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Research in Progress 14

**SHORT- AND LONG-TERM DETERMINANTS
OF CARDIOVASCULAR MORTALITY:
AN ECONOMETRIC ASSESSMENT OF THE WORKING
AGE POPULATION IN RUSSIA, 1965-95**

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This paper examines the short- and long-term determinants of the mortality due to cardiovascular diseases¹ (CVD) among the working-age population in Russia. Why Russia? Why CVD mortality? Why the working-age population? Russia is the Eastern European most affected by a rapid a sudden increase in mortality. A change which has hardly been recorded in peace time in the contemporary history. One of the main cause of such increase has been CVD mortality which has particularly been climbing rapidly among the working-age population in Russia ever since the onset of the transition and the dramatic economic and social shocks which accompanied it². Two possible explanations for this tremendous increase in adult mortality can be summarised as follows.

(1) The demographic crisis is merely a 'catch-up effect'³ of a past trend which was interrupted by the anti-alcohol campaign. The long-term factor is identified with a time trend which explains almost all variance. This approach is very unconvincing, because the authors have not provided any plausible econometric evidence that this phenomenon really occurred.

(2) The rise in mortality is due to short-term factors resulting from the problem-ridden initial phase of the transition, plus a long-term weakness in the offset variable, namely, health assets, exacerbated by the recent collapse in the provision of care through the health system. In this case, the long-term factor is not a trend, but a precise variable which could influence and constrain the short-term variation provoked by short-term shocks.

This paper belongs to the second theoretical family. It attempts to specify and assess econometrically a relationship among these components over time in a 1970-94 sample.

After a discussion of the theoretical model, the paper focuses on the evolution of both the dependent and explanatory variables, and then presents an estimate of the model.

1. The model

According to the Mosley-Chen approach (see Mosley-Chen 1985, Cornia,-Jolly and Stewart 1987; see Cornia 1996 for a review of the existing literature), we can distinguish between the underlying determinants and the proximate determinants of health status. The model is almost recursive. The underlying socio-economic variables do cause the proximate determinants which are more directly linked to the upsurge of the disease and to the final event of mortality. The key to this methodology is the role played by the proximate determinants as intermediate variables.

The relationship between proximate determinants and health status is mostly epidemiological, while the causal link between socio-economic determinants and proximate determinants reflects economic, social and institutional behaviours.

¹ This mortality group includes: Ischeamic Heart Diseases, Cerebrovascular Diseases and Circulatory Diseases

² The LE at 45 dropped by 5 years from 1989 to 1994

³ This is explanation is mainly put forward by russian and french demographers at INED (see Blum et al 1996, Andreev 1995)

The identification of this causal process is extremely important in the specification of a model for mortality, because different mechanisms are in operation in the proximate determinants (mostly biological) and in the exogenous variables and the proximate determinants (mostly behavioural). In the case of CVD mortality, the literature tends to identify the proximate determinants as risk-factors, namely, alcohol, smoke, diet, psychosocial stress and environmental pollution (onset variables) and disease control (offset variable).

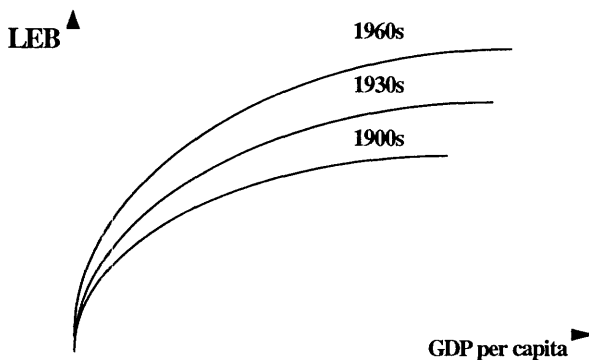
This is the initial methodology. Can one add to this?

1.1 The long-term factor

Two different pathways which affect mortality should be distinguished. The first is related to long-term effects⁴, and the second to short- and medium-term effects. This distinction has not received special attention in causation models of mortality, but it is a crucial one. Indeed, long-term effects constrain variations in mortality over the short run. The variations are provoked by proximate determinants, which in turn are influenced by shocks in underlying socio-economic variables.

In his seminal work, Preston (1975) points out that almost 80 per cent of the increase in life expectancy at birth (LEB) from 1900 to 1969 was due to a shift in the relationship between LEB and GDP (Graph 1). Of course, he does not deny that income growth plays a role in the improvement of LEB. Especially for countries at the lower end of GDP growth, rises in income definitely explain more than 20 per cent of LEB⁵. If per capita GDP constitutes a good predictor of the relative position of countries in LEB in the short run, there is a constant upward shift of the relationship between GDP and LEB⁶ which enables one to make a good forecast of the in the long run.

