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MORTALITY AND ECONOMIC FLUCTUATIONS

Theories and empirical results

from Spain and Sweden

by

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ABSTRACT. This dissertation examines the short-term impact of economic fluctuations or "business cycles" on mortality rates. The first chapter is a review of the historical development of ideas regarding the influence of economic conditions on death rates, from the early Malthusian controversies to the modern discovery of the association of economic expansions with spurts of mortality. The second chapter analyzes data from the 50 Spanish provinces in the years 1980-1997. Annual death rates for all causes and sex- and cause-specific deaths rates for particular causes are modeled as functions of the demographic structure of the population and indices of economic conditions at the province and national level, in fixed-effect regression models with the variables in levels, in first differences or in rates of change, and also in specifications with economic indicators lagged up to four years. The results reveal an increase of the rates of death due to the major causes (cardiovascular disease, cancer, injuries, respiratory diseases) coinciding with economic expansions (i.e., a procyclical fluctuation of general mortality) and a countercyclical oscillation of death rates due to suicide. The third chapter is a study of

the relationship between fluctuations of the economic activity and mortality oscillations in Sweden in the last two centuries. The relationship is assessed with spectral analysis and other techniques applied to a variety of economic indicators and death rates. The results show an intense relationship, specially strong in the mid decades of the 19th century, between oscillations of death rates and the quality of the harvest in the preceding year. In the last quarter of the 19th century a procyclical oscillation of mortality emerges which persists throughout the 20th century, though considerably weakened in the second half of the century. These empirical results from Sweden and Spain confirm the procyclical fluctuation of death rates found in previous research by Thomas in the 1920s, Eyer and Higgs in the 1970s and Ruhm recently. No evidence is found in favor of Brenner's hypothesis that attributes the increase of mortality during times of economic expansion to lagged effects of economic slowdown 10 to 15 years before.

When different fields of inquiry have been separately cultivated for a while, the borderland between them often provide fertile ground for new investigations.

Allyn A. Young, 1924

Analyzing business cycles mean neither more nor less than analyzing the economic process of the capitalist era. Most of us discover this truth which at once reveals the nature of the task and also its formidable dimensions. Cycles are not, like tonsils, separable things that might be treated by themselves, but are, like the beat of the heart, of the essence of the organism that display them.

Joseph A. Schumpeter, 1939

Here we come upon the greatest of all economic questions, but one that in fact is never asked: what is growth for? (...)Do we want renewed growth in order to maintain and enhance disparities in consumption? Have we not become disillusioned with the doctrine that 'disease, squalor and ignorance' will soon be cleared away by the 'trickle down' from ever-growing conspicuous consumption?

Secondly, many kinds of consumption that are chosen by some individuals generate *disutility* for others. The leading case is the spread of motor car—the higher the level of consumption, the more uncomfortable life becomes; this fact is painfully obvious but orthodox doctrine has not been able to accommodate it.

Joan Robinson, 1980

Work drives a wedge of paradox into the core of modern life. At times the indispensable adhesive binding a fragile life together, at other times, like a caustic acid, work corrodes interpersonal relations and poisons the wells of identity, motivation and self-worth. If it is often less extreme than this in its impact on individual experience, work is seldom any less central an element in the human condition. As many struggle simply to tolerate a day at a time in menial, routine, and dehumanizing jobs, nearly all workers believe at some level that work ought to be stamped with deeper meaning.

Diana C. Walsh, 1987

This dissertation is dedicated to the memory of Dorothy Swaine Thomas, who in the 1920s discovered the procyclical fluctuation of mortality with respect to business cycles; and to Joe Eyer, who fifty years later understood the whole business.

PREFACE

I became interested in the relationship between economic fluctuations and death rates when I saw Eyer's work cited in Hernán San Martín's *Economía de la salud*. I read Eyer's work in the early nineties, when I was working for the Pan-American Health Organization (PAHO/WHO). At that time I tried to contact Eyer, but I never was able to discover about his whereabouts, nobody knew where he was. He did not publish anything after 1984.

When in the late nineties I began looking for a theme for my dissertation I returned to the issue of business cycles and mortality. I thought it could be a good theme for a dissertation bridging the economic and public health fields. Duncan Foley gave me a push for doing it when I showed him a two-pages outline.

Just when I was beginning to do some preliminary research I found LaPlante's dissertation on the same issue and Ruhm's paper on mortality and business cycles in the US states. At first I thought that LaPlante's dissertation and Ruhm's paper did not leave space for my thesis. It seems it was not so. In fact, on one hand LaPlante's conclusions

were basically disproved by Ruhm's results; on the other hand, Ruhm's work gave me ideas on how to proceed with my own research, while his results, showing once again the procyclical oscillation of mortality, encouraged me and gave me some strength against skeptical views.

Finally I managed to write the three papers that form this dissertation. They are quite different, but the three of them address directly the issue I wanted to study, that of the relationship between short-term fluctuations in death rates and "cycles" of expansion-contraction of the economy. While the first paper (chapter 1) is basically a review of the literature on the issue, the other two (chapters 2 and 3) are empirical papers, one about Spain in recent years, the other about Sweden during the past two centuries.

In the first chapter I develop the basic ideas of Eyer's model of stress-mediated mortality fluctuations (figure P-1, page *vii*). I give more details about that complex model in the discussion of my results from Spanish data. However there was no place in this dissertation to discuss the controversial issue of unemployment-mortality. Valkonen and Martikainen's contributions to it have been substantial, but I think the issue is not totally closed.

Results from Scandinavian countries might not be applicable to other industrial economies, not to mention nations in the Third World. I think this is an open field for research.

I hope the material in this dissertation will find an outlet for publication and be a stimulus for other people to continue investigating the ground that Dorothy Thomas plowed in her *Social aspects of the business cycle*. Economic fluctuations have a strong impact on many aspects of social life. Or perhaps it would be better to say that economic fluctuations express themselves in multiple ways, from crime rates to exercising, from investment to fertility and nupciality. Schumpeter was right, business cycles are like the beat of the heart of the organism that displays them; or, we could say, like the heart beat of the beast.

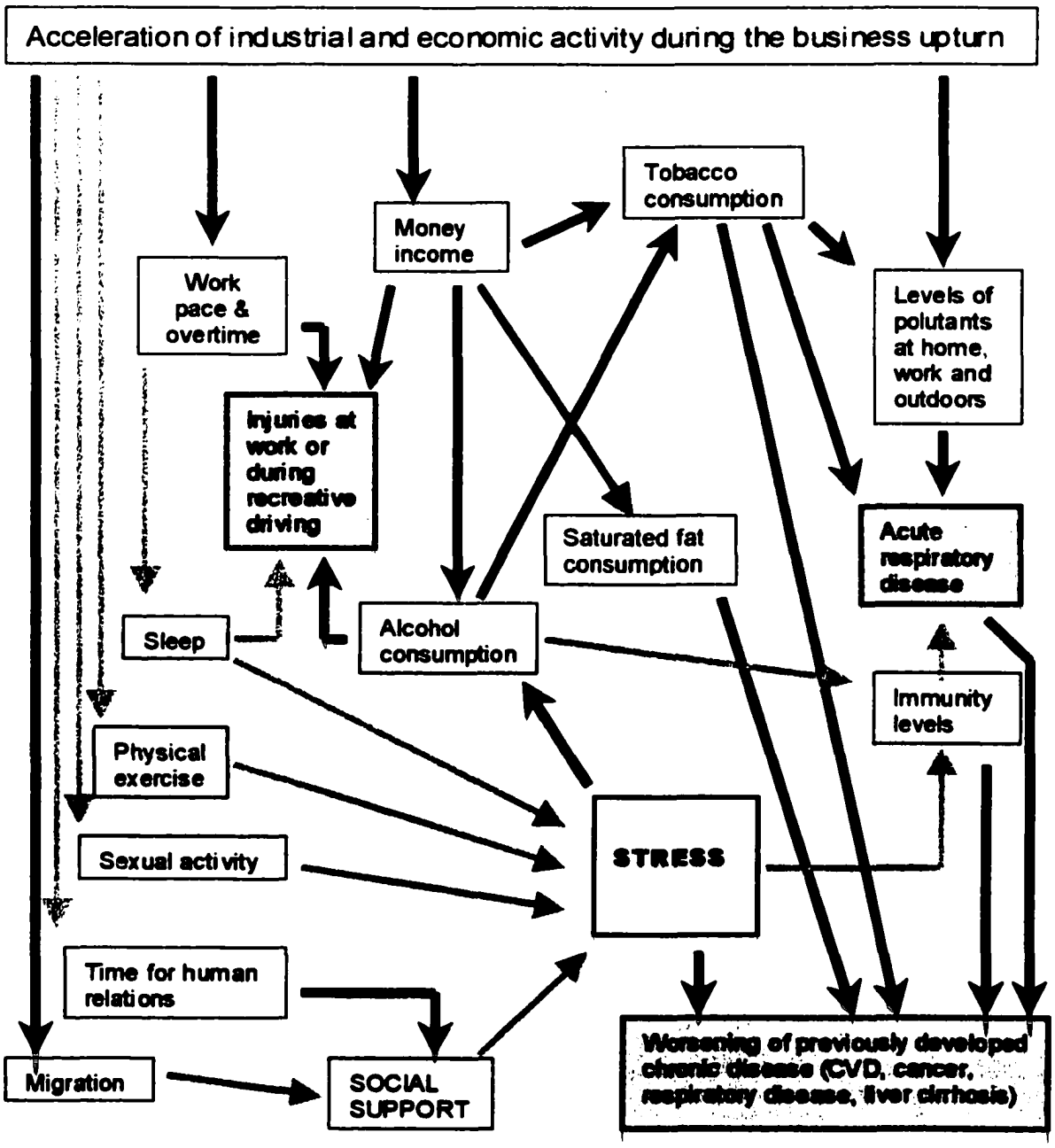

Figure P-1. Causal pathways in Eyer's stress-mediated model of procyclical mortality. Black thick arrows and thin gray arrows represent positive and negative effects, respectively. For instance, a business upturn increases work pace and overtime which in turn tend to reduce sleep, which raises both injury risk and stress levels, etc. For many of these pathways there is strong empirical evidence. The three shaded rectangles represent the final steps leading to death—through worsening of a chronic condition, a new acute disease or an injury. Some direct links (e.g. stress increasing tobacco consumption) and bi-directional pathways that are known (for instance alcohol consumption increasing tobacco consumption *and vice versa*) have been omitted to simplify the scheme.

Acknowledgements

I would have never written this dissertation or studied economics, if not because of my wife, Ana Diez Roux. During the writing of this dissertation she provided all kind of support—statistical, affective, culinary. She helped me with SAS, with the analysis, editing most of this manuscript, and, moreover, she put up with me all these years. She must be the first in the acknowledgements section. Duncan Foley encouraged me to write this dissertation from the first time I showed him an outline. Both him and Eugene Canjels provided good suggestions for the analysis and criticisms on drafts of the chapters. Guido de Marco helped me with very practical ideas to process with Excel hundreds of downloaded files, and Friedrich Huebler was crucial in teaching me some procedures for automatic downloading of data that saved me $3400. He helped me to overcome, though only temporarily, *la tacañería institucional* of the Spanish Instituto Nacional de Estadística, that made my work difficult, in spite of the fact that I was trying to use data that are public. Olle Krantz was kind enough to provide valuable

data to an unknown person (me) who got his email from Statistics Sweden. To both the person and the institution I am extremely grateful. Christopher Ruhm made some useful comments on my results on Spain and Naomi Sager helped me with the editing of chapter 1. My thanks to all of them.

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CHAPTER 1

FROM POLITICAL ARITHMETIC AND POLITICAL ECONOMY TO DEMOGRAPHY AND EPIDEMIOLOGY: IDEAS ON THE IMPACT OF ECONOMIC ACTIVITY ON DEATH RATES IN THE HISTORY OF SOCIAL SCIENCE

The bi-directional relationship between the economic activities of a society and the health of its people is presently a field of study for economic historians, epidemiologists, demographers and economists. This chapter examines the historical development of the ideas about the influence of economic conditions on death rates, from the early Malthusian controversies and the social medicine school to the modern debates on McKeown's nutritional determinism and the repeated discovery of the association of economic expansions with spurts of mortality.

I — From Petty's political arithmetic to the Malthusian controversies and the sanitary ideas of Engels and Marx

The relationship between the health of the population and the production and consumption activities of the society is an old concern. In ancient Greece, Hippocrates, when discussing the impact of air, water and living place on the health of the inhabitants of a town referred to "whether they are heavy drinkers, taking lunch, and inactive, or athletic, industrious, eating much and drinking little." In the modern age the origins of social science are clearly linked to the consideration of population phenomena and economic activities, and sometimes the relationship between them. Indeed, William Petty (1623-1687) and John Graunt (1620-1674) are claimed as founders of demography, epidemiology and economics, the three disciplines that directly deal with economic and population issues (Rosen 1958, Last 1988, Roncaglia 1987, Smith 1987).

Both *political arithmetic* and *Kameralismus* (*cameralism* or *cameral science*) have been considered early precedents of political economy (Heller 1941). *Cameralism*, practiced in the German principalities in the 17th and

18th century, was the practical art of how to govern an autonomous territory keeping plenty the State's treasury. The term is etymologically related to the State's or Prince's treasure (*Kammer, camera principis*) and for some authors cameralism is a specific German version or mercantilism (Recktenwald 1987). William Petty, "a sort of an English cameralist" (Hull 1900), invented the term *political arithmetic* to refer to the collection and interpretation of basic quantitative data of the national life, in the belief that such knowledge could be used to increase the power and prestige of the State (Rosen 1958). Precedents of this idea are to be found in the Italian Renaissance, when Florence and Venice began to consider important the collection of quantitative data on the population and other aspects of the city-state. In fact the term *statistics* seems to be derived from the Latin *status*, i.e., state (Oxford English Dictionary 1971) and therefore the roots of statistics and economics (through cameral science and political arithmetic) come this way down to the State and the Prince. Petty himself was convinced of the importance of a healthy population—as a factor of national opulence and power. He insisted in the necessity of collecting data on population, education,

diseases and any related topics, for the analysis of these data would throw light on important matters of national interest and policy. The first solid contribution in the field of political arithmetic has been considered John Graunt's *Natural and political observations upon the bills of mortality*, published in 1662 (Hull 1900, Rosen 1958). It seems however that Petty, who was Graunt's close friend, was the real author of the book, or at least parts of it (Roncaglia 1998). At any rate, the book includes important remarks on regularities in mortality and the first attempt to build a life table and for that reason it has been considered as the founding stone of demography (Smith 1998).

The most famous political economist who focused on population issues was Thomas Malthus, who brought up the idea that resource scarcity was the limiting factor arresting population growth—a theory that Schumpeter (1954) refers to as already proposed by Giovanni Botero in the 16th century.

In 1798 Malthus published anonymously *An Essay on the Principle of Population as it affects the future improvement of Society, with remarks on the Speculations of Mr. Godwin, M. Condorcet, and other writers*. As the

title itself indicates, Malthus's population theory began as a controversy against thinkers who were suggesting that the human lot could be improved. The ideas of Condorcet and Godwin were particularly hideous to wealthy British, since they contradicted the "common knowledge" that individual selfishness led to general good, and both writers proposed to improve society by abolishing private property (Routh 1975:107-108). In successive versions of his *Essay* Malthus developed his population theory that was a tough denial of any illusion about improving living standards. The *Essay* of 1798 (Malthus 1798:89) noticed that in the principal states of modern Europe population increased very slowly, or was indeed stationary or declining.[1] The passion between the sexes had not decayed, wrote the anonymous author, and was indeed a natural propensity that

> exists still in undiminished vigour. Why then do not its effects appear in a rapid increase of the human species? An intimate view of the state of society in any one country (...) will enable us to answer this question, and to say that a foresight of the difficulties attending the rearing of family acts as a preventive check, and the actual distresses of some

[1] This of course is true now, but it was not in 1798 (see for instance McEvedy & Jones 1980).

> of the lower classes, by which they are disabled from giving the proper food and attention to their children, act as a positive check to the natural increase of population.

The positive check resolved basically in early death because of hunger and epidemics. The preventive check would make people to delay marriage or never marry at all, so reducing fertility and population growth (Flew 1970, Schofield 1983, Fogel 1994a).

At the end of his life Malthus emphasized that in modern Europe "the principal check which at present keeps the population down to the level of the actual means of subsistence is the prudential restraint on marriage." Nevertheless, he had previously stated that this preventive check would come into play very slowly, and therefore high mortality due to scarcity of land and capital would be the regulating mechanism balancing human needs with natural resources in most periods of history. If so, any improvement in living conditions would generate in the short-term population growth and "overpopulation," eventually checked by the subsequent increases in death rates due to overuse of resources (Flew 1970, Routh 1975). This made Malthus an obvious target for socialist

thinkers, who generally did not pay too much attention to his rather weak mentions of the "preventive" check.

Frederick Engels already attacked Malthus in his *Outlines of a Critique of Political Economy* in 1844, when he was only 24 years old and Malthus had been dead for ten years. In Engels' view, Malthus

> maintains that population is always pressing on the means of subsistence; that as soon as production increases, population increases in the same proportion; and that the inherent tendency of the population to multiply in excess of the available mean of subsistence is the root of all misery and vice (437).

With respect to the Malthusian idea of a disproportion between an arithmetical increase of means of subsistence and a geometrical increase of population, Engels accepted Malthus's objection that land was limited, but only to remark that "[t]he labour-power to be employed on this land surface increases with population", and even assuming that the increase in yield due to the increase in labour does not always rise in proportion to the labour, there still remains a third element, science, "whose progress is as unlimited and at least as rapid of that of population" (440).

One year later, in *The condition of the working class in England* (1845) Engels wrote extensively on what in modern parlance would be epidemiological issues. He was a clear supporter of the so called miasmatic theory, that viewed "putrefying vegetable and animal substances" and "filth and stagnant pools of the working-people's quarters" as producing "precisely those gases which engender disease" (108). Engels attributed the larger mortality and morbidity in cities compared to the countryside to insufficient oxygen and excess of "carbonic acid gas" (CO_2 in modern terms) due to crowding and lack of proper ventilation (107). Besides these attributions, clearly wrong in the light of modern knowledge, Engels wrote many sound observations, often based in medical reports. He noted the high incidence of tuberculosis ("scrofula") and rickets ("rachitis") among children of workers and the frequency and severity of typhus among undernourished people. Engels also cited official statistics showing death rates in cities (specially in industrial districts) much larger than the average in England and Wales and a clear gradient of mortality in streets and houses classified in three classes, with mortality in the poorest class of houses and streets

doubling that of the richest categories (117). From the "Report on the Sanitary Condition of the Working Class" Engels cited average longevity in Liverpool that was 35 years for the upper classes, gentry and professional men; 22 years for business men and better-placed handicraftsmen; and only 15 years for operatives, day-labourers, and serviceable class in general (118). In passing, he mentioned the opinion that commercial crisis or bad harvest often triggered typhus epidemics (110-112). Close to the end of the book Engels claimed that "the most open declaration of war of the bourgeoisie upon the proletariat is Malthus's Law of Population and the New Poor Law framed in accordance with it" (289).

Engels and Marx did not see any necessary connection between the level of wages and the size of families. They considered the "surplus population", i.e., unemployment, as a result not from the vicious habits of people, but from capitalist development producing a continuous tendency to make sectors of working population superfluous (Bottomore & Himmelweit 1983).

Following Engels' interest in population health, Marx included numerous references to sanitary issues in the first volume of *Capital*. He mentioned the positive impact

of legislation on the health of workers and pointed out how mortality in the manufacturing districts was much higher than in agricultural ones (Marx 1864:406). He attributed to the large extension of the working day not only a deterioration of human labour-power but also "the premature extinction and death of this labour-power itself" (Marx 1864:376). Citing medical authorities who asserted that overworked labourers "die off with strange rapidity," Marx concluded that capital "takes no account of the health and the length of life of the worker, unless society forces it to do so" (381). Further blame was brought up by the shortening of life expectancy among industrial workers:

> The consumption of labour-power by capital is so rapid that the worker has already more or less completely lived himself out when he is only half-way through his life (...). It is precisely among the workers in large scale industry that we meet with the shortest life-expectancy. Dr. Lee, Medical Officer of Health of Manchester, stated that the average age at death of the Manchester upper middle class was 38 years, while the average age at death of the labouring class was 17 while at Liverpool those figures were represented as 35 against 15 (p. 795).

Citing Ramazzini's work *De morbis artificum* on diseases of the workers, Marx commented how some crippling of body and mind

> is inseparable even from the division of labour in our society as a whole. However, since manufacture carries this social separation of branches of labour much further, and also, by its peculiar division, attacks the individual at the very roots of his life, it is the first system to provide the materials and the impetus for industrial pathology (484).

In his inaugural address to the International Working Men Association Marx amazedly cited medical opinions asserting that the work interruption in Lancashire textile districts during the cotton crisis of the American Civil War had had some advantages from the sanitary point of view (Marx 1864b). Though subjected to a poor alimentation because of their exclusion from the factories, workers had stopped being exposed to the factory atmosphere and were actually improving in health. Mortality of children was decreasing because their mothers were now at last allowed to give them their own breasts, and Godfrey's cordial (a "pediatric" syrup made with opiates) was no longer bought.[2]

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[2] Marx briefly mentioned these health-improving effects of the cotton crisis in *Capital* (1864a:517). Recently, writing on fluctuations in

Remarkably, Marx was referring to beneficial effects of breast feeding a century before the World Health Organization launched international campaigns to discourage the nursing bottle and promote mother's milk. He was also suggesting potential healthy effects of economic slumps decades before social scientists began to look astonishingly to statistics showing drops of general mortality coinciding with economic depressions (see below).

In the second and third volumes of *Capital* Marx continued considering health questions when dealing with capital circulation and accumulation. When discussing cost minimizing in fixed capital, Marx wrote that it was "well enough known how much economy of space and therefore on buildings, crowds workers together on cramped conditions. A further factor is economy on means of ventilation." These and long working hours were responsible for a great increase in respiratory diseases and consequently increased mortality, as shown by statistics on higher

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height, body mass index and mortality in the US and England, Richard Fogel referred to the use of opiates in elixirs as one of the potential causes retarding physical development of children under 3 (Fogel 2000).

death rates and excess of lung disease in districts with large indoor industry (Marx 1894:185).

In the 1840s, two decades before publishing the first volume of *Capital,* Marx had paid attention to the link between suicide and economic recession. In his article "Peuchet: vom Selbstmord", published in 1846 in the German magazine *Gessellschaftspiegel* ("Mirror of Society") Marx presented to the German public the ideas of Peuchet, an economist, statistician and police archivist who in 1838 caused a sensation with his *Memoirs from the Police Archives,* posthumously published (Plaut and Anderson 1999). In the *Memoirs,* extensively quoted by Marx in his 1846 paper, Peuchet stated that:

> The yearly toll of suicides, which is to some extent *normal and periodic,* has to be viewed as a symptom of the fundamental defect of our society. For, in times of industrial stagnation and its crises, in times of high food prices and hard winters, this symptom always becomes more prominent and takes on an epidemic character. At these times, prostitution and theft increases proportionately. Although *penury is the greatest source of suicide,* we find it in all classes, among the idle rich, as well as among artist and politicians [emphasis added, JATG].

Perhaps this was the first time when the countercyclical character of suicide mortality with respect to business cycles was stated, some sixty years before Emile Durkheim (who did not cite Peuchet) asserted in his *Suicide* (1898) a relationship between *anomic conditions* (sudden changes in personal or economic life) and suicidal behavior. Peuchet's early observations of an association between increased frequency of suicide and industrial stagnation is remarkable, taking into account that the countercyclical character of suicide mortality has been clearly proved by statistical evidence in the 20th century (Dooley et al. 1996, Ruhm 2000).

II — Villermé, Virchow and Chadwick: the miasmatic theory and the social medicine movement

The peculiar mix of social knowledge and overall condemnation of capitalism that was created by Marx and Engels and then developed away from the mainstream of social science and economics thus included population issues from its start. At any rate, whatever the evaluation of their contributions to social knowledge, Engels and Marx were out of mainstream economics and

during the second half of the 19th century no major economist focused on population issues. However, the idea that poverty and bad living conditions cause disease and high mortality had already been popularized by Virchow, Villermé and others. Rudolph Virchow, considered as the founder of pathology, emphasized the need of hygienic and social reforms to prevent new epidemics when he was sent in 1848 to Silesia to investigate a typhus outbreak—in a report that was badly received by authorities. Louis Villermé proved that morbidity and mortality rates in Paris where related to the living conditions of the different social classes (Rosen 1958:189). In his *Tableau de L'Etat Physique et Moral des Ouvriers Employés dans les Manufactures de Coton* (1840) he made interesting observations on the health effects of professions and work:

> *The working day is very long except in times of crisis.* Workers employed in cotton and wool mills work between 15 and 15½ hours each day, of which an average of 13 hours are required (...). When there are large or rush orders, work at the mills sometimes goes trough Saturday night. This is an exception, however, and rather than have the workers spend the night at the factory, they are normally made to come

> back Sunday morning and work until noon (...). Professions most often affect the health of persons and their families in an indirect, mediate way through the conditions involving food, clothing, fatigue, duration of work, customs, etc. in which the worker exists. *This rule should be regarded as a general one* [my emphasis, JATG].

Villermé noted the noxious effects of crowding in factories and houses for pulmonary diseases, the unhealthy effects of working in cramped positions, the contrast of the rosy complexion and liveliness of children of countryside farmers compared with the pitiful aspect of those of urban factory workers. He also pointed out how the proportion of fit for military service among affluent classes was almost double that among the poorer.

It seems that Villermé's report proving the links between poverty and disease where highly influential on Edwin Chadwick, who was appointed as secretary of the Poor Law Commission, instructed by the British government in 1839 to collect information on the health condition of the working population in England and Wales. After three years a huge report was produced and Chadwick summarized the results. In his report he emphasized the connections between conditions of districts and rates of disease and

mortality. In Chadwick's view filthiness had to stop being considered an aesthetic problem and be viewed as a public health issue that had to be taken care of by engineering works and government actions. He thought that everything pointed to the conclusion that epidemic fevers were due to "miasmas" arising from decaying animal and vegetable matter; so water supply, sewerage and collection of garbage were basic preventive measures (Rosen 1958:190-191).

Chadwick had been deeply influenced by Jeremy Bentham and was friends with the economist Nassau Senior. From the point of view of modern economics Chadwick's contributions have been considered years ahead of his time. For instance he showed insight about what today would be termed externalities, when he emphasized the need to internalize industrial accident cost, making railways companies cover the heavy social costs of accidents that occurred during railway construction (Atiyah 1987). Through his positions in a variety of public administrative duties and his pamphlets on sanitary issues Chadwick not only contributed to popularize the miasmatic theory but became one of the founding figures of public health in Britain and in the world.

At the end of the 19th century time series of health statistics and census data were available and authors began paying attention to them. In his *Science of Statistics* Mayo-Smith (1896) commented on how the death rate maintained "a wonderful regularity" in Germany from 1841 to 1885, with an average annual variation from the average death-rate of only 3.9%. He also presented detailed statistics of age-adjusted mortality in England according to occupation. Setting mortality of clergymen, priests and ministers equal to 100, mortality was 143 for fishermen, 189 for tailors, 152 for lawyers, 397 for inn and hotel service workers, 175 for blacksmiths, 314 for earthenware makers, 331 for Cornish miners and 308 for costermongers, hawkers and street sellers. Working in a cramped or constrained attitude, exposure to poisonous or irritating substances, excessive work, fatal accidents, breathing foul air, drinking alcoholic beverages and inhalation of dust were factors considered causes of high mortality. For Mayo-Smith it was evident "that economic and social position must have great influence on the mortality as well as the occupation itself."

So during the second half of the 19th century the miasmatic theory of disease in which poverty, social

misery and filthiness were considered clearly linked to disease and death became "common knowledge." On the other hand, as political economy transmuted into economics, epidemics and health were mostly forgotten by economists and the study of population issues began to thrive in the newly developed fields of demography and epidemiology. From its beginning demography had some links to economics but, contrarily, epidemiology and public health were born from the medical, statistical and sanitary field, did not developed any important link with economics and they even reacted against *laissez-faire* theories, which were often considered by public health reformers as "the paltriest of all philosophies, in sanitation—as in everything else" (Easterlin 1999). In fact, between 1779 and 1817 Johann Peter Frank had published the six volumes of the monumental *System einer vollstäaanding medicinishen Polizey* in which basically health was considered a responsibility of the State, and the "medical police" functions were outlined in the fields of population policy, child education, food, veterinary health, garbage disposal, sewage and accident prevention (Rosen 1958-137-141). Frank, Villermé, Virchow and Chadwick are considered founders of the social medicine movement, a school of

thought emphasizing the links between living and working conditions and rates of disease and death.

In spite of early antagonisms between economics and public health the idea that health problems has to do with poverty permeated economic thought. The textbook in which many generations of economists learned their trade in the late 19th century and early 20th century asserted that "physical, mental, and moral ill-health is partly due to other causes than poverty: but this is the chief cause" (Marshall 1920).

III – From Pasteur's microbiology to McKeown's nutritional determinism

The 20th century was to be the century of biology. Starting with Pasteur, the list of discovered pathogenic germs enlarged at an increasing pace and the miasmatic theory of disease soon passed away. In the first half of the century procedures of hygiene, antibiotics and other specific means to prevent and to treat disease were developed and generalized. But also statistical data on population issues were now available showing that approximately from the mid 18th century death rates had been more or less steadily declining in England, France,

the Scandinavian countries and North America. The continuous growth of population and the sustained reduction of mortality at all ages largely disproved the Malthusian theories. The attribution of the fall in mortality mostly to the advances of medical science in preventing death became "common sense" (Fogel 1994). It was a doctor, Thomas McKeown, who strongly questioned this belief.

McKeown is probably the author who contributed more to the view that the long-term decline in mortality has been a direct consequence of "economic progress." In his early contributions (McKeown and Record 1962, McKeown, Record and Turner 1973) he showed that most of the reduction in mortality in modern times had taken place before specific therapeutic means were available, even before causal germs were known. Therefore it could not be possible that the decline in mortality was due to medical progress. Considering the different factors that could have contributed to the long-term reduction of mortality during the period of industrialization, he concluded by exclusion that the main one had to be the improvement in nutrition associated to the general increase in standard of living. A diet scarcely sufficient in proteins and

energetic requirements to fulfill physiological needs would generate a state of low immunity favorable to the development of infectious diseases (McKeown 1983). The fall in mortality rates in countries like China or the Indian state of Kerala during the 20th century was for McKeown evidence supporting the lack of importance of other factors such as improvements in the supply of drinking water, sanitation and personal medical and immunization services (McKeown 1988).

In the opinion of Simon Preston (1996), a leading author in demographic studies of mortality, McKeown's view on the lack of importance of specific therapeutic medical treatments has for the most part stood the test of time. McKeown's ideas were highly consistent with the economic paradigm of development (Kuznets 1979) and were quite attractive for economists. However, McKeown "nutritional determinism" was soon harshly criticized and largely challenged from the fields of economic history and demography (Frank & Mustard 1995). Contrarily to McKeown, Szreter (1988, 1997) attributed a leading role in the reduction of mortality in England and Wales during the second half of the 19th century to the public health movement and its locally administered sanitation programs

and preventive health measures combating the urban congestion created by industrialization.

Szreter partly based his conclusion and his criticism of McKeown on the demographic reconstruction of the period 1540-1870 in England by Wrigley and Schofield (1981). These authors could not find any specific evidence "for the view that long-term movements toward lower wages provoked higher mortality, nor that a steady rise in real wages brought a saving of life". On the contrary, "[t]o the degree (…) that rising real wages may justly be linked to urban growth, they exerted an influence upon mortality, though in the opposite direction to which is usually assumed to exist." In fact, all along the 17th century real wages had a steady growth associated to a long-term increase in mortality. "It is reasonable to suppose that in certain circumstances improving living standards will tend to raise mortality rather than reduce it. If higher real wages (...) concentrate more people in cities, higher death rates may result" (Wrigley & Schofield 1981:415).

From the field of economic history Easterlin (1977, 1999) has recently exposed similar views on the negligible impact of economic growth on the reduction of mortality, assigning a substantial adverse effect to the process of

urbanization linked to industrialization in Western countries. Sanitation and disease-preventive technologies would be the determinant factors in the reduction of mortality. Easterlin partly based his views in the early demographic work by Preston (1976). In a recent review on population studies on mortality, Preston (1996) himself has pointed out vaccination against smallpox and purification of milk as two specific health procedures undervalued by McKeown. Preston has also reasserted his conclusions of the 1970s, when using time series and cross-sectional data for nations with relatively good demographic data he concluded that only about 20% of the massive international improvements in mortality between the 1930s and the 1960s could be attributed to improved standards of living.

Robert Fogel received the 1993 Alfred Nobel Memorial Prize in Economic Science because of his pioneering studies in cliometrics. From a stance that perhaps is an intermediate position between McKeown's nutritional determinism and the "sanitarian" view of Szreter and Easterlin, Fogel (1994a, 1994b) has emphasized the importance of low caloric intake during pre-industrial times for underdevelopment during the prenatal period and the childhood, with the consequent small body size, high

incidence of chronic disease in the adult and high mortality at all ages. Looking at the relationship between economy and health in the other direction, he has pointed out how low caloric intake in pre-industrial times would also be responsible for a very low productivity of work. In Fogel's view, despite "all the different ways in which Malthus misread the future," his central proposition in *An Esssay on the principle of population*, "that much misery was caused by the pressure of population against available resources—remains valid today." He has also considered as due to the unprecedented increase of population after World War II the degradation of the environment that "threatens not only to impede progress in the Third World but to impede, if not reverse, the progress in the OECD world" (Fogel 1994b:232).

Interestingly, McKeown was also quite explicitly Malthusian. In one of his last contributions McKeown (1983) accepted that hygienic measures had had a role in the reduction of infections, that medical advances had some influence in the 20th century reduction of mortality and that a safe milk supply was the main reason for the reduction of deaths from gastroenteritis and contributed substantially to the fall in infant mortality from 1900.

However he explicitly asserted that "the slow growth of the human population before the 18th century was due mainly to lack of food, and the rapid increase from that time resulted largely from improved nutrition." In the 20th century, for the first time "it could be said that numbers and resources were in reasonable balance, so that the Malthusian adjustment through high mortality no longer operated."

For Richard Wilkinson social inequalities are a major determinant of mortality. In a number of papers and in his book *Unhealthy societies* (1996) Wilkinson stated how among the developed countries not the richest ones have the best health, but those which have the smallest income differences between rich and poor. For Wilkinson inequality and relative poverty have an absolute effect in rising death rates. Bringing together evidence from the social and medical sciences, Wilkinson's theory of mortality in modern societies has generated a whole controversy (Judge 1995, Judge et al. 1998, Gravelle 1998, Muntaner & Lynch 1999, Wilkinson 1999, Muntaner et al. 1999). Wilkinson's ideas will surely provide an important field of research in the next years.

IV – Thomas, Eyer, Brenner and the repeated discovery of the procyclical fluctuations of mortality

The former sections basically referred to the attempts to understand in broad terms the long-term impact of economic transformations on the changes in mortality. However, even from long ago there have been attempts to consider whether short-term variations in mortality are related to short-term variations in economic activity. As was mentioned, already in the 18th century Peuchet stated that periods of economic depression were associated with high incidence of suicide. One century later Durkheim attributed the higher incidence of suicide to social disruption because of sudden social changes.

Some late 19th century authors such as the Russian Tugan-Baranowsky and the German Arthur F. von Firks pointed out connections between the ups and downs in economic activity and changes in the death rate (Huntington 1920, Thomas 1925). But it was not until the 20th century that this relationship began to be more closely examined. From the beginning there were authors who thought that mortality was countercyclical, increasing in recessions, while others thought it was procyclical,

increasing during economic expansions.[3] Probably the geographer Ellsworth Huntington was the first who closely examined the relationship. In his *World-power and evolution* (1920), Huntington examined death rate series and business cycles indicators for the US, Great Britain, France and Germany. He detrended the series and examined the movements of the residuals, concluding that in the US and in Germany there was a relationship between business cycles and death rates, though he could not find the relationship in Great Britain and France:

> During hard times many people are out of work; the children of the laboring classes are often ill nourished, there is no money for medicines, delicacies, and doctor bills; even the more prosperous parts of the community are under a nervous strain. Hence at such times there must be more deaths than during the prosperous times which are supposed to follow good crops. Hence I looked for a rise in the deathrate during hard times and a fall in good.
>
> This line of thought may sound reasonable, but it is fallacious. The statistics from 1870 to the Great

[3] ***Procyclical, countercyclical*** **and** ***acyclical*** **are used meaning directly, inversely or not associated to economic expansion. The term *cycle* in *business cycle* (or *trade cycle* for the British) is misleading, as it seems to imply that economic activity fluctuates in a *regularly* recurrent way, which is not the case. For that reason the terms *business fluctuations* or *economic fluctuations* are to be preferred (Brandis 1964). However, *procyclical, countercyclical* and *acyclical* are the terms commonly used. Since no equivalent derivatives from *fluctuation* exist, they will also be used here.**

> War show that a high deathrate regularly *precedes* hard times, while a low deathrate precedes prosperity. By no possibility can the reverse be made to appear the case (Huntington 1920:28-29) .

Huntington asserted that alcohol consumption increases coincided with death rate peaks but he discarded the idea that the increase in liquor consumption could be the main reason for the increase in death rates. In his opinion, there was another factor controlling both: "Economic cycles of adversity and prosperity in the United States depend upon health far more that upon any other factor. And health depends largely upon the weather" (42). So he supported the idea, advanced by Jevons and very popular for a while, of a business cycle triggered by natural causes, concretely by weather. The peculiarity of Huntington's business cycle theory is that weather acted on economic activity through intermediate health variables. In France and England the relationship between death rate oscillations and economic cycles was not observable because "the mildness of both the summers and winters causes the variations in nervous activity to be so slight that their effect is concealed by other influences" (49).

In 1922, in "The influence of the business cycle on certain social conditions", William F. Ogburn and Dorothy Swaine Thomas examined the relationship between changes in death rates and economic ups and downs in the US. They observed that mortality increased in times of prosperity, with reductions in mortality coinciding with unemployment upturns. As this seemed illogical to them, they questioned if this proved some connection between economic activity and death rates. Trying to verify the effect they checked British mortality data against a very rude index of British business cycles. As they did not find any relation in these data, Ogburn and Thomas concluded the procyclical increase in mortality shown by US data was perhaps some kind of unexplained artifact.

Three years later, in *Social consequences of business cycles* Maurice B. Hexter analyzed monthly data from Boston, Massachusetts, during the period 1900-1920. He compared the death rate series from Boston with the series of unemployment data from the state of Massachusetts, concluding that "the death rate varies *directly with unemployment* (...) ten months following cyclical fluctuations in the death-rate, cyclical variations in

unemployment occur in the opposite direction" (Hexter 1925:149). Hexter knew Ogburn and Thomas's 1922 paper and he commented how strange were their results on mortality. "Our own findings of an inverse relationship [between the death rates and the business cycle] are closer to expectations" (p. 149). He also knew about Huntington's *World-power and evolution,* but perhaps his mental fighting with these ideas made him misquote Huntington. Where *World-power and evolution* asserts that "a high deathrate regularly *precedes* hard times, while a low deathrate precedes prosperity (Huntington 1920:29), Hexter quoted this idea asserting that a "high death-rate precedes prosperity" (p. 150). Curiously in a different page (p. 169) Hexter correctly quoted Huntington as "a high deathrate regularly *precedes* hard times, while a low deathrate precedes prosperity."

Dorothy Thomas did not mention either Huntington or Hexter in her PhD dissertation (London School of Economics), published in 1925 as *Social aspects of the business cycle.* In this book she reexamined the US data and analyzed British data on mortality. Her conclusions were as follows:

> The connexion between the general death-rate and the business cycle is contrary to expectation. Deaths, both in the United States and England, show a strong tendency to increase with prosperity and diminish with depression. The many and diverse factors influencing the death rate may possibly affect the correlation (...) so, although it is difficult to find a satisfactory explanation of the cause of this phenomenon, the conclusion must be drawn that, in both England and the United States, a high death-rate is associated with periods of prosperity, a low death-rate with periods of depression (Thomas 1925:158-159).

Huntington's, Hexter's and Thomas's findings seem to have passed without much impact in economics and the social sciences in general, as they were mostly forgotten during fifty years in which neither demographers nor economists looked in detail at the relationship between mortality and business fluctuations. When interest in this issue came up again in the 1970s, researchers were coming form the field of public health and epidemiology.

Perhaps Thomas herself was convinced that the idea of a procyclical fluctuation of mortality was wrong, as in her *Social and economic aspects of Swedish population movements 1750-1933* (1941) she did not mention her

previous finding, nor Huntington's or Hexter's books. Reviewing the excellent Swedish population statistics she asserted a clear correspondence between peaks in death rates and slumps in the harvest index during the second half of the 18th century (p. 83). But the positive correlation she found between percentage deviations from trends in business cycles (measured by an indicator based in investment in fixed capital) and crude death rates (r = 0.13 ± 0.14 for 1865-1913, r = 0.14 ± 0.19 for 1865-1892 and r = 0.06 ± 0.22 for 1893-1913) she dismissed as statistically not significant and therefore showing no connection between mortality and business cycles in Sweden (pp. 162-163 and 196). She mentioned that

> the slow progress in urbanization and the location of major industries in rural areas, unquestionably lessened the unfavorable effects of the Industrial Revolution observed for example in England, where disease spread rapidly because of unsanitary factory conditions and excessive urban congestion. Furthermore, the total death rate is an extremely complex phenomenon: excessive consumption of alcoholic beverages is a concomitant of prosperity and will tend to increase the death rate from alcoholism at these periods; tuberculosis is a concomitant of overcrowding, which may increase in

> times of prosperity, but increases in deaths from this cause may lag by several years after the prosperous phase; death from malnutrition will probably increase in depressions, as will suicides, etc. As a result of conflicting variations in these and other specific rates no correlation may appear in the total rates (pp. 162-163).

