have studied the health effects of irradiation from the Chernobyl accident. Abundant evidence has been gathered for the health damages of iodine and cesium isotopes which contributed mostly to radiation doses. Diseases of thyroid glands have been attributed to iodine irradiation. Thyroid concentrates up to 70 per cent of iodine isotopes in children, with those in-utero and infancy at the highest risk (Dedov et al, 1993). In the long term, thyroid dysfunction and related dysfunction of endocrine and immune regulation have been associated with abnormalities in physical and sexual development and in the reproductive function (Tronko et al, 1995). Another widely recognized impact of ionizing is the development of neurological and mental disorders. The researchers identified links between prenatal and postnatal iodine irradiation and encephaloparhy (Loganovskaya, 2004), mental retardation, and psychiatric abnormalities (Gourmelon et al, 2005). The irradiation with iodine isotopes is the most damaging for mental health of fetuses, even at low doses. These disorders may become apparent in childhood, or be associated with cognitive decline in adulthood (Hall et al, 2004). Cesium radionuclides cause damage to digestive and circulatory systems. Atrophy of stomach tissues and related diseases, such as chronic gastritis, liver and gall-bladder disease, and billinary tract and pancreatic disease, are induced with cesium irradiation (Shestopalov et al, 2004). Cesium causes non-

malignant illnesses of the blood-forming organs and circulatory system, such as vascular dystonia or hypotonia (Savanevsky & Gamshey, 2003), which are a recognized prerequisite for cardiovascular disease in adulthood. Infants have been identified as the most vulnerable to such organ damage, but due to the ability of cesium isotopes to penetrate the placenta, fetuses are also at a high disease risk (Lukyanova, 2003). Studies show that qualitative changes of the skeletal system structure at an early age, such as development of osteopenia and osteoporosis, may be also associated with cesium irradiation (Lukyanova et al, 2005). In relation to all radioisotopes, researchers report a statistically significant effect of small doses of radiation on the development of malignant neoplasms in tissues or organs, with a minimum latency period of several years (Komissarenko et al, 2002). The studies also document an increase in susceptibility to infections and toxicoses as a result of a systemic damage from irradiation (Busuet et al, 2002). In all, medical studies have shown that any radioisotopes may cause severe long-lasting health damage to different body systems during the development and growth in early life.

The discussion of the life course has identified two models, - accumulation of risks and critical periods. They both emphasize the importance of factors at distinct periods of life for future health and well-being. However, the crucial difference in the models is the timing of the periods when health is most vulnerable, and the extent to which it is possible for later experience to modify the environmental damage. A critical period model assumes that early events are wholly or partially irreversible, and later health experience may act as a simple additive to early health damage. In contrast, the model of risk accumulation highlights the importance of factors throughout the whole life, and views health behavior as a potential modifier of earlier experiences. To date, researchers have identified several environmental factors which are potentially damageable for future health, such as nutrition, infectious diseases, and poor living conditions. They all strengthen each other in the negative influence on health, or may eliminate each other, such as the combination of favorable socio-economic conditions and severe infectious environment.

The problem that remains un-solved in the literature is which of environmental factors that is crucial and thereby potentially more influential than the others. Historical studies based on macro data and epidemiological cohort and cases studies have provided abundant support for the influence of each of these factors. Epidemiologists have proposed different biological mechanisms linking early events and future diseases. The advocates of the importance of the infectious environment assume that infectious diseases in early life, and especially during infancy, generate chronic inflammatory markers which lead to future diseases. Non-infectious inflammogens, or pollutants, have been suggested to influence future morbidities in the same way. Another mechanism associates intrauterine nutrition experience, or fetal programming, with health in later life. In both interpretations, poor socio-economic conditions are secondary, and accentuate the impact of infectious diseases or malnutrition through the availability of resources and proper treatment. Armed with these concepts, contemporary historical and epidemiological studies, by using longitudinal individual level data, began to establish causal relationships between fetal and infancy events and later diseases. The bulk of this literature has pointed to the importance of inflammatory conditions during infancy, but there is also some evidence of the influences of fetal nutrition. The findings consistently support the assertion that environmental susceptibility is likely to peak or emerge during one

period and then gradually decrease by the end of the early childhood. Whether this critical period is in-utero or infancy, the question that is addressed in this study.

This current study adheres to a critical period model. In line with the historical period literature, we incorporate child socio-economic conditions into the analysis. By using a natural experiment in exposure to non-infectious inflammogens in the form of radionuclides we are able to observe exogenous shock in early development and life and match it to health status in adulthood. In this framework, socio-economic conditions at the time of the early event may potentially eliminate the negative consequences, but only during a particular critical period. Noteworthy, we are not able to answer how this compensatory effect occurs. Whether parents intentionally are redistributing more resources to an affected child, or whether children from poor families are initially more vulnerable to early shocks, is question that will be left open. What this study is able to establish is the timing of this critical period. In order to avoid any polarizations between the critical period hypotheses, we incorporate different birth groups in the model, including fetal, infantile stages, and the rest of the early childhood.

2.3 Hypotheses

Based on the theoretical model, the empirically testable hypotheses can be formulated as follows:

i) Individuals who were not exposed to radiation in early life have better health in adulthood;
ii) Individuals originating from more affluent families have better health in adulthood;
iii) Individuals who were not exposed to radiation in early life and originated from more affluent families have better health in adulthood.

Within the framework defined for this study, the socio-biological mechanism of long term health outcomes is major importance. Thus, we gradually test the purely biological hypothesis (i), the socio-economic hypothesis (ii), and then move towards testing the socio-biological, or interaction, hypothesis (iii). Following the critical period literature, our focus among exposed age groups will be on particular stages of individual's early life when environmental vulnerability is the largest. We adopt the infancy inflammation hypothesis and assume that individuals are most environmentally vulnerable to the inflammation-related events during infancy, and this vulnerability can be eliminated by favorable socio-economic conditions in childhood. Following the fetal nutrition and inflammation literature, we also suppose that this vulnerability may emerge in-utero and persist, albeit gradually decreasing, after the age of one. The latter group is limited up to the end of early childhood, at the age of five. Taken together, birth cohorts which adult health outcomes are particularly examined in this study include the following: exposed in-utero, exposed during infancy, and exposed between the ages one and five. The control group is established as those who were born after nine months of the exposure period, and therefore conceived after the end of the natural experiment in early-life irradiation.

3. Data and methods

3.1 Background

Chernobyl accident provides a natural experiment in radiation exposure of the population in Ukraine. The Chernobyl accident that occurred in 1986 allows us to match varying exposure to radiation throughout Ukraine among different birth cohorts to their health outcomes observed at least 18 years later. In establishing the timing and duration of exposure to Chernobyl radiation, main assertions of the natural experiment should be fulfilled, though. According to Almond (2006), they are the following: (i) the event should not be planned or anticipated; (ii) when it is occurring, there should be no means to avoid it and no treatment; (iii) there should be no other factor that takes place at the same time that could influence the outcome of the event. In what follows, the exposure periods are defined based on these requirements.

The exposure took place when a nuclear explosion occurred and caused contamination. On April 26, 1986, the nuclear power plant in Chernobyl in Ukraine exploded leading to the biggest civil nuclear accident. After the initial explosion, during the first days, a nuclear cloud contaminated substantial areas of Belarus, Ukraine, Russia, and in a week, western and northern parts of Europe, at the distance up to 3000 km from the place of accident (OECD, 2002). In the active phase, within the countries, radiation contamination was formed in an unpredictable pattern by wind direction, rainfalls, and natural landscapes. Chernobyl emissions were characterized by a wide spectrum of radionuclides, with different forms and composition (see Appendix D). Due to the specificity of the nuclear reactor destruction and weather conditions, contamination of the majority of the former Soviet Union and European territories has been caused mainly by highly mobile radionuclides, with different biological half times. Among significant contributors with high radiological value were the following: (i) 131I, iodine isotopes, which are short-lived elements with half life of 9 days that disappeared completely in a three-months-period; (ii) 134Cs and 137Cs, cesium isotopes with a half-life of 2.1 and 30 years respectively, which were available in the topsoil during approximately eight months. Other radiologically dangerous elements either fell in areas close to reactor (e.g. 90Sr), or were not available in large deposition at distant areas (e.g. 241Am). It has been estimated that during the first days Ukraine received 18.8 per cent of the total fall-out in overall Europe which covered the whole territory of the country (Gerasimova et al, 2006). This became the source of the exposure of the entire population in Ukraine.

Due to the acknowledged differences in the impact of most influential Chernobyl isotopes on child health, in the study we distinguish two exposure periods regarding of the relative contribution of these radionuclides to the exposure. Several aspects should be considered here. Firstly, population got exposed dependent on the chemical characteristics of radionuclides and specificity of their absorption by bodies. It has been established that, following the nuclear fall-out, individuals intook the radioisotopes in both external way, from the air or the ground, and internal way, through inhalation, absorption by skin, or by ingestion of contaminated food, primarily of milk, meat and vegetables (WHO, 2006). According to estimations, for the exposed children, the main source of irradiation was the consumption of milk (Baloga et al, 2006). In addition to different ways of irradiation, exposure period has been dependent on the psychical lives of the isotopes. Thus, while gradually decreasing within

a three months term, physical activity of the iodine during its biological half life was the largest among all radioisotopes and it contributed to the largest extent to the irradiation of children (see Appendix E). The data on dosimetries collected in 1986 indicates a share of 70 per cent for the dose received due to the exposure to iodine in Ukraine (Baloga et al, 2011). Cesium, the impact of which was relatively smaller during the first several months after the accident for most polluted areas of Ukraine, has become the most relevant radionuclide later on and in some regions will continue to irradiate for decades to come (UNSCEAR, 2008a). Importantly, the impact of cesium isotopes has been decreasing since the original explosion in 1986. However, the pattern of irradiation from cesium has changed with a time as regards the entire territory of Ukraine. It has been indicated that long-lived radionuclides were surface-kind throughout the whole country only up to 1987, when they became bioavailable in soil (Baloga et al, 2006, 2011). Since 1987, the move of radionuclides has been highly determined by the transfer characteristics of soil prevailed in a particular region: an intense deep ground contamination has occurred only in the regions with extremely transferrable soils, and by coincidence, almost all of them were the originally most contaminated territories of Ukraine. As a result, individuals residing at those regions have continued to be strongly irradiated from 1987, whereas others received the largest portion of radiation before (UNSCEAR, 2008a). The recent dosimetry of ground and food contamination, including milk products, and numerous radiation dosage estimates taken annually in Ukraine proves that irradiation from cesium is indeed taking place primarily at the most contaminated regions (Baloga et al, 2006).

In defining the duration of exposure, any possibility of self-selection should be taken into account. This aspect concerns the possibility of the population to avoid radiation due to the availability of safety information. It has been documented that in the first five months after the accident, the governmental activities were concentrated on the issues of the emergency power unit and those individuals who had to be evacuated within a 30 km zone (Gerasimova et al, 2006). A May 1 parade, which was held as usual in 1986 in the capital of Ukraine only 90 km away from the burning power plant, illustrates an absence of any public information on nuclear accident (Medvedev, 1990). By the middle of May 1986, officers of the Ministry of Health and mass media were forbidden to inform the people about the evacuation activities, protection methods, and scales of the accident (see Appendix F). Prior to August 1986, the national documents did not mention radioactive contamination of the territories (Baloga et al, 2011). Only in September 1986, the first national decree on the radiation accident was introduced, with some information on the limits for the consumption of local agricultural production, albeit for the most contaminated regions. From this time, local authorities were able to make some local decisions, and the general public was informed. However, no information on the scale of irradiation was available at that time. Maps of Chernobyl radiation contamination and radiation doses were classified until 1990. It is still likely that the population got informed about the accident from unofficial sources much earlier than in 1987. However, considering the lack of knowledge in the society about appropriate behavior after accidents of such severity and scale, we can suppose that few prevention measures were undertaken by the population in 1986. Also, any endogenous migration in a response to accident is unlikely to have taken place in Ukraine. Migration in the USSR was extremely difficult because of the institution of mandatory registration, which required individuals to reside only at a registered address. Emigration processes were therefore nearly impossible. Given these considerations, exposure period should be limited up

to the end of 1986, and any later periods may generate self-selection of individuals into or out of the treatment group through family planning.

Some important events that took place in Ukraine should be discussed in regard to the duration of radiation exposure. In Ukraine, like in most other former Soviet Republics, the period since the mid-1980s and the early post-Soviet transition was characterized by dramatic political, societal and economic changes, so called Perestroika [reconstruction] period. However, only from the middle of 1987, actual reforms on liberalization and democratization took place nationally and locally. From 1990, these led to an economic downturn, and increased insecurities for the majority of the country's population (e.g. Velychenko, 2007). The available data proves this fact: the real Gross Domestic Product per capita was relatively stable till 1990, and afterwards dropped drastically (see Appendix G, a). Another potential confounding event is the change in socio-economic structure as a result of the launched reforms. As a part of the democratization program, the reforms captured primarily educational system. The major reconstruction was undertaken in the system of higher education, which in the 70 years preceding Perestroika was a privilege of the upper class. In 1987-1990, the access to the higher education was opened to lower classes by reductions of the entry requirements, and by widening of the student housing stock (Andriewsky, 2003). However, the results of the educational reforms became apparent much later, only in 1995 (see Appendix G, b). Therefore, neither macroeconomic nor social environment differs for years preceding Chernobyl accident up to 1990.

Considering all aspects, we can now define the exposure periods for the current study. In order to create a random experimental group, the period of exposure to iodine in Ukraine can be stated from the beginning of accident up to three months term, while cesium irradiation should be captured till the end of 1986. As previously discussed, any strict distinction between periods as regards exposure to the particular radioisotope for all Ukrainian individuals is too strong, given the compounded effect of all isotopes on health. The only unambiguity is that iodine isotopes irradiated population during the first exposure period, and at that time contributed chiefly to the radiation dose. Exposure doses during longer exposure period have been formed primarily with cesium radioisotopes. Because of the potential importance of the economic and societal environment in Ukraine since 1990 for the adult morbidity of the exposed, the reference group should then be covered with those born not later than in 1989. Given the exposure period and a control group for the Chernobyl case in Ukraine, the health outcomes for all individuals since 2007 onwards can be considered as adulthood health outcomes, since all individuals born up to 1989 have reached maturity by 2007. Based on exposure periods, treatment and control birth cohorts can be further selected.

3.2 Data and operationalization

In the current study, the nationally representative Ukrainian Longitudinal Monitoring Survey is used as a data source. This is the only available data for the most affected countries, Ukraine, Belarus and Russia, that allow to identify adult health outcomes and child socio-economic status, as well as individual location at the time of accident. Among all the survey waves, the 2007 wave contains the data fulfilling all the age requirements for the individuals which come from the research question and context. Additional regional-level data on the radiation dosage has been compiled from the national reports of Ukraine on the consequences of the Chernobyl disaster published in 2006 and 2011. The reports are the official national documents on the environmental and health outcomes of the accident conducted under the auspices of the United Nations' organizations.