Graph 1: The cross-country relationship between LEB and GDP per capita in the 1900s, 30s and 60s



Source: author draw from S. Preston (1975)

⁴ Which is different from a time-trend effect. In the latter case the effect is only due to the time evolution while in the first case the long-term effect is due to a particular long-term relationship amongst different variables

⁵ This is due also to the use of a logistic functional form.

⁶ There is a similarity with the shift in the relationship between the level of factors and the production due to technical progress.

Therefore another major component is influencing LEB. Sen and Drèze (1989) partly explain it when they distinguish two types of mechanisms in the production of health (or the reduction of mortality). The first one is a 'growth-mediated' mechanism which combines economic growth (available resources) and policy choices (in determining the new available resources to be devoted to the development of health care). This may explain the performance of South-Korea and Hong Kong in LEB, and it may also explain why Brazil could not achieve the same level of health status despite an almost identical level of economic performance.

The second mechanism is the 'support led process' typical of countries in which, despite low economic growth, public policies have played a most important role because they have assigned priority to expenditures on the provision of health care. This may explain why countries like Costa-Rica and Cuba have generally healthier populations despite low economic performance.

In a cross-sectional analysis, the policy variable may be decisive in explaining differences in LEB. Is the situation perhaps similar if we begin to think in terms of evolution over time?⁷ The answer is 'yes', if we include in the more comprehensive structure of health assets the flow generated by the support-led process and economic growth.

As Musgrave (1987) and Murray and Chen (1993) point out, the accumulation of health assets has a key role in the reduction of mortality. Actually, the definitions of these assets vary among the proponents. Here, we define the stock of health assets as the stock of physical capital (hospitals, medical equipment, drugs, infrastructures for improving access to safe water and sewage), human capital (doctors and nurses), research and development, the embedded technological progress, and organisation (the operation, efficiency and effectiveness of the public health care system). Variations in this stock are mainly due to two components. First are the investments in health care made by public authorities so as to maintain and improve physical and human capital, and second is technological change⁸. The first component is related to GDP, but also, probably in a more decisive way, to public policies in resource allocation. The second is mainly exogenous in most countries.

The determinants of the stock of health assets can be represented by using the following formula:

$$\text{HASS}(t) = f(\text{HASS}(t-1), \text{HI}(t), \text{TECH})$$

where HASS = the stock of health assets at time (t) and (t-1), HI = expenditure on health and education, and TECH = the change in health technology.

The accumulation of health assets is a very long process. which may bound the evolution of mortality if there are short-term shocks in proximate determinants. First, some health assets are preventive (the role of vaccinations in preventing some infectious and parasitic diseases or the role of new technologies in early diagnoses of CVD) and therefore foster a decline in morbidity. Second, it is quite well known that a major factor in the epidemiological transition

⁷ Indeed, Preston has suggested that a variable expressing income inequality might be included in the LEB function.

⁸ S.Preston mentioned the importance of the Technological change imported through drugs in explaining some improvements in health in some developing countries

has been the shift from an extensive system of health care towards an intensive one that is more appropriate for dealing with the pathologies related to the ageing of the population, having as main consequence a reduction in case fatality rates.

The problem is the way to measure this variable and include it in a short-run specification. There are two alternatives.

(1) The use of a proxy variable.

(2) The use of a functional form and an estimator which constrain the short-term specification by a function with a time-varying asymptote variable reflecting the evolution of the long-term factor.

The first alternative can be easily included in a ECM representation. The CVD model should therefore conform to the following specification:

$$\Delta \ln CVD_{(t)} = \alpha + \sum \beta_i \Delta \ln PD_{i(t)} + (1-\chi)(\ln SDR_{(t-1)} - \ln HASS_{proxy(t-1)})$$

The last term is the error correction term which represents the long-run relationship between overall mortality (SDR, standardised death rate) and the health assets proxied by a variable (HASSproxy). Since the coefficient of the last term is positive, as the last term becomes larger or smaller, ΔCVD also increases or decreases, thereby 'correcting' the long-term positive or negative gap. In the case of a constant long-run relationship, the same short-term shocks among the proximate determinants (PD) would have the same effect over time. If the relationship becomes smaller (as is occurring in Western countries), the same short-term shocks would provoke, *ceteris paribus*, a smaller increase in CVD.

Although this alternative is definitely the most appropriate one, nonetheless there is a practical problem which unable us to utilise it.

The main problem in this formulation is the definition of the proxy variable to represent the accumulation of health assets. Many indicators are available on the health system in Russia. Unfortunately, however, they are too scattered over time (for a review, see Davis 1997), so that it is impossible to develop a synthetic variable as a proxy.