The 1960 National Bureau of Economic Research Conference on Demographic and Economic Change in Developed Countries was obviously a golden occasion for examining the relationship between business cycles and mortality, more so taking into account that Dorothy Thomas was present. In the introduction to the proceedings of the conference (NBER 1960) Ansley J. Coale wrote that the conference had focused on the causes and effects of variations in fertility and mortality from the view of demographers and economists. But he explained that the conference examined fertility much more than mortality and speculated that "mortality changes in the next decades are not likely to be strongly influenced by economic forces." He stated that "[t]he effect of economic variables on risks of dying in the industrially advanced countries is probably weaker and less direct than effects on fertility," and exposed the general view that "of course,

there has been a fairly intimate causal association between increases of life expectancy and industrialization." In brief, the conference did not examine the relation between business cycles and mortality. However, one of the contributions of the volume, by Dudley Kirk, deals with the relationship between marriage rates and birth rates with business cycles (a procyclical one, once more verified). The methods Kirk used to study this relationship were the same Thomas had used almost forty years before, and they are briefly commented by Thomas herself in the same volume. Why this conference did not examine the mortality-business cycle issue is for me a mystery.

However, in the years following World War II period the wealth of data on demographic and economic indicators had to produce some observations on relationships between mortality and the economy. In his *General theory of population* Sauvy (1966:330) presented a graph showing clearly that high unemployment periods in France, Germany, England and Wales were not at all associated with increases of mortality. He commented that it is "often believed that the great unemployment crises are deadly,

but their influence is now negligible. In Germany, for instance, the number of totally unemployed was over six million in 1932-3 and there was also a high percentage of partially unemployed—yet mortality went on dropping at the same rate as before." When examining mortality trends in the US between 1900 and 1970 in their textbook of demography, Smith and Zopf (1970) remarked as their most striking features the "rather consistent manner in which the death rate fell over the period under consideration, and the way in which the rate tended to level off after 1955." They pointed out the pronounced peak in mortality due to the great influenza epidemic in 1919, but they considered that "[t]he various other fluctuations prior to 1930 should be discounted liberally, due to the fact that data are for the expanding death registration area." So the addition of data of southern states and black population "may very well be responsible for many of the gyrations." They commented about the 1937 peak, "probably truly indicative of a slightly higher mortality rate for that year, although the reasons for such an increase are not readily apparent", but they did not mention the higher peak corresponding to 1929, strikingly apparent in the graphs they provided. They did not mention Thomas when

dealing with mortality, though they mentioned her when dealing with migration.

In 1977 Joseph Eyer, a biologist in the University of Pennsylvania, reexamined the relationship between unemployment and mortality in the US from 1870 to 1975. With a very simple graphical approach, he showed a clear relationship between the fluctuations of mortality and unemployment ups and downs. But the "Thomas effect" appeared again: mortality did not increase in periods of economic slumps but in periods of economic expansion. Eyer summarized this striking fact in the title of one of his articles, "Prosperity as a cause of death." In this article, Eyer (1977a) explained how Dorothy S. Thomas had first

> described this relationship in the United States data in 1922, in a paper in which her response was to deny the validity of the death rate data, rather than affirm the obvious relationship (...). Thomas' ambivalence toward the data is typical of the difficulties that investigators have repeatedly evidenced in dealing with this phenomenon. Tugan-Baranowsky, for instance, simply asserts that the death rate rises during serious business crises, though he has the opposite data right before him. Another whole school of 19th-century investigators

attributed the major fluctuations of adult male death rates to alcohol consumption, without pointing out that alcohol consumption rose with the boom and fell during depressions.

Eyer (1977a) showed that the three main causes of mortality in recent decades—cardiovascular disease, cancer and unintentional injuries—increased during economic upturns. Other causes of mortality like liver cirrhosis, diabetes, bronchitis, emphysema and asthma were also procyclical. Causes of death that were countercyclical in the US data, increasing in the slumps, included suicide and homicide. As the share in total mortality of procyclical causes was much larger that the share of countercyclical causes, total mortality moved procyclically. The main mediators of the increase of mortality rate during the upturns would be the social disruption and the increase in stress, noxious consumption or hazardous exposures associated with the acceleration of economic activity. Flu epidemics coinciding with the economic boom and due to the general low level of immunity associated with an overstressed population would largely contribute to increase the mortality caused by underlying chronic processes—such as cardiovascular disease, neoplasm

or chronic bronchitis-emphysema (Eyer 1977b; Eyer & Sterling 1977, Sterling & Eyer 1981).

Contrarily to Eyer, Harvey Brenner (1971, 1973, 1975, 1976) had pursued the hypothesis that the rise of unemployment during depressions causes the greatest part of the business cycle variation in the death rate. Brenner criticized Eyer because of a lack of statistical analysis of the relationship between mortality and economic growth using proper statistical multivariate methods. In his comments on Brenner's work, Eyer (1977b), referred to cross-sectional data which showed the increased mortality of unemployed compared with employed, but he denied this effect as sufficient to explain the mortality waves associated with business fluctuations. In his last contribution, Eyer (1984) took into consideration the striking fact that mortality waves also were observable in East block countries coinciding with business cycles in the West (though he did not show any data on it). As East economies were not exhibiting business cycles of unemployment, Eyer proposed that flu epidemics once originated in Western countries coinciding with economic

booms, would spread to the whole world triggering peaks in mortality when interacting with chronic processes.

As Colledge (1983) pointed out, Brenner attributed the actual slowing down of the long term decline in mortality to high unemployment *or* rapid economic growth. Indeed Brenner maintained an ambiguous position about the relationship between mortality and the business cycle. He asserted the effect of unemployment in increasing mortality, but he dealt with this effect through convoluted lagged multivariate models. For Brenner "peak lags between unemployment and mortality rates are two to three years in many industrialized countries" (1983:617), but he also referred to the adverse effect of rapid economic growth (565), so it was hard to discover any clearly proposed relationship between mortality and the business cycle. Even in a more recent contribution Brenner has asserted that "business cycle recessions and economic decline are related to increased morbidity and mortality, while economic growth has been associated with increased health and longevity" (Brenner 1993:374). This would imply a pure countercyclical relationship between mortality and

business cycles as well as a direct long-term link between economic growth and mortality decline.

In his most recent contribution (to my knowledge) Brenner asserted again that economic growth has "overwhelmingly beneficial effects on health, especially in reductions in acute infectious disease and occupational injuries as causes of death" (1995:211). He cited Kuznets's idea that as the modernization sector becomes dominant, inequalities decrease, and stated that business cycles "and specifically the unemployment rate, is the second macroeconomic influence on income inequalities. Inequalities widen as the business cycle contracts, specifically as unemployment grows" (221). As inequality is specifically linked to mortality, Brenner concluded that recessions increase inequality and this in turn raises mortality. However, "for the first time in American history in fact, the economic expansion of the 1980s was accompanied by deterioration in the standard of living for a majority of households" (222). All the evidence on business cycle influences over suicide, mental health, alcohol abuse, heart disease and infant mortality show in

Brenner's view the noxious effect of recession. About the overall influence of economic change, he wrote (227):

> The principal long-term declines in overall mortality, as well as age-specific mortality for all age groups, are associated with long-term improvements in the national economy, specifically real per capita income. The business cycle is heavily influential in the short- to medium-term fluctuations in mortality rates. Such variables as unemployment and business-failure are typically associated with increases in mortality *two to three years following the lowest point in the business cycle* [my italics, JATG] and extending for at least the next 10 to 15 years. The relationships have been most fully explored with data for the United States, United Kingdom, and Sweden (Brenner, 1976a, 1979, 1980b, c, 1984a, 1987b, c, 1991, 1993). Middle-aged mortality rates have also been examined from this viewpoint in at least 10 additional countries (Brenner, 1989).
>
> These types of relationships for overall mortality—with long term trends inversely related to economic growth and short to medium-term fluctuations significantly associated with economic cycles—have also been observed for the principal cardiovascular diseases, even when the models included controls for tobacco, fat, and alcohol consumption patterns (Brenner, 1976b, 1984a, 1987a, b, c; Brenner and Mooney, 1982).

As it can be seen, all references provided by Brenner to support his ideas are Brenner's references themselves. In a section titled "Macroeconomic change and mortality: the economic-change model of population health" (p. 232), Brenner explains that:

> Because, on average, economic cycles tend to be 4-5 years in length (Mitchells, 1951) this classic 2- to 3-year lag in mortality after the peak in the unemployment rate means that the first peak in mortality following recession approximately coincides with the subsequent peak in the business cycle—that is, the peak of "recovery" or "expansion." Given this observation, one should also note that the zero-lag relationship between unemployment and mortality rates is actually inverse. The *first peak* in the lagged mortality rate at 2-3 years after recession is not accidental: the delayed impact of economic distress interacts with chronic-disease mortality in such a way that the first wave of increased mortality peaks at about the same time as growth in the economic cycle.

Moreover, economic recession, "as measured by the unemployment rate, and/or by the business failure rate (...) is positively related to mortality over 0-10 and 1-7 years, respectively" and "[d]uring the recessional year itself, however, overall mortality declines, followed by a

sharp increase (with no lag) during the initial year of recovery. Important exceptions include suicide, homicide and other mental-health indices and infant mortality, whose positive relation to unemployment rates *begins* at the zero year" (237-238). These assertions, that basically reproduce what Eyer said in 1977, are included in a chapter with approximately 200 references, 22 of them by Brenner himself but no one by Joseph Eyer or Dorothy Thomas. Not one of the many critiques that time-series analysis by Brenner received from the seventies are there referenced. This is in my opinion a paradigmatic case of selective use of published references.

From the late seventies to the early nineties criticisms against Brenner multiplied. Kasl (1979), Gravelle et al. (1981), Winter (1983), Wagstaff (1985), Watkins (1985), Cook (1986), Forbes and McGregor (1984), and Søgaard (1992) criticized different aspects of Brenner's convoluted statistical methods. It was often mentioned that Brenner's effort in not showing his primary data and using multivariate lagged models make it impossible to follow closely the relations between variables. Brenner's work on mortality began to be cited

almost exclusively by himself and it is not even mentioned in recent books on mortality like the one by Wilkinson (1996).

After the Eyer-Brenner controversy empirical studies on the relation between the economy and mortality were published providing very inconclusive evidence. Higgs (1979) found increases in death rates associated to phases of economic expansion in 30 large US cities, in the period 1870-1900. He attributed this to high virulence bacteria brought by immigrants during prosperity times, a view which was disputed by Chernomas (1984), who attributed the effect to overcrowding and overwork. Analyzing statistics for Scotland in the Post-World War II period, McAvinchey (1984) concluded that mortality rates were negatively associated with the business cycle, with higher mortality in recession and lower mortality in booms. However Forbes and McGregor (1984) found in the same data little evidence of a consistent association between unemployment and male mortality. Both studies used multivariate regressions to investigate the relationship between mortality and economic variables.

Though Eyer's ideas were always referred in the literature with more respect than Brenner's, they were also criticized and even plainly rejected as invalid by Bunn (1979), LaPlante (1985), Watkins (1985), and Goff (1990). Watkins (1985:30) examined very superficially the same US data used by Eyer and concluded that "the Thomas effect dominated the effects of recession on health until the second quarter of this century, but sometime in the second quarter of the century it disappeared." Watkins considered that Brenner's econometrics were lousy, but he concluded that Brenner's main ideas were sound.

In a time-series analysis of Canadian data Adams found an inverse association between unemployment and mortality. The view of this author was that this perhaps could be due to the decrease of social activity, the lower risks of work-related deaths and the fall in alcohol and tobacco consumption in times of reduced income (Adams 1981, cited by Jin et al. 1995).

LaPlante and Goff are probably the only two modern authors who considered mortality not related to macroeconomic oscillations, i.e., acyclical. Mitchell P. LaPlante's PhD dissertation in sociology (1985) studied

the relationship between mortality and business cycles. Reviewing the former studies by Ogburn and Thomas, Brenner and Eyer, he concluded that Brenner results linking directly unemployment with mortality increases were spurious, due to his ad hoc specification of models and use of inappropriate detrending methods. In LaPlante's view, Eyer had also failed in proving statistically the link between economic booms and mortality upturns, mediated by flu epidemics. LaPlante examined Ogburn and Thomas's data from the first quarter of the century and concluded that they proved an increase in mortality associated to economic expansion. However this effect, that he tentatively attributed to migration to towns with the corresponding crowding and stimulus for infectious disease, was in his view no longer observable in the data after the 1930s.

Goff (1990) studied US data for the period 1948-1982 and found positive and significant signs for the coefficients for unemployment, per capita GNP, and Dow Jones average in a regression with cardiovascular disease mortality (the first cause of mortality) in males as dependent variable. This "implies inconsistent results in

that economic prosperity has both good and bad effects on cardiovascular disease." He therefore concluded that the result "indicate no systematic influence of aggregate economic measures on health," and presented his model in which "choice variables" are the real determinants of the health outcome.[4]

Taking into account all the former, probably it is not too bold to say that in the last decade of the 20th century there was no clear idea about the relationship between business cycles and mortality. Business cycles were not taken into account by most researchers working in mortality issues and Brenner's ideas about noxious effects of recession were sometimes quoted, generally by people not working in public health (Sorkin 1988, Mattick 1983, Sherman & Kolk 1996:21, Snyder 1996:97).

The possible contribution of business fluctuations to mortality was not mentioned by Robert W. Fogel in his speech on the occasion of receiving the 1993 Alfred Nobel Prize in Economic Sciences (Fogel 1994). In that speech, in which Fogel extensively presented connections between

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[4] "An individual will consume cigarettes to the point that the perceived marginal benefits of present satisfaction are equaled to the perceived marginal costs imposed on long-run wellness." This quotation from Goff's paper reveals how neoclassical economic theory illuminates human behavior, including drug (tobacco) addiction.

economic and demographic variables, he commented on the fact that during the decade of the 1930s the US unemployment rate was never less than 16% and for half of the period reached levels between 20% and 35%, in spite of which life expectation between 1929 and 1939 increased by 4 years. For Fogel this was the payoff of social investment in biomedical research and public health made during the 50 years before the great depression.

Neither was the short-term impact of economic fluctuations on mortality considered by Wilkinson (1996), who has tried to prove a direct relationship between the degree of social inequality and the level of mortality in different countries. Wilkinson was mainly concerned with explaining the long-term evolution of mortality and his data are highly suggestive of a faster decline of mortality wherever social indexes of inequality are lower. As it was mentioned, Brenner tried to outline a theory linking inequality changes, supposedly growing during recessions, with short-term mortality variations associated with the business cycle. However, Davis (1941) analyzing data from the first decades of the 20th century showed that inequality *increases* during economic expansion

and not during recessions, as the concentration of income is clearly procyclical.

In the 1990s research on business cycles and mortality (Starrin et al. 1990, Blane 1990, Short 1996) did not provided either clear models or definite answers. But recent contributions by Ruhm (1999, 2000, 2001) have provided new evidence on the relationship between business cycles and mortality. Ruhm has produced strong statistical evidence, based on panel data of the states of the US, that increases in unemployment coincide with drops in general mortality and mortality due to major causes of death. The only exception was suicide, which increased countercyclically during recessions, a result that replicates that of 19 of 20 studies in which the recession-suicide issue was investigated in recent decades (Dooley et al. 1996). As possible explanatory mechanisms of the procyclical fluctuation of mortality Ruhm (1999, 2000) has pointed to larger consumption of tobacco and alcohol, increases in obesity, reductions in sleep time and reduced exercise during times of economic expansion and falling unemployment.

Ruhm's results clearly imply a procyclical oscillation in mortality like that observed by Thomas and Eyer, and do not provide any support of Brenner's hypothesis (although Ruhm examines Brenner's idea of lagged effects of unemployment only marginally). Further evidence in the same direction based in data from Spain is provided in the second chapter of this dissertation.

A piece in the puzzle that apparently does not fit is the large body of literature (that will not be reviewed here) proving that in short follow ups of one or two years mortality in unemployed (adjusted for age, social class, etc.) is higher than in employed. This fact, verified in many investigations would seem to suggest that mortality had to be highest during recession, or shortly after. But this is directly contradicted by the data. Bi-directional causation—with ill health promoting loss of the job, and loss of employment inducing disease—, and confounding issues—unemployment causing reduction of income, this in turn generating bad health (Stern 1983)— are probably factors in this association. That increased mortality among unemployed is probably due in a large extent to third causes or reverse causation seems to be suggested by

the fact that the differential in mortality between employed and unemployed is strongly reduced during recessions, when unemployment surges (Martikainen & Valkonen, 1996, 1998).

V — Conclusion

In the last decade of the 20th century the fall of the Soviet bloc brought with it a demographic phenomenon without precedent in modern history. Coinciding with the breakdown of the USSR and the fall of the allied regimes in Eastern Europe death rates strongly rose in all these countries (Cornia & Paniccià 1995). Between 1989 and 1994 the annual death rate in Russia increased from 10.7 to 15.7 per thousand (Semashko Research Institute 2001), a 47% increase due mostly to an enormous growth in adult mortality (Nell and Stewart 1994, Brainerd 1997). In East Germany mortality surged in men, particularly between 1989 and 1991, in some groups up to 30%. Riphahn & Zimmerman (1998) consider that this increase in mortality of East Germans "can be explained by the increase in individual stress levels after the economic, cultural and political consequences of reunification." Explaining the breakdown of the centrally planned regimes and the surge in mortality in East Europe in terms of business cycles would be obviously wrong, since the 1990s transition in the Soviet bloc countries was much more than a "business fluctuation." But perhaps stress can be the link that puts together death rate fluctuations in both East and West.

The concepts of "stress" (Antonovsky 1979) and "social support" (Cohen & Syme 1985) seem to be key pieces in the relationship between the functioning of societies and the rate at which individuals die. Authors like Eyer, Johnson and Hall (1995), Graertz (1993) and Wilkinson (1996) have provided elements that probably will be pieces in the construction of a model to explain the complex issues involved here. In my opinion, stress and social cohesiveness can be the links that join together procyclical mortality oscillations in the West and the mortality explosion associated to the social breakdown in East Europe during the 1990s. But this is a field in which much investigation is still needed.

CHAPTER 2

DEATH RATES AND BUSINESS FLUCTUATIONS IN SPAIN AT THE END OF THE 20TH CENTURY: FURTHER EMPIRICAL EVIDENCE OF THE MORTALITY INCREASE DURING ECONOMIC EXPANSIONS

CHAPTER SUMMARY. Using data from the 50 Spanish provinces during the 18 year period 1980-1997, yearly death rates for all causes and cause-specific deaths rates for particular causes (for men and women combined and for each sex separately) were modeled as functions of the demographic structure of the population, general and sex-specific unemployment rates and employment ratios, and other proxies of economic conditions at the province and national level, in fixed-effect regression models. Regressions were also run for specifications with unemployment rates lagged up to four years and for specifications with the variables as first differences or relative change. The results show that mortality fluctuates procyclically, deviating up from its secular trend during economic expansions, and down during recessions. Regressions with lags do not support Brenner's theory that attributes procyclical mortality to lagged effects of unemployment.

I — Introduction

The present chapter is an empirical study of the relationship between short-term variations in mortality and short-term fluctuations in the economy, the so-called business cycles. In brief, the question is to decide if, in the particular study sample, over and above secular trends, mortality (1) increases during recessions, (2) increases during economic expansions, or (3) is unrelated to economic fluctuations; or, in other words, whether mortality is *countercyclical, procyclical* or *acyclical*. Research in this field can suggest pathways for better understanding the causes of disease and death in modern societies. On the other hand, whether macroeconomic fluctuations are related to changes in death rates is an important question directly linked to economic policy issues.

In chapter 1 the contributions by Hexter, Thomas, Eyer, Higgs, Brenner, Kasl, Wagstaff, Junankkar, Ruhm and other authors have been already discussed. From that background, in this chapter I analyze the relationship between mortality and economic fluctuations in Spain between 1980 and 1997. The strong fluctuations of unemployment in Spain in this period provide a "natural experiment" in which to attempt the empirical replication of previous studies. Theoretical and empirical support for possible pathways for this relationship is presented in the discussion.

II — Background, data and methods

The Spanish economy at the end of the 20th century. After the 1978 Constitution Spain was organized into 17 autonomous communities, each one including one or several of the 50 provinces in which the country had been politically structured during general Franco's dictatorship (1939-1976). The political transition in the mid-seventies coincided with the start of severe and protracted recessions. From rates of growth of real GDP of around 8% in the early seventies, the economy slowed down to reach negative growth rates in the early eighties. The growth of GDP recovered throughout the mid eighties, reaching more than 5% in 1987, to drop to negative figures again in the early nineties (figure 2.1) and recover to positive figures from the mid nineties.

Unemployment, which had been below 4% during the sixties and early seventies (though this is contested by some authors, as will be subsequently explained), began growing in the mid seventies. The late seventies and early eighties was the time of the so called *reestructuración industrial,* which implied the closing of many industries in the public sector. Unemployment grew to a peak of 21% in 1985 (figure 2.1). Despite some recovery during the late eighties unemployment reached 24% in 1994. The joblessness rate dropped steadily throughout the second half of the nineties, although figures remained well over 10%.

Discussing the reasons for the upsurge in unemployment in Spain since the mid-seventies is out of the scope of this investigation, but it is important to provide at least some context. Spanish recessions in the last quarter of the

20th century are clearly connected to the recessions in western Europe (figures 2.2 and 2.3). On the other hand, immigration, age-structure changes and gender factors also played a significant role in the upsurge of unemployment in Spain.

It has been argued that the low levels of unemployment during the sixties and seventies in Spain are somewhat fake as unemployment was camouflaged by intense migratory flows to other European countries (Martín 2000:52). The estimates of the number of Spanish workers migrating to European countries are quite disparate, but in general Spanish emigration is qualified as "massive" (Tortella 1994:389). While Román (1997:51) mentions that 2 million workers were gone from Spain in the late sixties, Martín (2000:194) states that the negative migratory balance during the sixties totaled 0.8 million. According to Sáez (1975:16, 121, 301-302), on the basis of statistics of the countries of arrival, 1.9 million Spaniards migrated to European countries between 1960 and 1967, among which 1.0 million were "permanent" (more than 6 months) emigrants (economically active population in Spain were 9.6 million men and 2.3 million women in the 1970 census). There was a narrow correspondence between the curves of GDP growth of Germany and Switzerland and the changes in the migration flow of Spaniards to Europe, so that the variation of labor demand in the Western European countries (as proxied by the rate of growth of GDP) was a good explanatory variable of the Spanish migration flows. Sáez also saw some correspondence between the unemployment rates in Spain during the sixties (oscillating around 2.5%, figure 2.2) and the oscillations in migration, what would be a

manifestation of the integration of the labor markets in Western Europe. The Spanish emigration to Europe had a temporary nature and there was a "return flow that is not registered and can be only hardly and indirectly estimated" (Sáez 1975:121). Tortella (1994) locates the return of the Spanish emigrants loosely "after the crises of 1973". Martín (2000), however, states that there was a significant repatriation of emigrants during the 1980s.

Age-structure was also a determinant factor in the upsurge of Spanish unemployment. In the period 1970-1995 the percentage year to year increase in population aged 15 to 64 years was much higher in Spain than in other European countries (figure 2.4). Between 1973 and 1979, when unemployment began surging in Spain, the yearly average increase in the population aged 15-64 years was 1.2% in Spain, 0.3% in Germany and 0.7% in France.

Gender factors were also involved in bringing extra labor supply to labor markets. The average proportion of females in the total labor force averaged only 23% in 1960-1973, increasing to 28% in 1975-1979, 31% in 1980-1989, and 36% in 1990-1995 (OECD Historical Statistics database). In the period 1976-1997 the proportion of females older than 16 years who were economically active rose from 28% to 40% (figure 2.5). The rate of integration of women in the labor force was strongly linked to the business cycle, with two peaks clearly coinciding with the years of high GDP grow, the early seventies and the late eighties (figure 2.6). The labor market in Spain appears to be specially flexible for women, pulling them in when labor is needed and expelling them quickly when the labor demand drops.

In summary, beginning in the mid- and late seventies the slump in the Spanish economy—part of a general economic downturn in Western Europe—was compounded by the return—probably throughout the late seventies and early eighties—of around a million Spanish immigrants formerly working in Germany and other European countries. The significant growth in population of working age and the increasing participation of women in the labor market were extra factors explaining the massive joblessness rate—more than 30% among active women and 20% among active men in 1994 (figure 2.5). In the early nineties Spain was the OECD country with the highest and most persistent unemployment. A well-known Spanish economist, Francisco Mochón, estimated in 1992 that the Spanish NAIRU was 17% (Román 1997:5-6), a rate that seems, perhaps, somewhat too large.

National mortality rates.[1] Following a dramatic drop in mortality during the fifties, in the 1960s the Spanish crude death rate did not show any clear trend (figure 2.7). From the early seventies through the early eighties crude mortality dropped, coinciding with the political transition and the upsurge in unemployment. Starting in the early eighties (the period of interest for this investigation)

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[1] In demography and epidemiology "death rate", "mortality" and "mortality rate" are generally used as strict synonyms. Crude mortality is the ratio of deaths throughout the year to midyear population, and reflects the real rate of dying in a population. Since crude mortality is strongly dependent on the age-structure of the population, age-specific death rates are often used in epidemiology and demography. Computation of age-specific mortality requires data both on deaths and population in the corresponding age-interval. Age-standardized mortality (age-adjusted mortality is a strict synonym) refers to mortality data for a whole specified population (can be sex-specific, for instance), keeping constant the age-structure of that population.

throughout the end of the century mortality had several oscillations overimposed on a general increasing trend.

A comparison of the paths of crude mortality and unemployment in Spain during the last three decades of the 20th century does not suggest any clear relationship between both variables, though the drop in mortality with rising unemployment in the period 1973-1982 and the coincidence of the 1991 peak in mortality with a trough in unemployment could be interpreted with some phantasy as suggestive of a procyclical pattern of mortality, with mortality oscillations mirroring those of unemployment like recent US data seem to reveal (figure 2.8). At any rate, crude mortality is largely dependant on the demographic structure and, in fact, the trend of the age-standardized mortality of Spaniards was declining all along the 1980s and 1990s (figure 2.9). In the case of Spain in this period the comparison of the oscillations of age-adjusted mortality with the level of economic activity as proxied by GDP growth does not provide any obvious pattern (figure 2.10).

So it would be wrong to consider that the upsurge in crude mortality from the eighties reflects a general decay of the health of the Spaniards. Crude mortality is an average of age-specific mortality rates, weighted by the proportions of population in each age stratum. Since the higher the age, the higher the age-specific death rate, the crude mortality usually goes up when the proportion of population in advanced age grows, even with age-specific death rates being stationary or declining at all ages.

In fact, most (but not all) of the age-specific death rates actually declined throughout the eighties and

nineties (figures 2.11 to 2.14), as did the age-standardized mortality (figure 2.15). Life expectancy, which is independent of the age-structure but incorporates age-specific death rates for all ages, was 72.5 years for males and 78.6 for females in 1980-1982, went up to 73.2 and 79.7, respectively, in 1985-1986 and 73.4 and 80.5 in 1990-1991 (United Nations, undated). Age- and sex-specific death rates show severe increases in mortality in young age-groups starting in the early 1990s and peaking in the early or mid 1990s (figures 12 and 14). However, because of the large weight of old ages in crude mortality the small deviation upward of mortality in 1990-1991 in these groups (figures 11 and 13) translates into the prominent peak in crude mortality (figure 2.7) and the bump in age-standardized mortality (figure 2.15) in 1990-1991.

Data. Data for the statistical models in this paper are demographic and economic statistics for the 50 Spanish provinces in the period 1980-1997. The 50 provinces analyzed in this paper—including 47 provinces in the Iberian peninsula, two provinces in the Canary Islands and the one in the Balearic Islands—amounted to 39.2 million people in 1997, more than 99.5% of the total Spanish population. The only excluded populations are only those of the North African towns of Ceuta and Melilla, colonial enclaves (totaling around 135,000 inhabitants) with special political status and which do not form part of any province.

The period of data observations (figure 2.1) that were used in the regressions comprises 18 years. The period begins with a deep recession in the early eighties. GDP

growth was almost zero. From there economic growth recovered to reach its maximum in 1987, although this was accompanied by a rise in unemployment which reached 21% in 1985. After 1987 growth dropped precipitously. Unemployment, which had dropped to 16% in 1990-1991, rose to nearly 25% in 1995. After 1993 economic growth recovered, finally bringing down unemployment in the second half of the 1990s. In summary (stylized facts) the period was characterized by a 1980-1987 expansion, a 1988-1993 contraction, and a 1994-1997 expansion.

The 18 year period of the study includes the 12 years in which the Spanish Workers Socialist Party (PSOE) was in power—with Felipe González as Prime Minister—, and also a few years of more conservative parties in power, before 1983 (UCD governments) and after 1996 (People's Party governments).

Statistical data for the study come from the TEMPUS database of the Spanish Instituto Nacional de Estadistica (INE). Crude data were directly downloaded from the INE website (http://www.ine.es) and were processed with Excel and analyzed with SAS. Total mortality rates and eight cause-specific death rates (cardiovascular disease, cancer, respiratory disease, infectious disease, external causes, traffic injuries, suicide, homicide) were computed from crude data (annual number of deaths in the province), for the whole population and for males and females separately. The denominators of the mortality rates were also taken from TEMPUS, which provides intercensal estimates of population for each province and each year in the last decades. Census are taken in Spain in ten years intervals. Specific data for population in various sex and age groups

were used to compute several indicators of age structure: the proportion of population under five years and the proportions over 64, over 74 and over 84.

Unemployment rates and employment ratios, both at the province and national levels, were used as indicators of economic activity. Data on unemployment and rates of activity derived from the so called *encuesta de población activa* (EPA), i.e. survey of active population, were taken directly from TEMPUS, which provides unemployment rates for each quarter at the national and province levels and both men and women. The annual average unemployment used as covariate in the regressions is the mean of the four quarterly data included at TEMPUS for each year.

Unemployment rates are computed in Spain as a percentage of those who answer in the EPA to be working in a paid job or looking for one. Activity rates are computed as the percentage of those older than 16 who are working or looking for a job. So the product $(1 - u) \cdot a$, where u and a are the rates of unemployment and activity, respectively, is the proportion of employed among all those older than 16. This employment ratio was computed from the original data and was used in some regressions.

Province real GDP per capita was included as covariate in some regressions and can be interpreted as an indicator of average income and also as an indicator of economic activity. Data on province GDP per capita adjusted for inflation were computed from the 50 series of province GDP at nominal prices taken from the TEMPUS database and a price deflator. This deflator was computed connecting two series of price deflators taken from TEMPUS: a consumer price index series comprising 1939-1992 (with 1983 as base year) for

the province capitals, and a CPI series comprising 1993-2001 (with 1992 as base year) and referring to the whole province.[2]

Epidemiological and statistical considerations. The criticisms against many previous studies of the relationship between mortality and business cycles clearly suggest that investigations based on single series of national mortality data have important limitations (Kasl 1979). In this type of studies a large number of explanatory variables—required to avoid spurious results due to omitted variables or, in epidemiological jargon, confounding—is often used to predict death rates for a series of only a few decades. So the number of covariates can be quite close to the number of observations, which posits grave questions regarding the statistical strength of the results. On the other hand, longer time series may be considered inappropriate for the analysis, as changes in regime could lead to different relationships between the variables in different sub-periods (McAvinchey 1984). In replications (Søgaard 1992), the longer the series of mortality rates the more likely was to get statistically significant coefficients relating mortality to a stationary series randomly produced. On the other hand, any lengthy time series is likely to suffer from substantial omitted variables bias (Ruhm 2000).

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[2] **The two series were just connected by a simple transformation of the CPI 1993-2001 to the base of the other series. To check the compatibility of the two CPI series I just graphed the resulting series for the 50 provinces. All them revealed a smooth growth with some weak fluctuations. I did not observe any kind of inflexion or singularity around 1992-1993.**

One way to overcome to some extent the problems of traditional time-series analysis is to use panel data. With a sufficient number of units to be analyzed statistical techniques can be used to take out time and geographical effects, making spurious results due to misspecification or omitted variables less likely. Panel models with fixed effects reduce the probability of bias coming in cross-sectional analysis from characteristics spuriously correlated across locations with the dependent variable. Panel models reduce also the likelihood of bias obtained from time-series analysis because of characteristics spuriously correlated with the dependant variable over time (Ruhm 1996). These considerations are the base for the general approach taken in this investigation, which uses the 50 Spanish provinces as units to provide the panel data to be analyzed.

Death rates are a ratio of two counts, deaths and population. Counts follow the Poisson distribution, which is asymmetrical and with variance equal to its mean (Zar 1984). As ratios of counts, death rates are not normally distributed. The logarithmic transformation of the rates improves the normality of the rate distribution, and it is a common way to "normalize" a distribution skewed to the right (Cody & Smith 1997). For this reason the dependent variable in most of the regressions in this paper is the natural log of the death rate. On the other hand, a logarithmically transformed death rate d/p is considered to be normally distributed with variance $1/E(d)$, where $E(d)$ is the expected number of deaths (Robertson & Ecob 1999). Since $E(d)$ is proportional to p, the variance of the logarithmically transformed death rate is inversely

proportional to the population size, ranging in the Spanish provinces from 100,000 to 5 million. This implies heteroscedasticity of the dependent variable. To compensate for it observations are weighted by the square root of the denominator of the death rate, a procedure considered accurate enough as to largely reduce heteroscedasticity (Ruhm 2000). Regressions with untransformed death rates (i.e. death rates in levels) are also computed.

Statistical models. The relationship between economic conditions and mortality is analyzed using fixed-effect models in which (transformed or untransformed) province crude death rates are regressed on vectors of variables for economic and demographic conditions, time-invariant province effects, province-invariant national time effects (year effects) and province-specific time trends. Regressions with both death rates and explanatory variables transformed into first differences or relative are also presented.

Five basic specifications for the regression models are used. All specifications include dummies for province effects and two demographic variables—the proportion of population under five and the proportion over 64—to adjust the results for age. Economic conditions at the province and the national levels are included in different combinations to yield five specifications, as follows.

In specification (*i*)

$$\ln M_{pt} = \psi_t + \pi_p + U_{pt} \cdot \beta + J_{pt} \cdot \gamma_1 + V_{pt} \cdot \gamma_2 + \varepsilon_{pt} \qquad [1]$$

ψ_t represents nationwide time effects (year effects), π_p are province specific time-invariant effects, U_{pt} is province unemployment as proxy for economic conditions, J_{pt} and V_{pt} are the age-structure or demographic variables (*jóvenes* and

viejos) and ε_{pt} is the error term. Specification (*ii*) adds to (*i*) the province real GDP per capita (I_{pt}) as an extra indicator of economic conditions:

$$\ln M_{pt} = \psi_t + \pi_p + U_{pt}\cdot\beta_1 + I_{pt}\cdot\beta_2 + J_{pt}\cdot\gamma_1 + V_{pt}\cdot\gamma_2 + \varepsilon_{pt} \quad [2].$$

In specifications (*iii*), (*iv*) and (*v*) the time-effect dummy is suppressed, so that results are adjusted for across-provinces influences but not for over-time ones. Specification (*iii*)

$$\ln M_{pt} = \pi_p + U_{pt}\cdot\beta_1 + I_{pt}\cdot\beta_2 + J_{pt}\cdot\gamma_1 + V_{pt}\cdot\gamma_2 + \varepsilon_{pt} \quad [3]$$

only differs from specification (*i*) in the absence of the time-effect dummy. Specifications (*iv*) and (*v*) model the effect of changes in the national economy on mortality by including yearly national unemployment as a covariate. Specification (*iv*) includes only national unemployment (U_t) as an indicator of economic conditions:

$$\ln M_{pt} = \pi_p + U_t\cdot\beta + J_{pt}\cdot\gamma_1 + V_{pt}\cdot\gamma_2 + \varepsilon_{pt} \quad [4]$$

while specification (*v*) includes both national unemployment (U_t) and province unemployment (U_{pt}) as proxies for the economy:

$$\ln M_{pt} = \pi_p + U_{pt}\cdot\beta_1 + U_t\cdot\beta_2 + I_{pt}\cdot\beta_3 + J_{pt}\cdot\gamma_1 + V_{pt}\cdot\gamma_2 + \varepsilon_{pt} \quad [5].$$

In a second batch of regressions, results are adjusted for province-specific time trends by adding to each of the five basic specifications the term $\tau_p\cdot t$, in which τ_p is the slope of a province-specific time trend. In a third batch the five basic specifications are used for regressions with the untransformed death rate as dependent variable. Therefore, a total of 15 regression specifications are examined for each of the mortality rates studied (all-cause and cause-specific mortality).

The five basic specifications model province death rates as functions of the demographic structure and

economic conditions (unemployment and GDP per capita) of the province. Specifications (*i*) and (*ii*) adjusting for over-time effects by using year dummies. In specifications (*iii*), (*iv*) and (*v*) no year dummies are included. In specifications (*iii*) and (*iv*) only province unemployment and national unemployment, respectively, are used as proxies for economic conditions. In specification (*v*) both province and national unemployment are covariates. The five specifications with province-specific time trends control the basic specifications for potential time trends with different slope in each province.

In a second set of 15 regressions, unemployment rates both at the province and national levels are substituted by a pair of sex-specific unemployment rates. In a third set of regressions, sex-specific unemployment rates are used as covariates, with a sex-specific death rate (for males *or* females) as dependent variable. In a fourth set of regressions employment ratios instead unemployment rates are the proxies for economic conditions.

Models with fixed-effects for the provinces (all specifications fitted) provide estimates of the impact on mortality of within-province variations of economic conditions. For these estimates of the impact of the economy on mortality being better than those derived from national-time series analysis the fluctuations of the proxy for the economy must be in a substantial degree independent across provinces. The correlations between the national unemployment rate and the 50 province unemployment rates in the period 1980-1997 (table 2.1) show that there is a substantial degree of independent fluctuation of unemployment across the provinces (*R* ranging from 0.30 to

0.96 for the 50 provinces). The correlations between other indicators of employment and unemployment at the national and province level in the whole population or in men and women separately show also a considerable degree of independent fluctuation in these variables. The correlation between the two sex-specific province employment ratios was negative in 36 provinces.

The global correlation between province output (proxied by province GDP per capita) and province unemployment in men is only -0.36 and negative in only 19 provinces. Female unemployment correlation with province output is 0.01 and negative only in 4 provinces. The global correlation between province output and province employment ratio in males is 0.14 but the correlation is negative in all the 50 provinces. The correlation between province output and province employment ratio in females is 0.32 and negative only in 8 provinces. Therefore rising province output is weakly associated with falls in unemployment for males and is not associated with unemployment reduction in females. On the other hand, province output is weakly inversely associated with men employment but tends to be positively associated with female employment. Therefore male unemployment is not a very good indicator of province economic growth though it is much better than female unemployment.

Given the considerable degree of independent variations in these sex-specific indicators of employment and unemployment, their use as explanatory variables in regressions to predict province mortality seems to be a step in the direction of improving the estimates of the impact of "the level of economic activity" on mortality.

The use of province-invariant fixed-effects dummies for time (year) provide estimates of the impact of the economy on mortality that are adjusted for nationwide time effects. The estimates derived from specifications in which time dummies are included must be free of biases due to the presence of linear or non-linear trends that affect the mortality data in the same way across provinces. The specifications in which province-specific time-trends are included imply an extra step to eliminate spurious effects due to time trends, in this case linear trends that are allowed to have different slopes for each province.

However, it is worth noting that the terms $\tau_p \cdot t$ (in which τ_p represents the slope of a province-specific time trend) and ψ_t (representing nationwide time effects, i.e., year effects) could be sources of overadjustment. Overadjustment implies adjustment for a variable so closely related to the variable of interest that no variability in the latter is allowed (Szklo & Nieto 2000). The dummies for year and the province-specific time-trends might eliminate all variability in unemployment, thus obscuring a true association. In other words, time-related changes in unemployment or employment levels could be the process driving time trends. Adjustment for time trends could thus obscure a true effect of unemployment or employment ratios and this possibility must be taken into account when interpreting the regression results.

Models with lagged effects. What is known about many modern causes of death (heart disease, malignancies, chronic lung disease) makes quite likely that economic conditions could have a *lagged* impact on mortality. It can be assumed that the state of the economy has both

contemporaneous and lagged effects on death rates. Therefore regressions were run including both unlagged and lagged terms for the economic indicators.

Regressions with differences. There is positive first autocorrelation both in death rates (0.947 in this sample) and in the economic and demographic indicators used as covariates (0.947 for unemployment and 0.988 for province output per capita). The inclusion of dummy variables for province and year in the panel data analysis attempts to solve this problem. Regressing first differences or the relative change of the dependent variable on first differences or the relative change of the explanatory variables is another way to deal with autocorrelation. The logarithmic difference of the death rate ($\ln M_{pt} - \ln M_{pt-1}$) and first differences of the death rates ($M_{pt} - M_{pt-1}$) were regressed on first differences of the explanatory variables. The relative change of the death rate ($[M_{pt} - M_{pt-1}]/M_{pt-1}$) was also regressed against the relative change of the explanatory variables. The time fixed-effect of the differentiated equation [1] will be $\psi_t - \psi_{t-1}$, therefore the regressions were run with dummies for year in the specifications (*i*) and (*ii*) and without year dummies for all the other specifications. No province dummies were used for these specification as the differencing makes redundant the term π_p (the province fixed effect). However, the result of differencing the specifications including province-specific time trends implies a province dummy, since $\tau_p \cdot t - \tau_p \cdot (t-1) = \tau_p$. Therefore specifications with province dummies τ_p and with or without year dummies were computed as differenced versions of the specifications with province-specific time trends.

III — Results

General averages for the whole 18 year period and the 50 provinces for total, cause-specific and sex-specific death rates were computed per 100,000 population, and weighted by the province population (table 2.2). The average crude mortality in the Spanish provinces during the period 1980-1997 was 827 per 100,000.[3]

Cardiovascular disease, cancer and injury deaths (largely due to traffic) are the three most important causes of death in affluent countries, accounting for a large proportion of total mortality. In this particular data set cardiovascular disease, cancer and traffic account for 67% of total deaths. The causes of death included in this study—cardiovascular disease, cancer, respiratory disease, infectious disease and external causes—account for 81% of total deaths (table 2.2). Suicides (0.7% of total mortality) and homicides (0.1% of total mortality) are a quite small subset of the so-called mortality due to external causes (5.1% of total mortality), but their special nature justifies studying them separately.