The sample selected for the study consists of approximately 1,100 individuals born between 1981 and 1989, chosen to represent the groups with varying radiation exposure. The descriptive statistics can be found in the Appendix H, and the list of recoded variables in the Appendix I. All the study groups are shown in Table 1. For two approaches as regards different exposure periods, individuals will be allocated differently among birth groups, albeit not for all groups. According to our research question, one treatment birth group is under the particular focus in this study – those children being during infancy at the exposure period. This study group is constituted with those who were born before, but not more than a year before April 26, 1986 (exposed during infancy). Other groups are also under attention, such as individuals who were exposed to radiation while in-utero and after first year of life. The in-utero group is selected within nine months after the radiation exposure period (exposed in-utero). To compose birth groups properly, those who were born during the radiation exposure period, which were subject to exposure both during infancy and in-utero, are also distinguished (exposed during infancy and in-utero). The remaining exposed birth group represents individuals who were between the ages of one and five at the exposure period and thus completes the groups of exposed during early life (exposed between the ages of one and five). The reference group consists of individuals who were born after nine months of the radiation exposure period and up to 1989, and therefore conceived after the end of the radiation exposure period. In recomposing the groups for two approaches, approximately 60 individuals treated as being not exposed within the first approach, will be allocated to the group of exposed individuals for the models with a longer exposure period. Regrouping of individuals will allow us to check the sensitivity of obtained results within both exposure periods. In fact, the control group that is formally defined as not exposed to iodine isotopes represents individuals irradiated to a lesser extent than others within the sample period, but still irradiated. Unlike, the control group of those conceived after the end of exposure to cesium are actually not irradiated, at least if they lived in low-contaminated regions of Ukraine. Therefore, if any, the larger effects are likely to be captured within the second approach.

Table 1 – Birth cohorts selected for the models

	First approach *Iodine irradiation* (exposure period 26 April 1986 – 25 July 1986)		Second approach *Cesium irradiation* (exposure period 26 April 1986 – 31 December 1986)	
	Birth period conceived	N of individuals	Birth period conceived	N of individuals
Radiation exposure between the ages 1-5	26 April 1981 – 25 April 1985	478	26 April 1981 – 25 April 1985	478
Radiation exposure during infancy	26 April 1985 – 25 April 1986	131	26 April 1985 – 25 April 1986	131
Radiation exposure during infancy and in-utero	26 April 1986 – 25 July 1986	39	26 April 1986 – 31 December 1986	94
Radiation exposure in-utero	26 July 1986 – 25 April 1987	109	01 January 1987 – 30 September 1987	118
Conceived after the end of radiation exposure period (not exposed)	26 April 1987 – 31 December 1989	352	01 October 1987 – 31 December 1989	288
Total number	26 April 1981 – 31 December 1989	1109	26 April 1981 – 31 December 1989	1109

The outcome variable used in the current study is constructed from self-reported health. Although the data contains information on the prevalence of chronic and some common diseases, and temporal disability of individuals due to health problems, a self-reported health variable qualifies the research question more properly. By its construction, self-perceived health variable measures in one single index the adult health experience, and it can capture the systemic influence of the radiation on the human bodies. In this study, self-estimated health from five-point scale is recoded into a binary variable (see Appendix I). Since a key distinction is drawn here between bad and good health, original observations for *very good* and *good* responses have been grouped as good health, whereas *average*, *bad*, and *very bad* as bad health. In addition to being intuitive, a decision on the recoding of responses on average health outcomes into negative ones is made on the basis of comparison of self-reported health variables with other health variables available in the dataset (see Appendix J). The data shows that individuals with self-reported fair health status respond on the prevalence and incidence of other health problems and diseases more frequently than on average across the sample. The pairwise associations of a constructed binary variable of self-reported health with other variables indicate the robustness of our decision (see Appendix K). Thus, from Appendix I, the evidence emerges that self-perceived health observations correlate stronger and more consistently with all other health observations than they do between each other. Noteworthy, when responses on average self-reported health are assumed as bad outcomes, any results obtained for the bad versus good health will be of lower bounds.

Among other hypotheses, the current study investigates the differences in adult health outcomes as regards individual socio-economic status in childhood which is measured by parental education. In a dataset, several potential variables for socio-economic status are available – recall of parental occupational status at the individual's age of 14 and parental education at observation. In line with previous discussion, parental occupational position cannot be used in the study due to potential confounding effects, namely the dramatic changes in the labour market and societal reconstruction occurred after the collapse of the Soviet Union in 1991. The variable on parents' educational status appears to be more appropriate for our analytical purposes. The choice of the parental education as an approximation for child

socio-economic position is rather common in the literature (e.g. Smith, 2009, Black et al, 2013). The distinction between those who had at least higher education and those with completed lower levels of education reflects particularly well the socio-economic structure of the Ukrainian society in 1980s. Originally, the variable on education of parents indicates the highest schooling attained at different levels, as well as information on whether the parent was not living with a respondent (see Appendix L). In the study, responses on incomplete higher education, higher education, and doctoral degree are recoded as high education, and other responses are considered as low education. We recoded incomplete higher education as a positive value due to several reasons. Primarily, incomplete higher education is regarded as high educational status due to specificity of Soviet educational system. Thus, in the USSR, individuals who passed interim attestation exams (after two years for internal studies, and three years for external studies) were given the diploma on incomplete higher education (Andriewsky, 2003). These diplomas provided access to stronger occupational positions nearly equally to diplomas on complete higher institute degree. We also observe parental educational status only in 2007, but not at birth or childhood of individuals. According to available data (see Appendix J), the average age of giving birth in Ukraine during the study period was 25 years, and for the first birth 23 years. Therefore, at the age of 23, at child's birth or childhood, at least non-full higher education can be assumed as being completed or close to completed.

In this study, early life socio-economic conditions are measured with either mother's or father's educational status. These sets of models are tested separately. In the models, we assume that families where mother (father) has a higher institute degree are wealthier than those where mother (father) has lower level of education. Subsequently, a control group in a study represents individuals with wealthier socio-economic status in childhood, who had parent with at least higher education, and a treatment group with poorer early conditions, whose parents did not have higher education. In terms of comparability between the two sets of models, given the strong gender gradient in the Soviet society, we suppose that distinction on the basis of father's education is likely to represent deepest social differentiation in comparison to that where maternal educational status is used. It has been widely acknowledged that the industrial composition of employment in USSR and Ukraine was gender-specific, with males dominating the construction and manufacturing industries and females working in healthcare, education, and service industries (Kalabekov, 2010). In the USSR, where labour earnings were the only source of income, among all occupational groups, occupation in production industries provided the highest wages and access to other resources. Also, for the model with father's education, we are able to distinguish a group with unknown father's education, which is mostly constituted by individuals who lived with single mothers (n=98). Hence, this group can be treated as of poor socio-economic origin. Given these, we are likely to capture the largest impact with father's educational status, and then the model with mother's education will yield estimates with lower bounds. Besides being of interest in the models as itself, a socio-economic position in childhood acquires particular importance regarding of the birth groups irradiated in their early life.

Within the major focus of the paper, we test the possibility of the individual's birth family to mediate the negative outcomes of the radiation exposure at critical periods, by introducing interaction terms in the models. One argument for these expectations comes from the fact that upper class families are capable of providing an affected child with additional resources. Also,

individuals from poorer families are likely to be initially more vulnerable to exogenous insults, or subjected to larger insults than those from wealthier families. All this can be captured with the estimates for the interaction terms between differently exposed birth groups and socio-economic groups. Table 2 shows the information on the differences in child socio-economic status and adult health outcomes for two exposure periods. A notable observation here is that individuals exposed during both in-utero and infancy came mostly from more affluent families, but otherwise have had relatively more cases with bad health, across all cohort groups. Unlike the data indicates, we would expect the directly opposite. This peculiarity of the data holds for both exposure periods, in the models with maternal education. For a longer exposure period, selection processes can be indicated only in the models with father's educational status as a socio-economic variable. The other birth cohorts indicate no considerable differences in terms of early socio-economic conditions and health outcomes.

In addition to the main hypotheses, the health outcomes for individuals with different intensity of the exposure will be tested by using radiation dosage. As previously mentioned, the noninfectious inflammatory responses are dose-dependent, and those individuals who were inflicted with larger radiation dose are likely to have worse health, in addition to common radiation-related health effects. Given this stronger insult for the more affected individuals, we also expect to observe health differences for them stemming from socio-economic conditions in early life. To obtain the dose-dependent effects, official regional radiation data is matched in the study to individual's place of residence (birth) in 1986. Average effective total exposure doses, which reflect the amount of energy absorbed by human body, are chosen for the study (see Appendix M). This variable captures not only a fallout contamination that was the source of the external irradiation for individuals, but also internal irradiation. In addition, it is based on the information for the different pollutants, such as 131I and 137Cs. As previously discussed, almost all territories most affected in 1986 with iodine isotopes experienced the most severe contamination by cesium after 1987, and therefore it is highly likely to capture the large impact with this measure. Additionally, the measure fits both exposure periods. Individuals who were born and resided in five out of twenty six regions are considered to be given the high radiation dose, whereas all others got exposed at lower levels. Those individuals who received a low radiation dose represent a baseline group. As expected, the geographical gradient in health outcomes can be observed collectively, for all birth cohorts. In addition, the collective influence of the early life conditions on health outcomes will be examined with interaction terms between radiation dosage and socio-economic status in childhood.

Table 2 – Distribution of cohort groups in relation to their adult health status and child socio-economic conditions, %

	First approach *Iodine irradiation* (exposure period 26 April 1986 – 25 July 1986)								Second approach *Cesium irradiation* (exposure period 26 April 1986 – 31 December 1986)							
	N	Bad health	Good health	Low mother's education	High mother's education	Low father's education	High father's education	Unknown father's education	N	Bad health	Good health	Low mother's education	High mother's education	Low father's education	High father's education	Unknown father's education
Radiation exposure between the ages 1-5	478	36.61	63.39	83.89	16.11	75.31	14.02	10.67	478	36.61	63.39	83.89	16.11	75.31	14.02	10.67
Radiation exposure during infancy	131	40.46	59.54	87.79	12.21	75.57	12.22	12.21	131	40.46	59.54	87.79	12.21	75.57	12.22	12.21
Radiation exposure during infancy and in-utero	39	53.85	46.15	64.10	35.90	69.23	17.95	12.82	94	44.68	55.32	75.53	24.47	79.79	13.83	6.38
Radiation exposure in-utero	109	34.86	65.14	83.49	16.51	82.57	12.84	4.59	118	32.2	67.8	78.81	21.19	70.34	17.80	11.86
Not exposed	352	30.97	69.03	78.13	21.88	68.18	17.61	14.20	288	30.56	69.44	78.82	21.18	69.10	17.01	13.89
Total number	1109	396	713	907	202	816	166	127	1109	396	713	907	202	816	166	127

To control for possible differences in group composition as well as possible confounding effects, several sets of variables are introduced in the analysis. Standard socio-demographic controls include age and gender. Since in the literature age has been found to exhibit a U-shaped impact on health (e.g. Blanchflower & Oswald, 2008), a quadratic term is added to improve the fit of the models. The study uses also some specific control variables, such as type of place of birth and migration status of individuals at the time of the accident. It is argued that individuals who resided at rural areas were likely to be exposed to a greater extent, through both external and internal ways (Baloga et al, 2011). However, since the major source of irradiation was through the food intake, and both rural and urban population consumed locally produced food, the expected differences can be negligible. Another specific factor accounts for the possible migration of individuals after the accident in 1986. In the models this control variable is likely to affect the estimates for the radiation intensity groups, but to much lesser extent, estimates for the birth groups. The argument here is that, as suggested and officially stated, regardless the changes in place of residence at the year of disaster, all the individuals in Ukraine were subject to radiation exposure during the exposure period.

3.3 Method

In our analysis, the focus is given to the estimates for the selected birth cohorts. Since the health outcome is defined as the self-reported bad health at a time of the interview, each individual will contribute with one observation. In this study, binary logistic regression models are used because of the dichotomous nature of the dependent variable.

Within the methodological framework of this study, we analyze the relationship between radiation exposure in early life and probabilities of having bad health in an individual's later life within two approaches. The approaches differ in the defined length of the exposure period and the prevalence of a particular isotope that caused irradiation during this period. First approach measures the effect from the early life exposure to *iodine* radiation, and the second approach to *cesium* radiation. As previously discussed, all radioisotopes contributed to irradiation of individuals in early life at the same time, but the activity of some of them was prevailing during particular periods. Iodine isotopes irradiated population chiefly during first three months, when contribution of cesium was much smaller. When the longer period of exposure is considered, during eight months, the activity of cesium was prevailing relatively to iodine radionuclides. After 1986, cesium continued to irradiate individuals at some regions of Ukraine, which are defined in this study as highly contaminated areas. At the same time, the exposure in the low contaminated regions stopped almost entirely. Therefore, it will be essential to include other important covariates into the models, such as migration and place of birth amongst others. Within both approaches, we describe results from a basic model, containing no other controls but birth cohorts and age, and proceed to extend it by adding other important covariates.

The models will stepwise test the purely biological hypothesis, the socio-economic hypothesis, and the socio-biological hypothesis while beginning with basic specification and then incorporating important controls. The biological hypothesis is examined with birth

groups of exposed individuals. The socio-economic hypothesis is tested by adding social groups. It has been previously stated that we proxy socio-economic status with either mother's or father's education, and this will be done in separate sets of models. In a given context, models based on father's higher education represent deeper social stratification than those with mother's higher education. To examine the socio-biological hypothesis, the final models will consist interaction terms between birth cohorts and early-life socio-economic conditions. Given that health damage from irradiation is dose-dependent, we incorporate radiation dosage into the models. The baseline effects will be measured for those who received low radiation doses and therefore for the majority of the individuals in the sample. In the final models, we will also test the persistence of the early life effect for individuals who received a high radiation dose and originated from less favorable conditions in early life, but obtain the collective impact for birth cohorts. Our focus in the study, nevertheless, will be on the individual effects for birth cohorts attributed to their socio-economic conditions in early life at the time of exposure.

The results are first presented in odds ratios, which measure the proportional size of the effect for treatment groups in relation to a control group. To estimate the absolute size of the effect from exposure to radiation, we then predict probabilities of having bad health in adulthood for an average individual in the sample with regard to different birth and social groups.

4. Results and Discussion

4.1 First approach

First approach measures the effect from the early life exposure to radiation within three-month term when *iodine* isotopes were contributing chiefly to radiation dose among all radioisotopes released after the Chernobyl accident.

In testing the biological hypothesis, the models 1 and 2 provide some evidence that the exposure to radiation in early life may directly affect health in adulthood. The estimates, which obtained from both basic specification and specification with controls, for the majority of exposed birth cohorts are consistent with the a-priori expectations, although only one of them significantly differs from that estimated for the non-exposed individuals (see Table 3). The results provide striking evidence for those exposed both during infancy and in-utero: probability of having bad health in adulthood for them is 2.3 times of that for the non-exposed individuals, and the effect is statistically significant. However, this result should be considered with some caution, as previously data indicated that individuals in this birth cohort might be positively selected as regards socio-economic origin. Further, individuals who were subject to radiation exposure during infancy are also likely to have the large negative impact on adult health, although the effect is not statistically significant. The effect increases substantially up to two-fold size relatively to a reference group with low radiation dosage when other important covariates are added into the model. The logged odds for those exposed in-utero is almost equal to zero, and then implicitly indicates the lesser damage from iodine radiation for health during intrauterine stage than during infancy, relatively to the non-exposed group. Similarly, individuals who were between the ages of one and five at the time of the accident show no signs of weaker adult health estimated with its probability in the models 1 and 2. Notably, albeit not significant, the coefficients for the age confirm our expectations concerning its U-shaped impact on adult health. Among other important covariates included in the model 2, estimates for those who received high radiation doses in childhood are large and significant. Those who were residing at the most contaminated regions have two times higher probability of bad health in adulthood in comparison to individuals living in low polluted areas. This effect is collective and therefore indicates the persistent exposure to radiation in Northern Ukraine after the exposure period established in this study. In all, the estimates in the models 1 and 2 produce some evidence on the biological mechanism thereby iodine irradiation affected long-term health during critical periods. Thus, individuals during both fetal and infantile stages and solely during infancy are likely to be the most environmentally vulnerable in comparison to the non-exposed cohort.