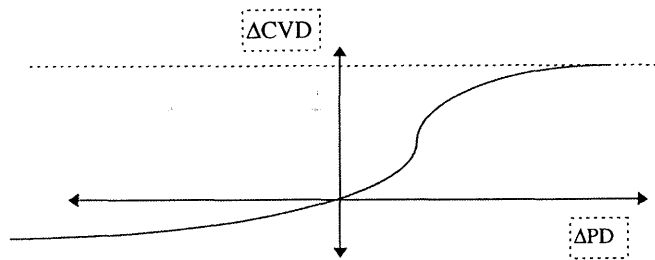
For the second alternative, by using a proper functional form and a related estimate procedure, we indirectly assume that:

(1) There is a constraint on the movement of the short-term relationship.

(2) This constraint varies over time and is supposed to be a random walk with negative drift.

The short-term representation can be synthesised as in Graph 2 and through a log-reciprocal functional form.

Graph 2: The short-term relationship between change in cardiovascular mortality and changes in proximate determinants



Where the asymptote is time-varying. The use of a time-varying coefficient in this case (in which omitted variables influence the constant) is suggested by Judge et al. (1985). This leads to the following model specification.

$$\Delta \ln SDR_{(t)} = \exp(\alpha_{(t)} - \sum \beta_i (1/\Delta PD_{i(t)}))$$

As we show in section 4, a Kalman filter estimator has been used to estimate the time-varying coefficient, and this has produced a result which is very close to the expected one. This alternative is at moment the most feasible one but there are two main points which should be explicated.

First, The imposition of an hard constraint like an asymptote could easily provoke a decrease in the explanatory capacity of the model.

Second, the time-varying asymptote could reflect not only the long term effect of the accumulation of health assets but also others omitted variables.

1.2 Internal demographic dynamics: the cohort effect

In explaining mortality, many authors (especially demographers) point out the importance of internal demographic dynamics which can play a significant role in the determination of the risk of a cohort to exposure to morbidity and mortality.

The main indicator of internal demographic dynamics proposed in the literature is the cohort effect (Grossman 1972, Murray and Chen 1992, Anand and Chen 1996). This refers to the health stock of an individual. Each individual is born with a health stock which is significantly influenced at birth by the nutritional and health status of the mother (maternal health) and by the nutritional conditions of the first living years, both of them are affected by external factors, such as exposure to disease, nutrient deficiency and environmental pollution, that in their turn are a function of socio-economic variables. Over time, this health stock can be reduced or improved, but the initial stock undoubtedly constitutes a significant starting point.

If a cohort has been born during famine or a period of great tension, such as during war or natural disaster, the theory states that it has received a poor health stock which can subsequently be improved only with extreme difficulty⁹. This cohort is therefore intrinsically weaker than other parts of the population. In the case of Russia, this argument has been

⁹ One of the most significant case of cohort effect is provided by the Dutch famine (1944) (see Lumey et al. 1991)

employed by many demographers (see Blum, Zacharov and Avdeev 1997) to explain the gap in mortality relative to Western countries. These demographers say that the cohorts born during the 1933 famine and during and just after World War II were provided with a poor health stock because of the strains provoked by the surrounding events. As a result of this particular initial condition, these cohorts may have been exposed to a higher risk of mortality. Therefore, they have exhibited death rates which are higher relative to other cohorts, and they are more vulnerable. et al. (1996) have calculated that the survival function of these cohorts indicates there is a structural weakness relative to other cohorts that increases with time.

In terms of our model, the presence of such cohorts in the sample age-group (15-60) should be viewed as an element tending to boost the constant (asymptote). This increase is exogenous and is caused by the structural weakness of these cohorts. On the other hand, if such cohorts are not present in the sample age-group, there should be no similar exogenous rise. This is the reason experts recommend the use of a dummy variable of 1 for years in which such cohorts are present and of a dummy variable of 0 for years in which they are absent. Unfortunately, the sample period (1970-94) does not permit this distinction among years to be made, since the working-age population during this period always contained such cohorts in the 15-60 age-group. The available demographic data would allow the study of mortality among a particular age-group, say, 35-39 (in which the World War II cohort was present only between 1975 and 1980), but the explanatory variables refer only to the more general group represented by the working-age population.¹⁰

2. The dataset

The demographic data, which seem to be the most reliable, are drawn by the book written by Mesle, Shkolnikov, Hertrich and Vallin (1996) often quoted in the text.