In each category of causes of death sex-specific crude death rates are higher for men, except for cardiovascular disease, where the higher rate for women may result from the fact that cardiovascular mortality is

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[3] Taking into account that crude death rates depend largely on the demographic structure this compares quite well for instance with US crude mortality, that was estimated to average 880 per 100,000 in 1972-1991. The favorable comparison is in spite of a younger population in the US, where 7.5% were aged 0-4 years and 11.5% over 64 in 1972-1991 (Ruhm 2000) against 6.0% and 13.3%, respectively, in Spain, in 1980-1997 (table 2).

strongly related to age, and overall women tend to be older than men.

In spite of the rapid aging of the Spanish population, cause-specific death rates for cardiovascular disease show a light long-term declining trend in the period of the study (figure 2.16), with the decline somewhat more pronounced for male cardiovascular mortality.

Cancer mortality (figure 2.16) grew steadily over the study period. This increase was more pronounced in men, among whom cancer mortality nearly equaled cardiovascular disease mortality in the late 1990s.

Respiratory disease mortality (figure 2.16) also reveals a slight increasing trend, both for males and females.

Mortality due to infectious disease (figure 2.17) shows a steady rise from 1986 in men and from 1982 in women.

Traffic-related mortality (figure 2.17) shows a prominent peak in 1989 and clear troughs in 1982 and 1994. This pattern coincides almost exactly with the GDP growth peak (1987) and troughs (1980, 1993) in the period.

Suicide mortality increased rapidly during the whole period, doubling between 1980 and 1997 for both men and women (figure 2.18). There are strong upward deviations in male suicide in 1984, 1986 and 1996.

Homicide rates (figure 2.18) show weak secular trends, decreasing for males, perhaps lightly increasing for females.

General mortality and cause-specific mortality as predicted by general indicators of economic activity. Using the whole

set of 900 observations (18 years over 50 provinces), estimates of the province unemployment effect on total death rates were negative and statistically significant for all the specifications (table 2.3, upper panel). Estimates of the "unemployment effect" with the logarithm of mortality as dependent variable ranged from -0.0011 to -0.0035, implying a fall in mortality ranging from 0.11% to 0.35% per percentage point of increase in province unemployment. Estimates from the regressions with mortality in levels imply that one percentage point increase in province unemployment is associated with a fall of between 1.13 and 2.12 units in mortality. As this is on average 827 per 100,000, these estimates imply a fall in mortality between 0.14% (= 1.13/827) and 0.26% (= 2.12/827). This is consistent with the estimates from the regressions with death rates logarithmically transformed. In gross terms it can be said that a percentage point increase (decrease) in unemployment will "produce" a decrease (increase) between one and three tenths of 1% in mortality. An increase of 5 percentage points in the unemployment rate (a quite moderate one for Spanish standards), for instance from 10% to 15%, will be associated with a fall between 0.5% and 1.5% in mortality. In absolute terms, since the population of Spain is 40 million, an increase in unemployment of 5 percentage points would "save" between 1600 (i.e, $0.005 \cdot [827/10^5] \cdot 40 \cdot 10^6 = 1654$) and 5000 (i.e., $0.015 \cdot [827/10^5] \cdot 40 \cdot 10^6 = 4962$) annual lives. Assuming linear trends for the death rate and the unemployment rate in the period 1980-1997, the average absolute value of the deviations of the actual rates from the trend are 1.16% for the death rate and 2.2 percentage points for the unemployment rate.

Therefore the average variation in mortality "caused" by the business cycle change in unemployment would be between 2.2 · 0.1% = 0.2% and 2.2 · 0.3% = 0.66%, which represents between 17% and 57% (since 0.2/1.16 = 0.17 and 0.66/1.16 = 0.57) of the mortality fluctuation.

The set of observations was split to study the robustness of the results (table 2.3, lower panel). In the "split-sample" estimates the negative signs for province unemployment remain in each specification but one, though the level of statistical significance diminishes, which is logical given the reduction in the number of observations and the subsequent increase of standard errors. The estimates show some strong oscillations in size, but most of them are not very different in size from the estimates of the "full sample." The estimates for the 11 provinces with population under 300,000, for the 15 provinces with lowest GDP per capita in 1990 and for the time-split period 1980-1988 (a period in which at the national level unemployment was always rising except two years) are indistinguishable from zero. However, in the case of the split period sample the addition of just one year (1989) causes the unemployment coefficient estimate to jump from -0.0005 to -0.0012 (with a standard error of 0.0007, which implies that the estimate is statistically significant at the 90% confidence level, though not at the usual 95%). Estimates for selected sets of provinces seem to suggest that the effect might be greater in more populated areas (estimates between -0.30% and -0.41%), more industrialized regions (estimates close to -0.5%) or richer provinces (estimate close to -0.4%).

Estimates for national unemployment effects are negative and statistically significant in those specifications where province unemployment is not included (specification (*iv*), table 2.3). In those specifications where unemployment at both national and province levels are included (specification (*v*), table 2.3) the coefficient estimates for the national unemployment are usually not statisticaly significant and are no longer clearly negative.

When cause-specific death rates are the dependent variable (table 2.4), the results are quite consistent with those for general mortality. Unemployment coefficients are consistently negative, though they are not statistically significant for cardiovascular disease mortality in two of the three specifications shown, for infectious disease mortality in one of the three and for homicide in the three specifications.

Sex-specific death rates and sex-specific economic indicators. When sex-specific death rates are the dependent variable (table 2.5) the results change very little. Province unemployment estimates are quite similar to those obtained when the general (male and female) mortality is the dependent variable (table 2.3): they remain negative and statistically significant in most of the specifications.

The regressions for sex- and cause-specific death rates (table 2.6) yield results similar to those for cause-specific mortality in the general population (table 2.4), and provide additional evidence of the robustness of the results. The coefficient estimate for province unemployment

is negative in all the regressions except in one of the specifications for female suicide. Estimates for external causes and its traffic mortality subset are large and strongly statistically significant but the fit of the regression is small in terms of $\bar{R}^2$ due to the low contribution of the age-structure variables to explaining this type of mortality. For cardiovascular disease, cancer and external causes, estimates of the unemployment effect are larger for female mortality than for male mortality while the opposite is true for infectious disease, traffic and suicides.

Results for sex-specific economic indicators (table 2.7, upper panel) provide further evidence of the relationship between death rates and economic activity. When the total death rate for men and women is the dependent variable, the effect of male unemployment rates is consistently negative and statistically significant most of the times. Estimates of the female unemployment effect are much lower and generally not statistically different from zero. Coefficient estimates for the effects of male and female national unemployment are always highly significant, though interestingly, opposite in sign, with those for female unemployment being negative and those for male unemployment positive. This may be a direct consequence of the high correlation between the two province sex-specific unemployment rates (median 0.65 for the 50 provinces, table 2.1), but perhaps may be related too with the quite different dynamics that economic activity imposes on the unemployment rate of men and women. Comparing the estimates of the effect of total unemployment rates and sex-specific unemployment rates (tables 2.3 and

2.7, upper panels) on total mortality, it is quite striking that the effect of overall unemployment on total mortality is largely reflected by male unemployment.

Results with male and female death rates (table 2.7, mid and bottom panel) as the dependent variables are similar to those reported above. The effects of province male unemployment are negative and larger than the effects of province female unemployment, and the effects of national unemployment are negative for male unemployment but positive for female unemployment

Death rates and employment ratios. If sex-specific unemployment rates have a statistically and demographically significant negative effect on mortality because they serve as proxies for the general level of economic activity (measured by the lack of involvement of "active" people of both sexes in economic activity), one would expect effect estimates to be even larger for an indicator such as the employment ratio, which is the proportion of persons with paid jobs or looking for one of all those older than 16 and is therefore referred to a more general population than the unemployment rate (this does not take into account those not looking for paid work because they are retired, exclusively involved in homework, students, discouraged about finding a worthwhile job, or any other reason). Therefore, one would expect coefficient estimates for sex-specific *employment* ratios to be larger in absolute value than those for sex-specific *unemployment* rates, and opposite in sign. This is exactly the case (table 2.8, compared to table 2.7). The impact of the province male employment ratio in males is strongly positive, for general

mortality and both male and female sex-specific mortality. On the other hand, the impact of province female employment ratio hardly ever differs from zero, even when the outcome is female mortality.

Using employment ratios to predict sex-specific mortality due to specific causes of death (tables 9 and 10) provides extra evidence of the robustness of the results. It seems that for many causes male employment ratios are much better predictor of both male and female mortality than female employment ratios.

Models with lagged effects. In the models with lagged effects (table 2.11) the specifications without province-specific time trends show a weakening toward zero of the effect of province unemployment on mortality as the time lag increases. However, in the specifications with province-specific time trends the coefficient estimates for the province unemployment lagged three or four years become positive and even statistically significant in some of the specifications. In the specifications (*iv*) and (*v*) where national unemployment is included, a bizarre pattern appears in the coefficient estimates for national unemployment both in the regressions with or without province-specific time trends. The sign of the coefficient estimate for unemployment is negative when unemployment is unlagged, positive when lagged one year, negative when lagged two years, positive when lagged three and negative when lagged four. The absolute value of these estimates is larger when the time lag increases and all of them are statistically significant. This pattern was also observed in a similar analysis of data from the states of the US

(Ruhm, personal communication). These estimates with switching signs may reflect instability of the coefficients rather than real effects. The high first order autocorrelation between unemployment rates (0.947 in this sample) perhaps can be the source of these results.

Regressions with differences. The unweighted specifications (left column, table 2.12) with mortality in first differences (logarithmically transformed or in levels) or in relative change show very poor fit (R^2 values drop considerably compared to non differenced equations, with R^2 between 0.3 and 0.4 and $R^2 < 0.1$ in regressions with and without year dummies, respectively). In most of these specifications the demographic variables have the expected (negative for the proportion under 5 and positive for the proportion over 64), but they are not statistically significant. The coefficient for unemployment is negative in 20 of the 24 unweighted specifications, but is not statistically significant in any of them. This can be at least partly due to the fact that annual changes in mortality show negative autocorrelation (- 0.201 in this sample), due probably to the fact that the death of many high-risk people one year leaves few high-risk people to die next year—this has been sometimes called "harvesting effect" (HEI 1997). The negative autocorrelations tend to enlarge standard errors (Søgaard 1992) reducing the efficiency of the estimates.

The Glejser test can provide information on the existence of heteroscedasticity and its form (Kotsotiannis 1977:186-189). When applied to the unweighted regressions with differences, the Glejser procedure suggests that the

residuals are correlated with the covariates, mostly with the second power of the differenced proportion of population over 64. A way to compensate heteroscedasticity is to use weighted OLS weighting by the inverse of the variance of the residuals, which amounts to using GLS (Gujarati 1995:362-365). The 30 specifications of weighted regressions (right column of the three sections of the table 2.12) show a much better fit than the unweighted ones (in terms of R^2). Only one has a "wrong" and statistically significant sign in the estimates of the age-structure variables and 23 had "right" signs in both demographic variables. Among these 30 regressions 24 include an estimate for province unemployment, which is negative in 20 specifications, 18 of them are statistically significant. These results seems to confirm that the coefficient estimate for the change in province unemployment is negative and therefore an increase in unemployment is associated with a drop in mortality.[4]

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[4] I also computed regressions weighted with the inverse of the square of the first difference in unemployment (results not shown). Though these regressions have better fit than the unweighted ones, most of them yield demographic effect estimates with "wrong" sign, so I discarded them. The sign of the unemployment estimate in most of these regressions was negative, but significant only in a few of them.

Taking into account the large differences in the size of the coefficient estimates in this set of regressions it does not seem worthwhile to pay attention to them beyond making an inference on its sign. But making heroic assumptions and assuming that the regressions weighted with the term related to the proportion or people over 64 (right column in table 12) produced unbiased estimates the size of the effect is quite consistent with that derived from the regressions with non-differenced variables. When the logarithmic difference (table 12, section I) is used, the coefficient estimate represents how the absolute change in province unemployment impacts on the relative change in mortality, since $\ln M_{pt} - \ln M_{pt-1} = \ln M_{pt}/M_{pt-1} = \ln (M_{pt-1} + M_{pt} - M_{pt-1})/M_{pt-1} = \ln [1 + (M_{pt} - M_{pt-1})/M_{pt-1}]$. When the fraction inside the brackets is small this is approximately equal to $(M_{pt} - M_{pt-1})/M_{pt-1}$, which is the relative change in mortality. If the coefficient estimate in the weighted regressions with logarithmic differences is approximately in the interval between -0.0013 and -

Real GDP per capita and mortality. The estimate of the effect of province real GDP per capita on general mortality or sex specific mortality of males or females was positive and statistically significant in 17 of the 18 specifications in which no province-specific time trends were included (tables 2.3, 2.5, 2.7 and 2.8). However, it was negative but not significant in all 9 specifications with province-specific time trends. With respect to the size of the effect, in specifications without province-specific time trends with death rates in logarithm an increase of one unit in province real GDP per capita is associated with an increase of about 1.1% in general mortality (1.02% for male mortality, 1.25% for female mortality, tables 2.3, 2.5, 2.7, and 2.9, right columns). Since GDP is measured in hundreds of thousand of 1983 pesetas, an increase of 100,000 pesetas in GDP per head (equivalent to about 14% of the average province GDP per head) is associated with a 1.1% increase in mortality (which is equivalent to a 9.1 unit increase in deaths per

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0.0047, then a percentage point increase in unemployment will reduce mortality between 0.1% and 0.5%. When the variables are in first differences (table 12, section II) the coefficient estimates for province unemployment are between -1.81 and -4.28, i.e., an increase of one percentage point in unemployment would reduce mortality between 1.81 and 4.28 per 100,000. Since the average mortality in the period is 827 per 100,000, this represents a reduction between 0.2% and 0.5%, quite consistent with that obtained from the specifications with logarithmic differences. When the variables are in relative change (table 12, section III), a unit increase in unemployment (a 100% increase in unemployment rate) is associated to a reduction between 2% and 9% in mortality. So an augmentation in one percentage point in the unemployment rate from 10% to 11% represents a 10% increase and will associate with a fall in mortality between 0.2% and 0.9%. The predicted changes derived from the three different sets of specifications are quite similar. However these estimates of the effect of absolute or relative differences in unemployment are almost double in size of those derived from the equations with non-differenced variables (table 2).

100,000). When mortality is measured in levels a unit increase in province GDP is associated with an increase of approximately 9.3 units in mortality (tables 2.3, 2.5, 2.7 and 2.8, right columns). Therefore the effect estimates from specifications with mortality log-transformed and from specifications with mortality in levels are quite consistent (its "large" size is only due to the units in which province GDP per capita is measured).

In regressions with cause-specific mortality as the dependent variable province GDP had positive and statistically significant effects on general and sex-specific mortality due to cancer, respiratory disease and infectious disease (tables 2.4, 2.6, 2.9 and 2.10). The GDP effect was negative for cardiovascular mortality and deaths due to external causes but only statistically significant for total (male plus female) mortality and male mortality. Province GDP has negative effect on mortality due to traffic injuries, but the estimate is only significant in one of the two specifications with male mortality as outcome. GDP effects were not statistically significant for suicide or homicide in any specification, but the estimate was negative in all specifications for suicide.

In the weighted regressions with variables in differences the effect of GDP was consistently negative and statistically significant.

It seems that the effect of province GDP per capita on mortality is not as robust as that of unemployment, with effects often gone in many specifications. The possible reasons for this behavior will be discussed below.

IV – Discussion

The persistent negative signs of the coefficient estimates for the effect of unemployment (or positive signs for the effect of employment) across a wide array of specifications and specific causes of death seem to provide substantial evidence of an increase in mortality when unemployment decreases. In other words, mortality is procyclical.

It should be noted however that these results reflect only ecological relationships: that age-adjusted mortality is higher when more people are involved in paid economic activities. These analyses do not provide any information on the effects of business fluctuations on the health status or the death rate of the employed or unemployed. Neither do they provide evidence on the effect of unemployment per se on health (i.e. the individual level effect of being unemployed). In these analysis unemployment serves only as a proxy for the state of the economy.

The size of the effect—a reduction of mortality between 0.11% and 0.35% per percentage point of increase in province unemployment, not taking into account the larger estimates from the difference equations—is approximately half of that found by Ruhm (2000), whose estimates from similar analysis for the US states ranged from 0.41% to 0.69%. Estimates for selected sets of provinces (table 2.3, lower panel) show a greater effect of unemployment in more populated areas (estimates between -0.30% and -0.41%) and in richer provinces (estimate close to -0.4%) or more industrialized regions (estimates close to -0.5%).

Comparing the estimates for the effect of province unemployment on general mortality and sex-specific mortality (tables 2.3 and 2.5), unemployment predicts male mortality much better than female mortality. In the specifications with province-specific time trends unemployment effect estimates are statistically significant predictors for male mortality but they are only marginally significant or not significant for female mortality (table 2.5). This suggest that female mortality is more linked to province-specific time trends and less dependent of general conditions of the labor market than male mortality.

The fact that employment ratios are better predictors of sex-specific mortality due to specific causes of death (tables 2.9 and 2.10) provides extra evidence that mortality fluctuations are caused at least in part by accelerations and decelerations of the economy (of which employment is an indicator). The fact that province unemployment is a much better predictor of mortality than national unemployment when both are included in regressions is consistent with the notion that the impact on population health of local economic conditions should be larger than that of national ones.

Coefficient estimates for province GDP are generally positive for specifications without province-specific time trends, which is consistent with the results for unemployment. However the GDP effect disappears or changes its direction when province-specific time trends are introduced and is consistently negative in the regressions with differenced variables. If the significant relationship between province GDP and province mortality observed in the basic specifications and in the specification with

mortality in levels were due to secular trends in both economic output and mortality, the sign of the unemployment coefficient estimate would have to be negative, as age-adjusted mortality was going down and province real GDP per capita was going up throughout the period of study. The specifications in which province GDP is included have also year dummies that would have to avoid a spurious relationship like that; moreover, the sign of the estimate is positive, not negative, hence a spurious correlation due to trends seems unlikely. The positive sign of the effect estimates of GDP in regressions unadjusted for province-specific time trends is consistent with an association between increasing economic activity and rising levels of mortality. This effect, however, is not consistent with regressions with mortality and GDP in differences or relative change, in which the GDP estimate is positive but not significant in the unweighted regressions and negative, and generally significant, in the weighted ones.

It therefore appears that province output per capita is in general a less consistent predictor of mortality than unemployment rates or employment ratios. This could be due not only to the impact of province output on mortality being related to province time trends, but also to measurement error. Large measurement errors in province gross output seems more likely than large measurement errors in province unemployment. On the other hand while province output per capita would be an indicator of the general level of economic activity, unemployment rates, or even better, employment ratios, capture the degree to which the level of economic activity (paid work) directly affects

the population. Low levels of joblessness imply that the flow of labor supply to firms is close to dry, while high levels of unemployment imply large availability of human labor for paid activities, but also for unpaid activities, as are most household ones. So if mortality changes procyclically with the business fluctuations because of variations in the average level of stress—as Eyer and Sterling thought—, unemployment/employment indicators would be better predictors of mortality than gross output indicators.

In regressions for cause-specific death rates (tables 2.4, 2.6, 2.8 and 2.9) the large differences in R^2 show that the models explain quite different portions of the variability for different causes of deaths. Since the proportions of people under 5 and over 64 are included in all models, R^2 values are high when advanced age is a strong predictor of death due to that cause—cardiovascular disease, cancer and respiratory disease, in decreasing order (table 2.4). For all other causes the models predict less than 60% of the variability in the death rate—for female suicide and homicide R^2 is as low as 30%.

In general, the R^2 for the different models is much better for male mortality than for female mortality. Also, as mentioned, unemployment coefficient estimates for male mortality are less sensitive to time-trends adjustment than estimates for female mortality. This can be interpreted as evidence of internal consistency of the models. Given the stronger involvement of men in economic activity and the greater role that the economic plays in men's lives compared to women's lives it is to be expected that an economic-demographic model will predict mortality in males

better than in females (compare R^2 for the specifications predicting cause-specific mortality in tables 2.6, 2.9 and 2.10). Ruhm (2001) has found variations in indicators of medical problems associated to the business cycle more pronounced in women. In the present investigation the better fit of the models for male respiratory disease mortality may be related to the much greater participation of men in production (steel and iron industry, chemical plants, mining, construction) and consumption activities (smoking) that are linked to the development of respiratory diseases. Once a chronic pneumopathy is well developed after years of exposition to tobacco, occupational insults or both, the carrier—a man more likely than a woman—will be more susceptible to the cyclical influences of the economy on his airways and immune system, and so the probability of death of the chronic patient will fluctuate with the level of economic activity.

Do these results represent a spurious statistical association? Regressing crude mortality on unemployment and age-structure variables, as was done in this study, is equivalent to regressing age-adjusted mortality on age-adjusted unemployment. Since age-adjusted mortality was generally falling during the period of study (figure 2.9) and unemployment was generally rising (both at province and national levels), it could be argued that linear regression will show an average "negative" relationship, which will be a spurious one resulting from secular trends in both variables. This interpretation is not sound for several reasons.

First, the negative effect estimates for unemployment appear in all types of specifications, even in those with year dummies or year dummies *and* province-specific time trends (table 2.2). If the negative relationship between unemployment and mortality was entirely dependent on secular trends, it would be eliminated by these statistical procedures.

Second, the negative relationship is also present when first differences (or relative change) in mortality are regressed on first differences (or relative change) in the regressors (table 2.12).

Third, if the results were only due to a spurious association, the negative relationship would become positive when crude mortality is regressed on unemployment, as both variables show an increasing long-term trend during the study period. However, when this is done (table 2.13, section I) the negative signs for the unemployment estimates remain in most of the specifications, showing that there is also a negative relationship between unemployment and *crude mortality*.[5]

A fourth reason to reject the idea of a spurious association due only to secular trends in the outcome and the regressors is that these results are consistent with other results from other countries and time periods in which also appeared a link between economic upturns and mortality increases (Ogburn & Thomas 1922, Thomas 1925, Eyer 1977a, Hicks 1977, Adams as cited by Jin et al. 1995,

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[5] **Interestingly this negative relationship becomes positive when time adjusting terms are dropped from the model specifications (basic specifications (*iii*), (*iv*) and (*v*) and specification (*v*) with death rate in levels). This positive sign disappears when some age-adjustment is added (table 13, section II).**

Ruhm 2000, Ruhm 2001). Even Brenner would agree that mortality rises in times of economic expansion, though his interpretation is that this is only a lagged effect of the unemployment in the previous recession.

Are the regressions results adequately adjusted for the age-structure of the population? A possible criticism of the results is that adjustment for the age-structure of population is poor, as only the proportions under 5 and over 64 have been used for the age-adjustment. To examine this possible criticism some regressions were run with less age-adjustment (table 2.13, sections I and II) and with finer age-adjustment (table 2.13, sections III and IV). These regressions show that the results obtained with the two demographic variables used throughout the study are quite robust. The inclusion of the proportion of population over 74 (besides those under 5 and over 64), scarcely changes the results (compare section III in table 2.13 with upper panel in table 2.2), and even strengthens them, increasing the negative effect of unemployment. The inclusion of the proportion over 84 (table 2.13, section IV), did not substantially change the results and the coefficient estimate for this proportion was not statistically significant in a number of specifications.

Causal links for an increase of mortality during economic expansions. The reasons for the paradoxical phenomenon of mortality increasing with economic prosperity and falling in "bad times" need to be examined independently for each cause of death. Eyer and Sterling (Eyer 1977b, Eyer & Sterling 1977, Sterling & Eyer 1981) provided a

comprehensive theory to explain the increase in mortality associated with economic expansions. The basic notion is that during economic expansion increased levels of work-hours, consumption of noxious substances, increased pace at work, and weakening of social support due to migration and overwork generate a general increase in stress levels and a subsequent decrease in immunity. This effect is not limited to the population directly involved in paid labor and may also be compounded by procyclical changes in general pollution and exposure to occupational noxas. The consequence is an increase in the risk of death among persons with underlying chronic disease, as well as an increased risk of mortality in the population generally due to incident acute cardiovascular events, infections, "accidents" or other pathological episodes triggered by stress and low immunity. Evidence is available on several pathways in this complex web of causation.

The effect of business upturns on working population. The increased risk of death associated with job stress and fatigue has been extensively studied. Industrials statistics showing the relationship between fatigue and injuries at work had already been noted in 1914 by J. A. Hobson, who considered the number of hours worked without intermission as a valid index of fatigue, related both to weakening in attention and muscular control. Hobson also pointed out to experiments attesting that fatigue reduce the power of the blood to resist bacteria and their toxic products and argued about fatigue associated to boredom leading to alcohol consumption. In the last decades of the past century Karasek and Theorell developed a two-factor

demand-control model in which high job demands associated with low control generate the highest risk of mortality, particularly cardiovascular mortality (Johnson & Hall 1995, Marmot & Feeney 1996, Peterson 1994, Bloomberg et al. 1994). Blood pressure (Landisbergis et al. 1977) and catecholamine levels (Kasl & Wells 1985) have been proposed as mediators in this causal path.[6] The effect of work stress seems to be much more pathogenic when maintained throughout some time (House 1986). The relationship between excess hours of work and health symptoms seems clearly proved by a variety of studies (Sparks et al. 1997). Overwork, work strain and effort intensity are clearly linked to business fluctuations. During economic upturns, firms try to cope in the short-term with the increased business rate by raising the rate of work and demanding overtime from the employees. Indirect evidence of the increase in job strain during economic expansion is also provided by data on work injuries (Kossoris 1938, Robinson 1988, Robinson & Shor 1989), that are clearly procyclical (Arno 1984). Some data are suggestive of an effect of work stress increasing the harmful impact of physical, chemical or biological exposures (House et al. 1979)

The degree of overwork and job stress that can be generated by business upturns was recently news in the US. The Maine legislature aproved in 2000 a bill establishing a cap of 80 hours in any two-week period for mandatory overtime. The bill finally passed (after two vetoes by the

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[6] Kalimo et al. (1987) collect a number of papers in which pathways between occupational factors and disease are explored. Psychological and cardiovascular disorders related to work stress and overwork are prominent issues in several chapters of the book.

governor of former versions with overtime limits of 96 hours) after a worker died because of an "accidental" electric shock after working continuously two and a half days on a total of five hours of sleep. Washington state considered a similar bill, but it did not pass. West Virginia and Pennsylvania debated but did not passed bills that would allow workers to refuse overtime without being punished. Firefighters in Connecticut challenged the constitutionality of mandatory overtime arguing unsuccessfully that it violates the 13th amendment ban on slavery. Federal statistics showed that in the late 1990s and 2000 US workers were doing record levels of overtime and according to the Bureau of Labor Statistics the average employee worked two more hours per week in 2000 than in 1982. This however is likely to be an underestimation, because the surge of women—many of whom often work part time— in the labor force is likely to have pulled down the average (Walsh 2000). Biddle and Hammermesh (1990) have provided data showing that sleep decreases with increasing wages and that each hour of additional work reduces sleep by roughly 10 minutes. As hours worked are clearly procyclical this suggests that sleep is countercyclical—more so if we assume wages are also procyclical.[7] Reduced sleep can increase stress levels, decrease alertness and

[7] Keynes asserted in 1936 that "an increase in unemployment can only occur to the accompaniment of a decline of real wages." However, after decades of half-heartened assertions and inconclusive interpretations of the evidence about wages and business fluctuations, modern economics seems to be arriving to accept that real wages are procyclical (Blachflower & Oswald 1994, Abraham & Haltwanger 1995). Almost 80 years ago, examining empirical data Dorothy Thomas had concluded that as full prosperity emerges "the supplementary costs of doing business increase disproportionately to the rise in selling prices. Wages rises, there is full employment, and the efficiency of labour tends to decline" (Thomas 1925:5).

raise the risk of injuries in and out of work. Overwork and lack of sleep might also reduce sexual activity, that has been suggested as protective of the risk of death in middle-aged men (Davey Smith et al. 1997).

Data from Spain show that overtime and extra work fluctuate procyclically—they fell during the contraction of the late eighties and increased after 1991 (figure 2.19). There was also an increase in occupational injuries and occupational disease during the business expansion. The yearly number of cases of occupational disease, accidents commuting to work, and injuries during time at work in the period 1976-1991 (figure 2.21) show a clear inverse relationship with unemployment (Tapia 1994). In the period 1988-1995 the rates of occupational injuries and occupational injury deaths increased first between 1988 and 1991, then decreased between 1991 and 1995, and increased again in 1994-1995 (Boix et al. 1997). This three periods coincide exactly with phases of economic expansion, economic downturn, and economic recovery, respectively. Occupational health problems tend to increase when "the economy" improves. This is consistent with the regression results in this paper, as a large portion of traffic mortality and mortality due to other external causes is directly related to business activity, and this causes appear intensely procyclical in the regression results.

Traffic and business fluctuations. The procyclical character of traffic mortality was mentioned in passing by Pelzman (1975), but was clearly pointed out by Eyer (1977a) and documented by experts in injury epidemiology who noticed a strong correlation between US motor vehicle

deaths and the index of industrial production (Baker et al. 1964). In cross-sectional comparisons among regions rates of collisions and traffic injury mortality are directly related to traffic volume (HLDI 1985) and GNP (Söderlung & Zwi 1995), which is itself highly correlated with traffic volume. A direct relationship between traffic volume and child pedestrian deaths is present in time series studies (Roberts & Crombie 1995). In the US, traffic mortality closely follows the fluctuations of the industrial economy (figure 2.21). Fluctuations in traffic deaths over the year and over the week (NSF 1991) strongly suggest that a large portion of driving is done for recreational motives and this portion is the one most connected with alcohol consumption. The fall in consumption of alcohol during recessions (Ruhm 1995, 1999, Freeman 1999) and the decline in excess alcohol consumption when levels of unemployment are high (Catalano, cited by Blake Turner 1995) is another factor that explains the procyclical character of traffic fatalities.

In Spain, traffic injuries between the early 1980s and the late 1992 (figure 2.17) followed closely the economic expansion and contraction, peaking in 1989 coinciding with the trough in unemployment. Regression results (tables 2.4 and 2.6) show that one percentage point increase in unemployment associates with drops of 1.3% and 0.9% of male and female traffic mortality, respectively. The coefficient estimates for the province GDP per capita effect on traffic mortality are consistently negative (tables 2.4, 2.6, 2.9 and 2.10). Though GDP effects are not statistically significant for general population mortality, they are nearly significant when male traffic death rate is

regressed on province economic conditions (table 2.6, upper panel), and become significant when male traffic mortality is regressed on sex-specific employment ratios (table 2.9). This may be explained by the fact that poorer regions that are generally the rural areas have higher traffic mortality than richer urban areas, due probably to differences in the occupancy of vehicles—much higher in rural areas—, and speed—lower in urban areas due to congestion (Robertson 1992:128, NCIPC 1989:115-116).

The effect of business fluctuations on the elderly and the general population. The mechanisms linking traffic mortality to economic activity clearly affect the whole population, not only persons of working age. On the other hand, mechanisms involving overwork, excess strain and lack of sleep can explain why cardiovascular disease or occupational injuries are expected to vary procyclically in people of working age. However, the majority of deaths in a country like Spain occur in the elderly. For general mortality to have an association with the level of economic activity there must be pathways for the economy to impact on the risk of death of elderly people, who are often retired. These pathways may involve social support and immunity, exposure to chemical pollution and noise, and changes in behaviors such as diet, exercise and alcohol and tobacco consumption associated with prosperity.

Epidemiological studies have shown that the risk of death is directly related to the degree of social isolation and inversely related to the degree of social support (Cohen 1985, Seeman & Berkman 1988) and social integration (Siahpush & Singh 1999). Well integrated communities where

people have stable lives, a number of friends and significant social relations have lower levels of mortality compared to disintegrated populations (Berkman & Syme 1979, Fuchs 1974, Zweifel & Breyer 1997, Wilkinson 1996). The importance of social support is confirmed by the well established fact that marital status is strongly linked to health, with an increasing gradient in mortality from married to single, widowed and divorced. The lower mortality of married persons appears to be independent of risky behaviors such as smoking and drinking and is likely to be attributable to higher levels of material resources, lower levels of stress, and probably to the beneficial effect of an intimate relationship and the social support it provides (Wyke & Ford 1992). Social support also appears to buffer the detrimental effects of stress on health. For example, the detrimental effect of previous job strain on mortality in retired men is intensified by having weak social networks and social support (Falk et al. 1992). It has also been suggested that overwork can have detrimental effects on *other* family members through a reduction of psychological and homework support (Sparks et al. 1997).

In Spain extended families are still frequent and it is common for married offspring and parents to live in the same town. Therefore moving to another town for a new job may not only produce social isolation in the migrant but will also affect the social support of elderly relatives who stay. Immigrants have been found to have higher mortality than non immigrants (Cassel 1963, Jenkins 1971, Fuchs 1974), in spite of the so-called "healthy immigrant effect" (immigrants initially have better health than non immigrants from the same source population, as healthy

persons are more likely to migrate than ill persons). At the international level migration has been shown to be procyclical with respect to the receptor country (Thomas 1925, 1941, Higgs 1979, Chernomas 1984, Sáez 1975). Migration between regions within a country is likely to follow a similar pattern. In fact, the finding of a larger than average negative effect of unemployment on mortality in the more industrial regions of Spain (table 2.3) could be very plausibly attributed at least in part to immigration, since Madrid, the Basque Country and Catalonia (the industrial regions) have been traditional receptors of immigrants from Andalusia, Extremadura, Murcia, Castile and Galicia (the less industrial, poorer regions).

Eyer (1977a, 1977b) pointed out the occurrence of microepidemics of mild infectious disease during times of increased economic activity. He attributed these epidemics to stress-related reductions in immunity. The link between stress, decreased immunity and higher incidence of infections has been verified in a number of studies. A higher incidence of infectious diseases during economic expansions has been also documented (Thomas 1925, Higgs 1979). Ruhm (2000) found that a one percentage point decrease in the state unemployment rate was associated with a 0.7% increase in state mortality due to flu or pneumonia. Though mild respiratory infections were not examined separately in this study, infectious disease mortality in the Spanish provinces increased between 0.3% and 0.8% per each percentage point reduction in province unemployment (table 2.4). Estimates for males (between 0.5% and 0.9%, table 2.6) were substantially greater than for females (between 0.1% and 0.6%). At the national level there was a

clear drop in infectious disease mortality during the recession in the early 1980s (figure 2.17), with a subsequent progressive increase that only looks to subside in the mid 1990s.[8]

Deaths related to acute infection constitute at least a fifth (and probably much more) of total deaths due to respiratory disease (see footnote in table 2.2) and regression estimates show that respiratory disease mortality rises procyclically between 0.1% and 0.2% per percentage point decrease in province unemployment (table 2.4). As with infectious disease mortality, the effect of economic activity on respiratory disease mortality is much stronger for males (tables 2.6, 2.9 and 2.10).

Chemical pollutants (SO_2, SO_3, NO, NO_2, NO_3, O_3, CO, etc.) and micro particles in suspension, related to transportation and industrial activity, have been shown to be associated with increases in rates of diseases and death (Gardner 1973, Dockery et al. 1993, Cifuentes et al. 2001, HEI 2001). The morbidity and mortality effects of pollution appear to be nonspecific. Air pollution for example has been associated with increases in a large range of conditions, including respiratory and cardiovascular disease, cancer and even skin disorders. Given the procyclical character of industrial activity and transportation, it is quite plausible that pollution is also procyclical. Up to the passage of the Clean Air Act in 1970, the only period since 1900 when the level of pollutant atmospheric emissions in the US stopped growing steadily was the depression of the 1930s (EPA 2001).

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[8] **AIDS mortality is not included in the infectious mortality data used in the regressions.**

Noise may also have detrimental effects on health and behavior, increasing aggressive behavior, diminishing mutual aid and contributing to tension-related illnesses (Monahan & Vaux 1980). Noise has been shown to be related to cardiovascular disease and psychiatric disorders (Hunt 1989, WHO 1992). Because traffic, transportation and industrial activity are major sources of noise, noise levels are also likely to be procyclical.

There is evidence that diet becomes healthier (because of a drop in consumption of fat) and time exercising increases when the economy weakens (Ruhm 2000). Both things can have a direct effect on health and risk of death. Alcohol consumption (which is also procyclical), impact on mortality through its effects on the risk of injuries and its independent effect on mortality due to chronic disease (lever cirrhosis, myocardial infarction, perhaps also cancer). Cirrhosis mortality responds very quickly to changes in price of alcohol beverages rates (Seeley 1960) and it has been shown to be procyclical (Eyer 1977, Ruhm 2000).

Tobacco consumption has long been known to be significantly related to personal income and variations in cigarette price. Smoking exhibits a strong procyclical fluctuation (Ruhm 2000) that can have a direct impact on mortality due to cardiovascular disease, cancer and respiratory disease.

Cardiovascular mortality. For cardiovascular mortality all specifications (tables 2.4 and 2.6, including also the specifications with province-specific time trends, not shown) rendered negative estimates for the effects of

province unemployment on general or sex-specific mortality; but only in specifications unadjusted for year effects the estimate is significant. In regressions with ER instead unemployment (tables 2.9 and 2.10) the coefficient estimates for sex-specific ER show a peculiar pattern. All of them, except one, are positive, but the standard errors are so large that the estimates are statistically indistinguishable from zero. However, in the regressions with province-specific time trends (not shown) the coefficient estimate for sex-specific ER increase lightly—though they are still not significant—with larger values for the male ER estimate when male cardiovascular mortality is considered and larger values for the female ER estimate when female cardiovascular mortality is the regressand. The estimates of the effect for province GDP and national sex-specific ER on cardiovascular mortality (tables 2.4, 2.6, 2.8 and 2.10), are significant in almost all the specifications shown (and with a peculiar pattern of signs), but drop in size to be not statistically significant in the specifications with province-specific time trends (not shown). All the former seems to suggest that the relationship between cardiovascular mortality and economic conditions as proxied by unemployment or employment indicators is particularly weak. The consistently negative signs for the unemployment estimates and the consistently positive signs for the ER estimates seems to suggest that cardiovascular disease is very weakly procyclical in this particular sample. With respect to the significant coefficients for the effect of province GDP and national male and female ER (that disappear when province-specific time trends are included) they may be perfectly

explained as consequences of the relationship of secular falling rates of cardiovascular mortality (figure 2.16) with secular falling male ER and secular rising female ER and province real GDP per capita.

Suicide and homicide mortality. In a considerable number of observations in the analyzed sample there are zero deaths due to suicide or homicide.[9] In these cases the corresponding cause- or cause- and sex-specific death rate is set to 10^{-11} (i.e., one per 100 billion) to avoid computational mistakes when natural logs are taken for the regressions. This might introduce some bias in the regression results and in any case reveals how far of normality is the distribution of the untransformed outcome variable in some regressions.

Regression results for *suicide* are quite complex. Taking into account regression results using unemployment as covariate (tables 2.4 and 2.6) it seems that female suicide is acyclical, while both total suicides and male suicides are procyclical with respect to local conditions and strongly countercyclical with respect to national conditions proxied by national unemployment. When specifications with province-specific time trends (not shown) are considered, female suicide remains as acyclical (all coefficient estimates for province and national unemployment are statistically zero) and total suicides and males suicides appear neatly countercyclical, as the coefficient estimates for province unemployment switch sign

[9] In the 900 observations, deaths are zero for total suicide and male suicide in 200 observations, for female suicide in 218 observations, for homicide in 257, for male homicide in 293 and for female homicide in 519.

(they are positive but statistically significant only in two of the 10 regressions) and the estimate for national unemployment estimate remains strongly positive and statistically significant.

When sex-specific suicide rates are modeled as functions of sex-specific ER, male suicide (table 2.9) appear procyclical with respect to province male employment and strongly countercyclical with respect to national female ER. In specifications with province-specific time trends (not shown) the effect of province male ER is no longer significant but that of the national female ER remains large, negative and significant. For female suicides the acyclical pattern seems no longer valid when sex-specific ER are considered. They appear strongly procyclical with respect to local female ER and national male ER and strongly countercyclical with respect to female ER. In specifications with province specific time trends only the national ER are still significant, but with opposite signs. As the effect of local economic conditions disappears in all the specifications with province-specific time trends and the effect of province GDP was zero in all specifications, it seems that total suicides tend to be countercyclical with respect to national employment conditions, with peculiar cross-effects between sex-specific suicides and sex specific national employment indicators—male suicide drops intensely when more women work and female suicide grow intensely when male employment grows and drop even more intensely when female unemployment grows. This finding of countercyclical suicide rates from Spain is consistent with many previous studies that show a direct relationship of suicide with unemployment levels

(Boor 1980) and rates of suicide increasing countercyclically during recessions (Dooley et al. 1996, Ruhm 2000).

Homicide mortality does not reveal any evidence to be related to economic conditions in this particular sample at least for total homicides and males homicides (in tables 2.4, 2.6 and 2.9 all coefficient estimates, including also those not shown– are statistically zero). However female homicides (around one fourth of all homicides) seem to be procyclical (tables 2.6 and 2.10) as they fall when unemployment grows and rise when *male* employment increase (coefficient estimates for unemployment are negative and marginally significant in specifications with province-specific time trends but estimates for the four indicators of employment are not significant when province time trends are adjusted for).

Mortality of the unemployed and Brenner's theory. The huge literature examining the effect of unemployment on health will not be reviewed here, but many authors in that field are now convinced that the clear association between ill health and unemployment probably reflects bi-directional causality (i.e. ill health causes unemployment and unemployment causes ill health) and may also result from a third factor (alcohol or drug abuse, social marginalization) that simultaneously increase the risks of getting jobless and becoming ill or dead (Valkonen & Martikainen 1995, Bartlet 1996). However, Brenner (1993, 1995) has argued that although mortality is procyclical, this results form the lagged effect of previous unemployment. However there is no evidence that mortality

increased coinciding with or following important business downturns like the Great Depression of the 1930s or the recession of the late seventies in Europe. In addition, business fluctuations are not periodical. Therefore, if procyclical mortality results form lagged unemployment effects, one would have to argue that the relevant lag is different for each cycle, which makes little sense from the point of view of the physiophathological mechanisms purported to link unemployment and health.