Table 3 – *Iodine irradiation.* Odds Ratios for self-reported adult health with mother's or father's education as a measure of child socio-economic status

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
no radiation exposure (ref)	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000
radiation exposure between the ages 1-5	0.815	1.042	0.791	0.816	1.028	1.024	0.446	0.451	0.551	0.612
	(0.407,1.633)	(0.432,2.511)	(0.394,1.587)	(0.407,1.637)	(0.426,2.481)	(0.424,2.475)	(0.182,1.094)	(0.189,1.075)	(0.192,1.576)	(0.219,1.715)
radiation exposure during infancy	1.234	1.739	1.177	1.236	1.681	1.713	0.500	0.178**	0.780	0.262
	(0.691,2.206)	(0.808,3.742)	(0.657,2.107)	(0.691,2.211)	(0.781,3.621)	(0.794,3.693)	(0.148,1.695)	(0.0445,0.712)	(0.199,3.053)	(0.0576,1.189)
radiation exposure during infancy and in-utero	2.249**	2.277*	2.370**	2.248**	2.413**	2.269*	4.127**	1.758	4.680**	2.089
	(1.142,4.430)	(1.133,4.575)	(1.199,4.686)	(1.140,4.432)	(1.196,4.867)	(1.127,4.565)	(1.408,12.09)	(0.446,6.936)	(1.520,14.41)	(0.480,9.091)
radiation exposure in-utero	1.072	1.071	1.042	1.104	1.035	1.095	2.388	1.372	2.662*	1.440
	(0.660,1.742)	(0.653,1.757)	(0.641,1.695)	(0.678,1.796)	(0.629,1.701)	(0.666,1.800)	(0.944,6.042)	(0.499,3.767)	(1.026,6.906)	(0.507,4.086)
age	0.896	0.824	0.937	0.912	0.865	0.836	0.941	0.842	0.830	0.772
	(0.277,2.902)	(0.247,2.743)	(0.288,3.044)	(0.281,2.962)	(0.259,2.893)	(0.251,2.793)	(0.289,3.063)	(0.257,2.760)	(0.246,2.806)	(0.227,2.622)
agesq	1.005	1.007	1.004	1.004	1.006	1.007	1.003	1.006	1.007	1.009
	(0.979,1.031)	(0.981,1.034)	(0.978,1.030)	(0.979,1.030)	(0.980,1.033)	(0.981,1.034)	(0.978,1.030)	(0.980,1.032)	(0.981,1.034)	(0.982,1.036)
mother's education high (ref)			1.000		1.000		1.000		1.000	
mother's education low			1.497**		1.583**		1.260		1.101	
			(1.126,1.989)		(1.180,2.123)		(0.786,2.019)		(0.663,1.827)	
father's education high (ref)				1.000		1.000		1.000		1.000
father's education low				0.949		0.961		0.566*		0.540*
				(0.706,1.276)		(0.705,1.310)		(0.347,0.924)		(0.320,0.912)
father's education unknown				1.305		1.267		0.839		0.869
				(0.873,1.950)		(0.837,1.916)		(0.440,1.602)		(0.433,1.746)
no radiation exposure*mother's education high (ref)							1.000		1.000	
radiation exposure between ages 1-5* mother's education low							1.977		1.971	
							(1.000,3.910)		(0.975,3.987)	
radiation exposure during infancy* mother's education low							2.635		2.366	
							(0.797,8.708)		(0.692,8.092)	
radiation exposure during infancy and in-utero* mother's education low							0.407		0.376	
							(0.120,1.384)		(0.105,1.351)	
radiation exposure in-utero* mother's education low							0.371*		0.322*	
							(0.139,0.989)		(0.117,0.882)	
no radiation exposure*father's education high (ref)								1.000		1.000
radiation exposure between ages 1-5* father's education low								2.210*		1.949
								(1.121,4.359)		(0.969,3.919)
radiation exposure during infancy* father's education low								9.486***		8.749**
								(2.393,37.61)		(2.150,35.60)
radiation exposure during infancy and in-utero* father's education low								1.426		1.241
								(0.321,6.325)		(0.255,6.031)
radiation exposure in-utero*father's education low								0.833		0.791
								(0.285,2.437)		(0.261,2.397)
radiation exposure between ages 1-5*father's education unknown								1.956		1.438
								(0.789,4.849)		(0.561,3.689)
radiation exposure during infancy* father's education unknown								8.470**		7.065*
								(1.668,43.00)		(1.342,37.19)

Extension of Table 3

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
radiation exposure during infancy and in-utero*father's education unknown								1.340		0.996
								(0.171,10.53)		(0.116,8.531)
radiation exposure in-utero*father's education unknown								0.807		0.560
								(0.126,5.162)		(0.0795,3.942)
radiation dose at place of birth low (ref)		1.000			1.000	1.000			1.000	1.000
radiation dose at place of birth high		2.044***			2.066***	2.063***			0.339	0.490
		(1.458,2.866)			(1.473,2.897)	(1.470,2.894)			(0.106,1.083)	(0.117,2.051)
radiation dose at place of birth unknown		2.057***			2.116***	2.029***			2.001	1.776
		(1.381,3.064)			(1.418,3.158)	(1.359,3.029)			(0.947,4.230)	(0.877,3.597)
radiation dose at place of birth low*mother's education high (ref)									1.000	
radiation dose at place of birth high*mother's education low									8.134***	
									(2.403,27.54)	
radiation dose at place of birth unknown*mother's education low									1.091	
									(0.485,2.453)	
radiation dose at place of birth low*father's education high (ref)										1.000
radiation dose at place of birth high*father's education low										4.556*
										(1.039,19.97)
radiation dose at place of birth unknown*father's education low										1.297
										(0.580,2.900)
radiation dose at place of birth high*father's education unknown										7.941*
										(1.180,53.44)
radiation dose at place of birth unknown*father's education unknown										0.906
										(0.309,2.661)
notresettled in 1986 (ref)		1.000			1.000	1.000			1.000	1.000
resettled in 1986		3.381			3.777	3.282			4.078	2.928
		[0.412,27.76]			(0.440,32.42)	(0.389,27.71)			(0.434,38.32)	(0.322,26.61)
resettled in 1986 info unknown		1.522			1.563	1.499			1.527	1.447
		[0.881,2.630]			(0.905,2.699)	(0.867,2.591)			(0.879,2.654)	(0.833,2.513)
urban place of birth (ref)		1.000			1.000	1.000			1.000	1.000
rural place of birth		0.840			0.821	0.851			0.800	0.842
		[0.663,1.065]			(0.647,1.042)	(0.670,1.080)			(0.628,1.020)	(0.661,1.072)
type of place of birth unknown		0.384**			0.381**	0.382**			0.340***	0.376**
		[0.207,0.712]			(0.205,0.710)	(0.206,0.709)			(0.181,0.639)	(0.201,0.706)
female (ref)		1.000			1.000	1.000			1.000	1.000
male		0.541***			0.540***	0.544***			0.514***	0.547***
		[0.437,0.671]			(0.436,0.670)	(0.439,0.674)			(0.413,0.640)	(0.440,0.680)
N	1109	1109	1109	1109	1109	1109	1109	1109	1109	1109
chi2	12.74	58.65	18.38	15.32	65.51	60.48	30.81	27.61	90.03	76.43

Exponentiated coefficients; 90% confidence intervals in brackets.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

The models with socio-economic variables provide clear evidence for the socio-economic hypothesis. The models 3-6 with both basic variables and other controls suggest that there is a direct impact of socio-economic conditions in early life on adult health outcomes. The models measure childhood environment with either mother's or father's education, and the latter specification presents presumably deeper societal differentiation. When prosperity of the individual's family is observed through maternal education, those individuals from families with less educated mothers have not less than 1.5 times higher probability of having bad health in adulthood, and the estimate is statistically significant. It can be seen from the Model 5 that the estimate for individuals originating from poor families where mothers had low education is not sensitive to any substantial extent other controls. Similar evidence is given if larger amplitude between social groups is considered on the basis of father's education, albeit with some important specificities. The negative impact of unfavorable conditions in childhood can be captured only for those individuals whose father's education is unknown, which primarily lived with single mothers. In contrast, in another disadvantageous group, whose fathers had low education, the individuals appear to display positive outcomes in both basic model and model with other controls. This may point to positive selection in the group. Unlike for the model with maternal education, all estimates based on father's education are insignificant. Regarding of the estimates for the birth groups, incorporating a socio-economic variable into the model does not change them to any substantial level. Like in the previous specifications, the probabilities of having adverse health outcomes in adulthood for those exposed during infancy and in-utero or during both periods jointly, are higher in comparison to individuals conceived after the end of the iodine exposure period, but only the latter estimate is significant. Similarly, the models with other controls suggest the higher negative outcomes for those who lived in high contaminated regions in childhood. For individuals whose place of birth is unknown, apparently those who migrated to Ukraine before 1986 or lived in rural areas, the disadvantage in adult health is also substantially larger. In all, the models with socio-economic origin provide an indication that there was a purely social pathway from family environment to the adult health of all birth cohorts, although this finding is sensitive to the choice of socio-economic variable.

The estimates for birth cohorts provide some evidence on that being exposed to iodine radiation due to the Chernobyl fallout at some critical periods in early life causes permanent health damage. The models indicate higher probabilities of having negative health outcomes in adulthood for those individuals who were exposed to radiation during infancy or during both infancy and fetal stage, and the latter estimate is significantly different from the estimates for individuals conceived after the end of the exposure period. The estimated effects, though, are solely biological. The results also show the great influence of social origin on adult health. According to our findings, those individuals who were born in poorer families are experiencing worse health in adulthood. When the social differentiation is fixed at a lower level of the family sufficiency, measured so with maternal education, this negative effect for individuals from less affluent early life conditions is statistically different from zero. Given this, we can suggest that the obtained individual effects for birth cohorts may be partially driven by varying child socio-economic environment within each birth group. As previously discussed, in terms of the critical period literature, there might be socio-biological pathways from early life to adult health outcomes. In what further follows, the interaction terms between birth cohorts and socio-economic status in childhood are incorporated into the models. As it has been done heretofore, we proceed in discussing the estimates within models

based on either mother's or father's education. The control group for the birth cohort then in coming models is individuals from wealthier families, where mother (father) had at least incomplete higher education. Subsequently, the treatment groups are those from poorer families, including individuals whose father's educational status is low or unknown.

Models testing the socio-biological hypothesis introduce substantial changes for collective effects of birth and social groups. Primarily, after incorporating the interaction terms between birth and socio-economic groups into the models 7 and 8, almost all collective estimates for socio-economic cohorts appear to be affected. Individuals from poorer families, measured with father's education, on average have a lower probability of having a bad health than those from wealthier families, and the estimate is statistically significant. This may potentially point to positive selection among these individuals. When social strata are measured with maternal education, like in the previous models, the log odds ratio for low income group remains positive, but loses its statistical significance. The original estimates for birth cohort are also changed, and for some groups inconsistently with the a-priori expectations. Among those who had an access to more resources in childhood, those individuals who were exposed during infancy and later childhood have lower chances to have negative health outcomes, and for one group this estimate is significant. In the model with father's education, individuals from rich families who were exposed to radiation during infancy have obtained 80 per cent lower probability of having adverse health outcomes in adulthood in comparison to those otherwise not-exposed and originated from another socio-economic group. In contrast, for other groups, either exposed during infancy and in-utero or solely in-utero, the data indicates adverse health outcomes. The size of this relative effect is stronger for the model with mother's education. Individuals who were exposed during infancy and in-utero from more affluent families have more than four times higher probability of having bad health in adulthood in comparison to the non-exposed from poorer families. This is a clear sign of positive selection on health outcomes in this particular group, that has been indicated previously. Therefore, the biological effect, previously found in the basic models, is driven by the overwhelming impact of the exposed during infancy and in-utero from wealthier group, and thus appears to be purely statistical.

Turning to the focus of this study, the models with interaction terms provide body of evidence for the socio-biological hypothesis. The data from the models 7 and 8 shows that some birth cohorts experience negative health outcomes later in life with regard to their less affluent socio-economic origin. According to our expectations, the size and significance of the effects appear to be more accentuated for the model with father's education. The produced estimates for those exposed to iodine irradiation during infancy and between the ages one and five with poorer origin indicate substantially higher probability of having negative health outcomes later in life, which are also significant for the model with father's education. A striking finding is that the estimates for the exposed during first year of life, either from families where father has low education or from one-parent families, are overlarge and significant. These individuals on average experience not less than 8.5 times higher probability of having bad health in adulthood than those who conceived after the end of the iodine exposure period and born in wealthier families. The model with father's education produces two times higher odds ratio for those exposed after the age of one and before reaching five, from poorer families, and the effect is also significant. In the model with maternal education as a measure of socio-economic origin, the log estimates for the exposed during infancy and in early

childhood are also positive, albeit insignificant. Similarly to the mirror estimates for births groups from wealthy families, the estimated lower probability for those who were exposed during infancy and in-utero or solely in-utero are a clear indication of the previously observed positive selection. For the model with father's education, though, the exponentiated estimate for the group that was exposed to iodine during infantile and fetal stages is more than one. Summing up on the critical periods, the models enabled to obtain strong effects for individuals who were exposed during infancy and before the age of five. According to the a-priori expectations, father's education, that measures social strata more deeply, substantially accentuates these effects. Presumably due to the presence of selection mechanisms in data, no clear impact can be captured for those who were exposed solely in-utero and both during infancy and in-utero.

The final models 9 and 10 extended with control covariates strengthen the evidence for the socio-biological hypothesis with the estimates for individuals who received low radiation doses. Even though controls only slightly changed the sizes of coefficients for birth cohorts, they do not affect their significance to any considerable extent. Likewise previously, the estimates provide sufficient evidence for selection processes in data for the exposed during infancy and in-utero or solely in-utero. This particularly holds when socio-economic origin is measured with mother's education. In contrast to the a-priori expectations, individuals from these groups with wealthier origin experience not less than two times higher probability of adverse health outcomes in adulthood in comparison to the non-exposed from poorer families. Also, some selection can be observed in the model with father's education for those who were not exposed to iodine radiation with less affluent socio-economic origin. Individual effects for study groups are also affected. Unlike to previous models, the only significant and overlarge estimate is produced for individuals who were exposed during infancy. The data indicates more than seven-fold higher probability of having bad health in adulthood for this group originating either from poor families or one-parent families in comparison to the non-exposed group with wealthy socio-economic background. In the models, the interaction effects for birth and social groups are measured for those who received low doses of iodine radiation. Socio-economic gradient is also present in health outcomes as regards iodine dosage. Interaction terms for individuals who were subject to a highest radiation dose in early childhood and born in less affluent families indicate significant and strong effects from exposure to iodine isotopes. Importantly, in size, this statistically significant collective impact from higher dosage intensity and socio-economic origin is not larger than that attributed exclusively to the interaction effect estimated for individuals with poor socio-economic conditions who were exposed during infancy.

Based on the models testing the socio-biological hypothesis, the absolute probabilities of negative health outcome in adulthood are predicted for all birth cohorts with regard to their socio-economic status in early life. Figure 2 displays the predicted probabilities for an average individual for the model with mother's education as a socio-economic variable. The predicted values indicate selection mechanisms in data for those individuals who were irradiated to iodine during in-utero and both during infancy and in-utero. As contrasted to the a-priori expectations, individuals for these birth groups who born in more affluent families obtain the highest probability of having bad health in adulthood. Among individuals from poorer families, the pattern of the effect from exposure to iodine radiation shapes an inverted U curve, with the most accentuated effect for the group that was exposed during infancy and the

least effect estimated for the exposed in-utero. Individuals who got irradiated both after birth and while in-utero and exclusively after birth and originated from poor socio-economic environment have not less than 0.5 as the probability of having adverse health outcome later in life. The predicted probabilities for those who were born in less affluent families suggest that disadvantages in adult health for the exposed in-utero are equal to those obtained for individuals who conceived after the end of the exposure period. Striking evidence is provided for birth cohorts which were exposed in postnatal stages and before the age of five as regards better socio-economic conditions in their childhood. The predicted values for these groups indicate probability of having bad health in adult life that is smallest among all birth groups, and even lower than that for the group which is assumed to be non-exposed to iodine irradiation. The latter probably indicates the positive selection in the group of the non-exposed from wealthier origin. More importantly, the largest gap between individuals from poor and wealthy families is observed for those exposed during infancy, as for them the average predicted probability of negative outcomes equals to 0.49 and 0.22 units, respectively. For the group between the ages of one and five, the data suggests slightly smaller possibility of this amelioration in adverse health outcomes generated with exposure to radiation. Therefore, the model with mother's education shows that the largest scope for the influence from the socio-economic origin opens up during first year of life and gradually decreases up to five years. Presumably due to the peculiarity of data, the evidence is inconclusive for the other birth groups.