Data on risk-factors namely: alcohol, smoking and fat are collected from different sources with significant problems of comparability and quality. For alcohol consumption the main problem is constituted by the unrecorded sales which became very important after 1992. It is universally recognised that the official data are heavily biased downward (Shkolnikov V. and Nemtsov A. 1996, Ryan 1995) . Unfortunately, these data do not take into account the significant share of the supply of alcohol represented by smuggling, the business of unregulated small private firms, illicit production, and home distillation by consumers¹¹. The difference between the two series was almost stable up to 1984, the relative distance was almost constant (4.7-5 litres). At the onset of the anti-alcohol campaign, the gap between the two estimates began to grow. As shown by Ryan (1995), the unrecorded sale of alcohol started to increase steadily before shooting up after 1992 (almost 2/3 of the whole quantity of alcohol consumed). Amongst the unofficial estimates of alcohol consumption, those ones made by Nemtsov (based on specific alcohol mortality) are widely recognised how the best proxy of the real alcohol consumption in Russia.

The situation is different for alcohol prices which are definitely more reliable apart from the proportion of consumption derived from smuggling.

¹⁰ We would assume, for instance, that the age-specific 35-39 unemployment rate is equal to the average.

¹¹ The unrecorded sales of alcohol account for almost 2/3 of the total consumption of alcohol

Smuggling is also the main problem in assessing the number of cigarettes smoked, and no “unofficial” estimates are available. Even in this case this consumption could seriously be affected by that phenomenon as well as its the relative price.

Regarding Fat and Kcalories intake they come from the FAOSTAT database, which assess the amount of food and nutrient intake available on the basis of both production and trade of food, adjusted by wastage, cleaning and cooking losses. It is quite well known that this tend to overestimate the quantity of food intake, even if, lacking other sources, it could catch the overall pattern of consumption.

3. Cardiovascular mortality in the context of the evolution of overall mortality in Russia, 1965-94

Table 1 introduces a description of the evolution of mortality in Russia.

Table 1: Contributions of the leading classes of causes of death to changes in life expectancy at birth, 1970-95

	Infectious	Neoplasm	CVD	Respiratory	Digestive	Other diseases	External	Total
Males								
1970-80	0.05	0.04	-1.05	0.09	-0.07	0.07	-0.79	-1.66
1980-82	0.07	-0.04	0.21	0.32	0.04	-0.01	0.30	0.89
1982-84	0.01	-0.04	-0.33	-0.03	-0.02	-0.11	-0.05	-0.57
1984-87	0.15	-0.05	0.68	0.48	-0.14	0.00	1.76	3.16
1987-91	0.05	-0.06	-0.11	0.13	-0.01	-0.10	-1.32	-1.42
1992	-0.05	0.07	-0.23	-0.08	-0.10	-0.21	-0.84	-1.44
1993	-0.12	-0.04	-1.09	-0.28	-0.10	-0.16	-1.29	-3.06
1994	-0.05	0.03	-0.68	-0.09	-0.09	-0.15	-0.46	-1.49
1995	-0.03	0.05	0.41	0.07	-0.02	0.04	0.19	0.71
Females								
1970-80	-0.01	0.17	-0.64	0.28	0.01	0.11	-0.30	-0.38
1980-82	0.03	-0.01	0.33	0.26	0.02	0.00	0.10	0.73
1982-84	0.02	0.00	-0.54	0.00	-0.01	-0.12	-0.06	-0.71
1984-87	0.09	-0.03	0.50	0.30	0.04	-0.04	0.46	1.32
1987-91	0.06	-0.03	0.38	0.14	0.00	-0.27	-0.31	-0.03
1992	0.01	-0.02	-0.15	0.02	0.03	-0.11	-0.25	-0.53
1993	-0.05	-0.00	-0.94	-0.13	-0.06	-0.15	-0.49	-1.81
1994	-0.02	0.01	-0.53	0.02	-0.07	-0.07	-0.17	-0.83
1995	-0.01	0.02	0.39	0.02	-0.01	0.04	0.08	0.53

Source: Shkolnikov(1997).

LEB climbed quite quickly in Russia from the end of World War II to 1965, more quickly than it did in other countries over the same period. It rose by around eight to nine years within two decades, thereby closing the health gap with Western Europe. This improvement was mainly due to the rapid pace of the first epidemiological transition (characterised by a sharp decrease in the mortality due to infectious and parasitic diseases) and to a significant drop in the incidence of respiratory diseases. The change in LEB was also accompanied by a more rapid decline in the infant mortality rate; a substantial drop was recorded especially in post-neonatal mortality. Nonetheless, despite the good initial trend, the health situation began to deteriorate during the 1970s, when LEB started to flatten and even slightly decrease. During this period, the gap widened between Russia and Western countries, in which LEB and health

status in general continued to improve steadily.¹² During the early 1980s, a slight increase was recorded, and in 1985 the anti-alcohol campaign led to the first massive change in LEB, which rose by almost two years. The effect of the anti-alcohol campaign was evident for only two years of actual implementation, and from 1988 to 1991 LEB fell at the same pace it had fallen in the early 1980s. In 1992, the onset of the transition coincided with a big drop in LEB, which reached the lowest point – 59 for men and 69 for women – in 1994.