Eyer's theory explains the procyclical fluctuations in mortality as a consequence of increased levels of stress. Of course many factors related to the development of chronic disease must necessarily operate over long periods, but once chronic disease is present, the concrete time of death can be determined by triggering factors with a lag of months, weeks, or even days. In the US heart attacks in working individuals peak on Mondays (Ruhm 2000) and suicides are generally highest on Mondays and on nonholidays (Bollen 1983). In Israel, Sunday is the day of the week when there are more deaths among Jews, while among Arabs there is no clear pattern (Anson & Anson 2000). These week patterns not only prove that death is a social event, but also that its concrete timing is subject to influences that have a very short lag.

Brenner (1993, 1995) suggested that the noxious effect of recessions on health could be partly related to increasing social inequality during economic downturns. In fact Davis (1941) showed with US data from the first decades of the 20th century that inequality increases during economic expansions and not during recessions, as the concentration of income is clearly procyclical. If the

procyclical nature of income inequality is confirmed it would provide a link between Eyer's and Wilkinson's theories, as economic expansions would cause deleterious effects on health in part through increasing social inequality.

Limitations of the present study. The use of linear regression methods applied to time series is always problematic, since the presence of autocorrelation creates the possibility of spurious results based in biased estimates. Though panel data regression is an important improvement, the sensitivity of some results in this study (particularly those for cardiovascular disease and suicide) to the adjustments for time trends reveals that there is still a large potential for bias and spurious results. A more systematic adjustment for time trends and the use of changes in mortality instead mortality in levels probably can overcome many of these problems and perhaps can be the proper approach for future research.

Many variables that probably have an effect on short term mortality have not been included in the present study. The most obvious are meteorological ones (temperature, humidity, atmospheric pressure, precipitation, etc.). Bad weather is consistently related to more deaths, mostly of elderly people (Gardner 1973, HEI 2001). Some activities and types of consumption are obviously noxious (smoking, drinking, excess total caloric intake, etc.), while others may reduce mortality (exercising, eating fruits and vegetable, medical care, pharmaceuticals, entertainment...). Educational level is also related to

health. Less educated people are more likely to adopt unhealthy behaviors and have risky occupations. If these variables are correlated with mortality and economic activity (but are not the *result* of economic activity) their omission will produce a spurious relationship between unemployment and mortality. The coefficient estimates will be biased and there will be what epidemiologists call confounding (Szklo & Nieto 2000). Weather is probably the only good candidate for confounding, as it could be correlated both with economic indicators and with mortality (as in Huntington 1920). Meteorological data are not included in the models in the present study because of the difficulty of choosing a good synthetic indicator of annual weather and also because of the lack of present credibility of business-cycle models à la Jevons, based on weather. It does not seem likely that education can vary substantially *with* business fluctuations. Exposure to risk factors and behaviors related to health and mortality (overwork, smoking, drinking, driving, exercising...) fluctuate with the level of economic activity. However, if the economy lead to changes in behaviors or exposures, these factors may be best thought of as mediating mechanisms rather than confounders of the business cycle-mortality relationship.

V – Some final comments

Previous studies and the results presented in this paper prove that, among the many influences determining mortality, the economy is an important one. Economic expansion seems to be causally associated with upturns in mortality. It has been estimated that world-wide 1.1 million die each year in work-related injuries and

illnesses, compared for example, to one million who die because of traffic injuries and half a million annual deaths due to war (Herbert and Landrigan 2000). Present results and Ruhm's results for a similar analysis of data from the 50 US states show that in modern industrial economies there is a small but significant increase in mortality associated with economic upturns. Therefore when dealing with policy questions alluding to the "unhealthy" effects of recessions may not be appropriate. Despite Brenner's complex and convoluted argument and the many criticism he has received, his work is often quoted (Sherman & Kolk, 1996:21, Sorkin 1988:83-87, Snyder 1996:97) as additional evidence for the need of expansionary economic policies. Obviously many links in the complex web relating economic and demographic issues have yet to be explored, but this must be done without preconceptions about the results that must be found to support one policy or another. Besides that, it seems that the procyclical character of mortality fluctuations is a proven fact. Therefore health effects are not a good reason to promote expansionary economic policies. Contrarily, the unhealthy effects of economic expansions must be a good reason to look for public health policies to dampen down these unhealthy effects.

Table 2.1. Correlation coefficients between unemployment rates and employment ratios at the national or province level, for the general population and for men and women population separately. Yearly data for the 50 Spanish provinces, 1980-1997

	NUR &			PUR & PER	PURM & PURW	NER &			PERM & PERW
	PUR	PURM	PURW			PER	PERM	PERW	
Max	0.96	0.97	0.96	-0.15	0.89	0.95	0.96	0.83	0.95
Median	0.85	0.76	0.69	-0.84	0.65	0.76	0.75	0.12	-0.20
Min	0.30	0.20	0.13	-0.99	0.03	0.32	0.43	-0.43	-0.79

NUR = national unemployment rate; PUR = province unemployment rate; PURM = province unemployment rate in men; PURW = province unemployment rate in women; PER = province employment ratio; NER = national employment ratio; PERM = province employment ratio in men; PERW = province employment ratio in women.

Table 2.2. Descriptive statistics of the dependent variables and covariates used in the regression models. Data for the 50 Spanish provinces in the period 1980-1997

Variable	Weighted mean	Standard deviation
Death rate per 100,000 population		
All causes, per 100,000	827.0	141.4
Men	891.6	147.7
Women	765.0	139.4
Cardiovascular disease (390-459)	345.6	73.9
Men	322.2	70.7
Women	368.0	81.1
Cancer (140-239)	196.0	39.9
Men	243.5	53.3
Women	150.3	29.4
Respiratory disease (460-519)	76.5	19.9
Men	94.3	25.6
Women	59.5	16.9
Infectious disease (001-139)	9.72	3.1
Men	11.3	3.8
Women	8.3	3.2
External causes (E800-E999)	42.1	9.4
Men	62.5	14.5
Women	22.5	6.3
Traffic injuries	15.7	5.7
Men	24.4	8.9
Women	7.3	3.4
Suicide	6.9	3.0
Men	10.5	4.6
Women	3.6	2.0
Homicide	0.9	0.7
Men	1.4	1.1
Women	0.5	0.5
Unemployment rates		
Yearly % average among all active	19.3	7.2
Yearly % average among active men	16.1	6.7
Yearly % average among active women	25.3	9.5
Employment ratios		
% employed among all aged >16	39.6	5.3
% employed among men aged >16	56.2	5.9
% employed among women aged >16	23.7	6.6
GDP per capita, hundred thousand 1983 pesetas	7.1	1.8
Population aged 0-4 years (%)	6.0	1.4
Population aged 65 and over (%)	13.3	3.8

Means weighted by province population. Death rates computed from crude data in the TEMPUS database, Instituto Nacional de Estadistica, Spain. Quarterly unemployment rates for every province directly taken from TEMPUS. Employment ratios computed from unemployment rates and rates of active population among persons older than 16 (from TEMPUS). Province real GDP per capita computed from province nominal GDP (from TEMPUS, data for 1994-1996 are provisional) and two series of consumer price index from TEMPUS. For most variables there are 900 observations (one for each year of the 18 year period for each of the 50 Spanish provinces). Bracketed codes refer to the Ninth Revision of the International Classification of Disease (ICD-9). The "cancer" category in this table corresponds in the TEMPUS database to deaths classified as caused by *tumores* ("tumors"), with ICD-9 codes 140-239. These codes include the solid malignant neoplasm (ICD-9 codes 140-199), myeloma, leukemia, lymphomas, Hodgkin's disease and other malignant neoplasm of lymphatic and hematopoietic tissue (200-208), carcinoma in situ (230-234), neoplasm of uncertain behavior (235-238) or unspecified nature (239), and benign neoplasm (210-229). Therefore the group includes a small group of deaths caused by tumors that are not malignancies (cancer) in the strict sense. As this number is tiny compared to total deaths in this category, the group is labeled as cancer mortality. Mortality due to respiratory diseases (460-519) includes death allocated to influenza, pneumonia, chronic bronchitis, emphysema and asthma, and a large miscellaneous group of chest disorders, including pneumoconiosis, empyema, lung abscess, pleurisy, atelectasia, respiratory failure, etc. For example in Barcelona, in 1990, 3% or this group of respiratory disease deaths were due to influenza, 19% to pneumonia, 18% to chronic bronchitis, emphysema or asthma, and 60% to a large miscellaneous group of chest disorders, including pneumoconiosis, empyema, lung abscess, pleurisy, atelectasia, respiratory failure, etc. Therefore about a fifth of this group of respiratory disease deaths are directly caused by acute infection and four fifths are fatalities in patients with a background of chronic lung disease; in these the concrete timing of death it is often determined by a mild infection. Mortality by infectious disease corresponds to "infectious and parasitic diseases" (ICD-9 codes 001-139)in TEMPUS. It includes cholera, typhoid fever, and other bowel infectious diseases, tuberculosis, meningococcal infections, tetanus, septicemia, measles, viral encephalitis, viral hepatitis, rickettsiosis, malaria, syphilis and other venereal diseases, and other infectious and parasitic diseases, excluding acute respiratory infections (460-466), pneumonia and influenza (480-487) and AIDS (279.5). Mortality due to external causes of injury or poisoning includes all deaths due to railroad or motor vehicle injuries, unintentional poisoning, falls or fires, all other unintentional injuries or adverse effects, suicides, homicides and other violence.

Table 2.3. Regression results for fixed effect models relating total mortality rates to economic conditions in the 50 Spanish provinces, 1980-1997

	Basic specification					With province specific time trends					Death rates in levels		
	(*i*)	(*ii*)	(*iii*)	(*iv*)	(*v*)	(*i*)	(*ii*)	(*iii*)	(*iv*)	(*v*)	(*i*)	(*ii*)	(*v*)
Province unemployment	-0.31* (0.05)	-0.23* (0.05)	-0.27* (0.04)		-0.35* (0.05)	-0.11* (0.05)	-0.11* (0.06)	-0.11* (0.04)		-0.12* (0.05)	-1.68* (0.39)	-1.13* (0.44)	-2.12* (0.43)
National unemployment				-0.16* (0.05)	0.15* (0.07)				-0.10* (0.04)	-0.01 (0.06)			0.74 (0.56)
Prov. GDP per capita		1.15* (0.36)					-0.19 (0.48)					9.18* (3.07)	
Year effects	Yes	Yes	No	No	No	Yes	Yes	No	No	No	Yes	Yes	No
N	900	850	900	900	900	900	850	900	900	900	900	850	900
Durbin-Watson *d*	1.01	1.03	1.18	1.12	1.20	1.54	1.59	1.82	1.81	1.82	1.1	1.1	1.2

"Split-sample" estimates

	1980-1988		1989-1997		Provinces over one million		Provinces under 300,000		15 prov. with lowest GDPpc in 1990		15 provinces with largest GDPpc in 1990		Historically industrialized regions	
	(*i*)	(*ii*)	(*i*)	(*ii*)	(*i*)	(*ii*)	(*i*)	(*ii*)	(*i*)	(*ii*)	(*i*)	(*ii*)	(*i*)	(*ii*)
Province unemployment	-0.05 (0.08)	-0.00 (0.09)	-0.15* (0.06)	-0.12 (0.07)	-0.41* (0.09)	-0.30* (0.11)	-0.12 (0.17)	-0.17 (0.19)	-0.001 (0.08)	0.02 (0.08)	-0.36* (0.13)	-0.39* (0.14)	-0.47* (0.17)	-0.55* (0.18)
National unemployment		9.83 (6.12)		-2.30 (6.70)		1.75* (0.88)		-0.09 (0.86)		-0.04 (1.05)		-1.00 (0.79)		-1.69 (1.09)
N	450	450	450	450	198	187	198	198	270	270	270	270	162	162
Durbin-Watson *d*	1.33	1.34	1.81	1.88	1.05	1.04	1.24	1.23	1.34	1.39	0.87	0.82	0.78	0.77

All regressions are weighted by the square root of province population. The dependent variable is the natural logarithm of the death rate per 100,000 in all the models, except the last three columns in the upper panel, in which the death rate is not log-transformed. Except in these three last columns of the upper panel all the coefficient estimates are multiplied times 100, so they

express the percent increase in death rates associated with a one unit increase in the covariate. In this and all the following tables, standard errors are bracketed under the coefficient estimates. If these are statistically significant at a 95% confidence level ($t > 1.96$, $P < 0.05$) they are marked *. All R^2 were larger than 0.94 and smaller (except one) than 0.98. All regressions in the table are adjusted for province effects and for the proportions of population under 5 or over 64. Year effects are included as specified. Province-specific time trends are only included in middle columns of the upper panel. In all specifications the coefficient estimates for the proportions under 5 and over 64 are both statistically significant (the size of the estimate for the proportion over 64 much larger than the other) and with negative and positive sign, respectively, as it is to be expected .

The 11 provinces with population over one million are Madrid (5.02 million), Barcelona (4.62), Valencia (2.14), Seville (1.67), Alicante (1.32), Málaga, Vizcaya, Cádiz, La Coruña and Asturias. There are also 11 provinces with population under 300,000: Álava, Ávila, Cuenca, Guadalajara, Huesca, La Rioja, Palencia, Segovia, Soria, Teruel, and Zamora. The 15 provinces with the lowest real GDP per capita, in order of decreasing GDP are Badajoz, Granada, Córdoba, Jaén, Orense, Sevilla, Cuenca, Cádiz, Málaga, Lugo, Albacete, Pontevedra, La Coruña, and Ávila. The historicaaly industrialized regions refer to the autonomous communities considered early industrial areas of Spain. They are the four Catalonian provinces, the four provinces of the Basque Country and Navarra and Madrid.

Table 2.4. Regression results for fixed-effects models relating cause-specific death rates to economic conditions (proxied by province and national unemployment and province real GDP per capita) and demographic structure in the 50 Spanish provinces, 1980-1997

	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)
	Cardiovascular disease			Cancer			Respiratory disease			Infectious disease		
Prov. unempl.	-0.08 (0.07)	-0.10 (0.07)	-0.20* (0.08)	-0.31* (0.06)	-0.19* (0.07)	-0.29* (0.06)	-0.24 (0.13)	0.07 (0.14)	-0.17 (0.14)	-0.76* (0.27)	-0.34 (0.30)	-0.69* (0.28)
Nat. unempl.			0.04 (0.10)			0.49* (0.08)			-0.37* (0.18)			-0.81* (0.36)
Prov. GDP per capita		-1.27* (0.52)			2.60* (0.48)			5.75* (0.98)			8.05* (2.12)	
R^2	0.95	0.95	0.93	0.95	0.95	0.95	0.86	0.87	0.83	0.55	0.57	0.51
	External causes			Traffic injuries			Suicide			Homicide		
Prov. unempl.	-0.53* (0.16)	-0.77* (0.18)	-0.66* (0.17)	-1.13* (0.31)	-1.39* (0.35)	-1.21* (0.34)	-1.20* (0.35)	-1.22* (0.40)	-1.50* (0.36)	-3.53 (3.71)	-4.86 (4.23)	-3.26 (3.66)
Nat. unempl.			-0.79* (0.22)			-1.82 (0.45)			2.79* (0.47)			6.00 (4.77)
Prov. GDP per capita		-4.08* (1.27)			-3.80 (2.48)			-2.85 (2.81)			9.11 (29.61)	
R^2	0.75	0.75	0.70	0.69	0.69	0.60	0.73	0.73	0.70	0.43	0.44	0.42

The dependent variable is always the natural log of the cause-specific death rate per 100,000 population. All coefficient estimates are multiplied times 100, so they express the percent increase in death rate associated with a one unit increase in the covariate. Specifications (*i*), (*ii*) and (*v*)

as in table 2.3. There are 900 observations in each model, except in some specifications where N = 850 due to missing values.

In specifications with province-specific time trends (not shown) coefficients for province unemployment tend to keep the same sign, sometimes they drop only a little (infectious disease mortality), sometimes they are no longer statistically significant (cardiovascular and respiratory disease mortality) and other times increase in absolute value (cancer, external causes and traffic mortality). In the regressions for suicide with province-specific time trends the estimate for province unemployment was always positive but only once statistically significant, while the estimate for national unemployment was always positive and significant.

The demographic variables were statistically significant and with the expected opposite signs except for external causes and traffic mortality, in which they were erratic in sign and often not significant.

Table 2.5. Regression results for fixed-effect models relating sex-specific mortality rates to economic conditions (proxied by province unemployment, national unemployment and province real GDP per capita) and the demographic structure in the 50 Spanish provinces, 1980-1997

	Basic specification					With province specific time trends					Death rates in levels		
	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(v)
Death rate for all causes, men													
Prov. unempl.	-0.31*	-0.24*	-0.28*		-0.33*	-0.16*	-0.16*	-0.15*		-0.16*	-1.81*	-1.33*	-2.15*
	(0.05)	(0.06)	(0.04)		(0.05)	(0.05)	(0.06)	(0.04)		(0.06)	(0.47)	(0.53)	(0.50)
Nat. unempl.				-0.21*	0.09				-0.12*	-0.02			0.26
				(0.05)	(0.07)				(0.05)	(0.07)			(0.66)
Prov. GDP per capita		1.02*					0.07					8.97*	
		(0.41)					(0.54)					(3.72)	
Death rate for all causes, women													
Prov. unempl.	-0.31*	-0.21*	-0.25*		-0.37*	-0.06	-0.06	-0.08		-0.07	-1.56*	-0.94*	-2.09*
	(0.05)	(0.06)	(0.04)		(0.06)	(0.06)	(0.06)	(0.04)		(0.06)	(0.41)	(0.47)	(0.46)
Nat. unempl.				-0.12*	0.22*				-0.07	-0.01			1.20*
				(0.06)	(0.08)				(0.05)	(0.08)			(0.59)
Prov.GDP per capita		1.28*					-0.50					9.36*	
		(0.42)					(0.59)					(3.27)	

Specifications as in previous tables. Except in the specifications with death rates in levels, all the coefficient estimates and their standard errors (in brackets) are multiplied times 100. Regressions were weighted by the square root of the population in the denominator of the mortality rate (i.e. province male population and province female population, respectively, in each panel).

All R^2 were in the interval 0.93 to 0.97 with very small differences between the R^2 values in the two panels (those in the lower one-female mortality-were larger). Coefficient estimates for population over 64 were positive and statistically significant in all regressions, those for population under 5 were negative and statistically significant in almost all the regressions, except some in which the estimate was positive and not statistically significant.

Table 2.6. Regression results for fixed-effects models relating sex and cause-specific death rates to economic and demographic conditions in the 50 Spanish provinces, 1980-1997

	Death rates in men											
	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)
	Cardiovasc. disease			Cancer			Respirat. disease			Infectious disease		
Prov. unempl.	-0.07	-0.11	-0.18*	-0.29*	-0.19*	-0.26*	-0.40*	-0.07	-0.31*	-0.87*	-0.48	-0.86*
	(0.08)	(0.09)	(0.09)	(0.07)	(0.08)	(0.07)	(0.13)	(0.15)	(0.14)	(0.32)	(0.36)	(0.33)
Nat. unempl.			-0.02			0.53*			-0.15			-0.79
			(0.11)			(0.10)			(0.19)			(0.43)
Prov. GDP per cap.		-2.01*			2.36*			6.09*			8.11*	
		(0.61)			(0.56)			(1.04)			(2.54)	
R^2	0.92	0.93	0.90	0.95	0.95	0.94	0.87	0.87	0.84	0.46	0.47	0.43
	External causes			Traffic injuries			Suicide			Homicide		
Prov. unempl.	-0.53*	-0.82*	-0.66*	-1.15*	-1.51*	-1.24*	-1.21*	-1.28*	-1.46*	-0.32	-0.28	-0.12
	(0.17)	(0.20)	(0.19)	(0.32)	(0.37)	(0.36)	(0.38)	(0.44)	(0.39)	(4.72)	(5.30)	(4.65)
Nat. unempl.			-0.78*			-1.93)*			2.78*			6.36
			(0.25)			(0.46)			(0.51)			(6.04)
Prov. GDP per cap.		-4.67*			-5.03			-3.72			39.55	
		(1.37)			(2.58)			(3.06)			(37.06)	
R^2	0.74	0.74	0.67	0.68	0.68	0.59	0.70	0.70	0.67	0.40	0.42	0.39
	Death rates in women											
	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)
	Cardiovasc. disease			Cancer			Respirat. disease			Infectious disease		
Prov. unempl.	-0.11	-0.09	-0.22*	-0.35*	-0.21*	-0.36*	-0.02	0.28	0.01	-0.57	-0.10	-0.45
	(0.07)	(0.08)	(0.08)	(0.08)	(0.09)	(0.08)	(0.17)	(0.19)	(0.18)	(0.38)	(0.44)	(0.39)
Nat. unempl.			0.11			0.45*			-0.68*			-1.02*
			(0.11)			(0.11)			(0.24)			(0.51)
Prov. GDP per cap.		-0.54			3.00*			5.41*			8.42*	
		(0.59)			(0.65)			(1.34)			(3.05)	
R^2	0.94	0.94	0.93	0.91	0.91	0.90	0.77	0.77	0.72	0.45	0.45	0.41
	External causes			Traffic injuries			Suicide			Homicide		
Prov. unempl.	-0.62*	-0.77*	-0.78	-0.99*	-0.86	-1.05*	-0.31	0.19	-0.52	-17.78*	-22.06*	-15.97*
	(0.24)	(0.27)	(0.24)	(0.46)	(0.52)	(0.48)	(2.05)	(2.38)	(2.04)	(7.21)	(8.29)	(7.14)
Nat. unempl.			-0.72			-1.54*			1.33			13.98
			(0.31)			(0.63)			(2.65)			(9.30)
Prov. GDP per cap.		-2.57			-0.12			-1.82			-30.93	
		(1.90)			(3.66)			(16.65)			(58.04)	
R^2	0.60	0.60	0.57	0.53	0.54	0.46	0.30	0.31	0.28	0.30	0.30	0.29

Regressions weighted by the square root of the population in the denominator of the mortality rate.

In specifications with province-specific time trends (not shown) the estimate for province unemployment is negative but generally not significant for male and female mortality due to cardiovascular

disease, cancer, respiratory disease and infectious disease, and for male homicide; for female homicide the estimate is negative and marginally significant. The estimate for province unemployment is negative and significant for mortality due to external causes and traffic in all specifications for male mortality and most of female mortality. For male suicide province unemployment estimates are positive but only once statistically significant; the estimate for national unemployment is always positive and significant. For female suicide the province unemployment estimate is always negative but not significant; the national unemployment estimate is only once positive, all the other times negative, never significant.

Table 2.7. Regression results for fixed-effect models relating general and sex-specific mortality to sex-specific unemployment rates at the province and national level in the 50 Spanish provinces, 1980-1997

	Death rate for all causes												
	Basic specification					With province specific time trends					Death rates in levels		
	(*i*)	(*ii*)	(*iii*)	(*iv*)	(*v*)	(*i*)	(*ii*)	(*iii*)	(*iv*)	(*v*)	(*i*)	(*ii*)	(*v*)
Prov.unemp.	-0.24*	-0.20*	-0.08		-0.26*	-0.13*	-0.14*	-0.07		-0.13*	-1.36*	-1.05*	-1.53*
men	(0.06)	(0.06)	(0.04)		(0.07)	(0.05)	(0.06)	(0.04)		(0.06)	(0.52)	(0.53)	(0.57)
Prov.unemp.	-0.08*	-0.05	-0.16		-0.10*	0.00	0.01	-0.04		-0.00	-0.41	-0.17	-0.64*
women.	(0.03)	(0.04)	(0.03)		(0.04)	(0.03)	(0.03)	(0.04)		(0.04)	(0.28)	(0.30)	0.31
Nat. unemp.				0.21*	0.44*				0.14*	0.25*			2.78*
men				(0.08)	(0.10)				(0.07)	(0.08)			(0.81)
Nat. unemp.				-0.44*	-0.34*				-0.33*	-0.33*			-2.40*
women				(0.09)	(0.09)				(0.09)	(0.09)			(0.78)
Prov. GDP		1.15*					-0.25					9.30*	
per capita		(0.37)					(0.46)					(3.08)	
R^2	0.96	0.96	0.95	0.95	0.95	0.98	0.98	0.97	0.97	0.97	0.96	0.96	0.95
	Death rate for all causes, men												
Prov.unemp.	-	-0.22*	-0.11*		-0.27*	-0.17*	-0.19*	-0.09*		-0.16	-1.52*	-1.29*	-1.65*
men	0.25*												
	(0.07)	(0.07)	(0.05)		(0.07)	(0.06)	(0.06)	(0.04)		(0.06)	(0.62)	(0.64)	(0.66)
Prov.unemp.	-0.07*	-0.04	-0.15*		-0.09*	-0.01	0.01	-0.06		-0.02	-0.40	-0.14	-0.58
women.	(0.04)	(0.04)	(0.04)		(0.04)	(0.04)	(0.04)	(0.04)		(0.04)	(0.34)	(0.36)	(0.36)
Nat. unemp.				0.21*	0.45*				0.13	0.27*			3.13*
men				(0.08)	(0.10)				(0.07)	(0.09)			(0.94)
Nat. unemp.				-0.50*	-0.41*				-0.36*	-0.35*			-3.38*
women				(0.09)	(0.10)				(0.10)	(0.10)			(0.90)
Prov. GDP		1.03*					-0.00					9.21*	
per capita		(0.41)					(0.54)					(3.73)	
R^2	0.95	0.95	0.94	0.94	0.94	0.97	0.97	0.97	0.96	0.96	0.95	0.95	0.94

Table 2.7 (cont.). Regression results for fixed-effect models relating general and sex-specific mortality to sex-specific unemployment rates at the province and national level in the 50 Spanish provinces, 1980-1997

	Basic specification					With province specific time trends					Death rates in levels		
	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(v)
	Death rate for all causes, women												
Prov. unemp. men	-0.23* (0.07)	-0.17* (0.07)	-0.05 (0.05)		-0.25* (0.08)	-0.09 (0.06)	-0.09 (0.07)	-0.04 (0.05)		-0.10 (0.07)	-1.19* (0.54)	-0.80 (0.57)	-1.38* (0.60)
Prov. unemp. women.	-0.09* (0.04)	-0.06 0.04	-0.18* (0.04)		-0.12* 0.04	-0.01 0.04	0.01 (0.04)	-0.03 (0.04)		0.01 (0.04)	-0.44 (0.30)	-0.20 (0.32)	-0.72* (0.33)
Nat. unemp. men				0.20* (0.09)	0.43* (0.11)				0.14 (0.08)	0.23* (0.10)			2.43* (0.85)
Nat. unemp. women				-0.37* (0.10)	-0.26* (0.10)				-0.30* (0.11)	-0.31* (0.11)			-1.47 (0.82)
Prov. GDP per capita		1.27* (0.42)					-0.54 (0.59)					9.35* (3.28)	
R^2	0.96	0.96	0.94	0.94	0.95	0.97	0.97	0.96	0.96	0.97	0.96	0.96	0.95

Regressions weighted by the square root of the population in the denominator of the mortality rate. Coefficient estimates for the proportion of population over 64 were positive in all 15 regressions in each panel and statistically significant in all 45 regressions but one. Those for population under 5 were always negative and almost always statistically significant.

Table 2.8. Regression results for fixed-effect models relating general and sex-specific province mortality to sex-specific employment ratios (ER) at the province and national level. Spain, 1980-1997

	Basic specification					With province specific time trends					Death rates in levels		
	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(v)
	Death rate for all causes												
Prov. ER, men	0.36* (0.06)	0.29* (0.07)	0.37* (0.05)		0.37* (0.07)	0.19* (0.06)	0.21* (0.06)	0.18* (0.05)		0.21* (0.07)	2.19* (0.54)	1.74* (0.56)	2.30* (0.58)
Prov. ER, women.	0.08 (0.06)	0.06 (0.06)	0.15 (0.05)		0.05 (0.06)	−0.07 (0.06)	−0.10 (0.07)	−0.08 (0.07)		−0.06 (0.07)	−0.25 (0.47)	−0.48 (0.49)	−0.41 (0.51)
Nat. ER, men				0.67* (0.09)	0.42* (0.11)				0.27 (0.20)	0.06 (0.21)			4.34* (0.88)
Nat. ER, women				−1.19* (0.17)	−1.43* (0.20)				−0.41 (0.43)	−0.41 (0.43)			−12.00* (1.65)
Prov. GDP per capita		1.11* (0.36)					−0.31 (0.48)					9.56* (3.02)	
R^2	0.96	0.96	0.95	0.95	0.95	0.98	0.98	0.97	0.97	0.97	.0.96	0.96	0.96
	Death rate for all causes, men												
Prov. ER, men	0.40* (0.07)	0.35* (0.08)	0.39*. (0.06)		0.41* (0.08)	0.25* (0.07)	0.26* (0.07)	0.21* (0.06)		0.26* (0.07)	2.70* (0.65)	2.28* (0.68)	2.78* (0.68)
Prov. ER, women.	0.02 (0.06)	−0.01 0.06	−0.14* (0.06)		−0.01 (0.07)	−0.11 (0.07)	−0.16 (0.08)	−0.10 (0.08)		−0.09 (0.08)	−0.86 (0.57)	−1.13 (0.60)	−1.06 (0.60)
Nat. ER, men				0.55* (0.09)	0.25* (0.12)				0.11 (0.22)	−0.16 (0.23)			3.40* (1.04)
Nat. ER, women				-0.80* (0.19)	−0.94* (0.22)				0.01 (0.48)	0.16 (0.48)			− 8.92* (1.96)
Prov. GDP per capita		1.01* (0.40)					−0.03 (0.54)					9.80* (3.65)	
R^2	0.95	0.95	0.94	0.94	0.94	0.97	0.97	0.96	0.96	0.96	0.95	0.95	0.94

Table 2.8 (cont.). Regression results for fixed-effect models relating general and sex-specific province mortality to sex-specific employment ratios (ER) at the province and national level. Spain, 1980-1997

	Basic specification					With province specific time trends					Death rates in levels		
	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(v)
Death rate for all causes, women													
Prov. ER, men	0.30* (0.07)	0.23* (0.08)	0.34* (0.08)		0.32* (0.08)	0.13 (0.07)	0.15 (0.08)	0.14*. (0.06)		0.16* (0.08)	1.68* (0.57)	1.20* (0.60)	1.82* (0.60)
Prov. ER, women.	0.15* (0.06)	0.13 (0.07)	-0.17* (0.06)		0.12 (0.07)	-0.03 (0.08)	-0.03 (0.08)	-0.06 (0.08)		-0.03 (0.08)	0.31 (0.50)	0.13 (0.53)	0.19 (0.53)
Nat. ER, men				0.81* (0.09)	0.63* (0.12)				0.47 (0.24)	0.31 (0.25)			5.33* (0.92)
Nat. ER, women				-1.64* (0.19)	-1.99* (0.22)				-0.88 (0.51)	-0.80) (0.51)			-15.06* (1.72)
Prov. GDP per capita		1.22* (0.41)					-0.63 (0.59)					9.37* (3.22)	
R^2	0.98	0.96	0.94	0.95	0.95	0.97	0.97	0.96	0.96	0.96	0.96	0.96	0.95

Regressions weighted by the square root of the population in the denominator of the mortality rate. The coefficient estimate for the proportion of population over 64 are positive and statistically significant in all 45 regressions (13 shown and 2 not shown in each panel) but one. Estimates for the proportion of population under 5 are negative all the times and statistically significant in all 45 regressions but one.

Table 2.9. Regression results for fixed-effect models relating cause-specific mortality in males to sex-specific employment ratios (ER) at the province and national level in the 50 Spanish provinces, 1980-1997

	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)
	Cardiovascular disease			Cancer			Respiratory disease			Infectious disease		
Prov. ER men	0.08	0.15	0.08	0.40*	0.26*	0.40*	0.48*	0.22	0.54*	0.65	0.23	0.60
	(0.11)	(0.11)	(0.12)	(0.10)	(0.10)	(0.10)	(0.18)	(0.19)	(0.20)	(0.44)	(0.47)	(0.45)
Prov. ER women	0.00	0.00	-0.09	-0.17	-0.20*	-0.10	-0.07	-0.24	-0.06	0.36	0.24	0.08
	(0.09)	(0.10)	(0.10)	(0.09)	(0.09)	(0.09)	(0.16)	(0.17)	(0.17)	(0.39)	(0.41)	(0.40)
Nat. ER, men			1.23*			-0.82*			-0.97*			1.22
			(0.18)			(0.15)			(0.30)			(0.69)
Nat. ER, women			-2.64*			0.40			2.29*			-0.5
			(0.33)			(0.29)			(0.57)			(1.30)
Prov.GDP per head		-1.99*			2.70*			6.21*			8.63*	
		(0.60)			(0.56)			(1.03)			(2.50)	
R^2	0.92	0.93	0.91	0.95	0.95	0.94	0.87	0.87	0.85	0.46	0.46	0.42
	External causes			Traffic injuries			Suicide			Homicide		
Prov. ER, men	1.24*	1.51*	1.26*	2.07*	2.39*	2.08*	1.54*	1.63*	1.58*	5.45	5.93	5.39
	(0.23)	(0.25)	(0.25)	(0.44)	(0.47)	(0.48)	(0.52)	(0.56)	(0.53)	(6.47)	6.81	(6.43)
Prov. ER, women.	-0.25	-0.06	-0.01	-0.52	-0.29	-0.04	0.60	0.66	0.76	-6.61	-9.42	-6.50
	(0.21)	(0.22)	(0.22)	(0.39)	(0.41)	(0.42)	(0.46)	(0.49)	(0.47)	(5.72)	(5.97)	(5.65)
Nat. ER, men			2.22*			3.67*			0.69			-14.42
			(0.39)			(0.74)			(0.81)			(9.80)
Nat. ER, women			-3.95*			-4.16*			-9.39*			11.50
			(0.72)			(1.38)			(1.51)			(18.39)
Prov. GDP per capita		-5.30*			-5.18*			-4.43			42.64	
		(1.33)			(2.52)			(3.00)			(36.43)	
R^2	0.74	0.75	0.69	0.68	0.68	0.61	0.70	0.70	0.69	0.40	0.42	0.39

Regressions weighted by the square root of the province male population. *In specifications with province-specific time trends* (not shown) the province male ER estimate goes up in absolute value close to be statistically significant (for

cardiovascular mortality) or is positive and significant (in most regressions for cancer and all for respiratory disease and traffic mortality). However, in the regressions for suicide the estimate for male ER goes lightly toward zero (enough to become non statistically significant) and the estimate for national female ER is always significant and negative, while the national male ER is always positive but not significant.

Table 2.10. Regression results for fixed effect models relating cause-specific mortality in females to sex-specific employment ratios (ER) at the province and national level in the 50 Spanish provinces, 1980-1997

	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)	(i)	(ii)	(v)
	Cardiovascular disease			Cancer			Respirat. disease			Infectious disease		
Prov. ER, men	0.08 (0.10)	0.09 (0.11)	0.09 (0.11)	0.37* (0.11)	0.19 (0.12)	0.37* (0.11)	0.09 (0.23)	-0.15 (0.25)	-0.17 (0.26)	0.73 (0.52)	0.42 (0.56)	0.65 (0.54)
Prov. ER, women.	0.16 (0.09)	0.16 (0.09)	0.14 (0.09)	-0.11 (0.10)	-0.15 (0.10)	-0.10 (0.10)	-0.24 (0.21)	-0.28 (0.22)	-0.28 (0.22)	0.20 (0.46)	0.11 (0.49)	-0.12 (0.47)
Nat. ER, men			1.35* (0.16)			-0.20 (0.17)			0.08 (0.39)			-0.11 (0.82)
Nat. ER women			-3.36* (0.30)			-0.50 (0.32)			1.46* (0.73)			2.94 (1.54)
Prov. GDP per capita		-0.71 (0.58)			3.41* (0.64)			5.34* (1.32)			7.70* (3.00)	
R^2	0.94	0.94	0.94	0.91	0.91	0.90	0.77	0.77	0.72	0.45	0.45	0.41
	External causes			Traffic injuries			Suicide			Homicide		
Prov. ER, men	0.83* (0.33)	0.95* (0.35)	0.86* (0.33)	1.84* (0.63)	1.89* (0.67)	1.84* (0.66)	-2.14 (2.80)	-1.63 (3.06)	-2.03 (2.80)	19.23* (9.91)	19.80* (10.7)	18.45 (9.87)
Prov. ER, women.	0.30 (0.29)	0.28 (0.30)	0.27 (0.29)	-0.07 (0.56)	-0.09 (0.59)	-0.53 (0.58)	4.58 (2.47)	4.35) (2.67)	4.96* (2.45)	-5.02 (8.74)	-2.22 (9.36)	-4.89 (8.66)
Nat. ER, men			2.07* (0.50)			3.36* (1.01)			10.37* (4.26)			-2.97* (1.51)
Nat. ER, women			-2.78* (0.94)			-5.00* (1.89)			-26.66* (7.99)			39.55 (28.2)
Prov. GDP per capita		-2.75 (1.86)			-1.21 (3.59)			-5.59 (16.4)			-6.37 (57.3)	
R^2	0.60	0.60	0.58	0.53	0.54	0.48	0.30	0.31	0.29	0.30	0.30	0.29

Regressions weighted with the square root of the province female population. *In specifications with province-specific time trends* (not shown) the estimates for male ER are positive and significant in the regressions for cancer, some of the regressions for respitatory disease mortality and all the regressions for suicide; the estimate is positive but not significant for most of the regressions for cardiovascular mortality. The estimate for female ER in specifications with

province-specific time trends are negative and significant for suicide mortality.

Table 2.11. Fixed-effect models relating total mortality to province and national unemployment lagged up to 4 years

	Basic specifications					With province-specific time trends				
	I	II	III	IV	V	VI	VII	VIII	IX	X
Regressors unlagged										
Prov.unempl. unlagged	-0.27* (0.06)	-0.21* (0.06)	-0.22* (0.04)		-0.32* (0.06)	-0.09 (0.06)	-0.10 (0.06)	-0.07 (0.05)		-0.11 (0.08)
Nat.unempl. unlagged				-0.10 (0.05)	0.18* (0.08)				-0.03 (0.06)	0.08 (0.08)
Prov. GDP per capita		0.90* (0.44)					-0.15 (0.52)			
Regressors unlagged and lagged 1 year										
Prov.unempl unlagged	-0.28* (0.06)	-0.24* (0.06)	-0.27* (0.05)		-0.33* (0.07)	-0.07 (0.07)	-0.07 (0.07)	-0.03 (0.06)		-0.06 (0.08)
Prov.unempl lagged 1 yr	-0.03 (0.04)	-0.02 (0.04)	0.02 (0.04)		-0.04 (0.05)	-0.02 (0.06)	-0.02 (0.07)	-0.08 (0.06)		-0.04 (0.07)
Nat.unempl. unlagged				-0.19* (0.06)	0.11 (0.08)				0.04 (0.08)	0.10 (0.11)
Nat.unempl lagged 1yr				0.11* (0.05)	0.15* (0.06)				-0.16 (0.10)	-0.12 (0.12)
Prov. GDP per capita		0.92* (0.43)					0.19 (0.51)			
Regressors unlagged and lagged 1 and 2 years										
Prov.unempl unlagged	-0.22* (0.07)	-0.20* (0.07)	-0.21* (0.06)		-0.27* (0.08)	-0.06 (0.06)	-0.06 (0.06)	-0.06 (0.06)		-0.08 (0.07)
Prov.unempl lagged 1 yr	-0.08 (0.07)	-0.08 (0.09)	-0.01 (0.09)		-0.07 (0.10)	-0.03 (0.06)	-0.03 (0.07)	-0.01 (0.07)		-0.02 (0.08)
Prov.unempl lagged 2 yr	-0.08 (0.09)	-0.04 (0.07)	-0.03 (0.06)		-0.08 (0.08)	0.06 (0.06)	0.02 (0.07)	-0.01 (0.07)		0.03 (0.07)
Nat unempl. unlagged				-0.01 (0.13)	0.23 (0.15)				-0.09 (0.12)	-0.01 0.14
Nat.unempl lagged 1yr				-0.21 (0.20)	-0.10 (0.22)				0.09 (0.19)	0.09 (0.20)
Nat unempl. lagged 2 yr				0.19 (0.14)	0.21 (0.15)				-0.11 (0.15)	-0.12 (0.16)
Prov. GDP per capita		0.70 (0.41)					0.19 (0.51)			
Regressors unlagged and lagged 1, 2 and 3 years										
Prov. unempl. unlagged	-0.20* (0.06)	-0.18* (0.06)	-0.24* (0.06)		-0.23* (0.07)	-0.05 (0.06)	-0.05 (0.06)	-0.14 (0.05)		-0.10 (0.07)
Prov. unempl. lagged 1 yr	-0.09 (0.08)	-0.09 (0.08)	0.00 (0.08)		-0.07 (0.08)	-0.03 (0.06)	-0.03 (0.06)	0.01 (0.06)		-0.01 (0.07)
Prov. unempl. lagged 2 yr	0.06 (0.08)	0.06 (0.08)	-0.07 (0.07)		-0.07 (0.09)	0.00 (0.06)	0.00 (0.06)	-0.09 (0.06)		-0.03 (0.07)
Prov. unempl. lagged 3 yr	0.01 (0.06)	0.00 (0.06)	0.01 (0.06)		0.03 (0.07)	0.13* (0.06)	0.13* (0.06)	0.12* 0.06		0.13* (0.07)
Nat unempl. unlagged				-0.38* (0.12)	-0.15 (0.14)				-0.34* (0.11)	-0.27* (0.12)
Nat unempl. lagged 1 yr				0.30 (0.20)	0.35 (0.22)				0.42* (0.18)	0.46* (0.19)
Nat unempl. lagged 2 yr				-0.27 (0.21)	-0.18 (0.22)				-0.52* (0.19)	-0.54 (0.20)
Nat unempl. lagged 3 yr				0.03 (0.13)	-0.02 (0.13)				0.30* (0.13)	0.21 (0.14)
Prov. GDP per capita		0.54 (0.39)					0.01 (0.48)			

Table 2.11 (cont.). Fixed-effect models relating total mortality to province and national unemployment lagged up to 4 years

	Basic stratification					With province-specific time trends				
	i	ii	iii	iv	v	ii	iii	iv	iv	v
	Regressors unlagged and lagged 1, 2, 3 and 4 years									
Prov.unempl. unlagged	-0.19* (0.06)	-0.18* (0.07)	-0.22* (0.06)		-0.19* (0.07)	-0.10 (0.06)	-0.14* (0.07)	-0.10 (0.06)	-0.50* (0.15)	-0.10 (0.07)
Prov.unempl. lagged 1 yr	-0.11 (0.07)	-0.10 (0.08)	-0.02 (0.07)		-0.08 (0.08)	-0.00 (0.06)	-0.00 (0.07)	-0.00 (0.06)	0.49* (0.20)	-0.01 (0.07)
Prov.unempl. lagged 2 yr	-0.09 (0.07)	-0.07 (0.08)	-0.08 (0.07)		-0.11 (0.08)	-0.05 (0.06)	-0.07 (0.07)	-0.05 (0.06)	-0.68* (0.23)	-0.03 (0.07)
Prov.unempl. lagged 3 yr	-0.01 (0.07)	-0.02 (0.08)	0.00 (0.07)		-0.03 (0.08)	0.01 (0.07)	0.03 (0.07)	0.01 (0.07)	0.65* (0.22)	0.02 (0.07)
Prov.unempl. lagged 4 yr	-0.02 (0.06)	0.01 (0.06)	-0.04 (0.06)		0.03 (0.07)	0.06 (0.06)	0.18* (0.06)	0.06 (0.06)	0.56 (0.16)*	0.17* (0.07)
Nat.unempl. unlagged				-0.64* (0.17)	-0.47* (0.18)					-0.40* (0.16)
Nat.unempl. lagged 1 yr				0.51* (0.23)	0.60* (0.23)					-0.47* (0.21)
Nat.unempl. lagged 2 yr				-0.60* (0.26)	-0.53* (0.27)					-0.60* (0.24)
Nat.unempl. lagged 3 yr				0.43 (0.22)	0.49* (0.23)					0.56* (0.22)
Nat.unempl. lagged 4 yr				-0.44* (0.16)	-0.50* (0.16)					-0.65* (0.17)
Prov. GDP per capita		0.78 (0.41)					-0.79 (0.52)			

The series of observations was trimmed four years starting with the last year (1997) in the regressions without lag, three years in the regressions with a one-year lag, two years in the regressions with a two-year lag, and so on. In this way there is a constant number of observations (N = 700) in all regressions and standard errors and statistical significance of coefficient estimates can be somewhat compared.