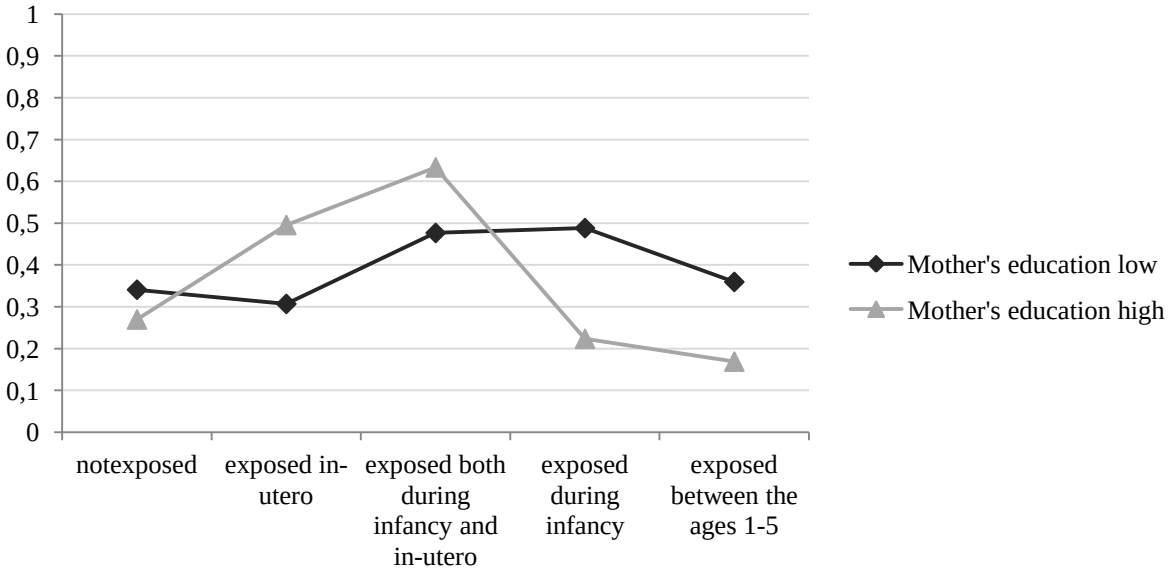


Figure 2 – Predicted probabilities of having bad health for an average individual as regards child socio-economic origin due to exposure to *iodine radiation* in early life.
Model with mother's education

The patterns of absolute probabilities from exposure during different periods in early life when socio-economic origin is measured otherwise with father's education appear to be similar. As previously discussed, in the models, poor socio-economic conditions are distinguished between those whose fathers had low education and those lived with single mothers. The data from Figure 3 shows that individuals who lived with single mothers and exposed at any age during early life experience on average higher probability of having bad health in adulthood than those

originated from families with less educated fathers. At most, this probability reaches 0.6 units for those exposed both during infancy and in-utero. As it has been found in the previous model, we observe here the largest probabilities of bad health in adulthood for the individuals who were irradiated both during infancy and in-utero and solely during infancy. Similarly to the previous model, disadvantage for the exposed in-utero for all poor groups is equal or lower relatively to the non-exposed individuals. Again, the predicted probabilities suggest possible selection mechanisms in data for the exposed in-utero, as they are larger for the more affluent group. For other birth cohorts the predicted values correspond to the a-priori expectations. Once more, the data suggest the lowest probability of having bad health for those who were irradiated to iodine during first year of life and originated from wealthier families. The predicted value goes down to 0.14 units and thereby appears to be the lowest among all groups in both models. Like in the previous model, this probability, that is substantially smaller than that predicted for individuals conceived after the end of the exposure period, points to positive selection in the control group. The average predicted probability for the individuals with more affluent origin and exposed between the ages one and five decreases now only to negligible levels, even though it is smaller in size than those estimated for individuals with poorer child conditions. In line with implications from the model with mother's education, the data provide sufficient evidence for the possibility to reverse negative outcomes of exposure during infancy and, to a minor extent, later in early childhood. Among individuals from poorer families, those who were exposed solely in-utero and both in-utero and during infancy also appear to be affected, and for the latter birth cohort the effect is largest. For wealthier groups, data for individual exposed exclusively before birth indicate selection mechanisms.

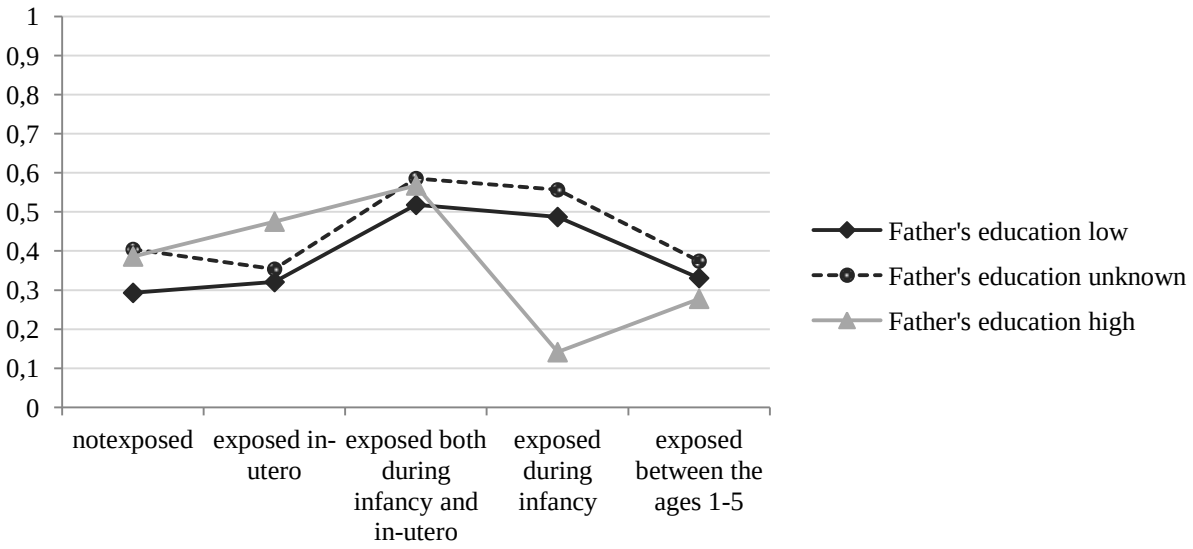


Figure 3 – Predicted probabilities of having bad health for an average individual as regards child socio-economic origin due to exposure to *iodine radiation* in early life.
Model with father's education

The first approach to selection of groups with varying exposure to iodine radiation allowed us to produce different patterns for birth cohorts and child socio-economic status and thereby to prove the study hypotheses. The purely biological hypothesis can not be confirmed. If any significant estimates are obtained in the models, like that for the group exposed both during infancy and in-utero, they appear to be driven by statistical selection mechanisms. The socio-

economic hypothesis is clearly supported. Those individuals who originated from families where mothers did not have higher education have much higher probability of having bad health in adulthood relatively to those otherwise from wealthier families. The first approach provided clear evidence for the socio-biological hypothesis. In both absolute and relative terms, the consistent evidence is suggested for individuals irradiated during first year of life. According to our expectations, the effects appear to be accentuated to a larger extent for the model with father's education than for the model with mother's education. For individuals originated from more affluent families and exposed exclusively after birth, negative outcomes from exposure appear to be ameliorated. The situation among individuals with wealthier socio-economic origin points to positive selection among those who were irradiated in-utero and both during infancy and in-utero. Similar selection mechanism can be observed for the individuals who were not exposed to iodine radiation and born in families with more educated fathers. Both poor groups defined either for individuals who had fathers with low education or who were born in one-parent families further indicate consistency of implications for the exposed after birth and later in childhood. From the model based on maternal education, average probabilities for the exposed during infancy and between the ages of one and five, which are smaller than the effect estimated for non-exposed individuals, may indicate the strictness and limitation of the defined exposure period. To account for this, we extend the exposure period and reallocate study groups.

4.2 Second approach

The exposure to radiation lasted, albeit gradually decreasing, during eight months after the accident throughout Ukraine that enables to investigate its potential link to negative health outcomes in adulthood by enlarging the exposure period. As previously discussed, any longer period of exposure to radiation can be stated from the beginning of the accident on April 26, 1986 until December 1986, and then irradiation during this period can be mainly attributed to *cesium* isotopes.

Data from the models for cesium irradiation provide some support for both biological and socio-economic hypotheses. Models 1-6 with birth and socio-economic groups suggest indication that there might be direct links from exposure to exogenous shock and poor conditions in early life to health in adulthood. The basic models 1 and model 2 with controls produces parameter estimates for birth groups which are in general consistent with the a-priori expectations, although none of them differs statistically from zero (see table 4). Individuals who were exposed both during infancy and in-utero and solely after birth experience on average greater disadvantage in health later in life than those who conceived after the end of exposure period. Unlike to the models for iodine irradiation, coefficient for those exposed both during infancy and in-utero, albeit being the largest among all birth groups and thereby indicating possible selection, is now insignificant. The estimates for the groups exposed while in-utero and before the ages one and five close to zero and insignificant. From the models 3-6, importance of the socio-economic status in childhood for adult health emerges clearly. The models suggest estimates, either with the mother's or father's education as a socio-economic variable, which are almost identical to those obtained with the first approach. Individuals with less educated mothers have 1.5 times higher probability of having bad health in adulthood

than those whose mothers have at least incomplete higher education, and coefficient is significant in both basic and extended specifications. Although the models 4 and 6 based on the father's educational status, does not allow to obtain any statistically significant estimate for socio-economic groups, they suggest the greater disadvantage in health outcomes for those individuals who lived with single mothers. The advantage in health outcomes observed for socio-economic group with low father's education may be due to the specificity of data, with this group typically having comparatively better health. Lower probability of bad health in adulthood among individuals originated from wealthier families on average can be indicated even when they limited to those who lived in low contaminated regions. Therefore, similarly to the models with iodine irradiation, parameter estimates obtained from basic specifications on the basis of exposure to cesium radioisotopes indicate that there is a direct influence of socio-economic environment on long term health. No statistically significant biological effects can be found, although their sizes suggest the major importance of infantile stage for later health status.

Models with interaction terms strongly support the socio-biological hypothesis for several birth groups. The baseline effects now for birth groups represent the responses for individuals from wealthier families, whose ether father or mother had higher education. The model 7 with mother's education produces parameter estimates for exposed groups which appear to form an inverted U-shape curve: individuals originating from poor families who were exposed in-utero and between the ages one and five have 3.5 and 2.8 times higher probability of having bad health in adulthood respectively, whereas those exposed during infancy experience 3.8 times higher probability, in comparison to the non-exposed to cesium irradiation and lived in wealthier families. When socio-economic status is defined more deeply, on the basis of father's education, the relative effects become accentuated to a much larger extent for some birth groups. For now, individuals irradiated during infancy whose fathers were less educated or did not leave with a family obtain at least eleven-fold higher probability of having negative health outcomes later in life in comparison to the control group. Importantly, for those originated from wealthier families, the estimate for the cohort exposed during first year of life, that is also significant, shows that these individuals have up to 6 times higher disadvantage in adult health than those non-exposed to cesium radiation. The model 8 also produces the estimate for the group between the ages one and five that is similar in size to that obtained from the model with maternal education. Unlike, the effect for the in-utero cohort decreased in size considerably, and lost its significance. In the models, for some birth and socio-economic groups, we can observe selection mechanisms in data. The positive estimate obtained for the individuals exposed both during infancy and in-utero appears to be strongly related to a process of positive selection. The same holds particularly for all baseline socio-economic groups, which according to data unexpectedly experience advantage in adult health outcomes. Given all, the models with interaction terms indicate the greater relative impact from irradiation in early life for those who were exposed during their infancy, and born either in rich or in poor families. Also, in comparative terms, the estimates for individuals with poor socio-economic background show that the environmental vulnerability possibly starts in-utero and decreases during early childhood.

Table 4 – *Cesium irradiation.* Odds Ratios for self-reported adult health with mother's education or father's education as a measure of child socio-economic status

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
no radiation exposure (ref)	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000
radiation exposure between the ages 1-5	0.912	1.046	0.893	0.906	1.029	1.026	0.378	0.430	0.403	0.523
	(0.379,2.197)	(0.373,2.939)	(0.370,2.151)	(0.375,2.185)	(0.366,2.892)	(0.364,2.891)	(0.131,1.088)	(0.153,1.204)	(0.122,1.332)	(0.162,1.686)
radiation exposure during infancy	1.350	1.731	1.299	1.342	1.670	1.701	0.414	0.165**	0.563	0.218
	(0.647,2.819)	(0.711,4.214)	(0.621,2.715)	(0.642,2.808)	(0.686,4.069)	(0.697,4.154)	(0.112,1.530)	(0.038,0.713)	(0.133,2.389)	(0.045,1.069)
radiation exposure during infancy and in-utero	1.679	1.597	1.693	1.707	1.600	1.621	3.503**	1.391	3.535**	1.387
	(0.858,3.287)	(0.806,3.165)	(0.864,3.317)	(0.870,3.347)	(0.807,3.175)	(0.816,3.219)	(1.285,9.553)	(0.441,4.385)	(1.255,9.962)	(0.408,4.716)
radiation exposure in-utero	1.040	0.964	1.035	1.038	0.952	0.962	0.375	0.613	0.348	0.555
	(0.624,1.734)	(0.572,1.624)	(0.620,1.727)	(0.622,1.733)	(0.564,1.606)	(0.570,1.622)	(0.130,1.078)	(0.238,1.577)	(0.118,1.023)	(0.211,1.456)
age	0.758	0.774	0.782	0.780	0.809	0.792	0.783	0.653	0.785	0.646
	(0.184,3.122)	(0.182,3.283)	(0.189,3.229)	(0.188,3.229)	(0.190,3.444)	(0.186,3.372)	(0.189,3.243)	(0.155,2.754)	(0.182,3.395)	(0.147,2.835)
agesq	1.008	1.008	1.007	1.007	1.007	1.008	1.007	1.011	1.008	1.012
	(0.978,1.039)	(0.978,1.040)	(0.977,1.038)	(0.977,1.039)	(0.977,1.039)	(0.977,1.040)	(0.977,1.038)	(0.981,1.043)	(0.977,1.040)	(0.981,1.045)
mother's education high (ref)			1.000		1.000		1.000		1.000	
mother's education low			1.460**		1.541**		0.877		0.753	
			(1.101,1.935)		(1.151,2.063)		(0.527,1.457)		(0.439,1.291)	
father's education high (ref)				1.000		1.000		1.000		1.000
father's education low				0.937		0.947		0.433**		0.397***
				(0.697,1.258)		(0.695,1.291)		(0.252,0.747)		(0.223,0.708)
father's education unknown				1.311		1.277		0.660		0.682
				(0.878,1.959)		(0.845,1.931)		(0.320,1.361)		(0.318,1.462)
no radiation exposure*mother's education high (ref)							1.000		1.000	
radiation exposure between ages 1-5* mother's education low							2.839**		2.881**	
							(1.399,5.759)		(1.389,5.976)	
radiation exposure during infancy* mother's education low							3.790*		3.477*	
							(1.130,12.71)		(1.001,12.08)	
radiation exposure during infancy and in-utero* mother's education low							0.373*		0.350*	
							(0.142,0.983)		(0.128,0.955)	
radiation exposure in-utero* mother's education low							3.451*		3.335*	
							(1.160,10.27)		(1.093,10.18)	
no radiation exposure*father's education high (ref)								1.000		1.000
radiation exposure between ages 1-5* father's education low								2.893**		2.638**
								(1.409,5.942)		(1.258,5.529)
radiation exposure during infancy* father's education low								12.40***		11.87***
								(3.064,50.16)		(2.855,49.38)
radiation exposure during infancy and in-utero* father's education low								1.472		1.462
								(0.474,4.564)		(0.437,4.897)
radiation exposure in-utero*father's education low								1.989		2.105
								(0.721,5.490)		(0.745,5.949)
radiation exposure between ages 1-5*father's education unknown								2.489		1.842
								(0.949,6.529)		(0.678,5.006)
radiation exposure during infancy* father's education unknown								10.71**		8.989**
								(2.043,56.13)		(1.648,49.04)