It is quite clear that the evolution of female LEB has been smoother than that of male LEB. Male LEB shows a higher variability, and this is also reflected in the LEB differentials. The differential was 9.22 in 1965. It climbed steadily to 11.63 during the 1970s, when there was an epidemic of CVD that struck more men than women. The anti-alcohol campaign provoked a return to the 1965 level in the differential. This trend was mainly driven by a drop in mortality due to violent causes (VIO, especially alcohol-related violence), which are an important factor in male deaths. However, during the most recent crisis, the differential shot back up, reaching 13.47 in 1995.

The available demographic dataset starts at the end of the epidemiological transition (1965). From it, one can see that CVD and, to a lesser extent, VIO bear substantial responsibility for the health crisis in the 1970s. While CVD mortality has been declining in Western Europe since 1970, in Russia (as in all Central and Eastern European countries) it has been growing steadily.

Among men, CVD and VIO have been the leading factors in determining the direction of LEB, while the rise in mortality due to neoplasm has been small but constant.

The role played by CVD and VIO in male deaths was decisive in the two post-1970 shocks: the mortality decline during the anti-alcohol campaign (1984-87) and the recent transition-related upsurge in mortality (1989-94). However, there is a difference between these two shocks in terms of the relative importance of CVD with respect to VIO. During the first shock, the major contribution to the decrease was furnished by VIO (60 per cent versus 21 per cent for CVD), while during the transition crisis CVD played the more determinant role (32 per cent versus 44 per cent for VIO).

Even among the male deaths due to VIO, there is a substantial difference between the two shocks. In 1984-87 the main drop in VIO was related to the decline in accidental alcohol poisonings. Meanwhile, during the transition crisis, there was a sharp increase in deaths due to homicide and undetermined intentional injuries, putting these, along with suicide, among the most important causes of violent death.

For CVD-related male mortality, during the first shock the drop in cerebrovascular diseases had the biggest impact, while during the 1989-94 shock heart diseases recorded the greatest rise.

¹² The ageing of the population as a result of the contraction in the mortality due to infectious and parasitic diseases provoked a structural rise in the exposure of people to diseases, like CVD, that are typical of the middle-aged and the elderly. This problem – related to morbidity – was evident in an increase in the mortality due to CVD. The Western countries tackled the problem by undertaking the second step in epidemiological transition (the reduction of adult mortality). This was not the case in Russia.

Neoplasm represent the third main cause of death among men. Mortality due to neoplasm does not show the same degree of variability as do the first two causes. This specific mortality rate has been mounting at a slow but steady pace. Remarkably, this was true even during the most recent mortality crisis. The contribution of mortality due to neoplasm to changes in LEB is important, though not dominant.

Among women, as among men, the deterioration in the 1970s was led by CVD, which accounted for around 50-60 per cent of the mortality during the entire 1970-94 sample period. Only during the 1984-87 shock did VIO match the contribution of CVD (37 per cent each). As a cause of death, neoplasm have been more important among females than they have among males, especially during the 1970s.

The age-group which showed the greatest variability and the greatest vulnerability to epidemic heart diseases during the 1970s and the subsequent shocks was middle-aged adults, both male and female, although, on average, there was a significantly higher gradient among males.

Figure 1a: Percentage change in age-specific standardised death rates: females

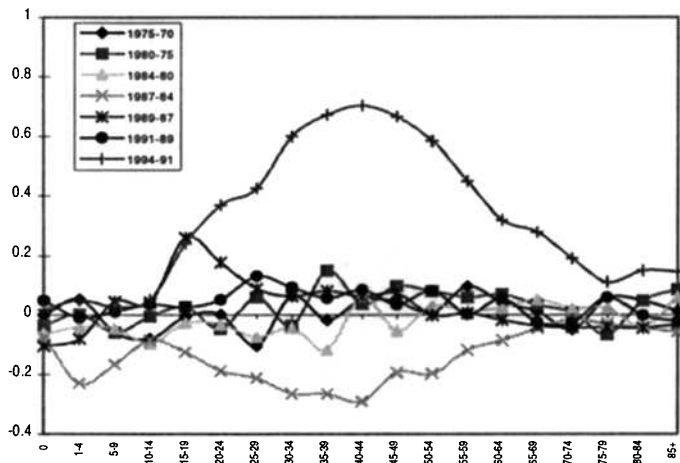
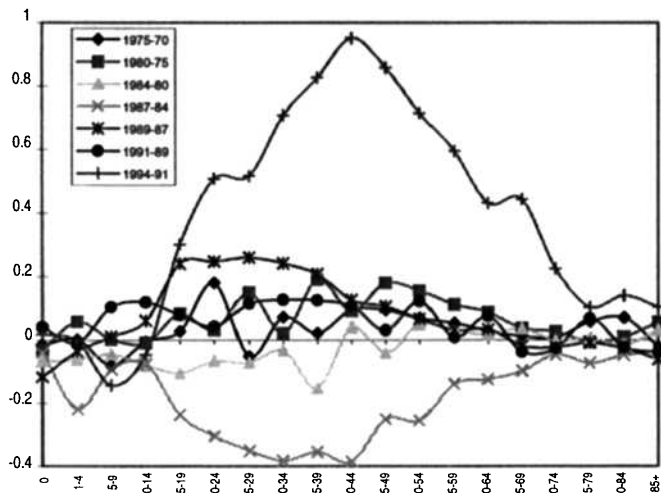