Table 2.12. Results for fixed-effect models regressing the changes in mortality on changes in economic and demographic variables. Section I: *logarithmic differences* in mortality regressed on first differences in economic and demographic variables. Section II: *first differences* in mortality regressed on first differences in covariates. Section III: *relative* change in mortality regressed on relative changes in covariates

	Unweighted regressions					Weighted regressions				
	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(iii)	(iv)	(v)
Section I – Log difference of mortality ($\ln M_t - \ln M_{t-1}$) as dependent variable										
Prov. unemployment	-0.02	-0.02	-0.05		-0.06	-0.13	-0.31*	0.25*		0.64*
	(0.06)	(0.07)	(0.06)		(0.06)	(0.06)	(0.06)	(0.12)		(0.12)
Nat. unemployment				-0.03	0.02				-2.09*	-2.56*
				(0.07)	(0.10)				(0.24)	(0.25)
Prov. GDP per capita		0.67					-4.21*			
		(0.50)					(0.52)			
R^2	0.349	0.354	0.023	0.022	0.023	0.788	0.806	0.036	0.110	0.141
Province fixed-effects included										
Prov. unemployment	-0.01	0.03	-0.04		-0.06	-0.30*	-0.39*	-0.47*		-0.33*
	(0.07)	(0.07)	(0.06)		(0.06)	(0.06)	(0.06)	(0.09)		(0.10)
Nat. unemployment				-0.02	0.03				-1.15*	-0.87*
				(0.09)	(0.11)				(0.22)	(0.23)
Prov. GDP per capita		0.60					-5.54*			
		(0.52)					(0.66)			
R^2	0.361	0.368	0.034	0.033	0.034	0.824	0.840	0.508	0.510	0.517
Section II – First difference in mortality ($\Delta M_t = M_t - M_{t-1}$) as dependent variable										
Prov. unemployment	-0.22	-0.17	-0.48		-0.63	-1.81*	-3.37*	1.05		3.86*
	(0.58)	(0.60)	(0.53)		(0.68)	(0.51)	(0.54)	(0.89)		(0.89)
Nat. unemployment				-0.20	0.34				-15.8*	-18.5*
				(0.72)	(0.92)				(1.84)	(1.93)
Prov. GDP per capita		7.13					-36.4*			
		(4.60)					(4.32)			
R^2	0.327	0.336	0.025	0.024	0.024	0.742	0.765	0.031	0.106	0.126
Province fixed-effects included										
Prov. unemployment	-0.18	0.20	-0.47		-0.63	-3.23*	-4.02*	-4.28*		-3.31*
	(0.60)	(0.62)	(0.56)		(0.71)	(0.52)	(0.52)	(0.73)		(0.78)
Nat. unemployment				-0.16	0.37				-8.89*	-6.01*
				(0.79)	(0.99)				(1.71)	(1.82)
Prov. GDP per capita		6.68					-48.62*			
		(4.81)					(5.54)			
R^2	0.338	0.348	0.036	0.035	0.036	0.783	0.805	0.477	0.472	0.484
Section III – Relative change in mortality ($[M_t - M_{t-1}]/M_{t-1}$) as dependent variable										
Prov. unemployment	-0.00*	0.01	-0.01		-0.01	-0.04*	-0.07*	-0.02*		0.10*
	(0.01	(0.01)	(0.01)		(0.01)	(0.01)	(0.01)	(0.00)		(0.01)
Nat. unemployment				-0.01	-0.00				0.21*	0.06
				(0.02)	(0.02)				(0.03)	(0.04)
Prov. GDP per capita		0.03					-0.24*			
		(0.03)					(0.03)			
R^2	0.351	0.358	0.023	0.023	0.023	0.778	0.800	0.081	0.086	0.085
Province fixed-effects included										
Prov. unemployment	-0.00	0.01	-0.01		-0.01	-0.06*	-0.09*	-0.03		-0.06*
	(0.01)	(0.01)	(0.01)		(0.01)	(0.01)	(0.01)	(0.01)		(0.01)
Nat. unemployment				-0.01	-0.00				0.11*	-0.18
				(0.02)	(0.02)				(0.03)	(0.04)
Prov. GDP per capita		0.03					-0.30*			
		(0.03)					(0.03)			
R^2	0.363	0.370	0.036	0.035	0.036	0.818	0.837	0.486	0.491	0.501

In all the panels only specifications (*i*) and (*ii*) include year dummies. In the lower panel of each section the regressions include a dummy for province. This is equivalent to including province-specific time trends in non-differenced models, therefore these models are analogous to the

specifications in the middle columns of upper panel in table 2.3. Since some data were missing and first differences were not computed for 1980, *N* was 849 in most regressions (in those in which province GDP is a regressor, generally *N* = 799).

In weighted regressions observations are weighted by the inverse of the squared change of the proportion over 64 in sections I and II. In section III weights are the inverse of the squared relative change in the proportion over 64.

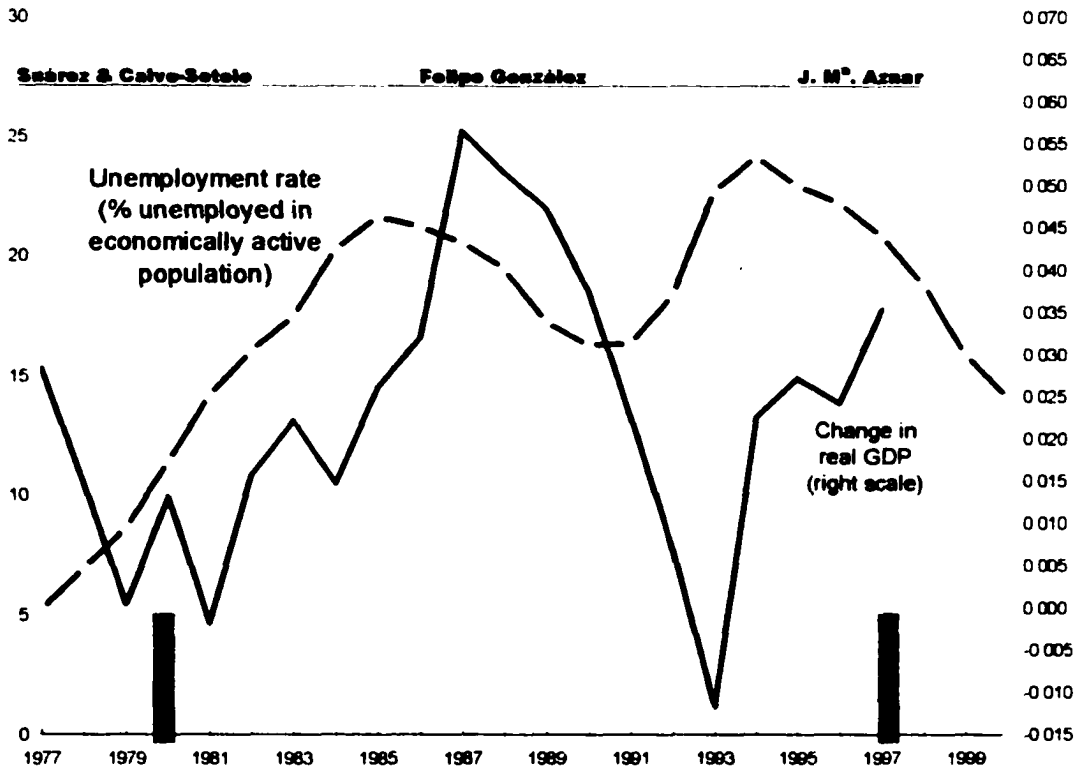
In section I all coefficient estimates and stardard errors are multiplied times 100.

Table 2.13. Regression results for fixed effect models relating mortality to economic and demographic variables. Each section shows a different structure of age-adjustment

	Basic specification					With province specific time trends					Death rates in levels		
	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(iii)	(iv)	(v)	(i)	(ii)	(v)
Section I – No demographic variables included													
Prov. unemployment	-0.43*	-0.39*	0.49*		-0.22*	-0.12*	-0.11*	-0.17*		-0.11*	-3.04*	-2.64*	-1.29
	(0.05)	(0.06)	(0.05)		(0.08)	(0.05)	(0.05)	(0.03)		(0.05)	(0.48)	(0.54)	(0.70)
Nat. unemployment				0.86*	1.07*				-0.18*	-0.08			8.57*
				(0.06)	(0.10)				(0.04)	(0.06)			(0.86)
Prov. GDP per capita		6.43					-0.88					9.14*	
		(4.12)					(0.46)					(3.57)	
Section II – Proportion under 5 as only demographic variable included for age-adjustment													
Prov. unemployment	-0.44*	-0.37*	-0.28*		-0.46*	-0.13*	-0.11*	0.18*		-0.13*	-3.06*	-2.57*	-3.23*
	(0.05)	(0.06)	(0.04)		(0.05)	(0.05)	(0.05)	(0.03)		(0.05)	(0.49)	(0.54)	(0.55)
Nat. unemployment				-0.07	0.34*				-0.20*	-0.08			2.64*
				(0.05)	(0.08)				(0.04)	(0.06)			(0.72)
Prov. GDP per capita		1.42*					0.21					11.94*	
		(0.04)					(0.48)					(3.81)	
Section III – Proportions under 5, over 64 and over 74 included for age-adjustment													
Prov. unemployment	-0.31*	-0.25*	-0.28*		-0.35*	-0.13*	-0.13*	-0.09*		-0.12*	-1.81*	-1.42*	-2.09*
	(0.05)	(0.05)	(0.04)		(0.05)	(0.05)	(0.05)	(0.04)		(0.05)	(0.38)	(0.43)	(0.42)
Nat. unemployment				-0.19*	0.12				-0.05*	0.05			0.27
				(0.05)	(0.07)				(0.04)	(0.06)			(0.55)
Prov. GDP per capita		0.90*					-0.50					5.33*	
		(0.38)					(0.47)					(3.10)	
Section IV – Proportions under 5, over 64, over 74 and over 84 included for age-adjustment													
Prov. unemployment	-0.29*	-0.22*	-0.27*		-0.32*	-0.13*	-0.13*	-0.09*		-0.12*	-1.66*	-1.23*	-1.90*
	(0.05)	(0.05)	(0.04)		(0.05)	(0.05)	(0.05)	(0.04)		(0.05)	(0.34)	(0.44)	(0.42)
Nat. unemployment				-0.21*	0.08				-0.05	0.06			-0.01
				(0.05)	(0.07)				(0.04)	(0.07)			(0.56)
Prov. GDP per capita		1.00*					0.53					5.96	
		(0.38)					(0.48)					(3.09)	

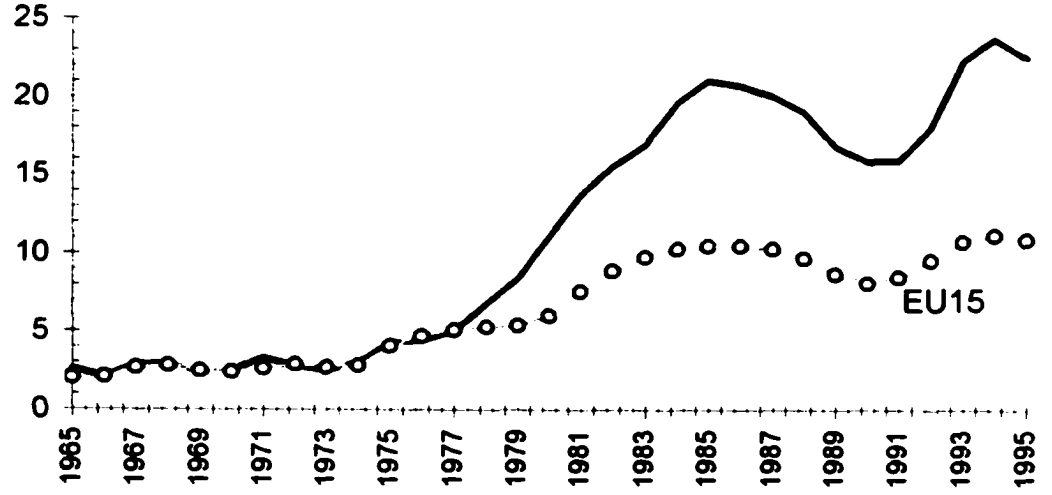
Specifications as in table 2.3. In all specifications $R^2 > 0.9$, except some specifications of section I which do not include year effects or province-specific time trends (for which R^2 is between 0.8 and 0.9). In section II the coefficient estimate for the proportion of population under 5 is negative in all specifications and statistically significant in all but one. In section III the coefficient estimate for the three demographic variables has the expected sign in all the specifications (< 0 for the proportion under 5 and >0 for the proportions over 64 and over 74) and is also statistically significant except for the population under 5 in two specifications. In section IV the coefficient estimates for the proportion under 5 were negative and statistically significant in 11 specifications, those for the proportions over 64 and over 74 were negative and statistically significant in all 13 specifications. However the estimate for the proportion over 84 was positive but not significant in all the specifications with province specific time trends and statistically significant and negative in all the other specifications.

Figure 2.1. Rate of unemployment, yearly variation in real GDP and prime ministers in Spain during the period of the study (between thick gray lines)



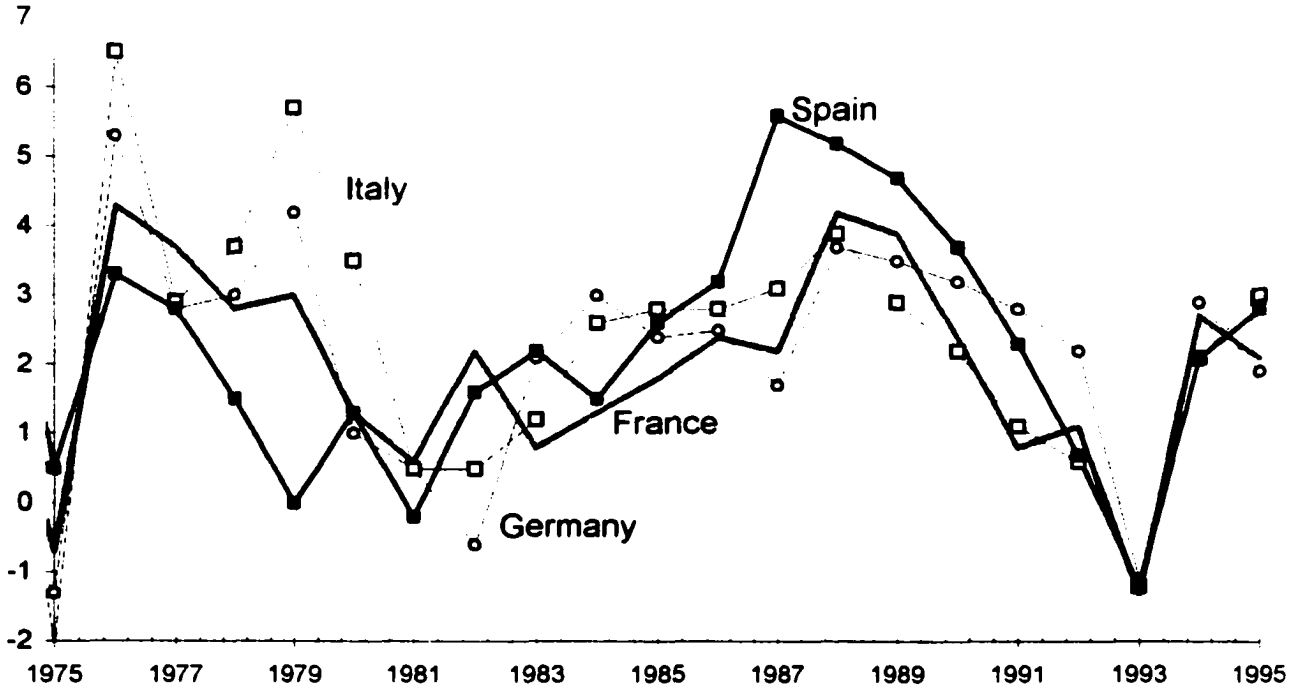
Source: Database TEMPUS, Instituto Nacional de Estadística, Spain

Figure 2.2. Unemployment as percent of total labor force in Spain and the 15 countries of the European Union



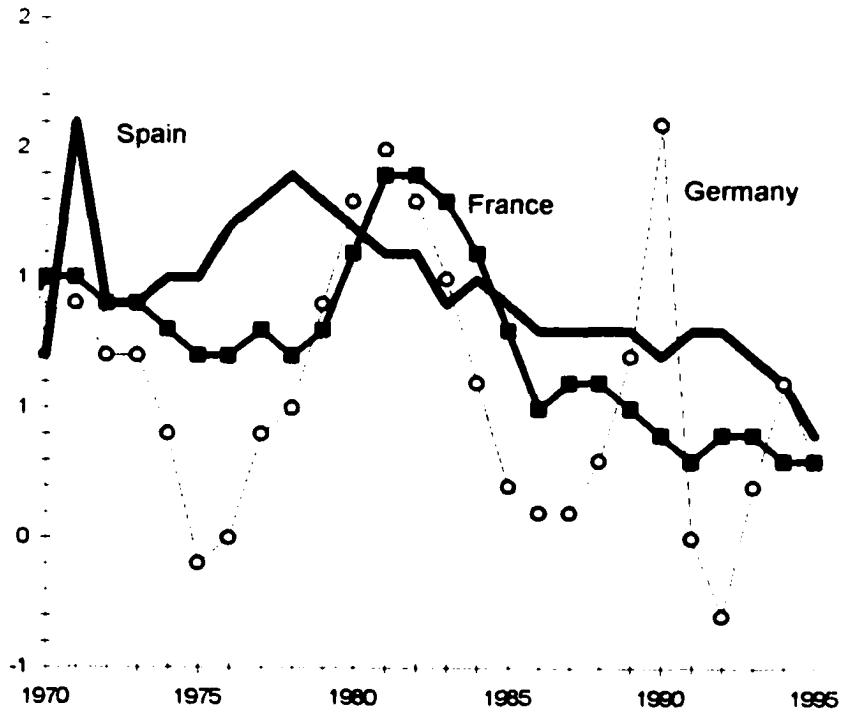
Source: OECD Historical Statistics database

Figure 2.3. Year-to-year real GDP growth (%) in four Western European countries



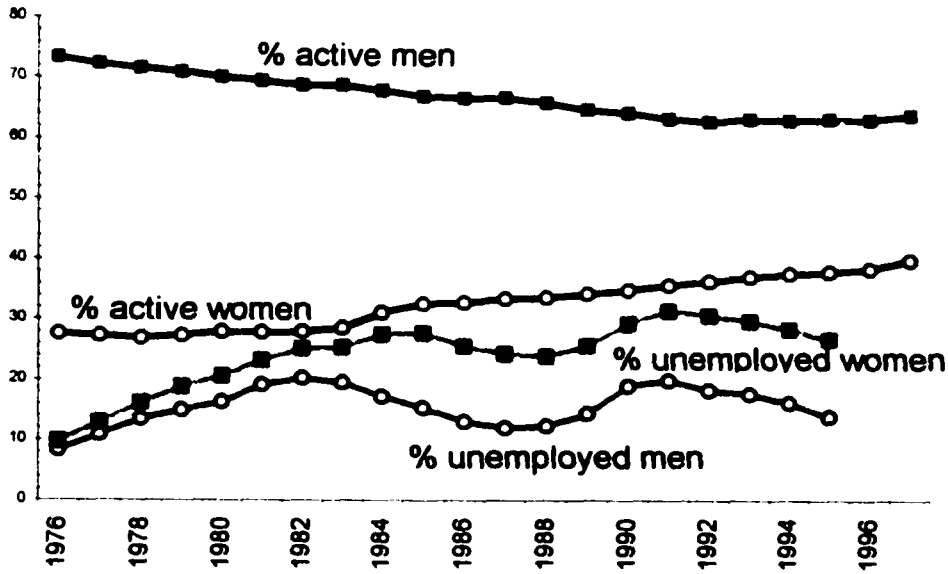
Source: OECD Historical Statistics database

Figure 2.4. Year to year percentage changes in population 15 to 64 in Spain, France and Germany



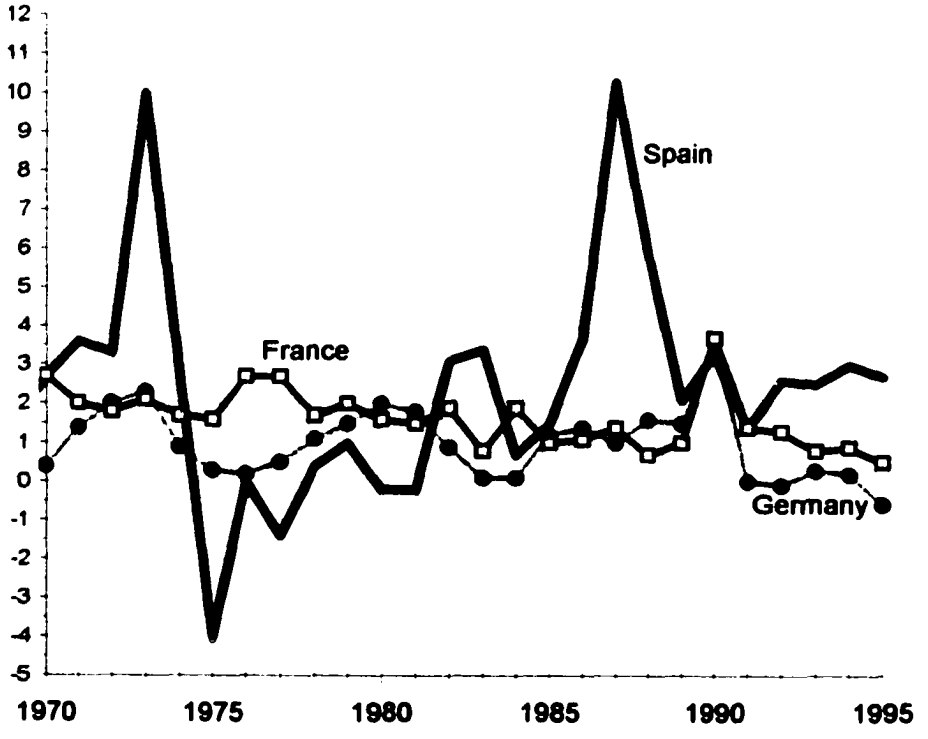
Source: OECD Historical statistics database

Figure 2.5. Proportion active among men and women older than 16 and sex-specific unemployment rates. Spain 1976-1997



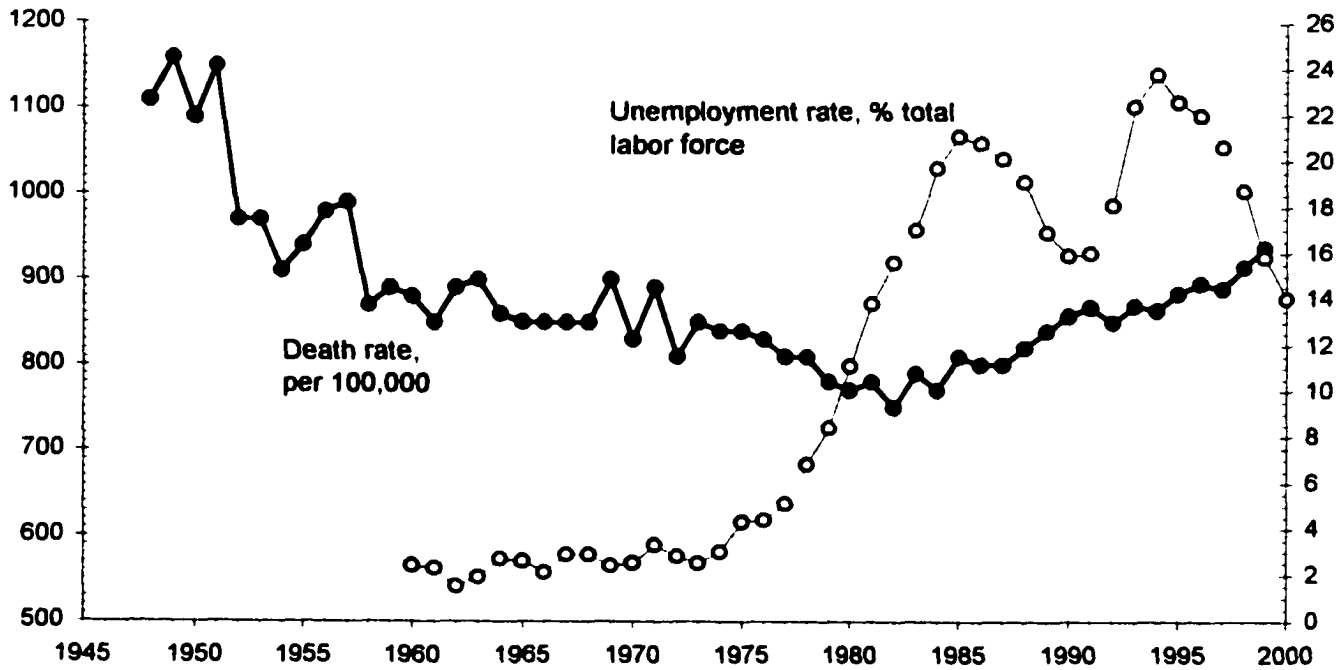
Source: TEMPUS database, Instituto Nacional de Estadística, Spain

Figure 2.6. Year to year percentage change in female labor force in Spain, France and Germany



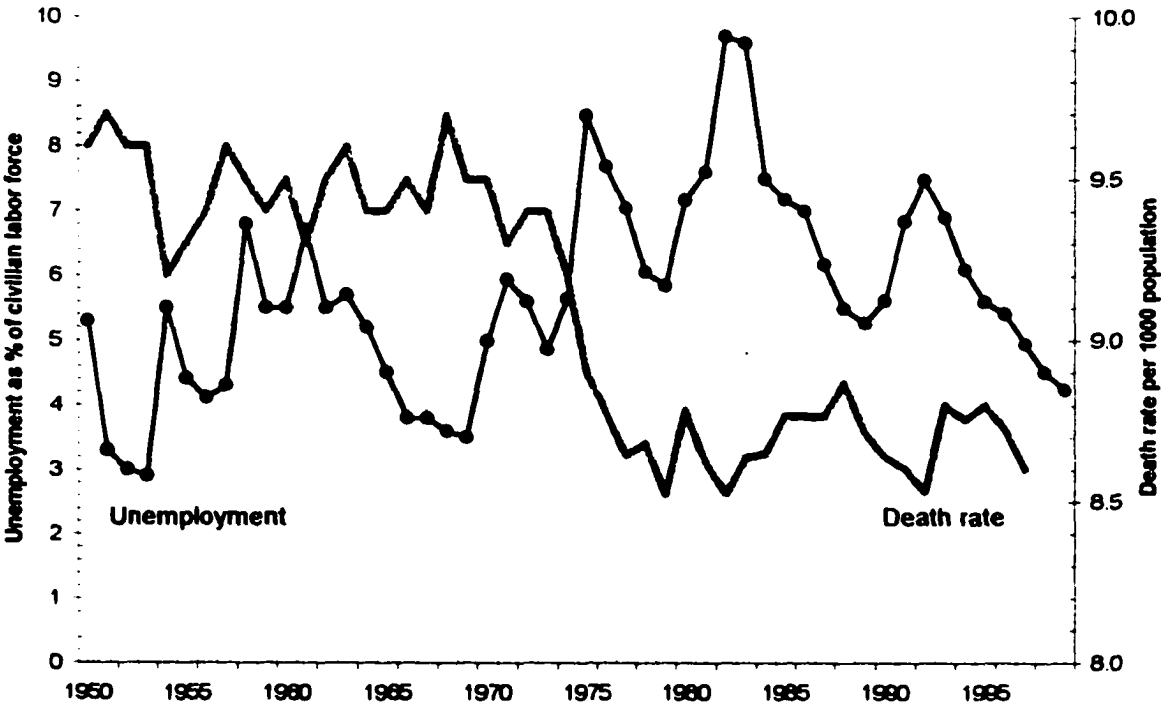
Source: OECD Historical Statistics database

Figure 2.7. Crude death rate and unemployment, Spain 1948-2000



Data sources: Mortality: *United Nations Demographic Yearbook. Historical supplement* (1960-1995) and computation from INE Tempus database (1996-1999). Unemployment: OECD online statistics.

Figure 2.8. Crude mortality and unemployment rate, USA 1950-1995



Source: Historical Statistics of the United States

Figure 2.9. Age-estandardized mortality and year to year change in real GDP. Spain 1980-1997

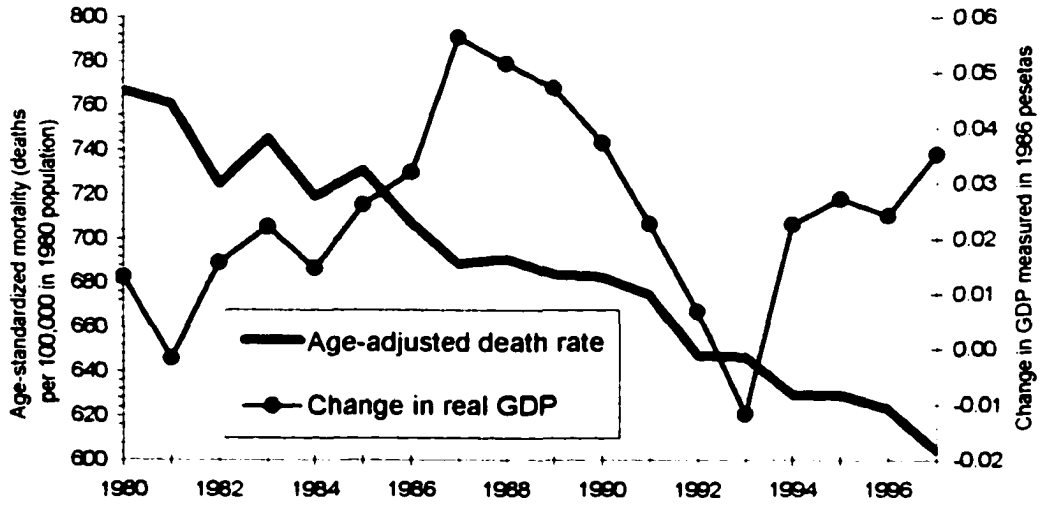
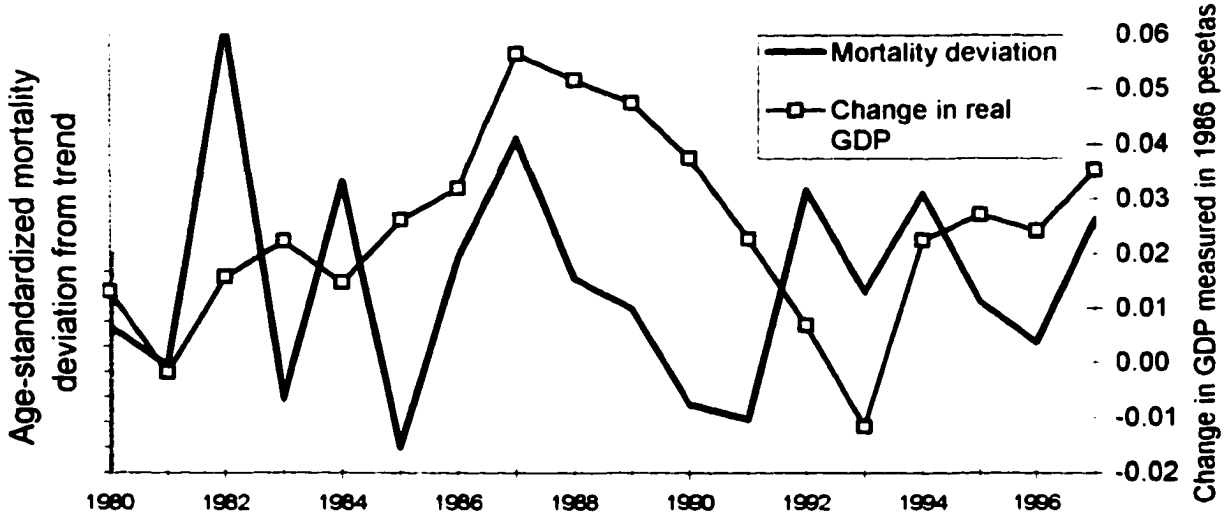
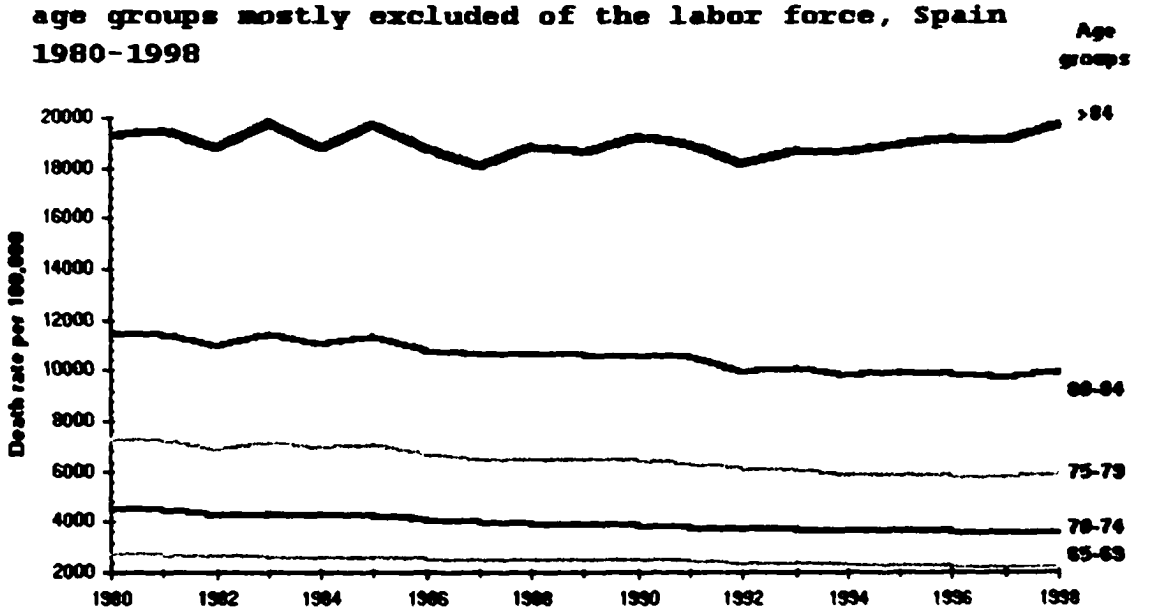


Figure 2.10. Age-estandardized mortality deviation from its linear trend and year to year change in real GDP.
Spain 1980-1997



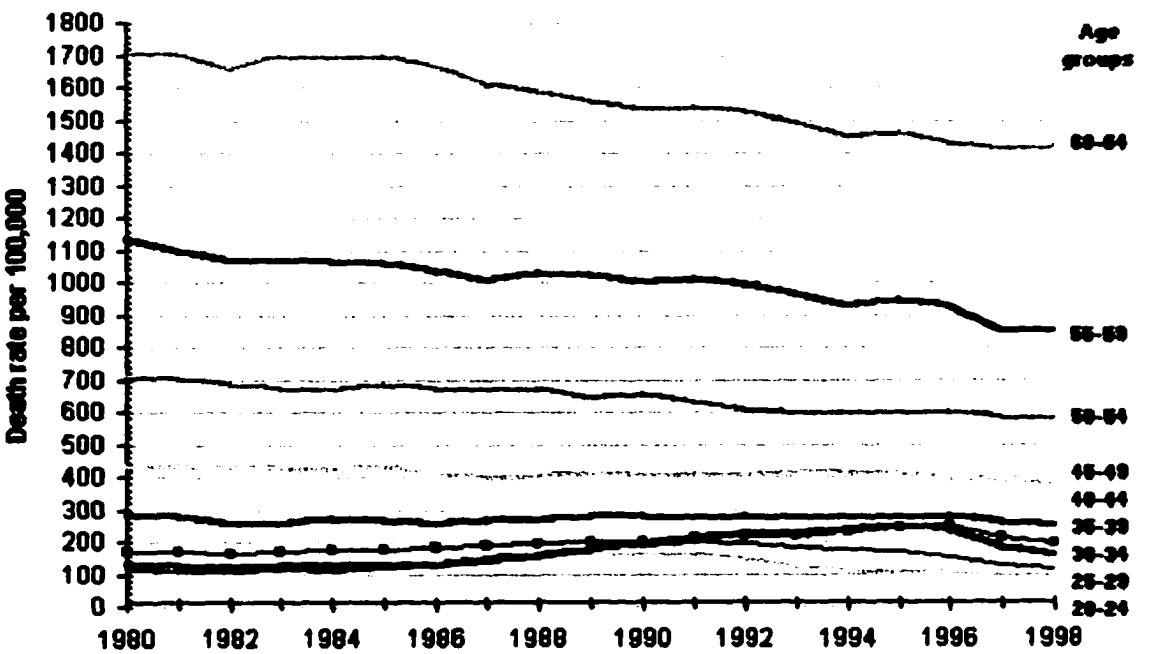
Source: Series computed from crude data taken from TEMPUS database, Instituto Nacional de Estadística, Spain

Figure 2.11. Age-specific death rates for males in age groups mostly excluded of the labor force, Spain 1980-1998



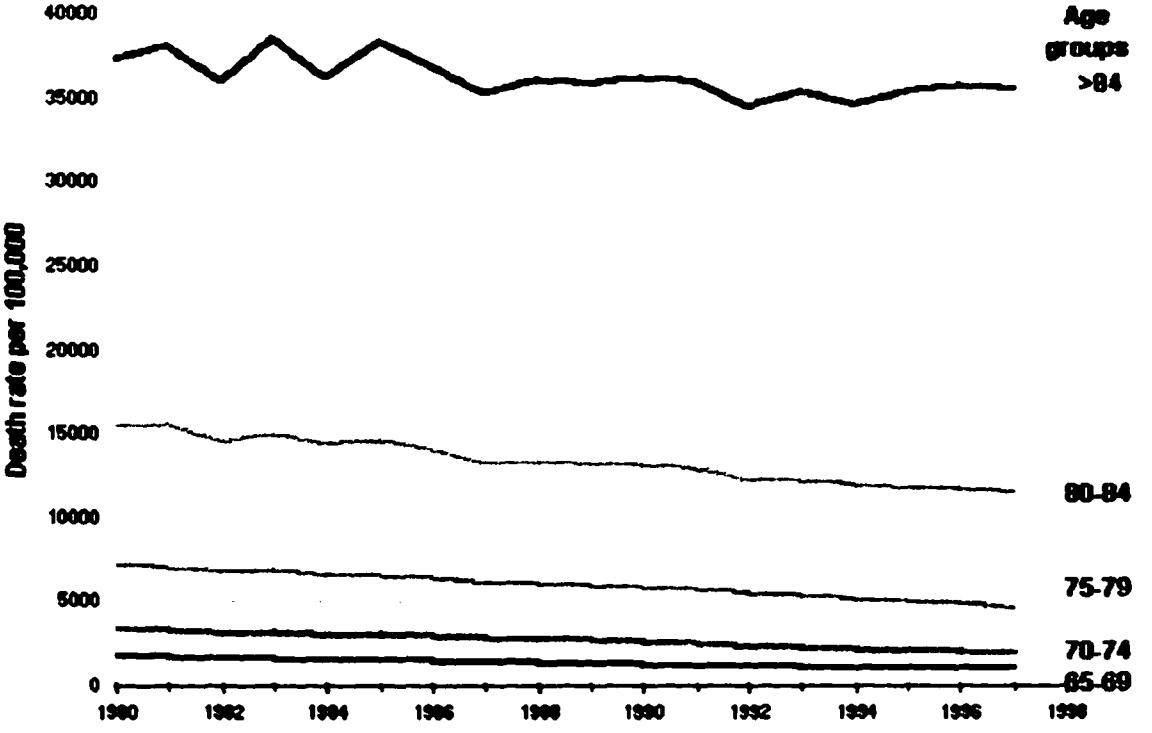
Source: United Nations: Demographic Yearbook, Historical supplement