Extension of Table 4

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
radiation exposure during infancy and in-utero*father's education unknown								1.299		1.147
								(0.219,7.694)		(0.181,7.256)
radiation exposure in-utero*father's education unknown								2.273		1.932
								(0.574,8.996)		(0.459,8.144)
radiation dose at place of birth low (ref)		1.000			1.000	1.000			1.000	1.000
radiation dose at place of birth high		2.067***			2.087***	2.010***			0.353	0.467
		(1.475,2.895)			(1.490,2.924)	(1.346,3.002)			(0.112,1.112)	(0.114,1.910)
radiation dose at place of birth unknown		2.044***			2.101***	2.091***			1.978	1.752
		(1.373,3.044)			(1.408,3.133)	(1.491,2.932)			(0.926,4.227)	(0.856,3.584)
radiation dose at place of birth low*mother's education high (ref)									1.000	
radiation dose at place of birth high*mother's education low									7.714***	
									(2.309,25.77)	
radiation dose at place of birth unknown*mother's education low									1.110	
									(0.487,2.529)	
radiation dose at place of birth low*father's education high (ref)										1.000
radiation dose at place of birth high*father's education low										4.870*
										(1.135,20.90)
radiation dose at place of birth unknown*father's education low										1.311
										(0.580,2.962)
radiation dose at place of birth high*father's education unknown										8.057*
										(1.217,53.33)
radiation dose at place of birth unknown*father's education unknown										0.894
										(0.300,2.663)
notresettled in 1986 (ref)		1.000			1.000	1.000			1.000	1.000
resettled in 1986		3.372			3.739	3.260			4.063	2.911
		(0.411,27.71)			(0.437,32.02)	(0.385,27.59)			(0.433,38.14)	(0.321,26.37)
resettled in 1986 info unknown		1.471			1.501	1.451			1.465	1.405
		(0.852,2.540)			(0.870,2.590)	(0.840,2.507)			(0.843,2.545)	(0.811,2.433)
urban place of birth (ref)		1.000			1.000	1.000			1.000	1.000
rural place of birth		0.836			0.818	0.848			0.803	0.835
		(0.660,1.060)			(0.645,1.038)	(0.668,1.077)			(0.630,1.023)	(0.656,1.063)
type of place of birth unknown		0.385**			0.383**	0.383**			0.341***	0.385**
		(0.208,0.715)			(0.206,0.714)	(0.206,0.712)			(0.181,0.643)	(0.205,0.723)
female (ref)		1.000			1.000	1.000			1.000	1.000
male		0.540***			0.539***	0.543***			0.515***	0.547***
		(0.436,0.669)			(0.435,0.668)	(0.438,0.673)			(0.414,0.642)	(0.440,0.679)
N	1109	1109	1109	1109	1109	1109	1109	1109	1109	1109
chi2	10.79	56.87	15.81	13.71	63.04	59.02	34.71	27.69	92.73	76.57

90% confidence intervals in brackets

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Introducing all available controls in models does not affect, to any substantial extent, individual estimates, and allows us to compare final results on the socio-biological hypothesis with relative effects obtained from two approaches. After incorporation of the other important covariates in the models 9 and 10, the sizes of parameter estimates for interaction terms appear to be only marginally affected. Among others, the baseline effects are now produced for those individuals who were irradiated at low doses. Like for the final models with iodine irradiation, we observe greatest relative disadvantage in adult health outcomes for those individuals who were exposed to cesium isotopes during infancy, and the effect is consistent among all specifications. The model for cesium exposure for the first time produces significant estimates for other birth and socio-economic groups. According to our findings, individuals with poor origin and exposed to cesium in prenatal stage experience larger negative health outcomes than those non-exposed and originated from wealthy families, albeit captured so only in the model with maternal education. The identical in size effect can be indicated for those from less affluent families who were irradiated with cesium between the ages one and five. As we expected, all the effects for interaction terms between birth and socio-economic cohorts are enhanced in the models based on cesium irradiation, which in its analytical strategy compares birth groups relatively more distinct in their exposure intensity. The same holds for the models with father's education, which distinguish socio-economic groups more deeply. Again, the models for cesium irradiation indicate larger collective effect for those who received high radiation doses and lived in less affluent families. Albeit overlarge, the size of the relative impact from radiation intensity is less than those obtained for birth cohort irradiated during first year of life, estimated for individuals from families with less educated parents or from one-parent families. Whereas in the models for iodine irradiation data pointed to positive selection in groups of exposed in-utero and both in-utero and during infancy, in the specifications for cesium exposure this process can be observed only for the latter group. Some selection bias possibly exists also in all baseline socio-economic groups. Therefore, the second approach in produced relative effects once again stresses the importance of the infancy for later life health outcomes, and provides new evidence on that for the in-utero stage and early childhood.

Absolute probabilities predicted for the model with mother's education for an average individual provide the pattern of gradually changing vulnerability to exposure among birth cohorts (see Figure 4). Because of the selection processes in some groups, the data differs as regards socio-economic origin. Among individuals with poor socio-economic background, all birth cohorts indicate the larger probabilities to have bad health in adulthood than those who assumed to be non-exposed to cesium radiation. More specifically, the disadvantage in adult health appears to emerge in-utero, reach the highest level of 0.49 units during infancy, and then decrease to in-utero levels during early life. Importantly, in line with previously discussed specificity of exposure to cesium radiation in Ukraine that persisting but gradually decreasing in some regions over time, those individuals who selected as conceived after the end of exposure period experience also large average probability of having negative health outcomes, which equals to 0.30 units. The evidence for groups with more educated mothers seems to be less consistent to the a-priori expectations. The predicted disadvantage for those exposed both during infancy and in-utero is substantially larger in comparison to those for any other group. This may point to the processes of positive selection in subsequent birth cohorts. Other birth groups demonstrate negative outcomes which correspond to theoretical considerations. Predicted probabilities of bad health in adulthood for individuals exposed

during prenatal stage and after birth up to five years old are much lower than that for non-exposed individuals. Among groups that behave expectedly, the difference in predicted probability between poor and wealthy groups, that is more than 0.2 units, are largest for those who were exposed to cesium radiation during infancy, and slightly smaller for those exposed in-utero and between the ages one and five. Therefore, even though the lowest average disadvantage in health in later life can be found for those irradiated in-utero, in terms of possibility to ameliorate the negative outcomes of exposure in early life with socio-economic environment, infancy appears to be the most important. The predicted probability that is largest for individuals who were exposed during first year of life and lived in less affluent families also supports this conclusion.

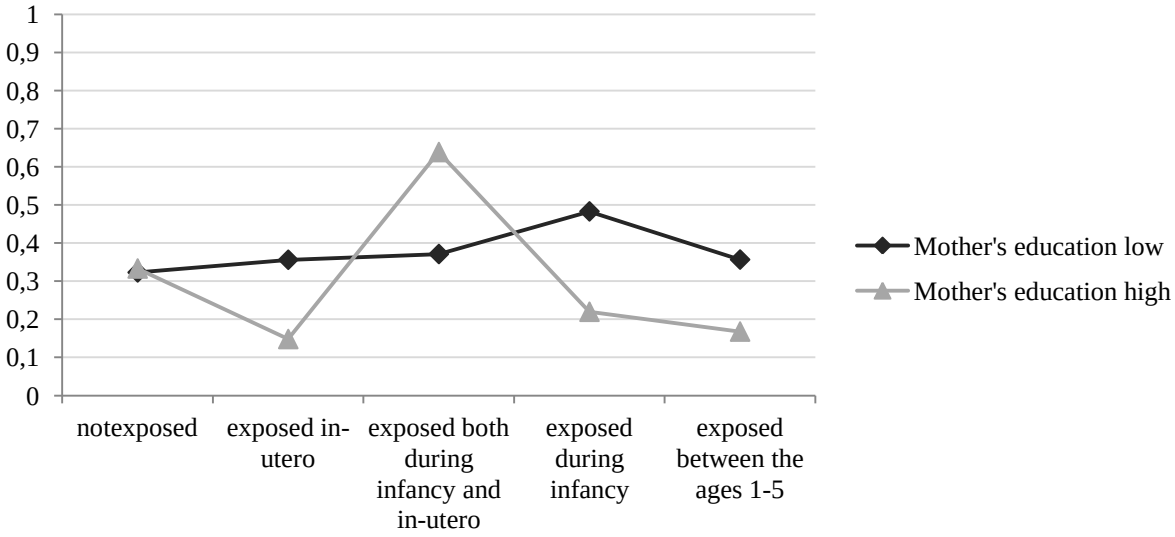


Figure 4 – Predicted probabilities of having bad health for an average individual as regards child socio-economic origin due to exposure to *cesium radiation* in early life.
Model with mother's education

The model with father's education allows to predict the probabilities of negative outcomes that appears to further accentuate the impact from exposure in early life for the birth cohort exposed during infancy. Like for the models with mother's educational status, the evidence demonstrated on Figure 5 is consistent for all birth cohorts for individuals with poorer socio-economic origin. Quite expectedly, individuals who lived with single mothers have systematically higher average probability of bad health in adulthood, which at most reaches 0.56 units. For both poor groups, whose either father had low education or did not live with a family, a pattern of vulnerability to exogenous shock indicates the large disadvantage only for individuals exposed to cesium especially during infancy and both during infancy and in-utero. As the predicted value for those exposed while in-utero is slightly larger than that for non-exposed individuals, we can state that the effect in the groups exposed both during infancy and in-utero is driven by the impact from the exposed solely during infancy. Importantly, the group with less educated fathers suggests the higher probability of negative health outcomes for irradiated between the ages one and five than for those affected in-utero. As it has been indicated previously with the relative effects, there are probably selection mechanisms in some birth groups among individuals with wealthier origin. Individuals who were not exposed to radiation or exposed both during infancy and in-utero seem to be selected on comparatively

worse health outcomes. For other birth cohorts, the evidence on disadvantage in adult health seems to be strongly supported for those exposed solely during infancy. According to our findings, the average probability of having bad health in adulthood for those exposed during their first year of life appears to reach at most 0.14 units, if individuals were born in wealthier families. This estimation for irradiated during infancy creates the gap between individuals with poor and wealthy origin of such size that never emerges in any other birth group. The data suggests that the negative outcomes of exposure between the ages one and five and exclusively in-utero can be ameliorated only from 0.39 to 0.29 units, while comparing families with more educated fathers and with no fathers. The difference in predicted probabilities between groups who lived with either more or less educated fathers points to the scope of mediation for the exposed in-utero and after the age of one that is almost negligible, if ever exists. In the model, in order to conclude on equal importance of all birth periods, one would expect to see an inverted U shape pattern for birth cohorts from wealthier families, as results for poor individuals suggest. However, our data does not provide this evidence. Taken together, the model with father's education accentuates the negative health outcomes only for those who were irradiated during infancy.

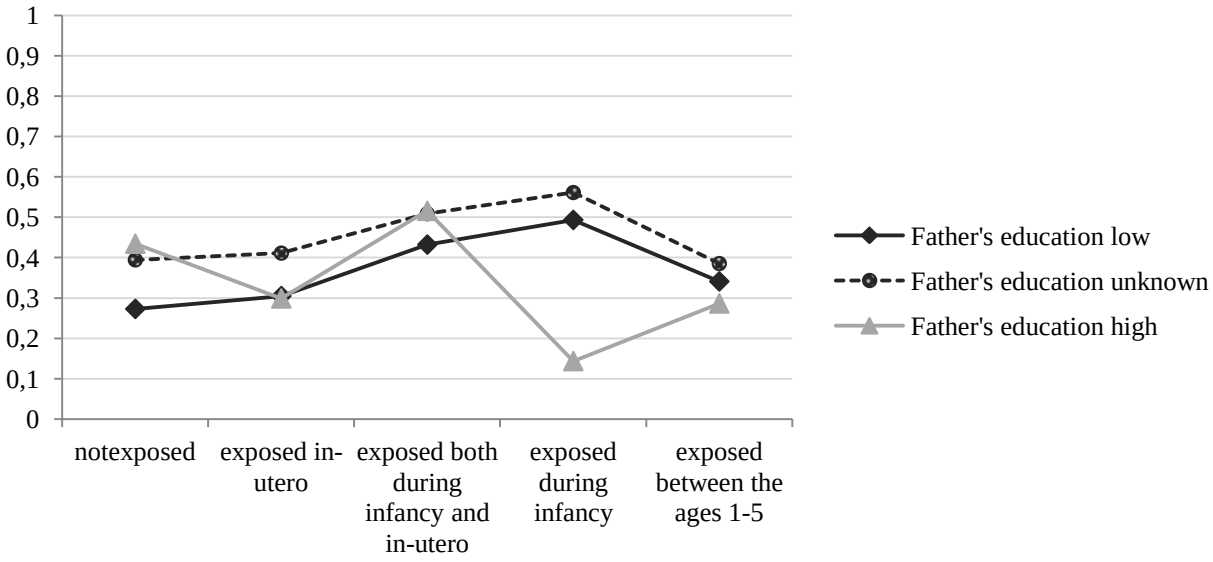


Figure 5 – Predicted probabilities of having bad health for an average individual as regards child socio-economic origin due to exposure to *cesium radiation* in early life.
Model with father's education

Turning to the birth cohort that is major focus of this study, the data from both approaches strongly supports the infancy inflammation hypothesis. The largest responses, in both relative and absolute terms, have been captured for those who were irradiated during first year of life, regardless of how long the exposure period is established. According to our expectations, the more distinctly a control group is selected from the time of exposure, as it is shown with the model for exposure to cesium, the stronger impact for the exposed during infancy can be indicated. Additionally, the results for this birth group are the only stable when socio-economic variable is changed. Again, the more distinct socio-economic groups are allocated, for instance with selection of a group whose fathers did not leave with individuals, the larger

is the effect for the irradiated after birth. The environmental vulnerability appears to gradually decrease when an individual ages. Thus, albeit being much smaller than those estimated for individuals who were exposed during infancy, the probabilities for the exposed between the ages one and five are found to be also large, in absolute and relative sizes. The estimates for this birth group keep statistical significance across different specifications.