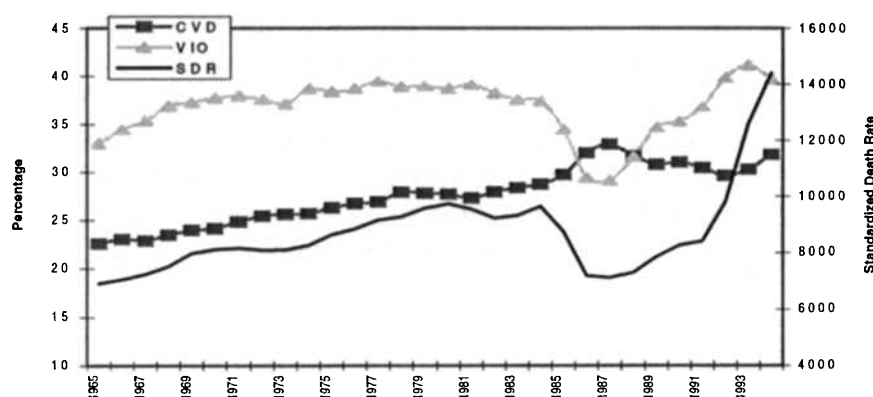
Figure 1b: Percentage change in age-specific standardised death rates: males



Source: author’s calculation from Mesle, Shkolnikov, Hertrich and Vallin (1996).

Figures 1a and 1b reveal that all the significant changes in mortality have been led by age-groups among the working-age population, especially middle aged adults (35-60) and more particularly the 35-39 and 40-44 age-groups. Of course, the gender differentials confirm that female mortality exhibits smoother variability even if the age-pattern is almost identical. If we compare age-specific and cause-specific mortality, we realise that the main causes of death among these groups are, as expected, CVD, VIO and, much less important, neoplasm and alcohol-related mortality. Also as expected, the first two causes are predominant among both males and females. Figure 2 clearly shows the relative weights of CDV and VIO in the total SDR among the male working age population. The incidence of these two groups of causes of death is almost 70 per cent over the entire sample period.

Figure 2: Percentage contribution of CVD and VIO mortality to total SDR



Source: author's calculation from Mesle, Shkolnikov, Hertrich and Vallin (1996).

Figure 2 shows the growing importance of CVD, starting in 1970, in determining mortality among the working-age population (epidemic heart disease), the drop in mortality – driven mainly by fewer deaths due to VIO, including alcohol related violent mortality – during the anti-alcohol campaign, and the dramatic rise in deaths at the outset of the transition that was led by these two sets of causes, CVD and VIO.

4. The proximate determinants

We can distinguish among four main proximate determinants which have short- and medium-term effects on CVD mortality.

4.1 Psychosocial stress

Psychosocial stress is the state of physiological and psychological arousal that is provoked when individuals are called upon to react to new and unexpected situations for which they do not know appropriate coping behaviours. The direct biological mechanism through which psychosocial stress affects an individual has been quite well analysed. It involves four pathways (Bobak and Marmot 1997):

- (1) cardiovascular reactivity (heartbeat, blood pressure, vasoconstriction, and oxygen demand) that produces ECG ischemia;
- (2) blood lipids which produce increased cortisol secretion;
- (3) haemostatic factors;
- (4) a direct effect on arterial endothelium, probably via immunological mechanisms.

There are also indirect negative effects of psychosocial stress. These are mediated through the intake of a 'perceived' stress reliever, mainly alcohol, or through high exposure to other risk-factors like smoking and a fatty diet.

Thus, psychosocial stress is quite narrowly linked to deaths due to heart diseases, ulcers, cirrhosis, alcohol psychosis, and suicide, that is, those specific causes of death that characterised the failure of the second step in the epidemiological transition during the 1970s and that explain most of the jump in Russian transition mortality.