Figure 2.12. Age-specific death rates for males in working ages. Spain 1980-1998



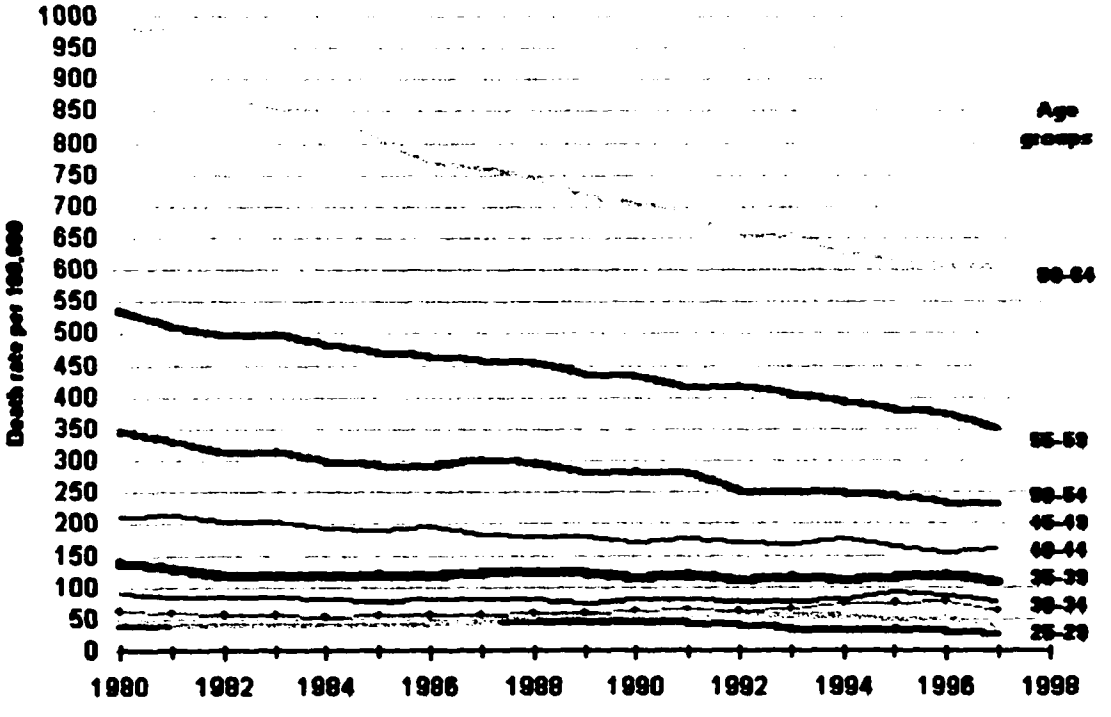
Source: United Nations: Demographic Yearbook, Historical supplement

Figure 2.13. Age-specific female death rates in age groups mostly excluded of the labor force. Spain 1980-1998



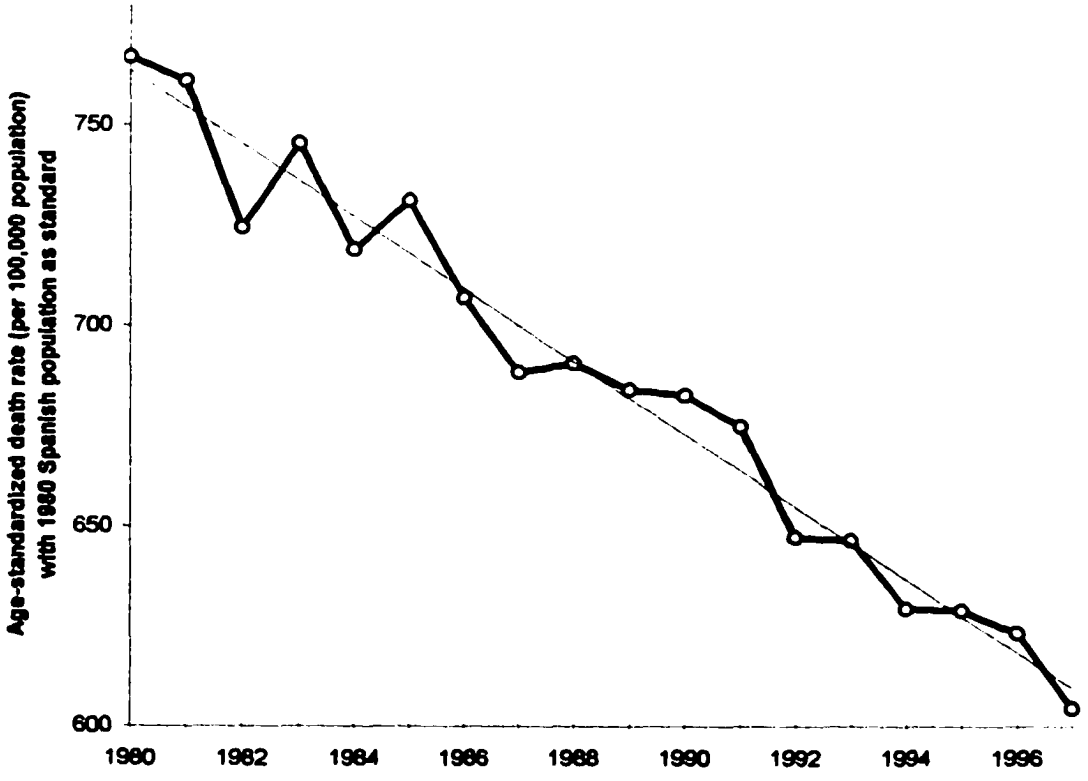
Source: United Nations: Demographic Yearbook, Historical supplement

Figure 2.14. Age-specific death rates for women in working ages. Spain 1980-1998



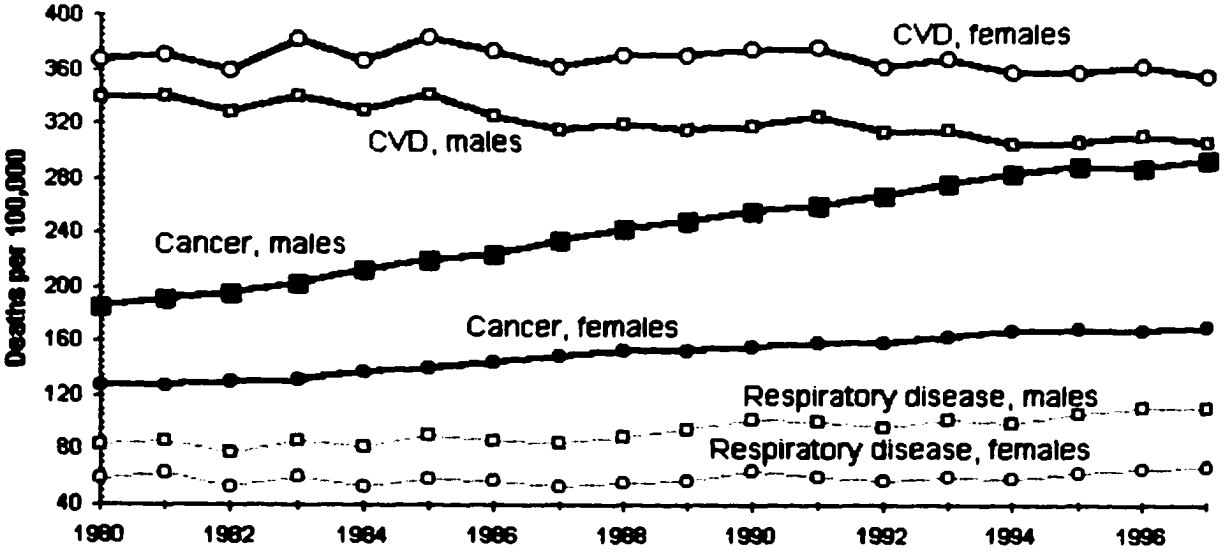
Source: United Nations: Demographic Yearbook, Historical supplement

Figure 2.15. Age-standardized mortality, Spain 1980-1997 (the thin black line is a linear trend)



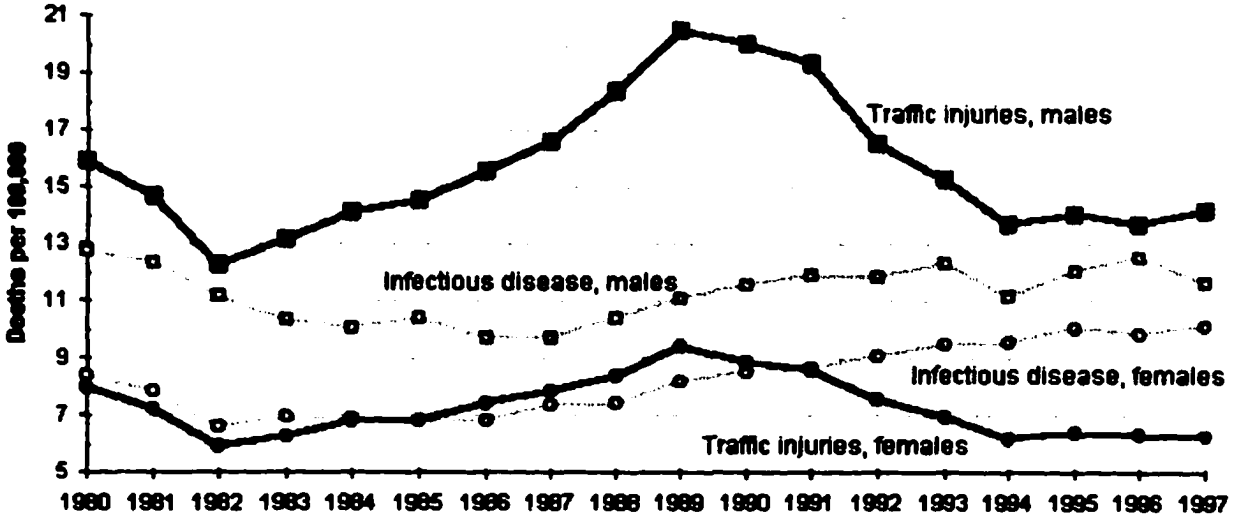
Source: Computed from crude data from TEMPUS database, Instituto Nacional de Estadistica, Spain

Figure 2.16. Crude mortality due to cardiovascular disease, cancer, and respiratory disease, in males and females. Spain, 1980-1997



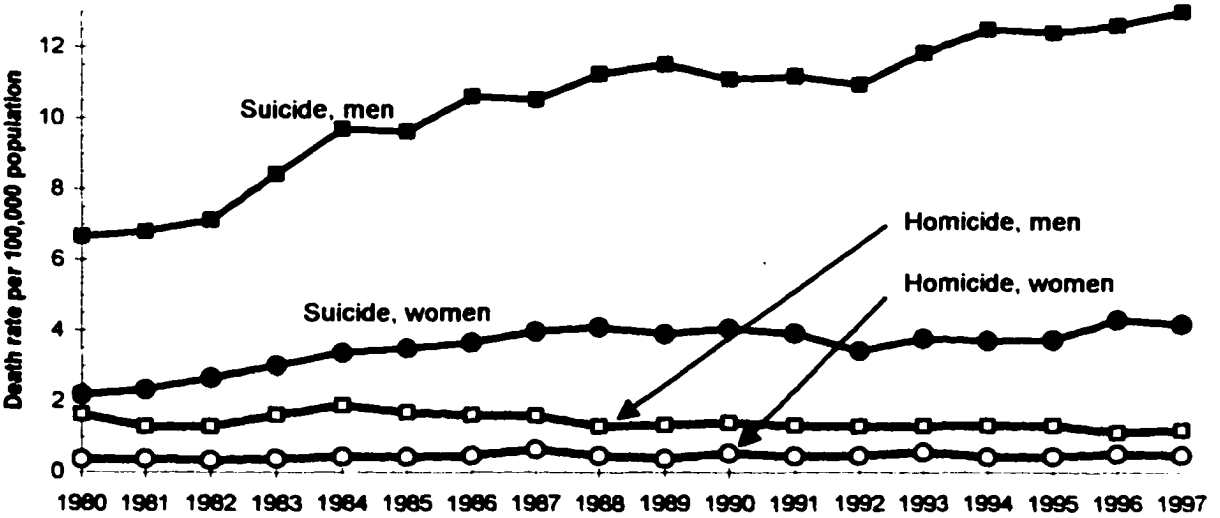
Source: Computed from crude data in TEMPUS database, Instituto Nacional de Estadística, Spain

Figure 2.17. Crude mortality due to traffic injuries and infectious diseases in men anD women. Spain 1980-1997



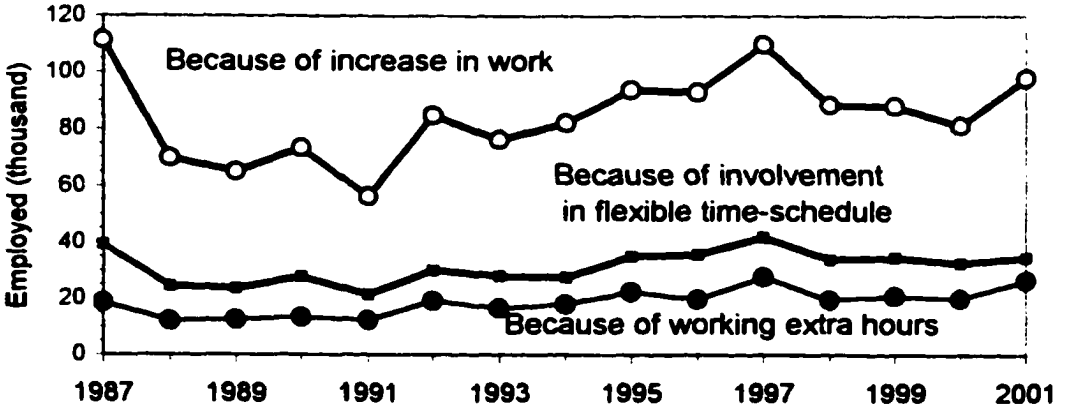
Source: Computed from crude data in TEMPUS database, Instituto Nacional de Estadística, Spain

Figure 2.18. Crude mortality due to suicide and homicide in men and women. Spain, 1980-1997



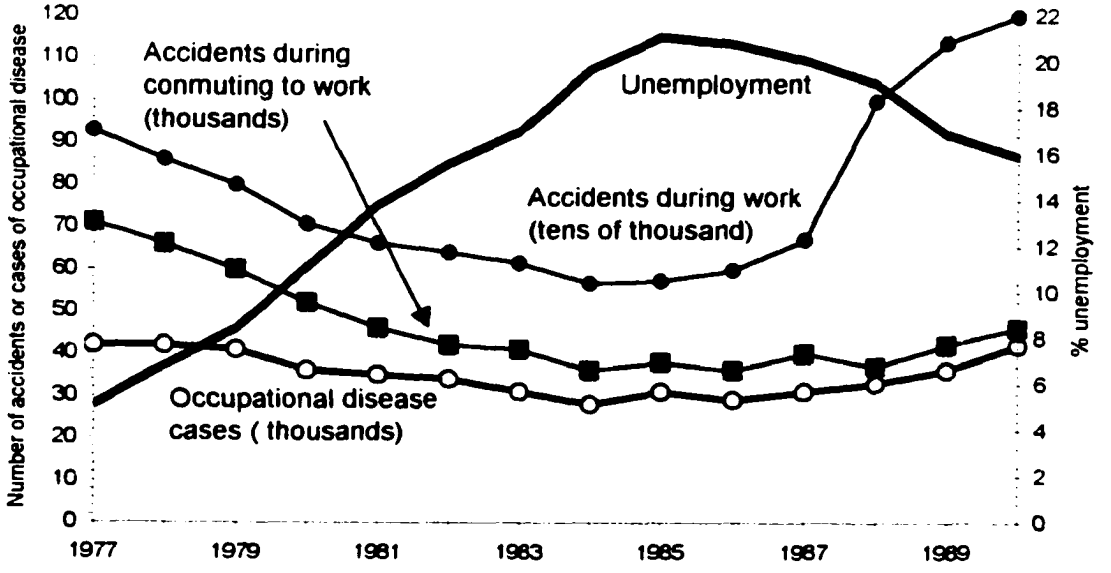
Source: Computed from crude data in TEMPUS database, Instituto Nacional de Estadistica.

Figure 2.19. Employed reporting to have worked more hours than usually, Spain, 1987-2001



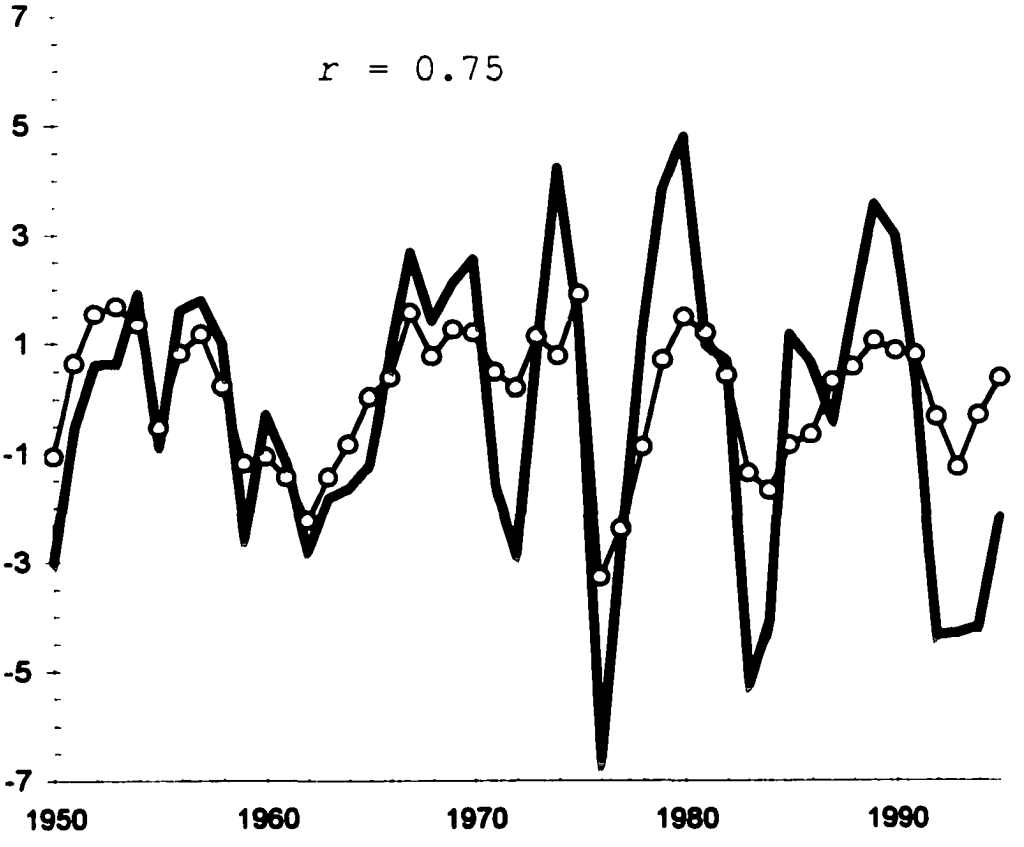
Data source: Database TEMPUS, Instituto Nacional de Estadistica, Spain

Figure 2.20. Unemployment and occupational injuries and diseases, Spain, 1977-1990



Data sources: Bulletin of Labor Statistics, Ministry of Labor, Spain (data reproduced in Falguera i Baro 1992). Unemployment data from OECD Historical Statistics database

Figure 2.21. Mortality due to traffic injuries and index of total industrial production (thick line), both series detrended (HP filter, gamma = 100) and normalized. USA, 1950-1995



Data from *Historical Statistics of the United States*

CHAPTER 3

MORTALITY AND ECONOMIC FLUCTUATIONS IN SWEDEN, 1800-1998

CHAPTER SUMMARY. The relationship between fluctuations of economic activity and mortality in Sweden in the period 1800-1998 is assessed with spectral analysis, local regressions, cross-correlograms and other statistical techniques applied to a price index (GDP deflator) and nominal and volume GDP estimates for the period 1800-1998. For subsamples of the whole period data on harvests, industrial investment, unemployment rates and several indices of manufacturing are also analyzed. The results show an intense relationship, specially strong in the mid decades of the 19th century, between death rates and the harvest yield the preceding year. The influence of harvests on death rates subsides in the last quarter of the 19th century, when death rates start to be strongly related to the up and downs of economic activity—as measured by inflation and capital investment—in a procyclical oscillation that persists throughout the 20th century, though considerably reduced in the latter half of the century. These results confirm the procyclical fluctuation of death rates found in previous research and don't provide any evidence in favor of the hypothesis that attributes the increase of mortality during times of economic expansion to lagged effects of economic slowdown 10 to 15 years before.

I — Introduction

In 1941 Dorothy Thomas published *Social and economic aspects of Swedish population movements*. In previous publications Thomas had shown a counterintuitive short-run association between periods of economic expansion and increases of mortality in Great Britain and the US (Ogburn & Thomas 1922, Thomas 1926).

Sweden probably has one of the best statistical registries in the world in terms of historical demographic data. In *Social and economic aspects* Thomas produced a meticulous picture of the socio-demographic and economic evolution of Sweden from 1750 to the 1930s. The method that Thomas used to study the relationship between fluctuations in economic, social and demographic variables was to compute correlations between deviations from trends. She detrended with a variety of methods (moving averages, lineal and polynomial trends, as necessary) and worked with the residuals as percent deviations from the trend or expressed in standard deviation units, usually reporting the Pearson correlation coefficient between residuals together with its standard error ($r \pm \sigma_r$).

In Swedish data Thomas found many of the effects of economic fluctuations that she had already seen in data from the US and Great Britain. Marriage rates were intensely procyclical, fluctuating "in such close connections with business conditions (...) that they might almost be taken as primary index of business cycles." Birth and fertility rates, too, were "strongly correlated with business conditions, but only in the earlier stage of industrialization, i.e., up to the middle [eighteen] nineties." Death rates showed a "strong tendency to rise after a [harvest] failure and decline in periods of abundance," during the second half of the 18th century. Though death rates "tended to be negatively correlated with the state of the harvest for every period and subperiod" [up to the early 20th century], "after the 1780s the coefficient attained probable statistical significance in only one subperiod, 1863-92."

For the percentage deviations from trends between a business cycle indicator (investment in mechanical equipment for manufacturing adjusted for price changes) and crude death rates, Thomas reported correlations of 0.13 ± 0.14 for the whole period 1865-1913, with correlations of 0.14 ± 0.19 and 0.06 ± 0.22, respectively, for the

subperiods 1865-1892 and 1893-1913 (Thomas, 1941, p. 196). So she concluded that

> In one respect the welfare of the people was independent of fluctuations in business conditions: depression—unlike harvest failure—brought no consistent increases in the death rate, and in fact, there was no significant correlation for either subperiod from the 1860s to the outbreak of the [first world] war, and *such insignificant correlation as existed was in the positive direction* (Thomas, 1941, p. 162; emphasis added by me – JATG].

In this paper I have tried to continue the work of Dorothy Thomas on business cycles and mortality, presenting an analysis of the relationship between different indicators of economic fluctuations and death rates in Sweden in the years 1800-1998. The results of the analysis confirm a clear association until the last quarter of the 19th century between mortality oscillations and harvest conditions, with mortality rising immediately after a bad harvest. Then industrialization started to develop quickly and a procyclical oscillation of mortality emerged and persisted throughout the 20th century. Prior work by Thomas, Eyer (1977, 1984), Brenner (1995) and Ruhm (2000) provides consistent evidence of a link between the fluctuations of economic activity called "business cycles"

and (statistically and demographically significant) fluctuations in death rates in a number of industrial countries. The results of this paper confirm this procyclical fluctuation of mortality in Sweden, but don't provide any evidence in favor of Brenner's theory that the procyclical oscillation of death rates is due to lagged effects of economic slowdowns. In fact most of the results in this paper tend to disprove that hypothesis.

II — Data and methods

The series of annual crude mortality rates (deaths per 1000 population) from 1800 to the present are available online from Statistics Sweden. As indicators of economic fluctuations for the two centuries I used four series of annual estimates of a general price index, nominal GDP, volume GDP and volume GDP per capita[1] (Krantz 2002). These four series and a series of national unemployment rates from 1911 to 1999 (figure 3.1) are unpublished data, kindly provided to me by Olle Krantz (Department of Economic History, University of Umeå). From Thomas (1926), I used

[1] **The price index is a GDP deflator, indexed to make the 1910-1912 price level index equal to 100. The nominal GDP is estimated in nominal million Swedish crowns or *kronen* (SEK), and the volume GDP is the nominal GDP adjusted for the price change and indexed to make volume $GDP_{1930} = 100$, so that, $V = (N/d) \cdot 100 \cdot (d_{1930}/N_{1930})$ where V is volume GDP, N is nominal GDP and d is the deflator.**

two series, a harvest index series (1753-1913) and an industrial investment series (1865-1913). Four indices of industrial manufacturing activity (aggregate hours, average hours, employment and output) are available online from OECD sources for the last five decades and were employed for the analysis of the most recent period.

As in most countries of Western Europe, in Sweden crude mortality declined considerably during the 19th and 20th centuries. From levels around 20 to 30 deaths per 1000 population in the early 1800s, mortality declined to death rates around 14 per 1000 in the early 20th century, reaching a minimum of 9.4 in 1955 (figure 3.1). There are however strong deviations from the trend, including for instance peaks of 40.3 in 1809 and 17.9 in 1917—the world influenza epidemics. Beginning in the mid-20th century, mortality starts to gradually increase, revealing the effect of population aging. Crude death rates that were used in the present analysis are strongly dependant on the population age-structure. However, the analysis of the relationships between the economy and mortality using crude death rates is not biased by age-structure changes as long as it refers to the short-term oscillations and not the long-term changes (Ohlsson & Bengtsson 1984). In the short-

run, age structure can be considered constant. In addition, detrending death rate series substantially eliminates the influence of age on mortality fluctuations.

The three indicators of national output (nominal, volume and per capita GDP) grow exponentially throughout the 19th and 20th centuries. The price index seems to oscillate without trend during several decades in the 19th century, and then increases rapidly with the other indicators.

Death rates and these four economic indicators are non stationary series not only in levels but also in first differences (figure 3.2). Heteroscedasticity is evident for first differences. Obvious differences in variance for different periods are also present when the series are detrended (for instance with the Hodrick-Prescott filter). However, rates of change (i.e., relative differences $[y_t - y_{t-1}]/y_{t-1}$ that can be approximated by the first difference in natural logs, $\ln x_t - \ln x_{t-1}$), seem to eliminate heteroscedasticity (figure 3.3). The augmented Dickey-Fuller test applied to the five series of rates of change of mortality, price index and nominal, volume and per capita volume GDP rejects the hypothesis of unit roots

at the 99% confidence level, so the series can be quite confidently considered stationary.[2]

Since the series of volume GDP and volume GDP per capita fluctuate in a very similar way (the correlation of the annual rate of change in volume GDP and volume GDP per capita is 0.963 for the whole period 1800-1998) and both series rendered almost identical results in the initial steps of the analysis, I did not continue analyzing the per capita series and no result for it will be presented here.

SAS and E-Views were used for the statistical analysis.

III — Cross-correlations

Cross-correlations between the rates of change (r_G) of the variables or their deviations from the secular trends (r_D) provide a starting point for the analysis.[3] The general profile of correlations between rates of change is quite similar to that of correlations between deviations from trend for the whole period of study and for the four half-century subsamples (table 3.1). Many of them are

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[2] Ohlsson & Bengtsson (1984) also transformed demographic variables into "yearly percentage changes" to eliminate the trend before applying spectral analysis.

[3] All the correlations reported in the paper are Pearson product moment correlation coefficients.

statistically significant. Mortality is positively correlated with the price index and negatively with volume GDP. In the 19th century subsamples there are high correlations between death rates and both the price index and the volume GDP. In the first half of the 20th century only price index movements seem to be substantially correlated with the death rate, then in the second half all correlations drop to zero.

These data imply that throughout the period 1800-1950 periods of inflation and deflation are associated with periods of positive and negative change in the death rate, respectively. Similarly, periods with inflation above or below its secular trend coincide with periods of mortality above or below its secular trend, respectively. The graph of the price index and the death rate for the period 1830-1900 (figure 3.4) is very illustrative of the clear association between oscillations of the two variables.

Consistent negative correlations between volume GDP and death rates are observed both for detrended series and for rates of change in the three subsamples before 1950. Growth of GDP is associated with drops in the death rate and also deviations of GDP above its trend are associated with mortality below its trend. These associations are

specially intense during the first half of the 19th century, much weaker in the second half and disappear in the 20th century.

For the whole period 1800-1998 and in the four subsamples both change and deviation from trend of nominal GDP are positively associated with change and deviation from trend of mortality. However the relationship is generally weak and only marginally significant for the deviations from trends (r_D = 0.24) in the two subsamples 1851-1900 and 1901-1950.

In summary, contemporaneous cross-correlations of the death rate and the price index show a positive relationship that grows stronger from the first to the second half of the 19th century to be somewhat reduced in the first half of the 20th century and disappear after 1950. Volume GDP significantly correlates negatively with mortality in the 19th century, particularly in 1800-1850. Nominal GDP shows a weak positive relationship to mortality between 1850 and 1950.

IV – Spectral analysis 1800-1998

Spectral analysis can provide evidence on the frequency components of time series and the relationship

between different oscillating time series, identifying lagged relationships that might be overlooked when looking at contemporaneous cross-correlations. For the death rate series and the three economic indicators series, smoothed periodograms (figures 3.5 to 3.8) clearly show important cyclical components at high frequencies, i.e., at periods under 20 years.[4]

For the death rate change series, most of the cyclical components in the smoothed periodogram (figure 3.5) correspond to oscillations with a period of 10 years or less.[5] In fact there are obvious peaks at a period of 10

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[4] For the smoothing of periodograms I used a Parzen kernel, a common option. The periodograms did not change much when using triangular, quadratic spectral or Tukey kernels, that I also tested. However the periodogram profile is quite different for different bandwidths of the kernel. After trying different bandwidths I chose a bandwidth $l = 1.2\ [F(n/2) + 1]^{1/2}$, where $F(n)$ represents the *floor* of $n/2$, i.e., the largest whole number $k \leq n/2$, and n is the number of observations in the series. *Grosso modo*, the bandwidth l of this Parzen kernel is equal to the square root of n, an option that Gottman (1981) considers appropriate, so approximately $l = 12$ for the periodograms of the whole period 1800-1998 and $l = 6$ for the subsamples of 50 years. These bandwidths substantially reduce the number of peaks of the unsmoothed periodogram, but not as much as to render a flat profile.

To check that only long-cycle and trends of the original series (in levels) are removed by the process of transforming the data into rates of change, I used the squared coherence k^2 of the cross-spectra of the two series (levels and rates of change), for different subsamples, as suggested by Bengtsson & Ohlsson (1985). The k^2 of mortality and the rate of change of mortality is very high, around 90%, for high frequencies corresponding to short-period oscillations. However, the results for the three economic indicators were worst, especially for nominal and volume GDP, not as much for the price index, implying a considerable distortion of short period oscillations of the variables in levels when the data are transformed into rates of growth.

[5] Ohlsson & Bengtsson (1984) also found this death rate oscillation with a period of 10 years in what they called stage II of the Swedish demographic experience, 1800-1869.

years in the subsamples 1800-1950. That peak seems to be displaced toward a higher frequency with a period of 6 years in the subsample 1901-1950 and 1950-1998. In general the spectral densities for high frequency components, i.e., oscillations with a period of few years, drop considerably from each subsample to the next (note different *y* axis scales in figure 3.5).

The smoothed periodograms of the rate of change in prices and nominal and volume GDP (figures 3.6 to 3.8) also reveal high frequency components corresponding to periods from a few to 15-20 years. In the three series the spectral densities for high frequency oscillations with periods around 10 years or less are much larger in the subsamples 1850-1950 than in the other subsamples. These frequencies correspond to the periodicity usually attributed to business cycles (five to eleven years, as suggested for instance by Roepke, 1937). Therefore the stronger oscillatory component of the series in the period 1850-1950 would be consistent with the emergence of an industrial business cycle in the second half of the 19th century and the known reduction of intensity of business cycles after World War II.

The squared spectral coherency k^2 of two time series may be interpreted as the proportion of variance that both series share at a particular frequency band. The cross-spectrum of rates of change in mortality and prices (figure 3.9) reaches a squared spectral coherency $k^2 = 0.7$ for business cycle frequencies of around 8 years in the 1851-1900 subsample. Since the periodograms of both series reveal a peak at that frequency, that implies that around 70% of a major business-cycle frequency is shared by the two series. This is strongly suggestive that one variable is causing the other or that there is a third factor causing both of them.[6] Squared coherence also peaks at business-cycle frequencies in the other three subsamples, but the corresponding k^2 is 0.6 in 1901-1950 and only 0.1 in 1951-1998. The cross-spectra of mortality with either nominal or volume GDP (figures 3.10 and 3.11) reveal much smaller values of k^2 for business-cycle frequencies. In the subsample 1951-1998 the cross-spectra of mortality with the

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[6] **This is the so-called method of concomitant variation, or fifth of the cannons derived by John Stuart Mill (in *A system of logic,* 1856) as logical strategies to infer causal relationships: "Whatever phenomenon varies in any manner whether another phenomenon varies in some particular manner, is either a cause or an effect of that phenomenon, or is connected with it through some fact of causation" (cited by Last, 1988).**

three economic indicators (figures 3.9 to 3.11) reveal very low values of k.

The phase of the cross-spectrum provides information on the degree of overlapping or lead-lag oscillation among the two series. It makes sense to look at it when there is a high degree of spectral coherence among the series (Warner 1998). In the case of mortality and the price index, the phase diagram for frequencies corresponding to a period around 10 years reveal values between 0 and 1 radians for the phase of change in mortality by change in the price index for the subsample 1800-1850. In the 1851-1900 subsample the phase is between -1 and 0, around 0.5 to 0.7 in the 1901-1950 subsample and around 2 in the last subsample 1951-1998. Since $f = \varphi/2\pi$, where φ is the phase in radians and f is the phase in fractions of a cycle, these values represent an approximate f in the interval from -0.2 to 0.3 cycles. This implies that peaks in the death rate change would tend to occur some two years after a peak of inflation in the second half of the 19th century, more or less coinciding with the peak in inflation in 1900-1950 and some two or three years before the maximum inflation in the last half of the 20th century. The result of the phase spectrum provides a quantitative estimate of

the impression provided by the visual examination of the series in the period 1850-1900 (figure 3.4).

In summary, spectral analysis reveals high spectral power for oscillations at business-cycle frequencies in all the series, with high coherences only between the oscillations of mortality and prices from 1850 to 1950.

V — Local regressions

The procedure of differencing is often considered to introduce noise in the potential relationship between variables. Yule (1921) showed that differencing an oscillating series selectively changes the amplitude of oscillations. Local regression, sometimes called *loess*, provides a means to study the relationship between variables of non-stationary series. The procedure yields a series of regression coefficient estimates with confidence bands that are dependant on the variability of the regressor along the whole time-sample.

Loess of death rates on the three detrended series of economic indicators provides confirmation of the results already presented.[7] The estimated regression coefficient

[7] For the local regressions I used a window of 51 observations symmetrically weighted from 0.05 to a peak of 13.9 for the 26th observation and back to 0.05 for the 51st observation. The profile of these weights resembles closely that of the Parzen kernel. The exact

relating death rates to the price index (figure 3.12, first panel) is positive and statistically significant at a 95% confidence level between the 1830s and the early 1900s. In the following decades the coefficient is much smaller, though still significant, then becomes indistinguishable from zero. When the regressor is the nominal GDP (figure 3.12, second panel) the regression coefficient estimate is positive for most of the 19th century, but it is only statistically significant at the 95% confidence level for a brief period towards the end of the 19th century. Throughout the 20th century the coefficient estimate is indistinguishable from zero. The volume GDP renders a different picture (figure 3.14, third panel), with a negative regression coefficient estimate until the 1920s or 1930s, statistically significant at the 95% confidence level for the whole 19th century and marginally significant in the first decades of the 20th century. These results as well as the weakening correlations between economic variables and mortality rates throughout the 20th century seem to suggest a dampening of the effect of the economy on death rates after the first decades of the 20th century. This will be discussed below.

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series of the weights that I used is as follows: 0.1, 0.2, 0.3, 0.4, 0.5, 0.7, 0.9, 1.2, 1.5, 1.9, 2.5, 3.2, 4, 5, 5.9, 7, 8, 9, 10.2, 11.3, 12.2, 13, 13.6, 13.8, 13.9, 13.8, 13.6, 13, etc., down to 0.1.

VI – Links between the economy and mortality fluctuations in the 1800s and early 1900s

The statistical evidence already presented shows a clear connection between death rates fluctuations and the up and downs of the Swedish economy between 1800 and the early decades of the 20th century. In the first half of the 19th century spurts of mortality coincided with periods of increasing prices. Mortality increases were even more strongly associated with years of real GDP growth below the trend (table 3.1). In the second half of the 19th century the connection of mortality fluctuations with real GDP considerably weakens, while the death rate moves quite consistently up and down with prices. This link inflation-mortality substantially remains during the first half of the 20th century, when the impact of GDP movements on death rates apparently disappears.

The harvest is a major factor influencing the economy and the death rate during the 1800s. Correlations of the harvest index with movements in economic indicators and death rates (table 3.2) show that during most of the 19th century bad harvests were associated with high or rising mortality one year later. During the period 1800-1850 there

is also a strong association between the harvest index and price movements. Years of better harvest than the previous year translate into accelerated growth of volume GDP ($r_{\square}$ = 0.59 in 1800-1840), though nominal GDP decreases ($r_{\square}$ = -0.49), probably because of the strong deflation. The effect of harvests on prices and nominal GDP is dramatically reduced after 1840, but there is still a strong association between periods of good harvest and volume GDP growth. The association of bad harvests with high or rising mortality and good harvests with low or diminishing death rates is maintained throughout the 19th century, achieving its highest level in the mid-decades of the century, when the correlation between the harvest index and the rate of change in mortality is r_D = - 0.38. This impact is not surprising given that persons involved in agriculture and subsidiary occupations constitute 79% of the total Swedish population in 1800, 78% in 1850 and still 55% in 1900 (Thomas 1941). The share of "agriculture and ancillaries" in GDP ranged between 35% and 40% from 1800 up to the 1870s, and was still 25% in the 1910s (figure 3.13). Before industrialization the inability to store food over long periods after good harvests generated overconsumption and waste and seriously limited the reserves that were

available to face the food scarcity following bad harvests (Thomas 1941). These periods of overabundance or dearth of food and agricultural commodities translated into dramatic deflation or inflation of prices respectively (table 3.2). For Ohlman & Bengtsson (1984) up to the 1880s there was a clear link between real wages and mortality fluctuations, but this link was largely mediated by the outcome of the rye harvest that was the major determinant of real wages by its impact on the cost of living. Thus, in the first half of the 19th century the positive association between price index and mortality and the negative association between real GDP and death rates can be interpreted as being driven by the strong dependence of mortality on harvests, together with the large impact of agriculture on GDP and the dependence of the price level on harvests.

From the mid-19th century "the standard of living increased in consequence mainly of improvements in agricultural productivity. The old mortality pattern was broken as a result of better nutritional standards" (Ohlsson & Bengtsson 1994). Judging by the relationship between mortality and the GDP deflator (table 3.1, figures 3.4 and 3.12) in the second half of the 19th century

periods of inflation were clearly associated with spurts in mortality. This connection between inflation and high death rates persists into the first half of the 20th century despite the fact that the agricultural link explanation seems less and less applicable as we advance into the final decades of the 19th century, when industrialization developed quickly. In fact during the period 1881-1913 the correlations between the harvest index and mortality become indistinguishable from zero (table 3.2).

Before World War II, the cycle of inflation-deflation was one of the known characteristics of the "trade cycle," with inflation developing in the upward swing of the cycle and prices falling with each business cycle contraction (Roepke 1937, Sherman & Kolk 1996). The wholesale price index of industrial commodities was in fact one of the series still included by the National Bureau of Economic Research in its 1950 list of roughly coincident indicators of the business cycle, though probably in answer to new realities, it was removed of the NBER list in the 1975 revision (Gabisch 1989). Therefore the association of inflation periods with spurts of mortality during the last decades of the 1900s and first decades of the 20th century

can be interpreted as strong evidence of a procyclical movement of mortality during these years. This is confirmed by the relationship between business cycle indicators and mortality deviations in the period 1865-1913. The correlations of manufacturing and mining investment (investment in fixed capital that increases when the economy thrives and expectations of profit are good) and mortality (table 3.3) clearly show that peaks and troughs of investment are associated, to a large extent, with peaks and troughs of mortality. The relationship is weak during the years 1865-1889 but becomes quite strong (r = 0.48) during the years 1989-1913 when we consider the correlation of mortality with investment in the previous year.[9]

In spite of the view that the connections between economic variables and death rate fluctuations was mostly gone already in the period 1869-1914 (Ohlsson & Bengtsson 1984), the association between fluctuations of various business cycle indicators and mortality rates leaves no

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[9] For the correlation of this index of investment with mortality Thomas (1941) reported coefficients of 0.13 ± 0.14 for the period 1865-1913 and 0.06 ± 0.22 for the subperiod 1893-1913. In my computations with SAS these values are 0.15 ± 0.15 and - 0.01 ± 0.21 (assuming that Thomas' standard error for r is the standard error for z in Blalock, 1960, where the skewed form of the sampling distribution of Pearson's r is discussed). Thomas would had found that the weak correlation 0.06 ± 0.22 in 1890-1913 increased to 0.48 ± 0.21 by simply lagging investment one year—as she had done with the harvest index. But you don't look for what you don't want to find, and judging by her publications, Thomas probably was quite troubled by her discovery of mortality going up in "good times."

doubt regarding the existence of a procyclical oscillation of mortality in Sweden during the final years of the 1900s and first decades of the 20th century. In the first half of the past century nominal GDP fluctuations show a weak association with death rate movements, with a marginally significant r_D = 0.24 (table 3.1), but the correlation of detrended volume GDP and mortality is zero. To interpret this contemporaneous correlations as evidence against the procyclical fluctuation of mortality is misleading, as the correlations are much higher with a short lag. For mortality and volume GDP lagged one or two years r_D goes up to 0.18 and 0.27, respectively. For mortality correlated with nominal GDP lagged one or two years, r_D is 0.50 and 0.53, respectively.

VII – Fluctuations of the real economy and mortality in the 20th century

The standard deviation of the detrended series of death rates is 1.49 in 1851-1900, 0.76 in the first half of the 20th century (0.46 discounting the year of the world flu epidemics, 1918) and only 0.17 in 1951-1998. As time passed there was an intense reduction in the amplitude of the fluctuations of mortality in Sweden (figure 3.1). Were

these fluctuations, in spite of their falling intensity, still related to the business cycle? The evidence already presented points to an affirmative answer for the first decades of the 1900s. The evidence is harder to assess, as we will see, for the remainder of the century.