Our results are inconclusive on the group exposed solely in-utero. The estimates for poor socio-economic group indicate some vulnerability of individuals irradiated during fetal stage that is smaller than for other birth groups, but comparisons across different socio-economic groups do not allow to draw any consistent implication. In the models with shorter exposure period, positive selection observed in data does not allow to capture properly the impact for the exposed in-utero. For the longer exposure period, any significant difference between individuals irradiated in-utero and originated from distinct socio-economic groups can be indicated only in the specification with mother's education. When no statistical selection processes are found in responses for the in-utero cohort in the models for longer exposure period, this appears to be puzzling. If any, one would expect the effect to be most accentuated in the model with father's education, as it happens with the cohort exposed during infancy, but not to disappear. Unlike the in-utero cohort, across specifications with either mother's or father's education, the estimate for the exposed after the age of one is keeping its size. The only explanation for instability of the effect for the exposed in-utero may be attributed to the socio-economic variable as itself. As previously discussed, parental education can affect health outcome of the children not only through access to resources, but also as a knowledge tool. Therefore, it is likely that maternal education for those exposed during fetal stage serves as a productivity shift factor, and then can be referred primarily to the mothers' possibility to process information and make decisions on health behavior after the accident like Chernobyl. When we are unable to observe on such characteristics, this possibility of influence stemming from the mothers' educational status *per se* should not be excluded.

4.3 Limitations

This study has some limitations which should be acknowledged when interpreting the results.

The primary limitation concerns the use of the self-reported health as a health measure. In the literature, there is a long-standing debate on the efficacy of self-reported health measures (e.g. Baker et al, 2004, Bjorner et al, 2005). In practice, this measure has been used in the field since the 1950s, and nowadays is recommended as a standard part of health surveys (Robine et al, 2003). In addition, self-perceived health is suggested to be related to the strongest biological indicator, death, and therefore is also recommended as a tool for disease risk screening and clinical trials (e.g. May et al, 2006). It is argued that in assessing own health, individuals take into account not only health experience and expectations, but also contextual information (Jylhä, 2009). In relation to the Chernobyl case, it can be of particular importance for those exposed to radiation. Thus, several studies mention that the psychological rather than the psychical damage of Chernobyl may ultimately affect self-perception (Bromet et al, 2002; Havenaar et al, 2003). It has been concluded that numerous stress symptoms accompanying exposed individuals were driven by the belief that their health was adversely affected by the disaster. However, even though for adults these effects were given no clinical

legacy, for those exposed during childhood any mental disorders were related by researchers to neurological and immune damages (WHO, 2007, Yablokov et al, 2009). In this vein, if there are any strong disturbances in self-rated health measures caused by purely psychological effects, are likely to persist only for those exposed individuals who were officially given in childhood the label *Chernobyl victim*. These persons were subject to additional medical treatment undertaken by authorities, and some monetary maintenance. The richness of data allows us to allocate those individuals. The data indicate that only five persons in a sample are receiving Chernobyl payments, and none of the individuals were resettled from an evacuation zone (see Appendix N). If some endogenous factors related to Chernobyl self-perceived affectedness are distorting health outcome estimates, measures of radiation dosage introduced in our models have controlled for this.

Additional limitation of this study is the possible selection bias for some exposed birth cohorts. It is acknowledged in the field that mortality caused by environmental insults is substantially higher during prenatal stage than during other periods in early life. As a result, survivors of negative fetal events are generally positively selected (Almond & Currie, 2011b). Hence, in order to be detected, the negative effects of the exposure have to be substantially strong to overwhelm the positive effect of selection. In other words, the analysis commonly tends to understate the negative outcomes of exposure for those exposed in-utero by increasing the number of healthier fetuses. The empirical literature on Chernobyl radiation exposure indicates that this selection mechanism might indeed have taken place, but only for the most contaminated regions. According to the results of the French-German Chernobyl Initiative (Bebeshko et al, 2003), a weak but otherwise significant correlation between stillbirth, in-utero, and postnatal mortality and radiation factors is identified for the most contaminated regions of Ukraine. A significant increase in frequency of spontaneous abortions and miscarriages has also been documented for the most polluted regions of Belarus and Russia (Yablokov et al, 2009). However, if any positive selection for in-utero cohort exists in our data, the models used here are capable of accounting for this drawback to some extent. Thus, the distinction between most and less contaminated regions by using the radiation dosage measure controls for the possible downward bias for those exposed during fetal stage.

Due to the specificities of data, any results yielded in this current study are likely to be understated. There might be several sources of this underestimation. Primarily, underestimation stems from the specification of the health variable. As indicated previously, self-reported health is recoded into a binary variable, with responses on average adult health placed to the group of bad health outcomes. It is done based on the evidence that individuals who perceive own health at observation as being fair, are likely to respond positively on the incidence of health problems and diseases more frequently than on average in a sample. However, given the fact that in the final group of negative outcomes, responses on average health greatly dominate the pure bad responses, we actually estimate probability of being in average health in relation to having good health. Had we had solely bad versus good outcomes, our results might have been substantially larger. Another source of underestimation comes from the specificity of exposure to radiation. Any individuals, which might be observed on average in Ukraine from the time of the accident till onwards, may have been exposed to radiation to some extent, and there is no ideal control group for the analysis of adult health outcomes. In fact, in the models we compare health effects of individuals who

received larger portion of radiation with those who were less irradiated on average with regard to geographical allocation. This particularly holds for highly contaminated regions, but not for low polluted areas. It creates downward bias in the estimates, which ideally should reflect results of exposed birth cohorts versus unexposed ones. However, even though radiation damage has still not vanished completely in some regions, it has been decreasing gradually over time. In this vein, the current study will benefit from the implementation of the exposure periods of different lengths. Individuals assumed as being not exposed in the longer period of cesium irradiation are certainly irradiated to much lesser extent than those selected for the control group of iodine irradiation. Given this, we expect to find greater effects for the longer exposure period. Two approaches – based either on iodine (3 months) or cesium (approximately 8 months) irradiation – serve then as a sensitivity test to each other. Therefore, all the estimates obtained in this study should be treated as of lower bounds.

Any extension of exposure to radiation for a long period potentially weakens the assertions of natural experiment. The irradiation pattern in Ukraine precludes the analysis of gradually decreasing exposure to cesium isotopes. However, the longer the duration of irradiation is established, the more likely self-selection into or out of the study groups due to family planning decisions to be indicated. Therefore, the period of exposure to iodine radionuclides limited to three months allows selecting the quasi-experimental groups more soundly than that defined for cesium irradiation with eight months. Even though there are good a-priori reasons to assume the random variations in exposure to cesium until the end of 1986, selection processes in data are still possible. In this vein, the major importance is which effect is stable across different specifications. Our findings indicate that the effect for those exposed in-utero is significant only for the models with cesium exposure based on the mother's educational status. The exercises with any longer exposure periods up to the middle or the end of 1987 (not reported here) indicate that the size and significance of this estimate is sensitive to the choice of period. The effect for individuals who were irradiated while in-utero almost disappears and loses its significance, and positive selection is likely to be present. In contrast, the parameter estimate for those exposed during infancy is keeping its significance and magnitude. Moreover, for the longer exposure periods the significant effects for the first-year birth cohort can be found for both originating from poor and rich families. Any recomposing with longer duration of irradiation produces also significant estimate for those exposed between the ages one and five, and its size is stable. Similarly to longer periods, any cut-off earlier than three months after the beginning of the accident does not change results for birth and social groups to any substantial extent comparatively to those obtained in this study. Like for the majority of the models, models with duration of exposure of one or one and a half months (not reported here) produce logged odds, which correspond to the a-priori expectations and significantly differ from zero only for individuals irradiated during first year of life. Taken together, the results for the in-utero group should be treated with some caution, i.e. keeping its sensitivity to duration of the irradiation period in mind. The estimates for those exposed especially after birth and between the ages one and five appear to be robust across different specifications.

5. Conclusion

Consumption of milk was the main source of irradiation for young children in Ukraine after the Chernobyl accident according to numerous reports. Radionuclides were available on the ground and in the topsoil from the beginning of the exposure period on April, 26 until the end of 1986 and had been causing the contamination of grass used for the production of milk throughout the whole country. National authorities introduced limits to consumption of food only in autumn 1986 and put them into practice much later. The availability of cesium radioisotopes in the Ukrainian milk produced in the most contaminated regions highly exceeds the today's radiological norms. Our results show that this dangerous environment on average has not had direct consequences for the adult health of those individuals who were irradiated in early life after the accident. However, their socio-economic status in early life mattered. According to our findings, among those who got irradiated during early life, only individuals from poorer families demonstrate negative effects of exposure in young adulthood. Being born in a less affluent family and exposed to the Chernobyl accident in early childhood is associated with having the probability of 0.5 to report the bad health two decades later. Among these negatively affected individuals, the impact of radiation load is the largest for those who were exposed during infancy. Only for these individuals, family access to resources allowed to lower the probability of negative health outcomes to almost negligible levels.

Our results do not allow to confirm the purely biological hypothesis. The exposure to iodine and cesium isotopes in early life does not have a direct effect on adult health, either for an average or for a low-irradiated individual. Even though the data points to the much higher probability of having bad health in adulthood for those individuals who were exposed to radionuclides during infancy relatively to those conceived after the end of exposure period, the effect is not statistically significant. However, damage from radiation is dose-dependent. Individuals who resided in the most contaminated regions of Ukraine at the time of the accident have two times higher probability of having bad health later in life in comparison to those from less polluted areas. These high doses of iodine or cesium radiation affected all age groups. The direct impact on adult health from high radiation doses was shown to be valid for individuals up to five years old. The two-fold difference in health outcomes between low and high contaminated areas exactly equals the relative radiation doses. Most importantly, the collective effect from high irradiation has not disappeared after the end of the exposure period. The estimated effects for birth cohorts in the model with longer exposure period are much larger, albeit insignificant. The predicted probabilities indicate that bad health can be identified in more than one fourth of all reported cases among individuals assumed to be non-exposed to irradiation. In this regard, our results are consistent with the current literature that provides abundant evidence of strong negative health effects for individuals residing in Northern Ukraine, i.e. the territories that were highly polluted with radioisotopes.

The purely social hypothesis can be confirmed. However, evidence strongly depends on what variable is chosen to measure socio-economic origin. In this study it has been argued that access to higher education reflects socio-economic differences between poor and wealthy groups in Ukraine in the 1980s. Since this distinction can be made on either's parent's education, the use of father's education represents deeper social stratification. Individuals whose mothers did not have at least incomplete higher education have the probability of 0.3 to

have the bad health in adulthood, that is one and a half times higher than that for individuals from families with educated mothers. This collective effect measured with mother's education is robust to the changes in exposure periods. Another group that shows negative signs of poor socio-economic background in adulthood is those individuals who lived with single mothers. However, no effects for social groups based on the father's education are significant. This does not hold for individuals who received high radiation doses in early life. Regardless of the measure of socio-economic origin, individuals born in poor families among those who resided in the most polluted areas, have at least fourfold higher probability of having bad health in young adulthood relatively to the group of originating from wealthy families which lived in low polluted areas. The obtained estimates point to the exponential increase of dose-dependent damage on health from radiation if it is enhanced by poor socio-economic conditions in early life. These socio-economic differences in adult morbidity for mostly irradiated individuals are collective in relation to birth groups as well as in the case of biological effects.

Our findings strongly support the socio-biological hypothesis. The exposure to radiation in early life affected health with regard to socio-economic background of individuals. Dependent on the socio-economic status, the size of the effect from irradiation has been different for birth groups. Our results suggest that individuals originating from families whose mothers did not have higher education and affected with radiation in early life have the probability of 0.4 to have bad health in adulthood. The model with mother's education shows clearly that the long-life effect from exposure to radiation emerges in-utero, peaks during infancy, and gradually decreases after the age of one, among those originated from poor families. According to the predicted probabilities, at least one third of these individuals will report bad health in adulthood. The use of fathers' educational status also allows to produce significant effects, but not for all birth and social groups. Among those whose fathers did not have higher education or live with a family, only individuals who were affected after birth and up to five years indicate negative outcomes in adulthood. Those who lived with single mothers have bad health in one fourth of all cases later in life. Since we expect any effects to be accentuated with the father's education, these results point to sensitivity of the effect for those who were exposed to radiation while in-utero.

The infancy inflammation hypothesis is strongly supported by our results. The effect for the group which was irradiated during the first year of life is stable across different specifications, and the largest among all birth groups. Regardless of the duration of exposure period, one half of these individuals would have bad health in adulthood. The probability of having bad health for individuals born in poor families where mothers did not have higher education and exposed during infancy relatively to the group of non-exposed from wealthy families is four-fold. Those who were also exposed to radiation after birth but lived in wealthier families, where mothers had at least incomplete higher education, report bad health in adulthood in one fifth of cases. The effect is strongly accentuated with father's education as a socio-economic measure. In the model with longer exposure period, the effect for those originated from poor families and irradiated during infancy increases twelve-fold, relatively to the non-exposed individuals from wealthy families. According to the predicted probabilities, only ten percent of them while born in wealthier families report bad health later in life. This is the lowest threshold of the effect in models for all birth groups with varying exposure. As it has been

expected, measure of the father's education accentuates the effect for those exposed during infancy as the longer exposure period does.

Our results for birth and social cohorts are estimated for individuals who were irradiated at low doses. Doses received by the population in low contaminated regions of Ukraine are nearly identical to those regions in other European countries affected by the Chernobyl radiation. In the current study this dose averages to 0.85 millisievert for the low-polluted areas, and for the whole Ukraine it equals to 0.98 on average. In the official sources, the dosage lower than 1 millisievert is assumed to be harmless for health (World Nuclear Association, 2011; Baloga et al, 2011). In this regard, our results constitute the concern for the public and academia. Our findings show that radiation in early childhood even at lower doses leads to severe long-life health damage if we consider social differentiation in the society. In poor families, children during early childhood up to five years old demonstrate high vulnerability to radiation, and especially infants are at the highest risk. It is also likely that this environmental vulnerability is formed in-utero. Wealthier families are able to eliminate this negative effect for infants to a considerable extent, and for the other birth groups this possibility is almost negligible.

The picture provided with our findings is that children during infancy can be permanently damaged, but this damage can be remediated. This has important implications for the timing of policy interventions. According to our findings, any preventive or ameliorative measures should be targeted to infants and women of child bearing age. The cohort designs in our study clearly show that insults and compensations are likely to be more effective during infancy than during the fetal period or between the ages one and five. It also establishes that exogenous shock from radionuclides causes severe damage that is already apparent in young adulthood, and presumably has lasted since early childhood, but this damage may be ameliorated by distributing additional resources to the affected infants. Given the fact that irradiation occurred mainly through the consumption of food and was strengthened by processes of normal metabolism in child bodies, any social program should be primarily directed to provision of better nutrients. It is likely that wealthy families through food of higher quality, that was rich in microelements, were able to alleviate the negative consequences of exposure. It is also possible that necessary knowledge and other resources allowed parents to process safety information and undertake some preventive measures, primarily through self-restriction in consumption of irradiated food. Therefore, policy measures of provision of quality childcare or dissemination of information about radiation and health behavior might be efficient.

The compensatory measures should be prescribed after any irradiation of newborn children. In the current conditions, the exposure to radiation may be caused naturally by the nuclear accidents (UNSCEAR, 2008b). The nuclear fallout at the power station in Fukushima due to the earthquake in 2011, contaminated some Japanese regions at levels identical to the Chernobyl's doses, is one of the recent episodes (WHO, 2012). In such environment, proper behavior during irradiation appears to be necessary to prevent the negative long-life consequences of exposure in early childhood. When poor families are incapable to provide the child with proper treatment and nutrition, local government initiatives aiming to supply the nutrients or medicine should be encouraged. In addition to comprising the natural radiation exposure, the use of radiation in some civil purposes causes inflammation at doses which are nearly equals to those estimated in this study as dangerous for infants. All experiences of

irradiation at similar levels, such as the use of radon in ventilation of basements, irradiation coming from medical scans or long-distance flights, should be avoided as irreversibly harmful for children in early life. If undertaken, post-incident immediate treatment is vital to eliminate the harm from irradiation. When this treatment is unlikely to stop entirely the generation of inflammatory processes in child bodies, it may at least clean the tissues and organs from biologically storable radionuclides which cause persistent deterioration while decay.