What events trigger psychosocial stress? The literature is vast (Bremner 1991, Whelan et al. 1992, Beale and Nethercott 1985, 1989). It seems to suggest that psychosocial stress is particularly associated with family breakdown, job insecurity and unemployment, migration, and difficult emotional states such as anger, depression and hopelessness.¹³ One of the key factors in explaining the level of psychosocial stress is the degree to which an individual can discount the stress-causing event. The presence or absence of this coping capacity may explain why the impact of unemployment on individuals is definitely much higher in Russia than it is in Western Europe and why it is particularly dangerous among middle-aged male adults. Interestingly, women seem to be less susceptible than men to stress-induced death, possibly suggesting that women are exposed to risks of lower intensity, that social conventions shelter women from stress more than they do males, or that specific physiological factors give women greater protection.

All the stressors – unemployment, uncertainty, migration, divorce, separation, hopelessness, and so on – have intensified in Russia during the past six years. The fact that the negative changes in the labour market have been both unexpected and significant has affected especially middle-aged males. Substantial internal migrations under very unfavourable conditions and entailing the redefinition of survival strategies and greater stress have been reported. Migration to urban areas has increased in a particularly dramatic fashion. The Russian military presence abroad has shrunk, and large numbers of soldiers have been returning home. There have also been large flows of refugees from ethnic strife. Meanwhile, family conflict, domestic violence, loss of self-esteem and alcoholism have become much more common. Divorce rates have been climbing rapidly. These effects have been reinforced by the extreme weakness of 'civil society', the network of social intermediaries (including the extended family) which can counterbalance the effect of economic and social hardship.

Psychosocial stress is not a new phenomenon linked only to the transition. Some authors (among others, Bobak and Marmot 1996, 1997) have pointed out that the epidemic in heart diseases in 1965-85 was likely due to endemic psychosocial stress (due to frustration, lack of control and rewards at the workplace and limited social interaction, that is, to the overall failure of the socialist system). With the end of the socialist system, this endemic psychosocial stress ought to have diminished, and its mortality impact ought to have been reduced. Nonetheless, the transition has witnessed a surge in psychosocial stress (reflecting the

¹³ For example, the death rates due to heart disease, suicide and other causes are two to four times higher among divorced men, widowers and migrants than they are among married men or men who remain in a stable community. The prevalence of suicide, stroke and ulcers has been found to be higher among the unemployed, as shown, for instance, by data on the US, Canada and Britain (Eyer and Sterling 1977). Likewise, a recent study has determined that myocardial infarction was more prevalent by a factor of 1.5-1.6 among adults suffering from depression, anger or inability to cope than it was in a control group (Anda et al. 1993).

unexpected, sharp and sudden adjustment crisis), the causes, intensity, duration, and mortality impact of which are quite different from those of endemic psychosocial stress.

Psychosocial stress is also likely to be responsible for violent or irrational reactions, non-compliance with regulations and illegal behaviours. Yet, increases in ‘violent’ mortality in Russia have definitely also been fostered by the collapse in law and order. The regulatory, supervisory and control apparatus of the state have become very weak and unable to function in a satisfactory fashion.

A quantitative variable, ‘psychosocial stress’, could be represented by several variables reflecting, for instance, family situation, dislocation and unexpected economic difficulties, each of which would carry a portion of the variance of the unknown variable, but all of which would be highly correlated together. The variable ‘psychosocial stress’ would thus reflect the joint impact of these other variables.

This is typical of a situation for which the econometric literature suggests the use of the principal component technique (see, among others, Theil 1971, Kennedy 1993). If the initial hypothesis (that the variables can catch different aspects of the one phenomenon) is confirmed, the variance extracted by the first component should be very high (the rule of thumb suggested by the literature is more than 70 per cent), and the component should be highly correlated with each variable. The variables available for the representation of the latent indicator reflecting the different components of psychosocial stress are, for family tensions, the rate of divorce (*dvr_rate*) and, for dislocation, the rate of internal rural-urban migration (*mig_rate*), and the unemployment rate (*unemp_rate*). Then this component is multiplied by a constructed variable, EXPT, which is an indicator of the gap between expectations and the actual situation (it could be interpreted like a degree of unexpectetdness)¹⁴. The unemployment rate has non-zero values only since 1991.

The result of the PC extraction shows that the variance extracted by the first component is fairly good (76 per cent). The scoring coefficients indicate that migration and unemployment are very good determinants of the value of the first component.