In most industrial countries the association between price movements and business cycles drastically changed during the mid decades of the 20th century. The inflation-deflation fluctuation linked to the traditional trade cycle disappeared, and more or less continuous inflation emerged, a phenomenon sometimes interpreted as a consequence of the hegemony of monopoly capital (Sherman & Kolk 1996) or, more realistically in my opinion, as an effect of the application of Keynesian policies to regulate the cycle (Mattick 1978). Sweden is one of the countries where Keynesianism (*avant la lettre*) was already orienting economic policy in the early 1930s, in the form of the ideas and practical suggestions of Wicksell, Myrdal, Ohlin, Hammarskjöld and others (Galbraith 1987). Continuous inflation began in Sweden in the mid 1930s and continued throughout the century (figure 3.1, second panel).

The disappearance of price fluctuations associated with the business cycle must seriously distort the

relationship between money-based business-cycle indicators and a variable like the death rate. This is probably part of the explanation of the different picture shown by the correlations of mortality with the GDP deflator and nominal or volume GDP in 1901-1950 and after 1950 (table 3.1).

Business-cycle indicators that are not money-based offer an alternative to explore the impact of economic indicators on mortality.

The Dickey-Fuller test applied to Swedish national unemployment rates 1911-2000 rejects the hypothesis of unit roots at the 99% confidence level, so the series can be quite confidently considered stationary and susceptible to spectral analysis. Both unemployment and mortality have peak spectral densities at frequencies corresponding to a period between 9.8 and 11 years (figure 3.14). For that frequency the spectral squared coherence of the two series is around 67% and the spectral phase of the cross-spectrum is -2.5 radians. Since $f = \varphi/2\pi$, where φ is the phase in radians and f is the phase in fractions of a cycle, $f = -2.5/2\pi$ = aprox. - 0.4. Therefore peaks in unemployment occur four tenths of a cycle, some 4 years before peaks in mortality (for cycles of about ten years). This implies

peaks in unemployment occurring at or slightly before troughs in mortality, i.e., peaks in mortality approximately coinciding or scarcely preceding the lowest unemployment (assuming a symmetrical raise and decline of it along the cycle). The maximum of spectral density at a common frequency, the high squared coherence and the spectral phase constitute strong evidence supporting the procyclical oscillation of mortality from 1911 to the end of the century.

Other business cycle indicators available for the last half of the 20th century also provide evidence of a link between economy fluctuations and mortality. Of four indicators of manufacturing activity that I checked, two, the indices of aggregate hours and average hours, show statistically significant correlations with mortality deviations at a 95% confidence level; a third one, the index of manufacturing employment, shows a significant correlation at a 90% confidence level (table 3.4).[9] The periodograms of the detrended series of these three indices show peaks of spectral density at frequencies around 10 years. At these frequencies the cross-spectrum with death rates (table 3.5) show squared coherences between 40% and

[9] The forth one is the manufacturing output index whose correlations with mortality deviations are positive for lags 0 to four, with a maximum $r = 0.20$ ($P = 0.17$) for a two-year lag.

55% and spectral phases implying that oscillations of aggregate hours and manufacturing employment are approximately synchronized with death rates while average hours precede by some 4 years (figure 3.15).

The indices of manufacturing aggregate hours and manufacturing employment fluctuate quite in parallel and both have a high correlation with the detrended volume GDP at zero lag (table 3.6). The highest correlation of the average hours index with GDP movements is at a two year lag. A diagrammatic representation of the relationships between oscillations in all these variables based on the patterns observed in the data (figure 3.16) perhaps can be illustrative. The death rate goes up with the ascending phase of the cycle, when hiring is increasing aggregate hours and reducing average hours in manufacturing.[10]

Therefore, in the second half of the century the association of the death rate movements with the fluctuations of the economy, measured with cross-correlations at zero or few-year lags, is statistically significant at the usual levels of confidence for two

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[10] It must be noted that in a country like Sweden most deaths correspond to retired people, so to think that there is direct link of this fluctuation to business and working conditions is wrong (though the link exists for occupational injuries and illnesses as well as traffic injuries).

business cycle indicators and marginally significant for other three indicators, including unemployment rate and volume GDP (tables 3.4 and 3.5). It can be concluded that death rates continued fluctuating with the business cycle during the most recent decades, though damped down.

For particular causes of death like liver cirrhosis and cardiovascular disease in given age-groups, Starrin et al. (1990) also found a procyclical oscillation of the death rate in the years 1963-1983.

VIII — The question of lags

An important problem in the analysis of the relationship between economic fluctuations and mortality is the issue of lags. In fact, in the results of major investigations in the field (Thomas, Eyer, Brenner, Ruhm) generally there is agreement about the procyclical oscillation of the death rate. What is highly controversial is Brenner's view that attributes the spurt in mortality during rapid economic growth to a long-lagged effect of the previous high levels of unemployment, that would have effects extending for 10 to 15 years. Though this theory has been strongly criticized, for many it has intuitive appeal, because it seems obvious that "recession must be

bad for health" and, therefore, that any relationship between economic growth and mortality must result from the lagged effect of a prior recession.

It seems reasonable to think that economic factors may have a lagged effect on mortality. In modern times most deaths are due to chronic diseases (cardiovascular disease, cancer, respiratory disease) that develop over years. However, the model of stress-related mortality that was developed by Eyer (details are in Tapia 2002) implies economy-mortality relationships that are coincidental or lagged a few years only. These are precisely the lags that spectral analysis or cross-correlograms reveal in these Swedish data.

In cross-correlograms of death rates with lagged volume GDP, in each 25-year period between 1800 and 2000 the maximum cross-correlation between mortality and lagged GDP is for lags no larger than five years, and usually much shorter (table 3.7). The correlations between detrended mortality and lagged detrended GDP seem to show a pattern of change around the 1870s. In the years 1851-1875 the relationship between economic activity, indexed by the GDP, and fluctuations in mortality is coincidental and strongly negative, probably reflecting the impact of good/bad

harvests on economic growth/slowdown and high/low mortality. However from the period 1876-1900 onward the relationship turns positive (with above trend mortality associated with above trend GDP), and lagged one or two years. After 1950 this relationship is weakened, although it remains positive and most intense for a one- or two-year lag.

In general, in cross-correlograms of death rates with lagged economic indicators (figures 3.17 and 3.18), for lags larger than a few years the cross-correlation drops to zero. The highest correlations are for zero lag or for a short lag of one or two years. For detrended mortality and lagged unemployment rates (figure 3.18) the highest correlation, $r_D = -0.34$, is between unemployment at year t and mortality two years later, at $t + 2$. However, as also shown by the cross-correlogram, mortality in a given year is highly correlated with unemployment three years later ($r_D = 0.61$). In the cross-correlograms of rates of change (figure 3.17) the highest correlation of mortality with prior unemployment is present for a lag of three years ($r_G = -0.33$), but the correlation of mortality with unemployment

three years later is much higher (0.62).[11] This surprising phenomenon of the relationship of death rates with economic variables in the future being stronger than the relationship of the economic variable with the future death rate is also present in the cross-correlograms of death rates with the index of manufacturing aggregate hours, the price index and the nominal and volume GDP (figure 3.18). I was unable to determine if this is an statistical artifact, if it has to do with the Easterlin hypothesis or if it results from some other phenomenon. In any case it is largely at odds with the possibility of the economy having effects on death rates with lags of 10 to 15 years, as Brenner maintains. The large peaks at zero- or one-year lag in the cross-correlogram of rates of change of mortality with rates of change of volume GDP and GDP deflator (figures 3.17 and 3.18) strongly suggest a coincidental relationship between the variables, not a lagged one.

Local regressions with lagged values do not support Brenner's hypothesis of lagged effects of the recession either. Regressing the detrended death rate on the

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[11] If we eliminate the 1921 observation for unemployment, which is a clear outlier, this result dissapears, so it is quite unreliable. The unemployment rate in the Olle Krantz series jumps from 3.0% in 1920 to 14.9% in 1921. In Maddison (1964) the unemployment estimate is 1.3% for 1920 and 6.4% for 1921. All these estimates are referred to total labor force.

detrended price index lagged one year renders a coefficient estimate not very different from that obtained in the regression using coincidental values (figure 3.12). The coefficient estimate reaches zero at previous years as the lag increases and with a four-year lag the coefficient estimate becomes negative for most of the range, though it is statistically significant only for a short interval. With longer lags the coefficient estimate becomes increasingly unstable crossing the zero line several times and being statistically non significant for practically the entire regression range. This is also the general pattern for local regressions of detrended mortality on detrended nominal or volume GDP, where lags above 4 or 5 years render coefficient estimates crossing the zero line several times in the range 1800-1998.

To further investigate Brenner's hypothesis of a long-lagged effect of the economy on death rates I ran a regression with detrended death rates as the dependent variable and sets of lagged values (from 0 to 15) of detrended volume GDP and GDP deflator as regressors. I also examined lagged unemployment rates, including and excluding different sets of variables, in different combinations. I also tried regressions with the variables in rates of

change. For variables with lags over five years all these regressions rendered coefficient estimates with *t* values under 2, except a few cases in which marginally significant coefficients suggested mutually inconsistent long-lag effects of the economy on mortality rates.

In Sweden the largest unemployment rates during the 20th century were present in 1921-1922 and 1932-1934 (figure 3.19), when joblessness reached levels of 15% and 13%, respectively. If Brenner's hypothesis fits these data, mortality should have deviated above its trend around the years 1931-1937 and 1942-1949. In these two periods mortality oscillated above and below trend, and was in fact below its trend most of the time.

IX – A final comment

Economic variables like GDP or unemployment estimates are largely inaccurate and conclusions based on them have to be viewed with skepticism. However, errors in measurement usually tend to minimize relationships between variables, not generate links between them. Therefore it is unlikely that relationships like those reported in this paper are spurious results. Moreover, they are consistent

with the findings of other investigations. The data presented here showed that, in Sweden, short-term oscillations of mortality in the two past centuries have been largely related to the harvest during the earlier years and to the business cycle during the years later. Mortality oscillations have been largely reduced in size in recent decades and in this investigation no attempt has been made to measure the degree to which the oscillations in the economy impact in mortality. That would require age-specific or age-adjusted mortality rates and other kind of data, probably disaggregated by geographical regions and adjusted for a number of possible confounders.

Table 3.1. Correlations (times 100) between three economic indicators and death rates, computed both among detrended series (r_D) and among year-to-year rate of change (r_G) of the four variables. Series detrended with the Hodrick-Prescott filter ($\gamma = 100$). Sweden, 1800-1998

	Nominal GDP		Volume GDP		Death rate	
	r_D	r_G	r_D	r_G	r_D	r_G
1800-1850						
Price index	89***	86***	- 73***	- 75***	24·	32·
Nominal GDP			- 40**	- 30·	1	10
Volume GDP					-53***	-44**
1851-1900						
Price index	81***	78***	13	- 21	46***	38**
Nominal GDP			63***	44**	24·	17
Volume GDP					-26·	-26·
1901-1950						
Price index	91***	89***	-43**	-35*	30*	31*
Nominal GDP			-17	12	24·	17
Volume GDP					- 1	-22
1951-1998						
Price index	56***	81***	-08	-26·	0	-1
Nominal GDP			69***	34*	5	3
Volume GDP					6	0
1800-1998						
Price index	49***	85***	-14*	-36***	8	27***
Nominal GDP			64***	18*	1	12
Volume GDP					-1	-28***

· $P < 0.1$
* $P < 0.05$
** $P < 0.01$
*** $P < 0.001$

Table 3.2. Correlations (times 100) of a harvest index of the previous year (0 = disaster harvest, 9 = excellent harvest) with a price index (GDP deflator), nominal GDP, volume GDP and death rates as detrended series (r_D), or in rates of growth (r_G). For the detrending of the four series it was used the Hodrick-Prescott filter (γ = 100)

	Price index		Nominal GDP		Volume GDP		Mortality	
Period	r_D	r_G	r_D	r_G	r_D	r_G	r_D	r_G
1800-1840	-68***	-60***	-49**	-33*	59***	69***	-17	-13
1841-1880	-7	-17	9	13	40*	43**	-28'	-38*
1881-1913	-16	-16	-12	13	10	34'	-18	6

' $P < 0.10$

* $P < 0.05$

** $P < 0.01$

*** $P < 0.001$

Source: Author's elaboration from data in Thomas, 1941 (harvest index), Krantz, 2002 (GDP deflator, nominal and volume GDP) and Statistics Sweden (death rates).

Table 3.3. Correlations (times 100) between detrended series of death rates and a business cycle index lagged up to four years. Death rates detrended with the Hodrick-Prescott filter ($\gamma = 100$). The detrended investment indicator data (investment for mechanical equipment in manufacturing and mining, in SEK adjusted by a price index of industrial raw materials) are percentage deviations from a third degree parabola. Sweden, 1865-1913

Lag	1865-1913	1865-1889	1890-1913
0	15	20	- 1
1	29*	23	48**
2	25·	20	44*
3	24	24	25
4	12	19	- 9

· $P = 0.087$

* $P < 0.05$

** $P < 0.01$

Source: Author's elaboration from data in Thomas, 1941 (detrended business cycle index) and Statistics Sweden (death rates).

Table 3.4. Correlations (times 100) between detrended series of death rates and four business cycle indicators (national unemployment and three indices of manufacturing) lagged up to five years. All series detrended with the Hodrick-Prescott filter, γ = 100. Sweden, 1950-1998, except last column on the right

Lag	Aggregate hours	Average hours	Employment index	Unemployment rate 1950-1998	Unemployment rate 1911-1998
0	13	-37**	-25'	-15	-24*
1	17	-11	20	-26'	-29***
2	31*	20	24'	-9	-34**
3	12	21	6	1	0
4	-14	7	-14	12	15
5	- 2	19	-08	16	17

* $P < 0.05$

** $P < 0.01$

' $P < 0.10$

' $P = 0.11$

Source: Author's elaboration from data collected online from the US Bureau of Labor Statistics (business cycle indicators, downloaded from http://data.bls.gov) and Statistics Sweden (death rates, downloaded from http://www.scb.se/eng/).

Table 3.5. Relationships between oscillations in death rates and oscillations in eight economic indicators, based on cross-spectra of rates of change (of mortality and the economic indicator) and detrended series. The spectral phase estimates the years that the oscillation in mortality precedes the oscillation in the economic variable. All data are referred to wave frequencies with periods around 11 years. Sweden, 1950-1998

	Squared spectral coherence k^2		Spectral phase in years	
	Rate of change	Detrended series	Rate of change	Detrended series
Price index	0.1	0.2	2	2
Nominal GDP	0.1	0.3	1	0
Volume GDP	0.1	0.2	- 1	-1
Unemployment rate	0.2	0.3	- 4	-5
Aggregate hours	0.1	0.4	1	-1
Average hours	0.2	0.4	- 3	-3
Employment index	0.1	0.4	1	0
Output index	0.1	0.3	- 1	-2

Source: Author's elaboration from industrial manufacturing data collected online from the US Bureau of Labor Statistics (indices of aggregate hours, average hours, employment and output) and data in Krantz, 2002 (GDP deflator, nominal and volume GDP, and unemployment rates).

Table 3.6. Correlations (times 100) between detrended series of three manufacturing indices, national unemployment rate and death rate with lagged volume GDP. All six series detrended with the Hodrick-Prescott filter, γ = 100. Sweden, 1950-1998

GDP lag	Aggregate hours	Average hours	Employment index	Unemployment rate	Death rate
0	74***	7	74***	-70***	12
1	51***	21	59***	-67***	27'
2	4	-42**	18	-42**	11
3	-31*	-38**	-19	-15	2
4	-39**	-25'	- 30*	16	-6
5	-35*	-12	- 30*	34*	-9

' $P < 0.10$

* $P < 0.05$

** $P < 0.01$

*** $P < 0.01$

Source: Author's elaboration from data collected online from the US Bureau of Labor Statistics (manufacturing indices), Statistics Sweden (death rates) and Olle Krantz (volume GDP and unemployment rate).

Table 3.7. Pearson coefficient for the correlation between detrended series of volume GDP and death rates, for the same year or for GDP lagged up to five years. Sweden, 1850-1998. Both series detrended with the Hodrick-Prescott filter, γ = 100

Lag	1851-1950	1851-1875	1876-1900	1901-1925	1925-1950	1951-1975	1976-1998
0	-4	-46*	18	-33	34˙	-4	18
1	7	-30	44*	11	39˙	-4	45*
2	15	12	35˙	62**	9	-26	25
3	4	32	5	32	-30	-6	3
4	2	36	7	30	38˙	-19	-3
5	4	6	-25	39˙	-12	-39˙	-1

˙ $P < 0.10$

* $P < 0.05$

** $P < 0.01$

Source: Author's elaboration from volume GDP data in Krantz, 2002, and death rates from Statistics Sweden.

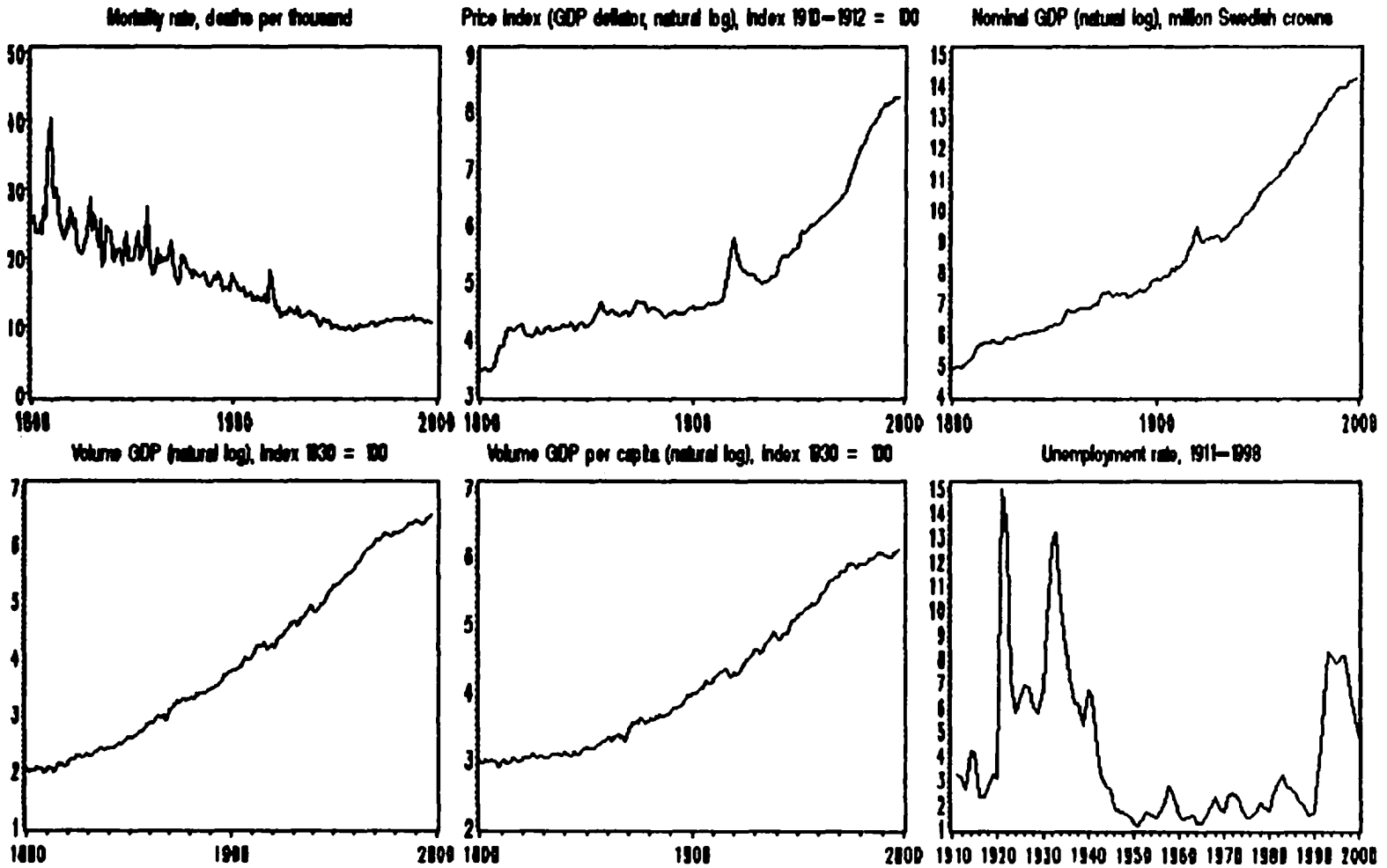


Figure 3.1. Death rates and five economic indicators, Sweden, 1800-1998. Data from Statistics Sweden and Olle Krantz (University of Umeå).

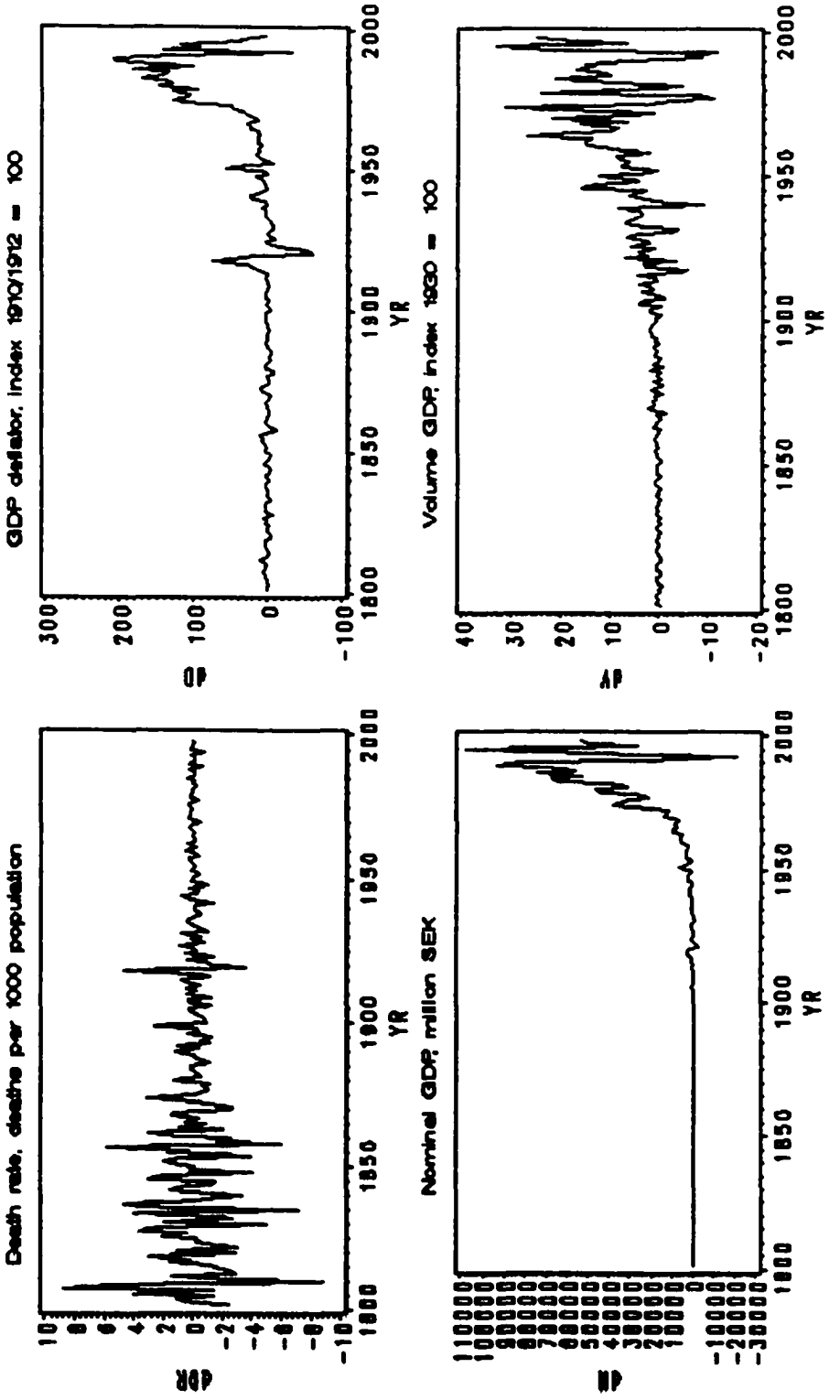


Figure 3.2. First differences in death rates, GDP deflator, nominal GDP and volume GDP. Sweden, 1800-1998.

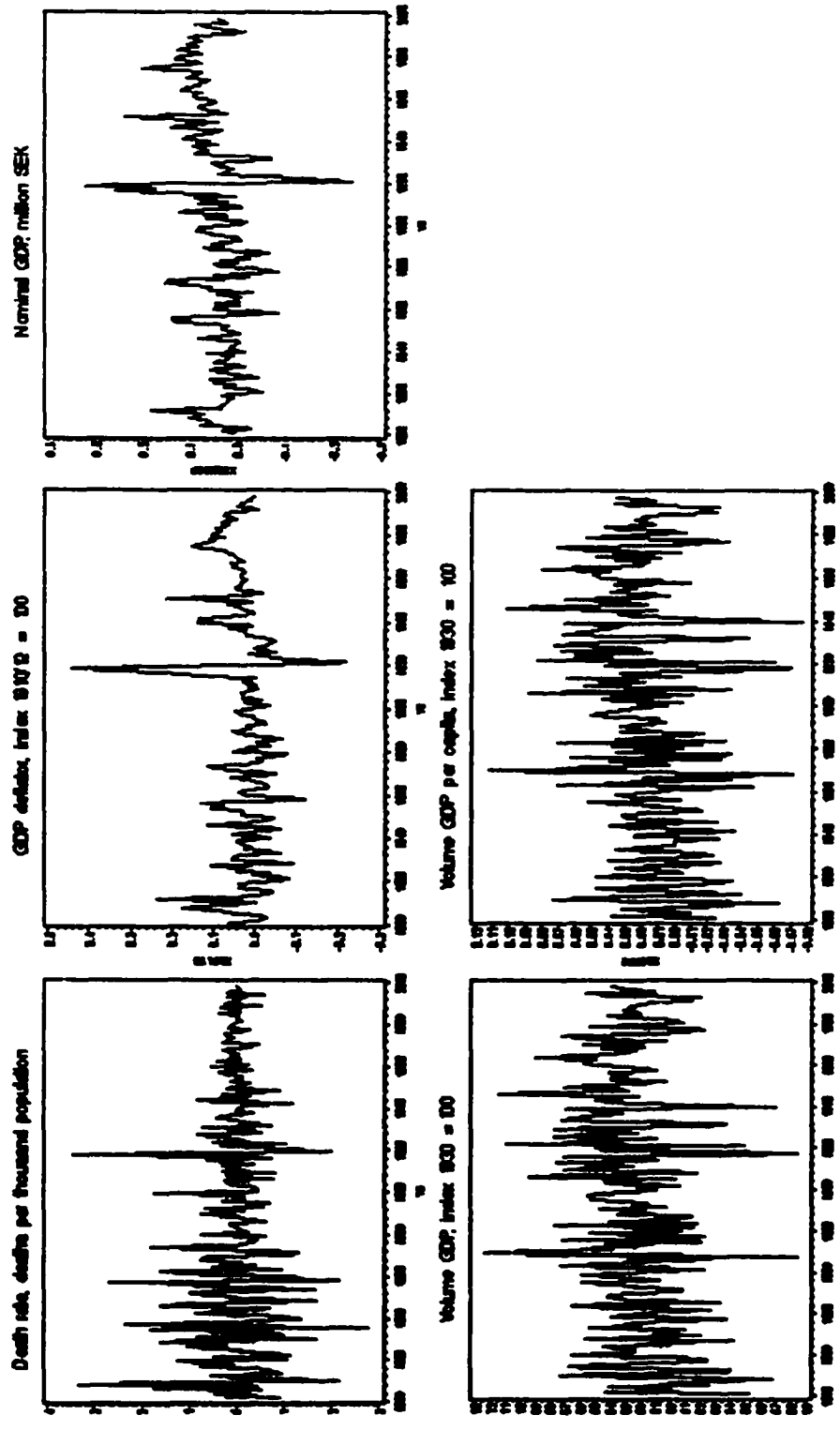
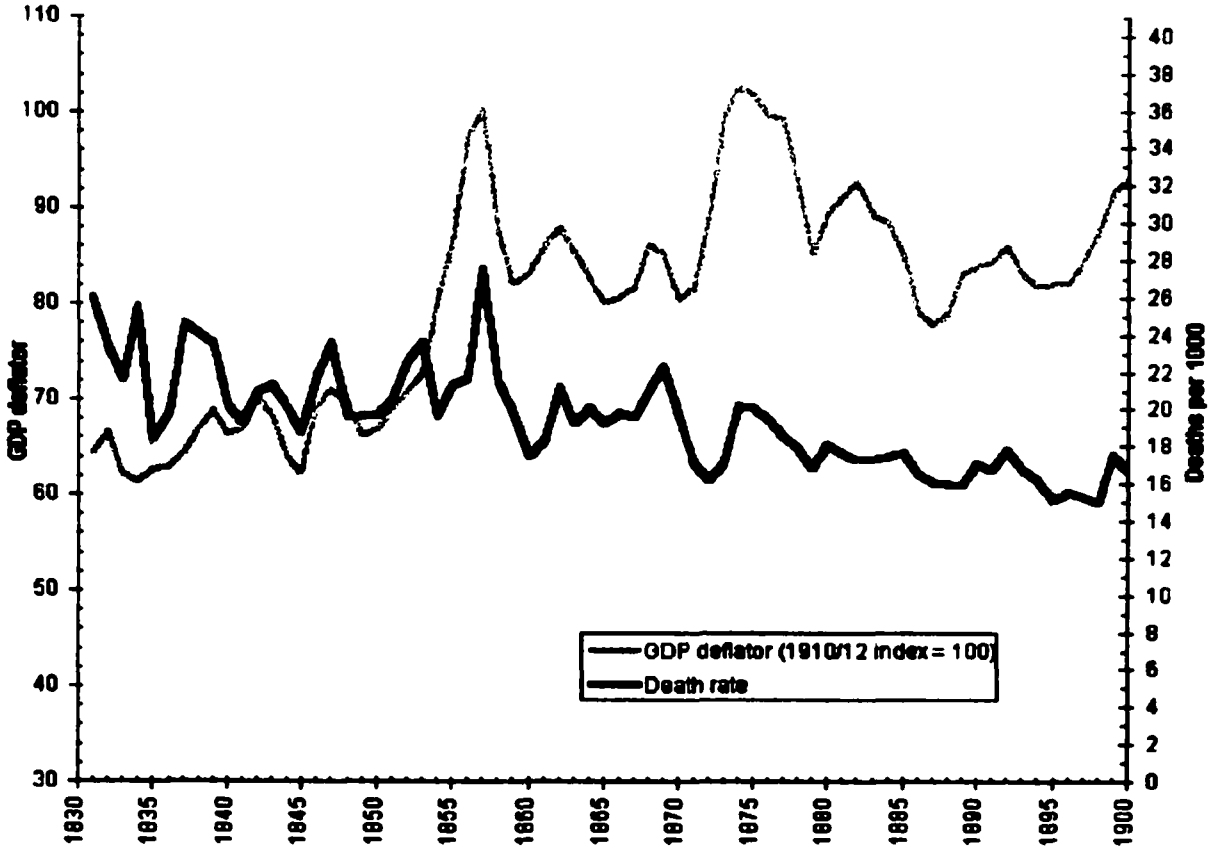


Figure 3.3. Mortality and four economic indicators, series in annual rate of change. Sweden, 1800-1998.

Figure 3.4. Crude mortality and price index (GDP deflator). Sweden, 1830-1900



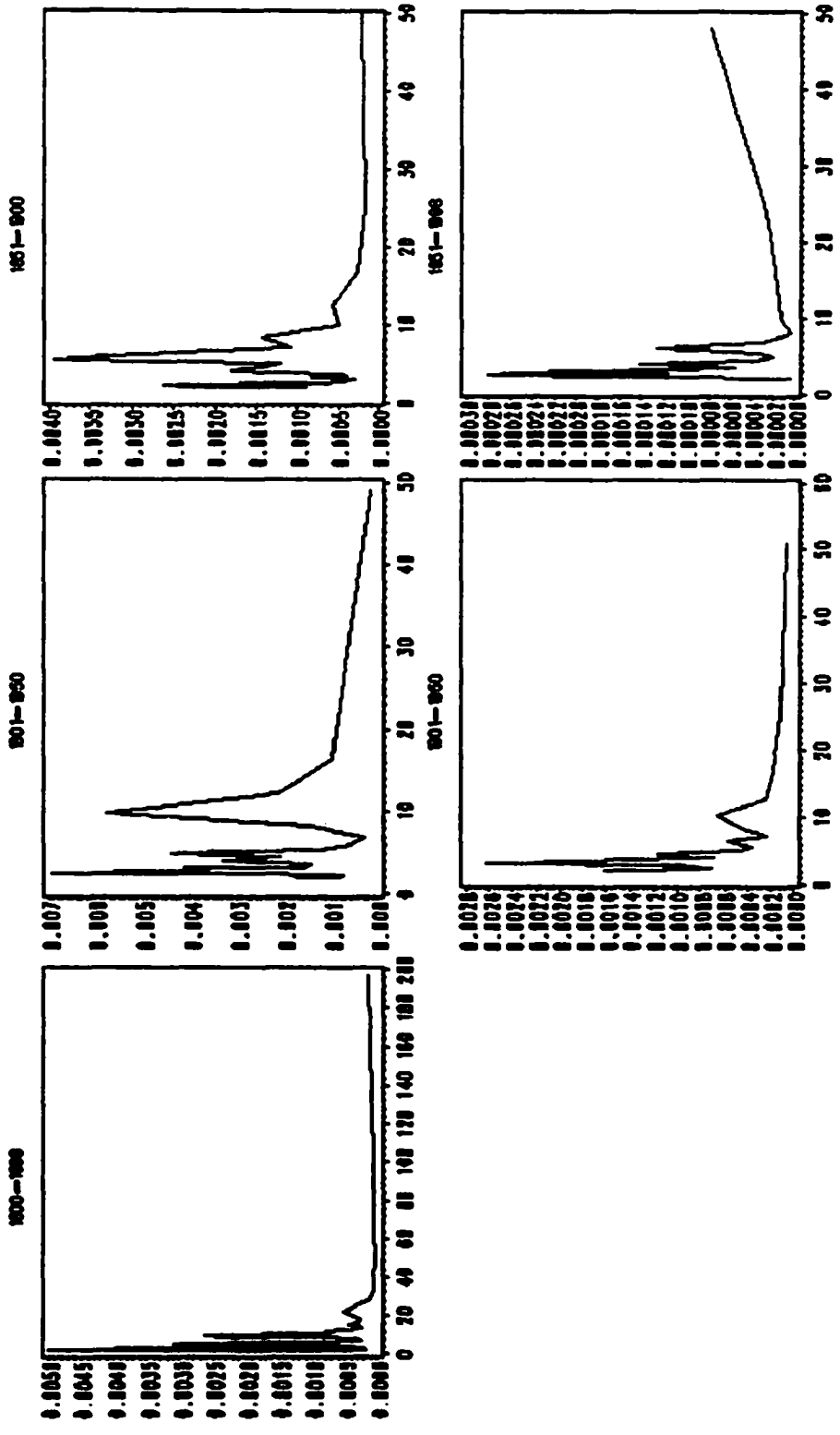


Figure 3.5. Spectral densities of the series of annual rate of change of mortality. Smoothing of the periodogram with Parzen kernel. Sweden, 1800-1998

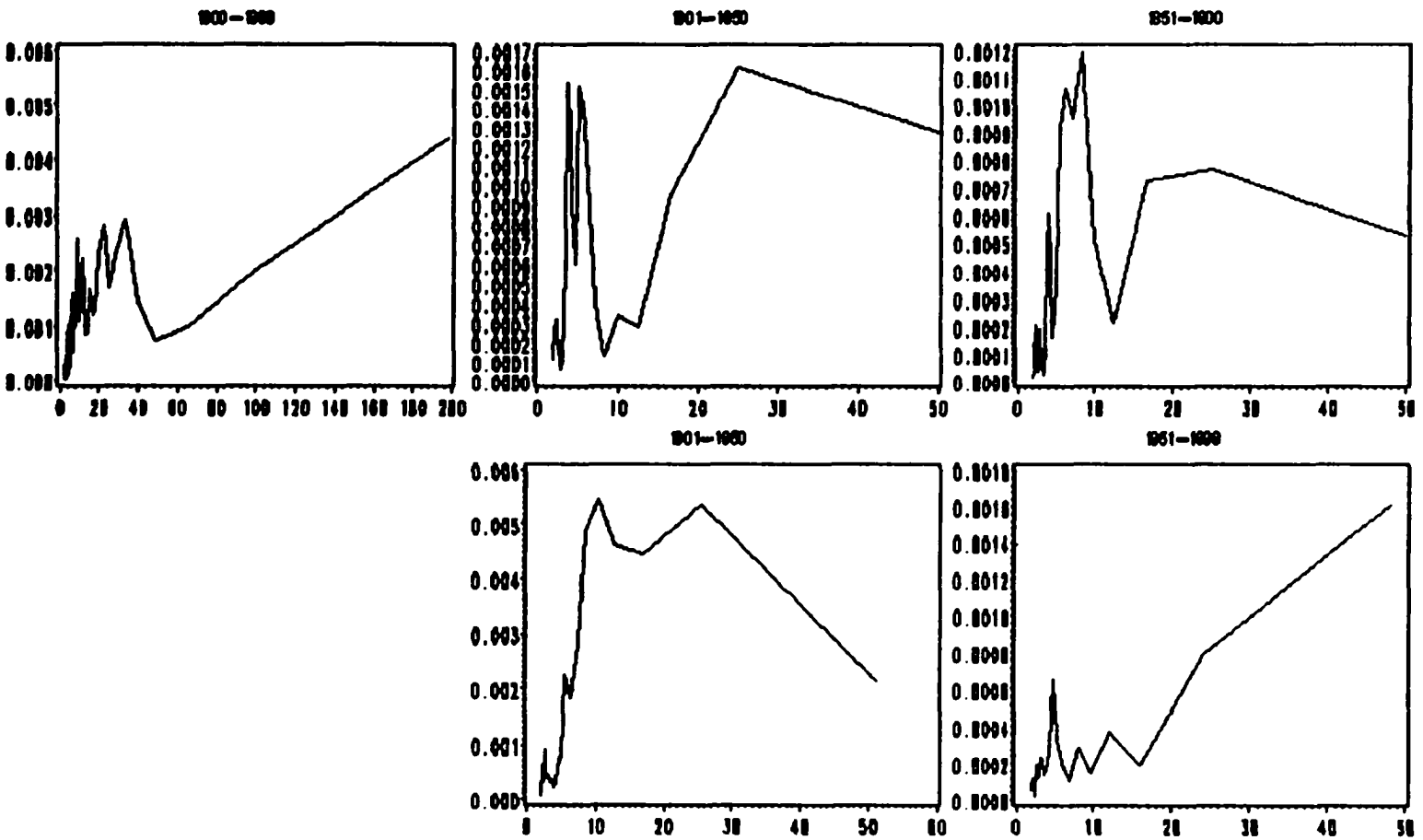


Figure 3.6. Spectral densities of the series of annual rates of change of the price index (GDP deflator). Smothing of the periodogram with Parzen kernel. Sweden, 1800-1998

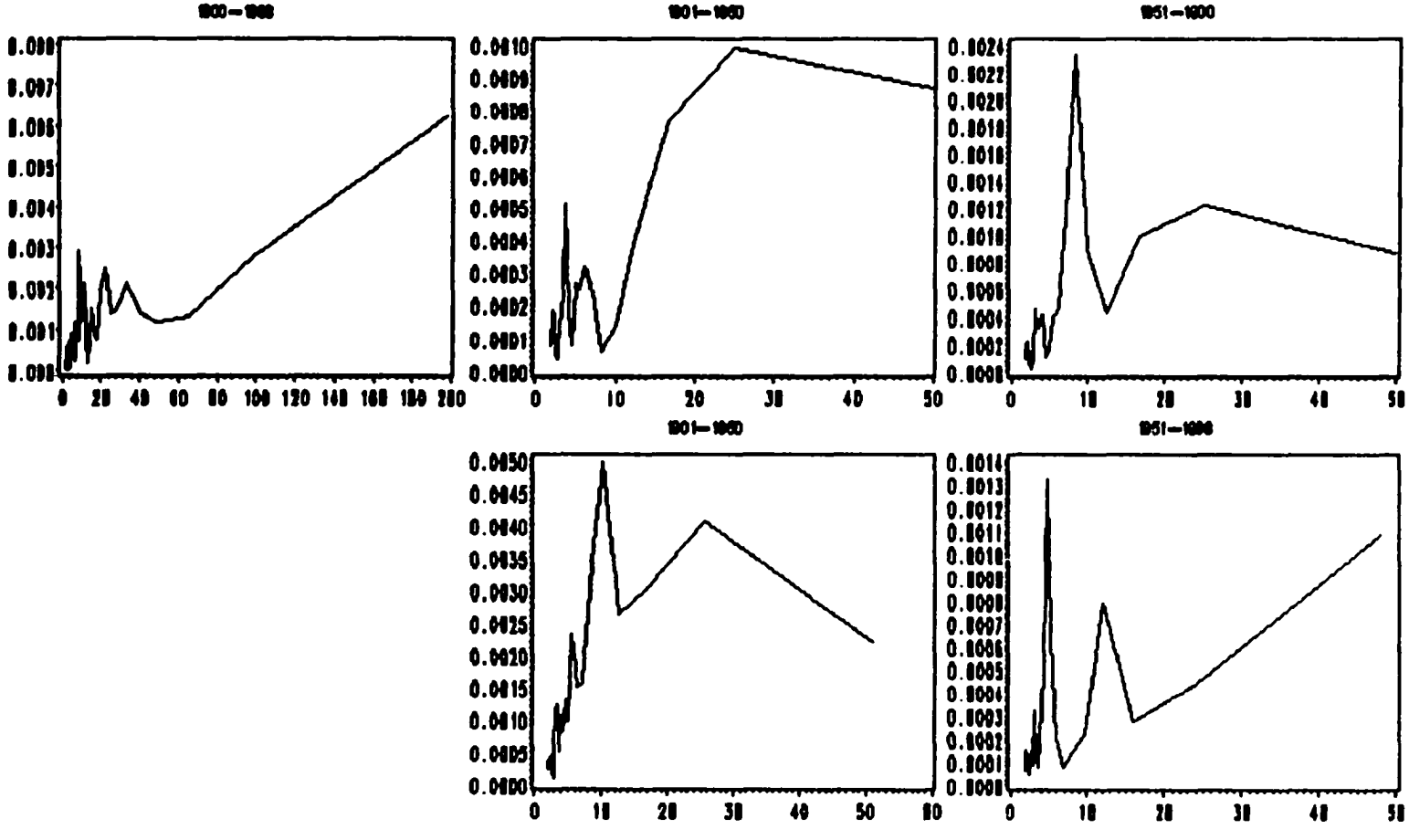


Figure 3.7. Spectral densities of the series of annual rate of change of nominal GDP. Smoothing of the periodogram with Parzen kernel. Sweden, 1800-1998