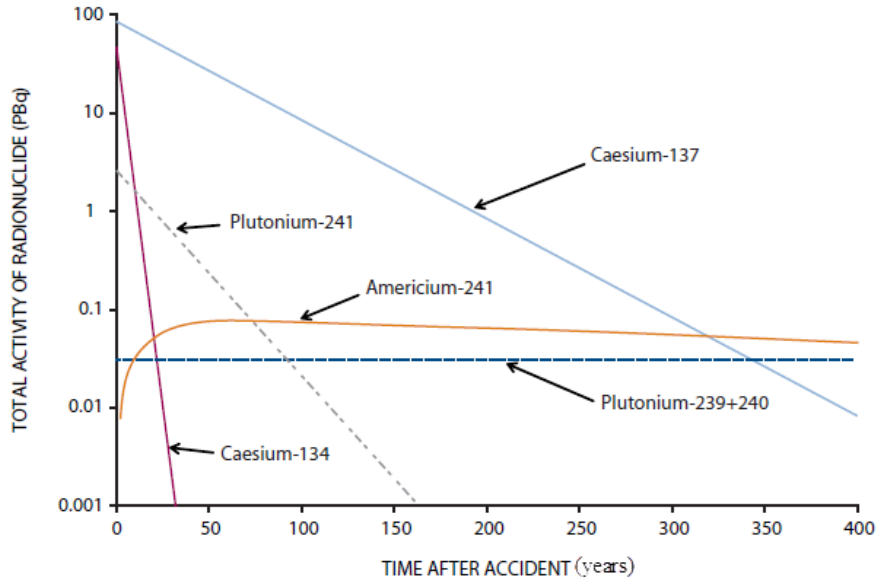
Our study stresses that any scars from irradiation in early childhood are persisting and irreversible with later experiences. The context information reinforces this finding of the current study with the following fact. Since 1991, numerous national programmes have been launched in Ukraine targeting those individuals who were exposed to Chernobyl radionuclides in early life (Baloga et al, 2011). These programmes covered all child cohorts exposed to radiation throughout Ukraine. The compensatory measures included thorough clinical examination of health, rehabilitation, and the release of vitaminized foodstuffs and pills for treatment and prophylaxis. The children have been followed up with this social care during late childhood and adolescence up to their maturity. Our results therefore are large and significant despite these compensatory measures undertaken beyond the critical period. Notwithstanding the treatment measures launched since late childhood, individuals who were irradiated especially during infancy and up to five years and lived in poor families appear to carry along damage from early-life events throughout life course. Our evidence is pessimistic for these individuals. However, it points promisingly to the necessity of particular attention to infants in the Ukrainian regions which still suffer from radiation.

The damage from non-infectious inflammogens in the form of radionuclides caused after birth and up to five years should be considered as wholly or partially irreversible similarly to the long-life impact of infectious diseases. Our study provides additional evidence on the infancy inflammation hypothesis thereby challenging the fetal origins paradigm. At least for exposure from radionuclides in cope with poor socio-economic environment, the period during infancy appears to be the crucial critical period. Our findings not only show clearly that this period exists, but also that it is limited to the first year of life. Either fetal stage or early childhood between the ages one and five are likely to represent sensitive periods which do not leave irreversible damage on health and can be modified by future conditions. Moreover, our results show that the period after the first year up to five years is relatively more vulnerable than in-utero. In this regard, our findings are in unison with those provided by literature on infectious diseases. Any inflammation-related events which are aggravated by severe socio-economic environment appear to cause persistent damage on health only during infancy and at much lesser levels during the rest of early childhood. The possibility to modify this damage to any considerable extent by economic resources exists only during infancy.

Appendices

Appendix A

Total amounts in the environment of various long-lived radionuclides released as a function of time after the Chernobyl accident*



Source: UNSCEAR, 2008a

*Cesium isotopes were available throughout the whole Ukraine until 1987, and then through the deep ground moved to the Northern regions. Americium and plutonium are deposited within a Chernobyl evacuation zone.

Appendix B

An extended Grossmann model with critical periods

Let us consider production of health capital *h* during a two-period early life.

$$h = A[\gamma I_1 + (1-\gamma) I_2], \quad (1)$$

where I_1 – investments during early life, before age 5, I_2 – investment during early life, after age 5, A – a productivity shift factor.

From the model (1), at the given level of total investments during early life $I_1 + I_2$, the allocation of child's investments between two periods will affect the level of human capital *h* for $\gamma \neq 0.5$. If $\gamma > 0.5$, then health at the end of period 1 is more important to *h* than investments in the period 2, and if $A > 1$, *h* may increase faster than I_1. Thus, model (1) allows the possibility of critical periods, namely that particular periods in early life may cause disproportional effects on adult outcomes which do not necessarily decline monotonically with age.

The model 1 implies perfect substitutability of investments between period 1 and 2. This means that as regards a depreciation rate, all child's investments should be concentrated in a period where the return is highest.

A more flexible human capital production technology is the function with constant elasticity of substitution:

$$h = A[\gamma I_1^{\phi} + (1-\gamma) I_2^{\phi}]^{1/\phi}, \quad (2)$$

In this model, the effect on human capital from the allocation of investments between period 1 and 2 is dependent also on the elasticity of substitution, $1/(1-\phi)$, and the share parameter, γ. For perfect substitutability of child's investments $\phi=1$, and model (2) reduces to model (1).

Let us consider the effect of exogenous shock μ_g to health investments that occur during the first childhood period. In this case, μ_g is negative.

For a fixed investments model, we assume that investments in the second childhood period do not respond to μ_g. Then, investments in period 1, during early life before the age 5, are: $I_1 + \mu_g$.

In the model 2, the impact of an early life environmental shock on adult health outcomes is as follows:

$$\frac{\delta h}{\delta \mu_g} = \gamma A\left[\gamma (I_1 + \mu_g)^{\phi} + (1-\gamma) I_2^{\phi}\right]^{(1-\phi)/\phi} (I_1 + \mu_g)^{\phi-1}. \quad (3)$$

For the model with the perfect substitutability (model 1), we have: $\frac{\delta h}{\delta \mu_g} = \gamma A$.

Therefore, damage to adult human capital is proportional to the share parameter on period 1 investments, and is unrelated to the investment level I_1.

For imperfect substitutability between periods (model 2), child's investments have diminishing returns. Thus, exogenous shocks experienced at different baseline investment levels have different effects on *h*.

In the model with responsive investments, we should assume that parents observe μ_g at the end of the period 1.

For the case of perfect substitutability between different early life periods, we should consider parental investments in child's human capital. Let us assume parent's utility U_p trades off their own consumption C against the child human capital h:

$$U_p = U(C, h)$$

Parents have budget constraints:

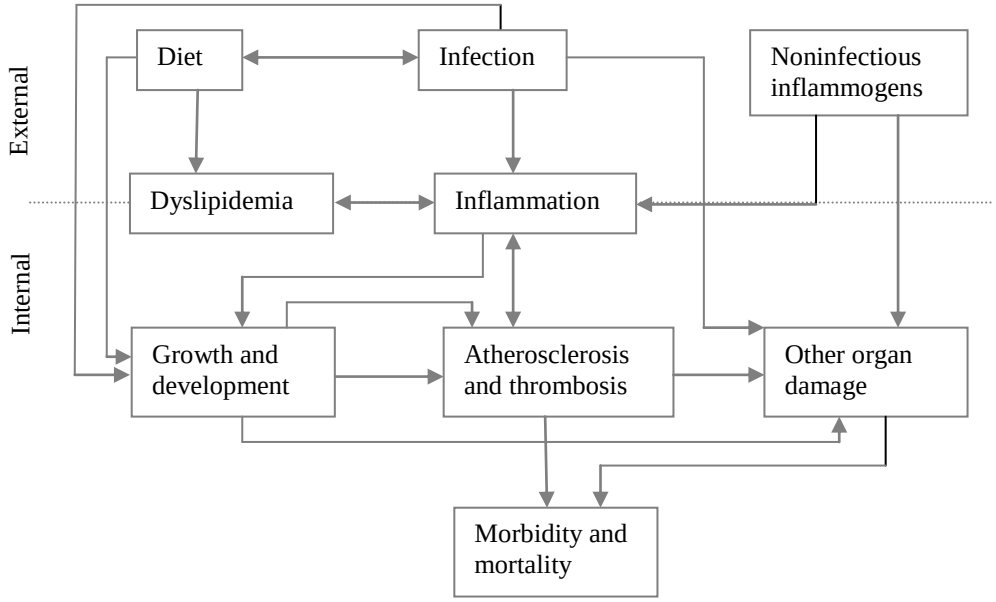
$$Y_p = C + I_1 + I_2 /(1+r)$$

The amelioration happens in the model at the cost of reduced parental utility. With the negative exogenous shock μ_g, the marginal utility of h becomes too high relatively to that in consumption C. Since the human capital production technology in (1) permits parents to convert some consumption into h at a constant rate, child's investments in a period 2 I_2 have to increase. This mediates the effect of the exogenous shock μ_g in the second childhood period.

Source: adapted from Almond & Currie (2011a)

Appendix C

A critical period model with inflammation as a central mechanism



Source: Finch & Crimmins (2006)

Appendix D

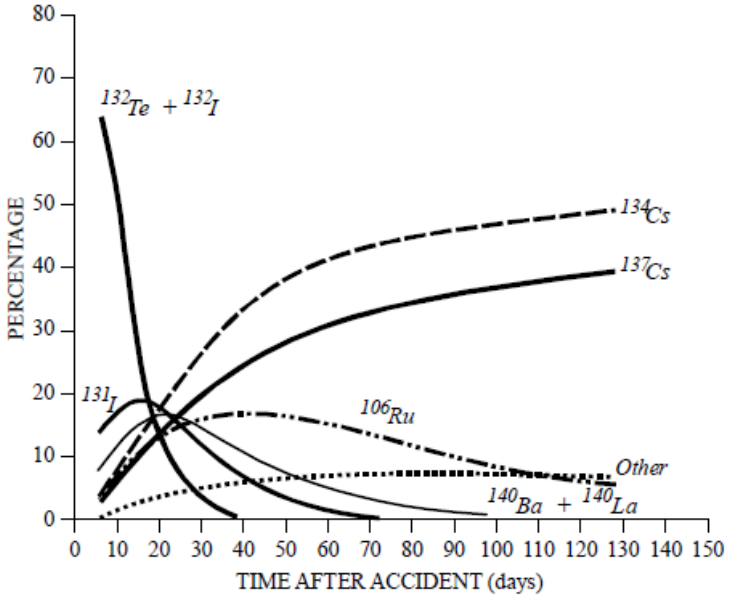
Chemical characteristics and physical activity of radionuclides released as a result of the Chernobyl accident

Radionuclide	Duration of half-life	Emission activity, PBq	
		26 April 1986	26 April 2006
Rare gases			
85Kr	10.72 years	33	9.058
133Xe	5.25 days	6500	0.000
Volatile elements			
129Te	33.6 days	240	0.000
132Te	3.26 days	1150	0.000
131I	**8.04 days**	**1760**	**0.000**
133I	0.81 days	2500	0.000
134Cs	**2.06 years**	**54**	**0.065**
136Cs	13.1 days	36	0.000
137Cs	**30.0 years**	**85**	**53.550**
Intermediate volatility elements			
89Sr	50.5 days	115	0.000
90Sr	29.12 years	10	6.210
103Ru	39.3 days	168	0.000
106Ru	1.01 years	73	0.000
140Ba	12.7 days	240	0.000
Heavy volatile elements			
95Zr	64.0 days	196	0.000
99Mo	2.75 days	168	0.000
141Ce	32.5 days	196	0.000
144Ce	0.78 years	116	0.000
239Np	2.35 days	400	0.000
238Pu	87.74 years	0.035	0.030
240Pu	6537 years	0.042	0.042
241Pu	14.4 years	6	2.292
242Pu	376000 years	0.001	0.001
242Cm	18.1 years	0.9	0.419
Total contamination		13936	72

Source: UNSCEAR (2008a)

Appendix E

Contributions of radionuclides to the absorbed external dose rate in some contaminated areas of the USSR*



Source: UNSCEAR (2000)

*Provided as an example of the irradiation pattern. For Ukrainian SSR, through both internal and external sources, iodine isotopes contributed largely to radiation doses during the first few months after the accident (up to 70 per cent). Cesium radioisotopes became the largest contributors later on (up to 90 per cent). Radiation dose has been decreasing over the period.

Appendix F

Examples of concealment of data on the consequences of Chernobyl accident by chief officers

1. " The data on patients' records related to the accident and collected in medical institutions should have a *limited access* status. And data collected in regional and municipal sanitary control institutions, … on radioactive contamination of objects, environment (including food) that exceeds maximum permissible concentration is *classified*." [From Order # 30-S by Minister of Health of Ukraine A. Romanenko on May 18, 1986]

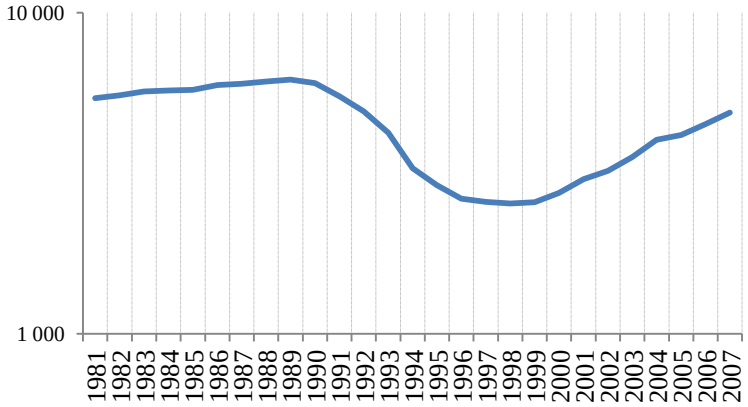
2. " To *classify* information on the accident… To *classify* information on results of medical treatment. To *classify* information on the degree of radioactive effects on the personnel who participated in the elimination of the consequences of the Chernobyl accident." [From the order by the Chief of Third Main Administration of the USSR's Ministry of Health E. Shulzhenko, #U-2617-S, June 27, 1986]

Source: Yablokov et al, 2009, p.55

Appendix G

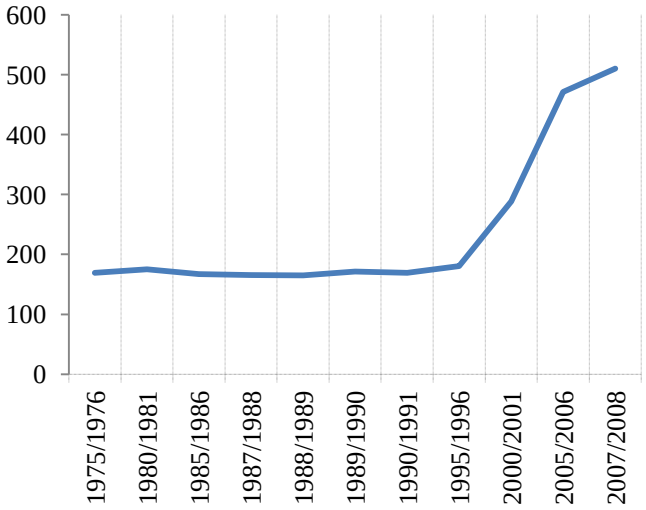
Potential confounders of the adult health outcomes in Ukraine

a) GDP per capita in Ukraine in 1981-2007, constant 1990 int. GK$, log values



Source: data adapted from Maddisson project database (2013)

b) Enrollment in tertiary education in Ukraine in 1975-2007, persons per 10000 of total population