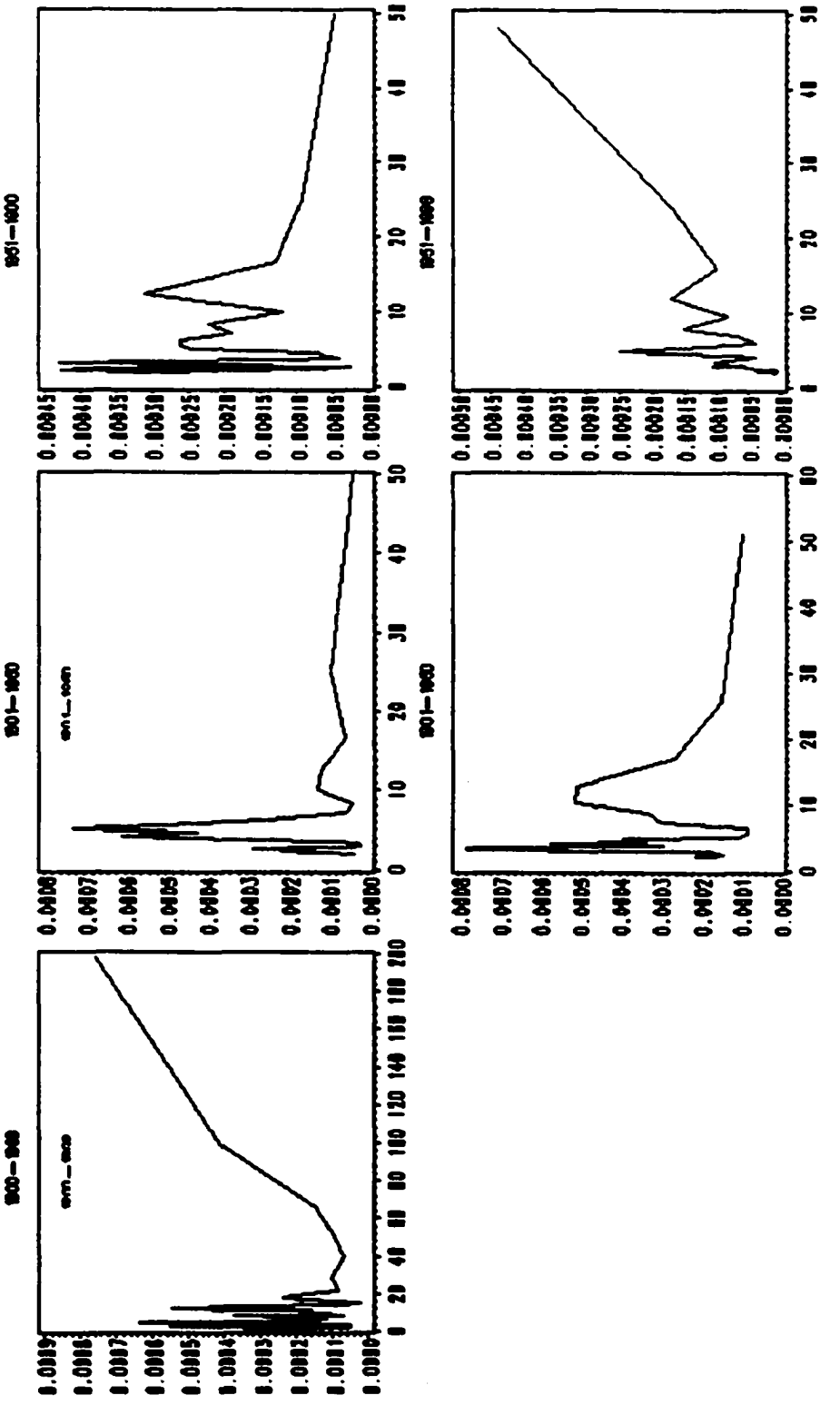


Figure 3.8. Spectral densities of the series of annual rate of change of volume GDP. Smoothing of the periodogram with Parzen kernel. Sweden, 1800-1998

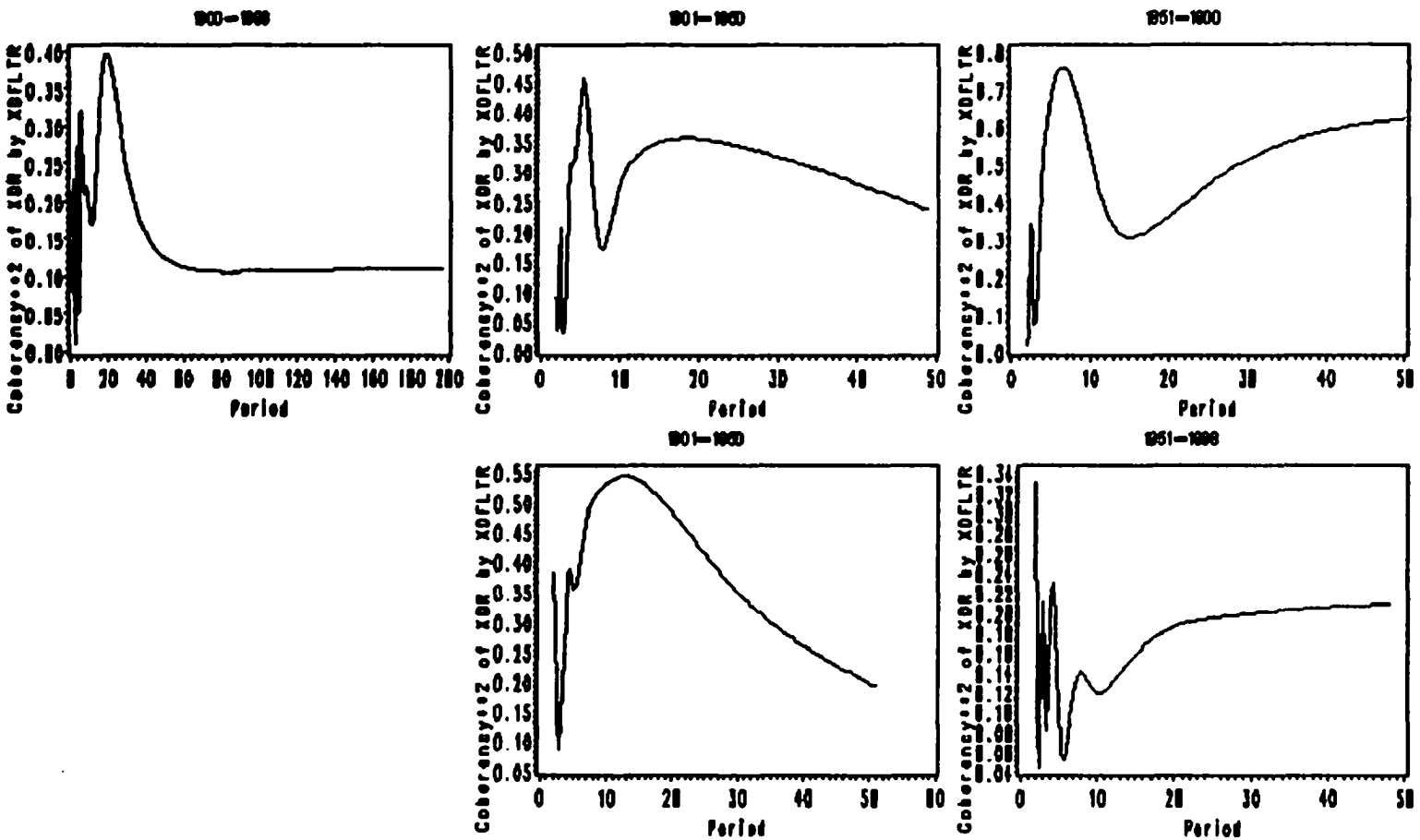


Figure 3.9. Squared cross-spectral coherence of annual rates of change of mortality and GDP deflator. Smoothing with Parzen kernel. Sweden, 1800-1998

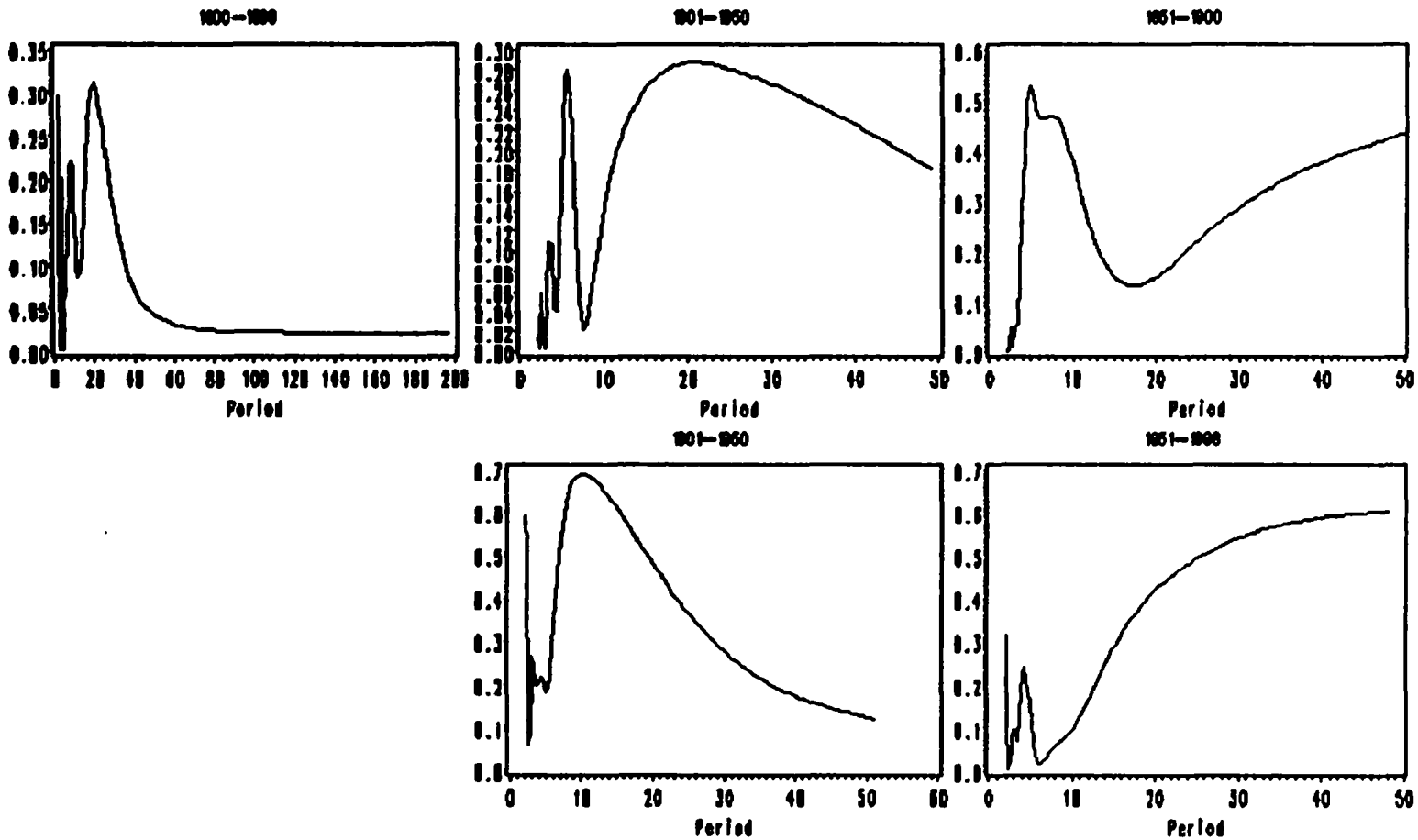


Figure 3.10. Squared spectral coherence of rates of change of mortality and nominal GDP. Smoothing with Parzen kernel. Sweden, 1800-1998

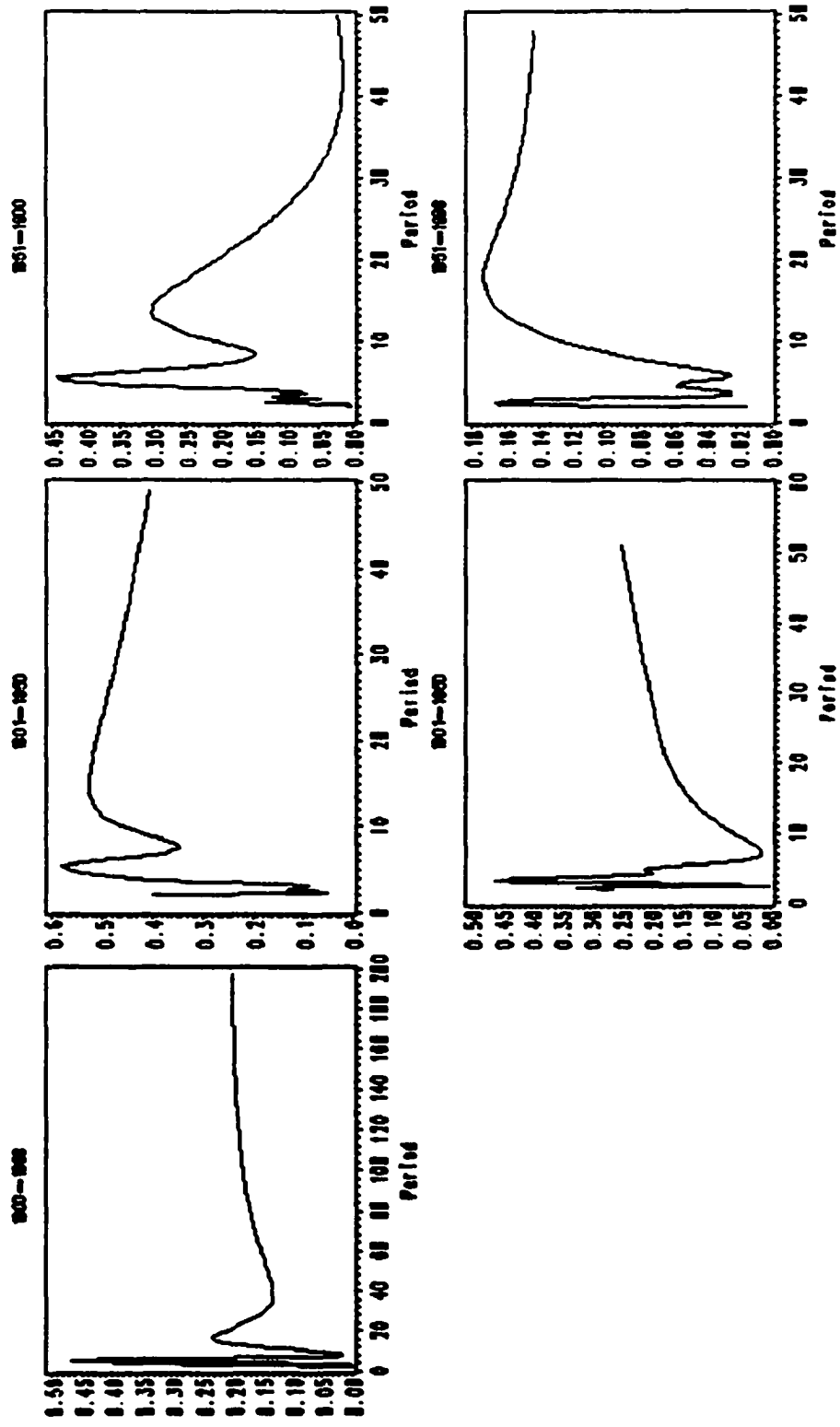
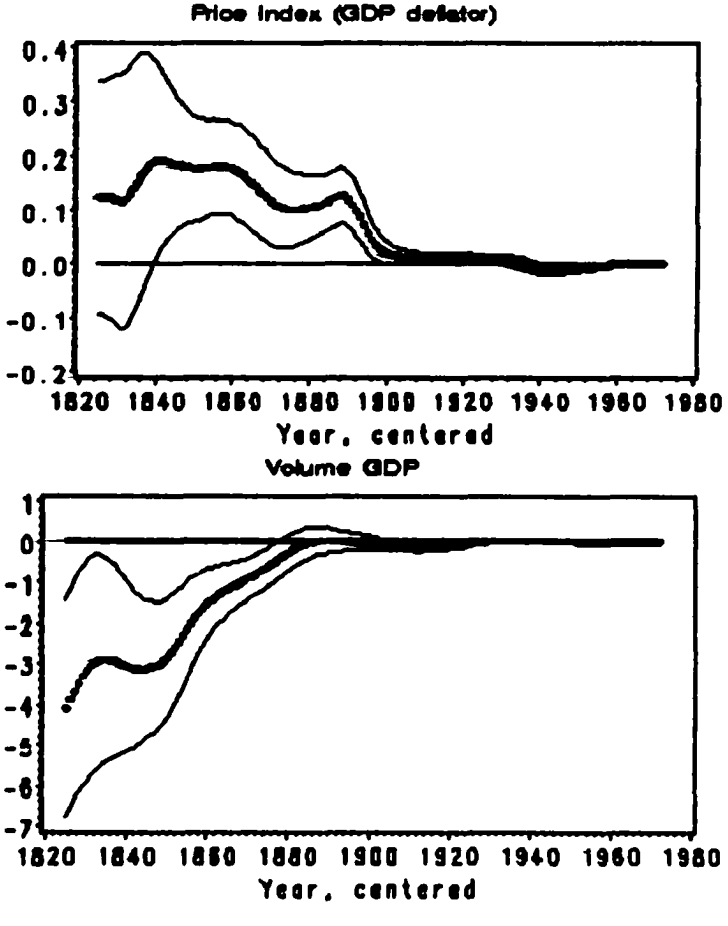


Figure 3.11. Squared spectral coherence of the rates of change of mortality and volume GDP. Smoothing with Parzen kernel. Sweden, 1800-1998



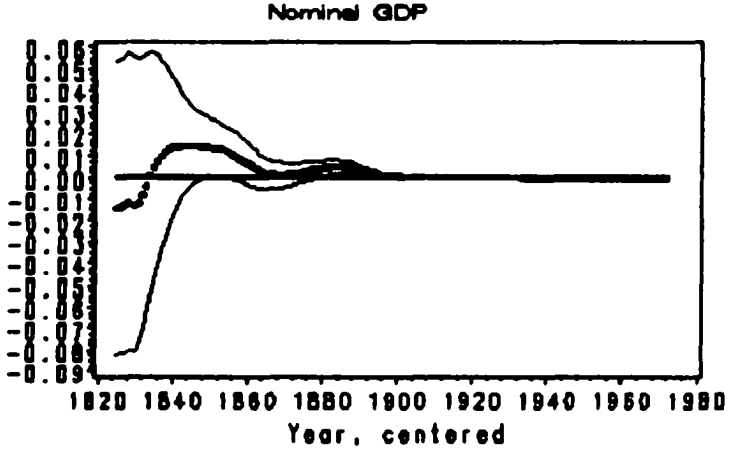
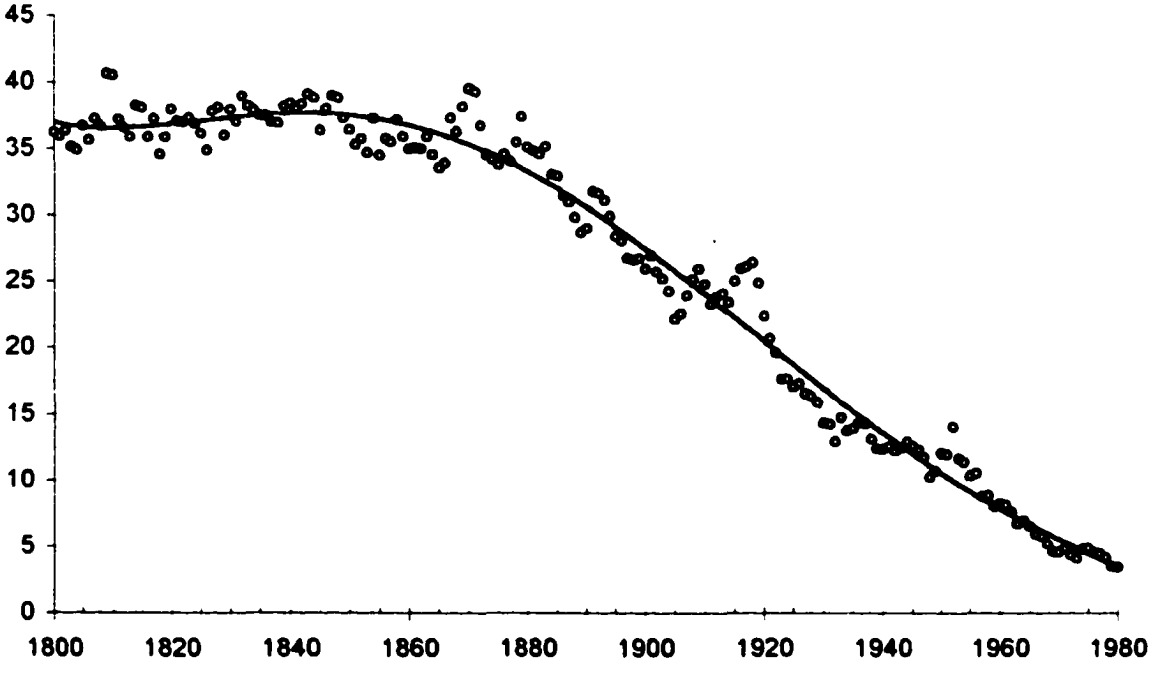


Figure 3.13. Regression coefficient estimates and 95% confidence bands obtained from local regressions of mortality rates on three economic indicators. All data detrended with the Hodrick-Prescott filter, gamma = 100. Local regression window of 51 years, weights Parzen-like. Sweden, 1800-1998.

Figure 3.13. Percent share of agriculture and ancillaries in Swedish GDP. The trend is a 4th degree polinomial



Source: Author's elaboration from data in Krantz, 2002

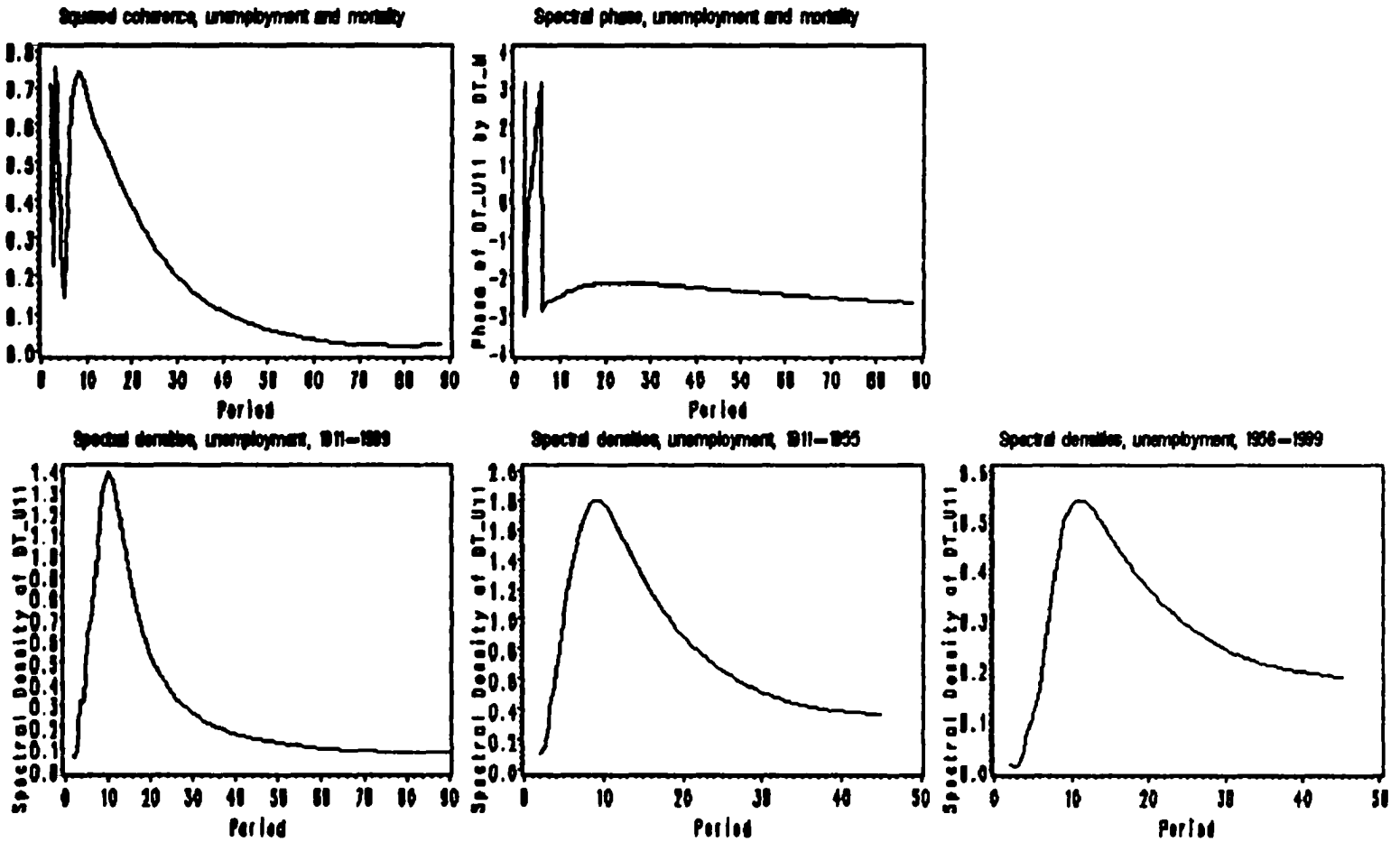


Figure 3.14. Spectral squared coherence and phase of detrended series of unemployment and death rates (upper panel) and spectral densities of the detrended series of unemployment rates. Sweden, 1911-1999

Figure 3.15. Death rate (solid line) and index of average hours in manufacturing, both series detrended (Hodrick-Prescott filter, gamma = 100). Sweden, 1950-1998.

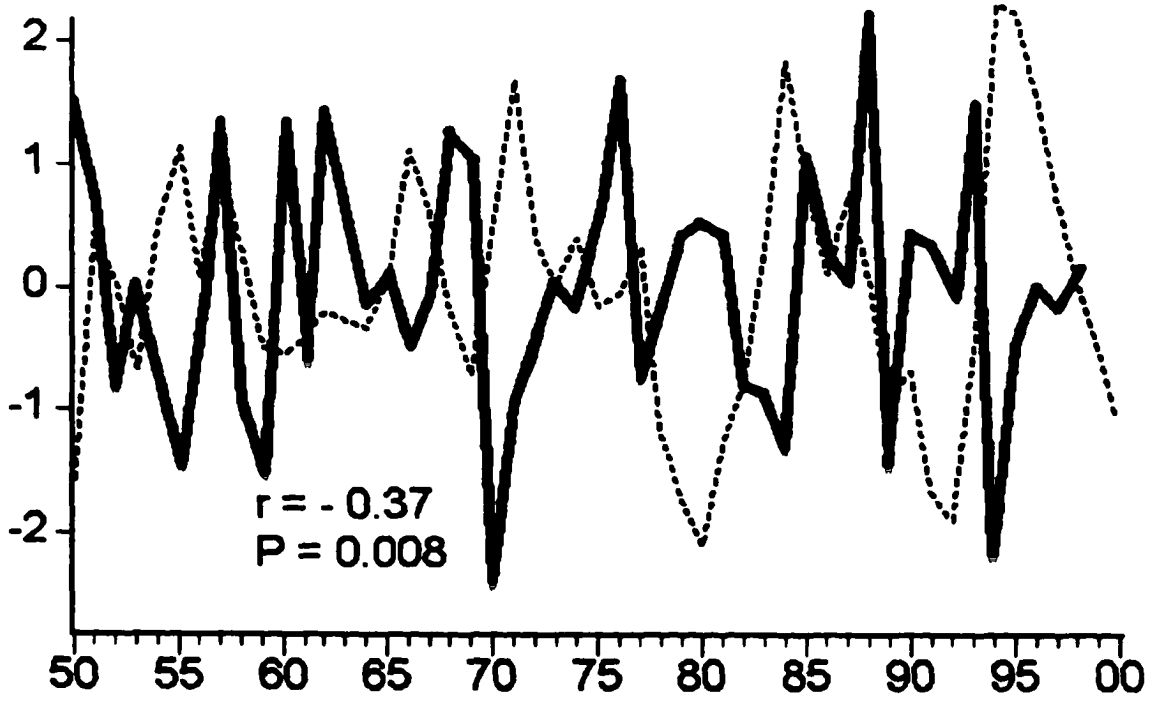
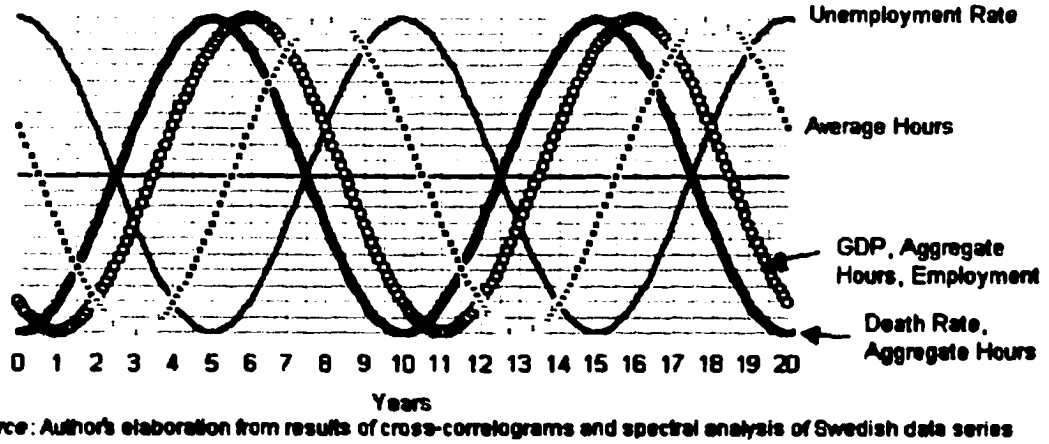


Figure 3.16. Diagramatic representation of some business cycle variables. Sweden, 1950-2000.



Source: Author's elaboration from results of cross-correlograms and spectral analysis of Swedish data series reported by the US Bureau of Labor Statistics (indeces of employment, average hours and aggregate hours in manufacturing), Krantz, 2001 (unemployment rates) and Statistics Sweden (death rates)

Figure 3.17. Cross-correlograms of detrended series of death rates and four economic indicators, all detrended with the Hodrick-Prescoott filter. A negative lag value -*n* represents the economic variable lagged *n* years with respect to mortality.

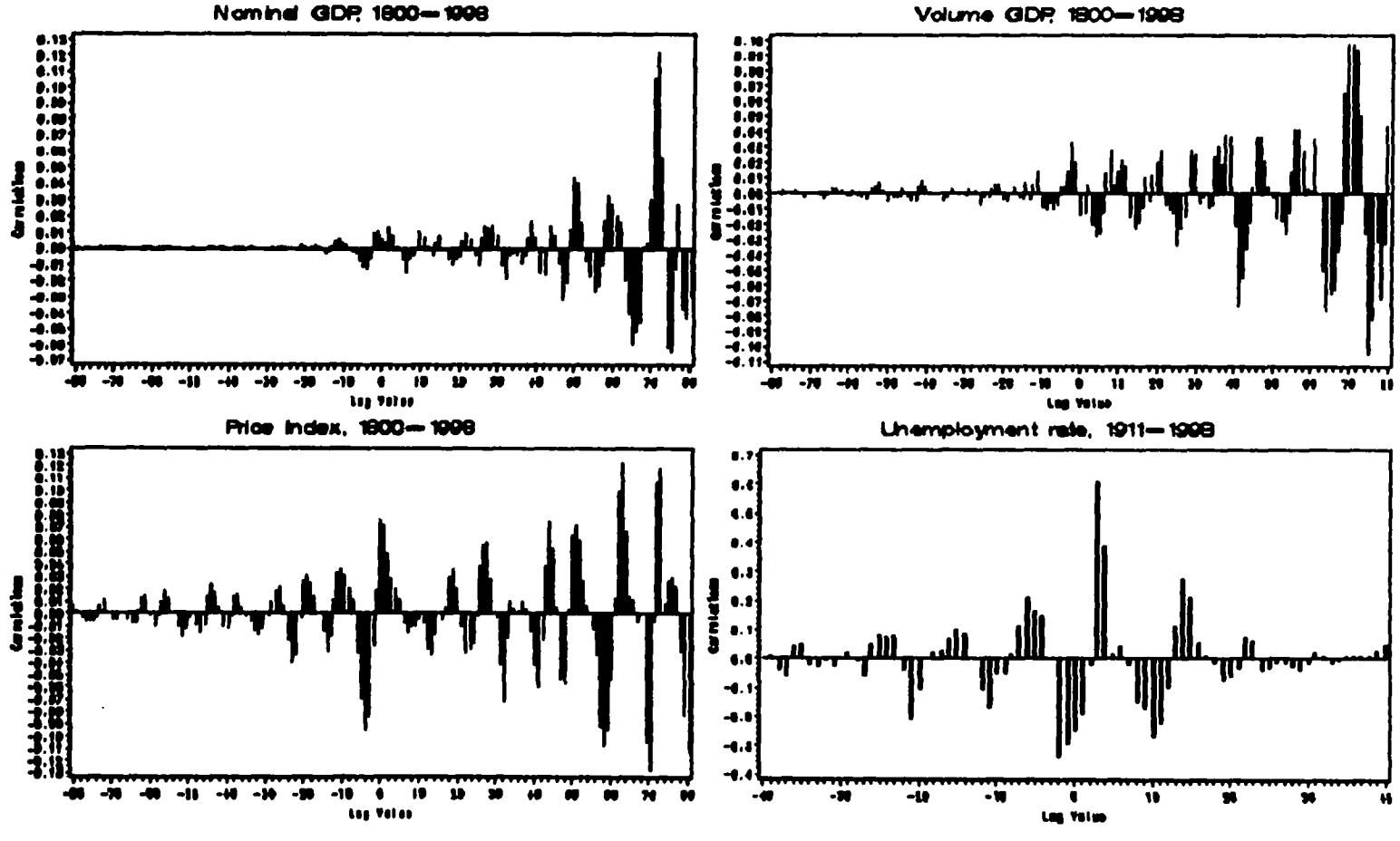


Figure 3.18. Cross-correlograms of the rate of changes of mortality with the rate of change of four economic indicators in Sweden, in the years and variables indicated in each panel

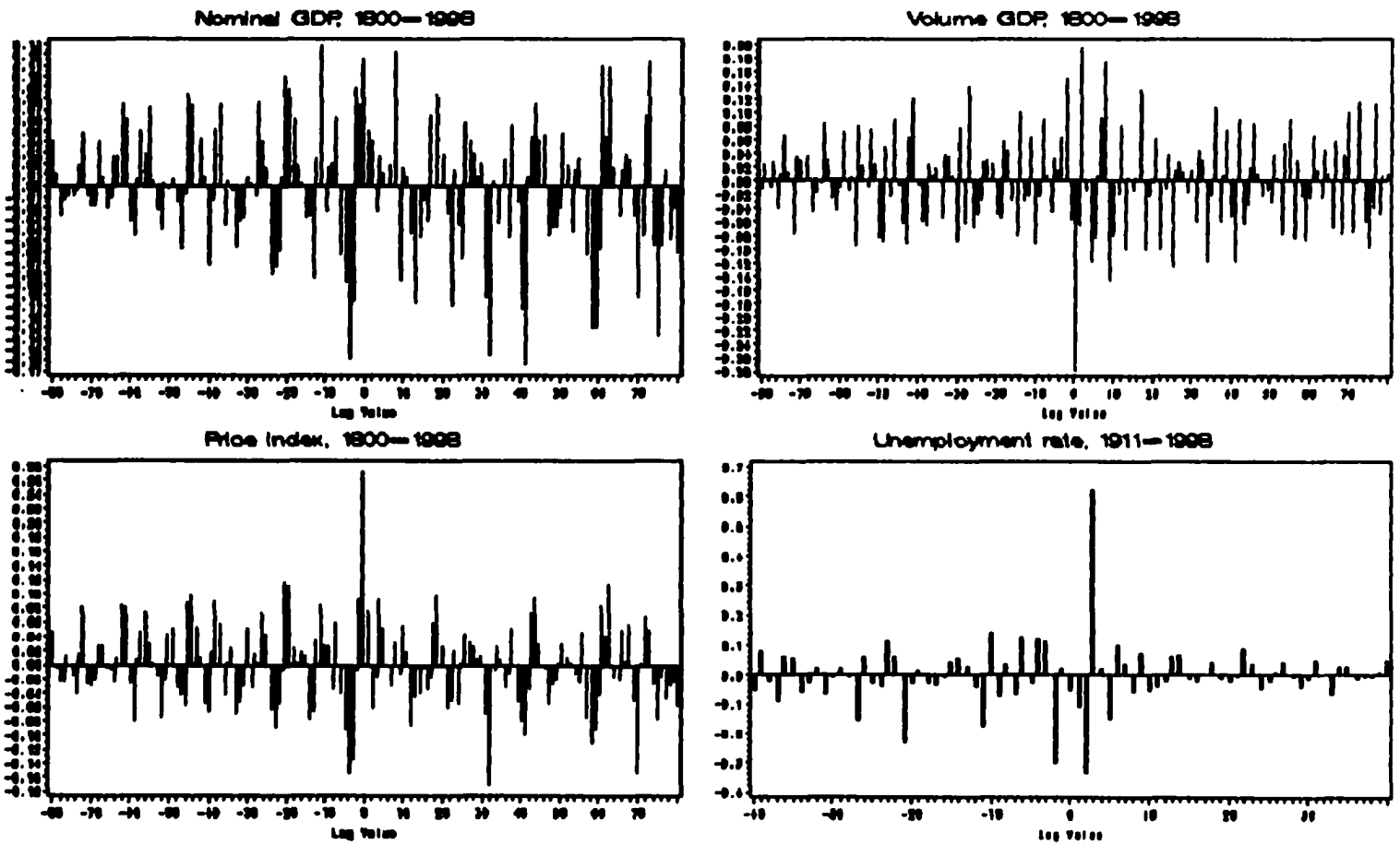
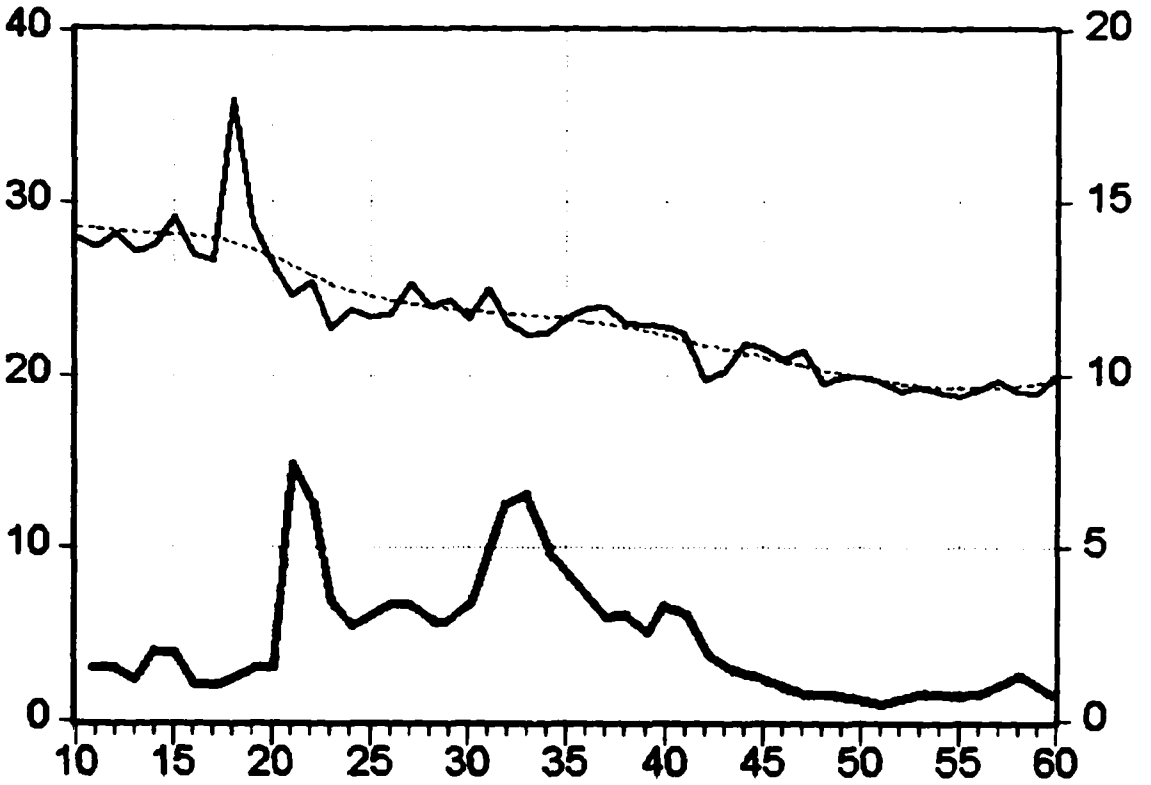


Figure 3.19. Death rate (upper black curve, right scale) and unemployment (lower gray curve, left scale) in Sweden, 1910-1960. The thin line on the death rate curve is a Hodrick-Prescott trend (gamma = 100)



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