Source: data adapted from National economy USSR (1991); Statistical yearbook of Ukraine (2009)

Appendix H

Descriptive statistics of the variables

Variable	Full sample		Mother's education high		Mother's education low		Father's education high		Father's education low		Father's education unknown	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Birth year	1985.274	2.417	1985.589	2.376	1985.204	2.422	1985.476	2.368	1985.195	2.411	1985.52	2.507
Age at observation	21.726	2.417	21.411	2.376	21.796	2.422	21.524	2.368	21.805	2.411	21.480	2.507
Health at observation, self-reported	0.357	0.480	0.287	0.454	0.373	0.484	0.355	0.480	0.348	0.477	0.417	0.495
Radiation exposure between the ages 1-5	0.431	0.495	0.381	0.487	0.442	0.497	0.404	0.492	0.441	0.497	0.402	0.492
Radiation exposure during infancy	0.118	0.323	0.079	0.271	0.127	0.333	0.096	0.296	0.121	0.327	0.126	0.333
Radiation exposure during infancy and in-utero (first approach)	0.085	0.279	0.114	0.318	0.078	0.269	0.078	0.270	0.092	0.289	0.047	0.213
Radiation exposure in-utero (first approach)	0.106	0.309	0.124	0.330	0.103	0.304	0.127	0.333	0.102	0.303	0.110	0.314
Conceived after the end of radiation exposure period (first approach)	0.260	0.439	0.302	0.460	0.250	0.433	0.295	0.458	0.244	0.430	0.315	0.466
Radiation exposure during infancy and in-utero (second approach)	0.035	0.184	0.069	0.255	0.028	0.164	0.042	0.202	0.033	0.179	0.039	0.195
Radiation exposure in-utero (second approach)	0.098	0.298	0.089	0.286	0.100	0.301	0.084	0.279	0.110	0.313	0.039	0.195
Conceived after the end of radiation exposure period (second approach)	0.317	0.466	0.381	0.486	0.303	0.460	0.374	0.485	0.294	0.456	0.394	0.491
Mother's education high	0.182	0.386	1.000	0.000	0.000	0.000	0.584	0.494	0.112	0.315	0.110	0.314
Mother's education low	0.818	0.386	0.000	0.000	1.000	0.000	0.416	0.494	0.889	0.315	0.890	0.314
Father's education high	0.150	0.357	0.480	0.501	0.076	0.265	1.000	0.000	0.000	0.000	0.000	0.000
Father's education low	0.736	0.441	0.451	0.499	0.799	0.401	0.000	0.000	1.000	0.000	0.000	0.000
Father's education unknown	0.115	0.319	0.069	0.255	0.125	0.330	0.000	0.000	0.000	0.000	1.000	0.000
Radiation dose at place of birth low	0.770	0.421	0.738	0.441	0.777	0.416	0.711	0.455	0.786	0.411	0.748	0.436
Radiation dose at place of birth high	0.110	0.313	0.099	0.299	0.113	0.316	0.054	0.227	0.126	0.332	0.079	0.270
Radiation dose at place of birth unknown	0.120	0.325	0.163	0.371	0.110	0.313	0.235	0.425	0.088	0.284	0.173	0.380

Male	0.479	0.500	0.485	0.501	0.474	0.500	0.506	0.502	0.479	0.500	0.441	0.499
Female	0.521	0.500	0.515	0.501	0.523	0.500	0.494	0.502	0.521	0.500	0.560	0.499
Urban place of birth	0.612	0.488	0.663	0.474	0.601	0.490	0.711	0.455	0.583	0.493	0.669	0.472
Rural place of birth	0.339	0.474	0.267	0.444	0.355	0.479	0.193	0.396	0.381	0.486	0.260	0.440
Type of place of birth unknown	0.049	0.215	0.069	0.255	0.044	0.205	0.096	0.296	0.036	0.185	0.071	0.258
Resettled in 1986	0.003	0.052	0.005	0.070	0.002	0.047	0.006	0.078	0.001	0.035	0.008	0.089
Not resettled in 1986	0.527	0.500	0.436	0.497	0.547	0.498	0.482	0.501	0.543	0.499	0.480	0.502
Resettlement information unknown	0.471	0.499	0.559	0.498	0.451	0.499	0.512	0.501	0.456	0.498	0.512	0.502
N	1109		202		907		166		816		127	

Appendix I

Recoded variables

Variable	
Health at observation, self-reported	=1 if individual has bad health, 0=otherwise. recoded from the original 5-point scale self-estimation. The respondents were asked 'how would you evaluate your health according to the 5-grade scale?' *Very Bad*, *Bad*, and *Average*, *not good*, *but not bad* =1 or Bad health; *Good* and *Very Good* =0 or Good Health
Birth year	continuous, gathered from a dataset
Age at observation	continuous, calculated on a basis of the birth information
Radiation exposure between the ages 1-5	=1 if exposed between the ages 1-5 at the exposure period, 0=otherwise
Radiation exposure during infancy	=1 if exposed during infancy at the exposure period, 0=otherwise
Radiation exposure during infancy and in-utero	=1 if exposed during infancy and in-utero at the exposure period, 0=otherwise
Radiation exposure in-utero	=1 if exposed in-utero at the exposure period, 0=otherwise
Conceived after the end of radiation exposure period	reference group
Mother's education high	reference group, mother has a higher, incomplete higher education, or a candidate/doctorate degree. Recoded from the multipoint scale; original values include: no formal education, basic compulsory education (grades 1-6), compulsory education (grades 7-9), incomplete general secondary education (grades 10-11 without diploma), completed general secondary education (grades 9-10 with diploma), vocational elementary education, vocational secondary education, professional secondary education, incomplete professional higher education (at least three years), complete professional higher education, candidate or doctor of sciences, information is unknown, and mother not living with a respondent.
Mother's education low	=1 if mother has less than incomplete higher education, 0=otherwise
Father's education high	reference group, father has a higher, incomplete higher education, or a candidate/doctorate degree. Recoded from the multipoint scale; original values include: no formal education, basic compulsory education (grades 1-6), compulsory education (grades 7-9), incomplete general secondary education (grades 10-11 without diploma), completed general secondary education (grades 9-10 with diploma), vocational elementary education, vocational secondary education, professional secondary education, incomplete professional higher education (at least three years), complete professional higher education, candidate or doctor of sciences, information is unknown, and father not living with a respondent.
Father's education low	1 if father has less than incomplete higher education, 0=otherwise
Father's education unknown	1 if father was not living with an individual (n=98) or father's education unknown (n=29), 0=otherwise

Male	=1 if male, 0=otherwise
Female	reference group
Urban place of birth	reference group
Rural place of birth	=1 if type of place of birth is urban, 0=otherwise
Type of place of birth unknown	=1 if type of place of birth is unknown, 0=otherwise
Not resettled in 1986	reference group
Resettled in 1986	=1 if individuals changed place of residence after the Chernobyl accident in 1986, 0=otherwise
Resettlement information unknown	=1 if resettlement information on 1986 is unknown, 0=otherwise
Radiation dose at place of birth low	reference group
Radiation dose at place of birth high	=1 if place of birth coincides with the most contaminated regions, 0=otherwise
Radiation dose at place of birth unknown	=1 if information on place of birth is unknown, 0=otherwise
Abnormal bmi	=1 if bmi <18.6 or >25.0, 0=otherwise bmi is calculated as bmi=body height in kg/squared body weight in sqm
Any health problems last 3 months	=1 if individual has had any health problems in the last three months, 0=otherwise
Any health problems that limit amount of work/studies	=1 if individual has any impairment or health problem that limits the kind or amount of work he/she can do, 0=otherwise
Any chronic diseases	=1 if individual has any chronic illnesses, such as heart disease, illness of the lungs, liver disease, kidney disease, gastrointestinal disease, spinal problems, other chronic illnesses; 0=otherwise
Any listed diseases	=1 if individual has suffered from any listed illnesses, such as diabetes, myocardial infarction, high blood pressure, stroke, anemia, and tuberculosis; 0=otherwise

Appendix J

Distribution of individuals from different self-reported health groups across groups based on other health variables

Health at observation, self-reported		Bmi group			Any health problems last 3 months			Any health problems that limit work or studies			Any chronic diseases		Suffered from any of listed diseases		Total
		missing	normal, 18.6-25	abnormal, >18.6 and <25	missing	yes	no	missing	yes	no	yes	no	yes	no	
Very good	N	4	51	16	1	5	65	1	1	69	3	68	3	68	71
	% within group	5,63	71,83	22,54	1,41	7,04	91,55	1,41	1,41	97,18	4,23	95,77	4,23	95,77	100,00
	% in total	10,00	6,61	5,37	14,29	2,46	7,23	1,89	0,91	7,29	1,15	8,02	5,77	6,43	6,40
Good	N	25	467	150	5	44	593	22	13	607	47	595	8	634	642
	% within group	3,89	72,74	23,37	0,78	6,85	92,37	3,43	2,02	94,55	7,32	92,68	1,25	98,75	100,00
	% in total	62,50	60,57	50,34	71,43	21,67	65,96	41,51	11,82	64,16	18,01	70,17	15,38	59,98	57,89
Average, not good, but not bad	N	9	232	118	1	128	230	26	75	258	184	175	36	323	359
	% within group	2,51	64,62	32,87	0,28	35,65	64,07	7,24	20,89	71,87	51,25	48,75	10,03	89,97	100,00
	% in total	22,50	30,09	39,60	14,29	63,05	25,58	49,06	68,18	27,27	70,50	20,64	69,23	30,56	32,37
Bad	N	1	18	13	0	21	11	4	16	12	23	9	3	29	32
	% within group	3,13	56,25	40,63	0,00	65,63	34,38	12,50	50,00	37,50	71,88	28,13	9,38	90,63	100,00
	% in total	2,50	2,33	4,36	0,00	10,34	1,22	7,55	14,55	1,27	8,81	1,06	5,77	2,74	2,89
Very bad	N	1	3	1	0	5	0	0	5	0	4	1	2	3	5
	% within group	20,00	60,00	20,00	0,00	100,00	0,00	0,00	100,00	0,00	80,00	20,00	40,00	60,00	100,00
	% in total	2,50	0,39	0,34	0,00	2,46	0,00	0,00	4,55	0,00	1,53	0,12	3,85	0,28	0,45
Total	N	40	771	298	7	203	899	53	110	946	261	848	52	1	1109
	% within group	3,61	69,52	26,87	0,63	18,30	81,06	4,78	9,92	85,30	23,53	76,47	4,69	95,31	100,00
	% in total	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00	100,00

Appendix K

Pairwise correlations between recoded self-reported health and other health variables[†]

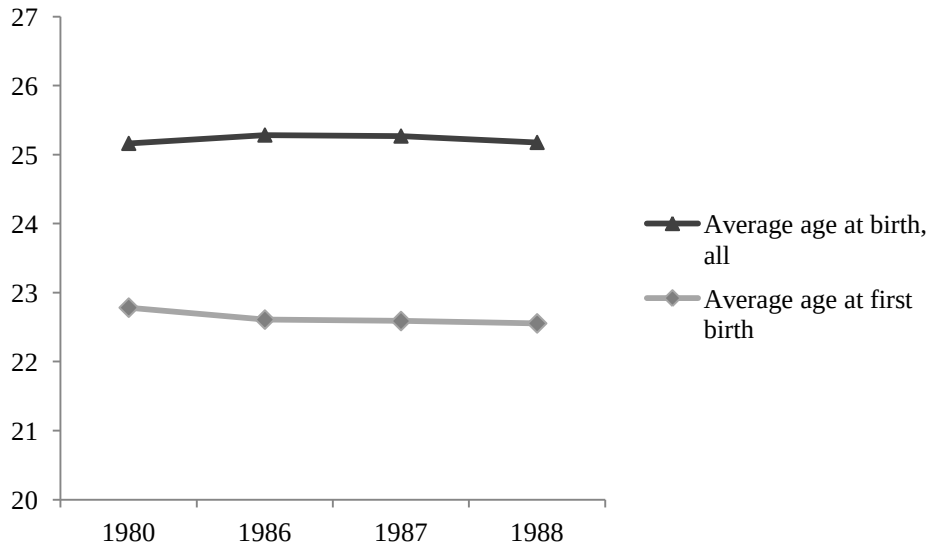
	health, self-reported	abnormal bmi	any health problems last 3 months	any health problems that limit amount of work/studies	any chronic diseases	any listed diseases
health, self-reported	1.000					
abnormal bmi	0.107***	1.000				
any health problems last 3 months	0.397***	0.010	1.000			
any health problems that limit amount of work/studies	0.377***	-0.0003	0.233***	1.000		
any chronic diseases	0.523***	0.031	0.373***	0.272***	1.000	
any listed diseases	0.200***	0.047	0.177***	0.164***	0.249***	1.000

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

† all variables are binary, see Appendix F for definition

Appendix L

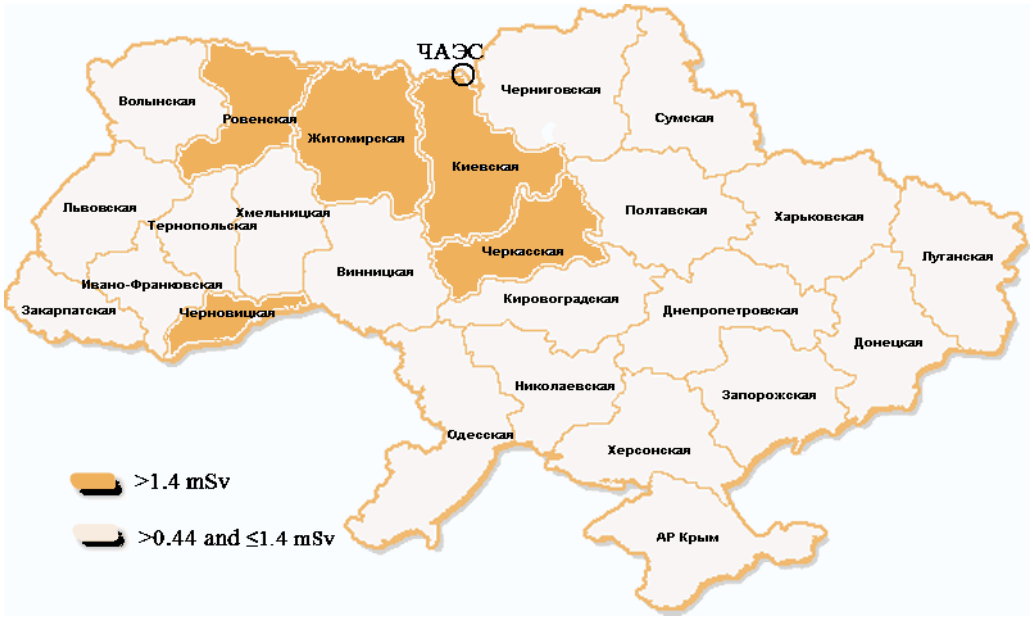
Average age of female at birth in total and at first birth, in Ukraine in 1980-1988



Source: data adapted from Population USSR (1989)

Appendix M

Distribution of the radiation contamination in Ukraine due to the Chernobyl accident in 1986, measured with the average total effective dose (iodine and cesium isotopes)



Source: data adapted from Baloga et al. (2006)

Appendix N

Distribution of individuals treated as *Chernobyl victim* in a sample*, %

Variable	N	Personally receiving a Chernobyl assistance	Not receiving a Chernobyl assistance
Radiation exposure between the ages 1-5	478	0,42	99,58
Radiation exposure during infancy	131	1,53	98,47
Radiation exposure during infancy and in-utero	94	0,00	100,00
Radiation exposure in-utero	118	0,00	100,00
Conceived after the end of radiation exposure period	288	0,35	99,65
Total number	1109	5	1104

*None of the individuals in a sample was evacuated from the 30-km zone of contamination.

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