Table 2: Output of the principal components extraction

<i>Component</i>	<i>Eigenvalue</i>	<i>Difference</i>	<i>Proportion</i>	<i>Cumulative</i>
1	2.2702	1.67705	0.7568	0.7568
2	0.5932	0.45668	0.1977	0.9545
3	0.1365		0.0455	1.0000
<i>Variables</i>	<i>Scoring coefficients</i>	<i>Correlation Indices</i>		
DVR_RUS	0.53812	0.8259		
URM_RUS	0.54304	0.8182		
MIG_RATE	0.63612	0.9585		

source: author’s calculation

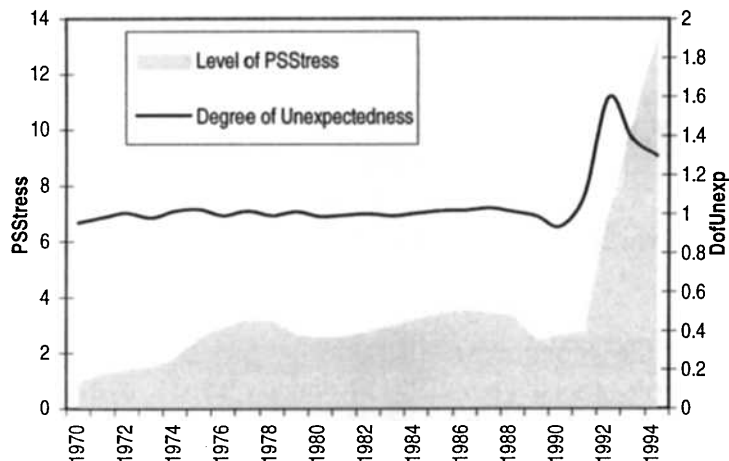
¹⁴ EXPT is calculated through a comparison of adaptive expectations based on an 11-year exponential smoothing forecast and the actual value of real consumption per capita. Formally:

$EXPT(t) = CONS(t)/CONS^*(t)$

in which CONS*(t) represents a one-year forecast of an esponential smoothing model computed in the range (t-12)-(t-1).

The evolution of the stress variable after the correction by the degree of unexpectedness is represented in Figure 3.

Figure 3: The psychosocial synthetic variable



Source: author's calculation

The steady increase during the 1970s, the stabilisation in the early 80s, the small drop after 1985, and the push forward of the variable after 1991 are all quite noteworthy.

4.2 Alcohol consumption

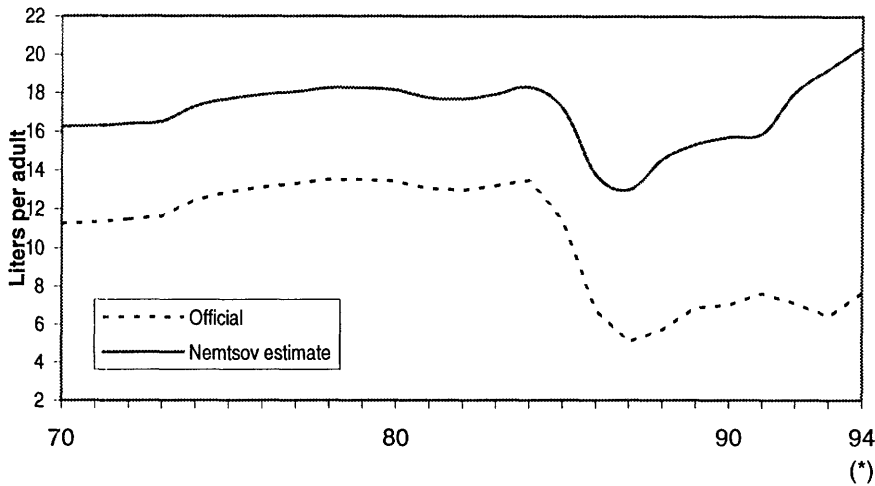
Alcohol consumption is widely recognised as a prominent risk-factor in Russia, where it is a traditional practice. Alcohol intake rose quite steadily during the pre-transition period. This provoked an upsurge of the alcohol prevalence among all population strata.

Epidemiologically speaking, alcohol consumption has a serious effect on cerebrovascular mortality, cirrhosis and all violence-related mortality (suicide, homicide and undetermined voluntary injury). However, its role in determining mortality because of heart disease is questionable. The most widely accepted epidemiological theory states that only the consumption of very large quantities of alcohol could constitute a serious threat in terms of ischemic heart mortality.

In any case, the importance of this proximate determinant is obvious given the effect of the anti-alcohol campaign undertaken in Russia in 1985 for two years. Thereafter occurred a sharp and sudden decrease in mortality among the working-age population that was mainly driven by an abatement in the mortality due to violent causes and cerebrovascular diseases.

One might have expected that the belt-tightening imposed by the massive reduction in real household incomes would have entailed a significant drop in the purchase of alcohol. Actually, the official data on alcohol sales seem to confirm this hypothesis. Figure 4 shows two time-series: the official one (Goskomstat 1996) and the estimates of alcohol consumption in Shkolnikov-Nemtsov that are the most reliable among the unofficial data sources.

Figure 4: Alcohol consumption in Russia according to different data sources: litres per adult equivalent



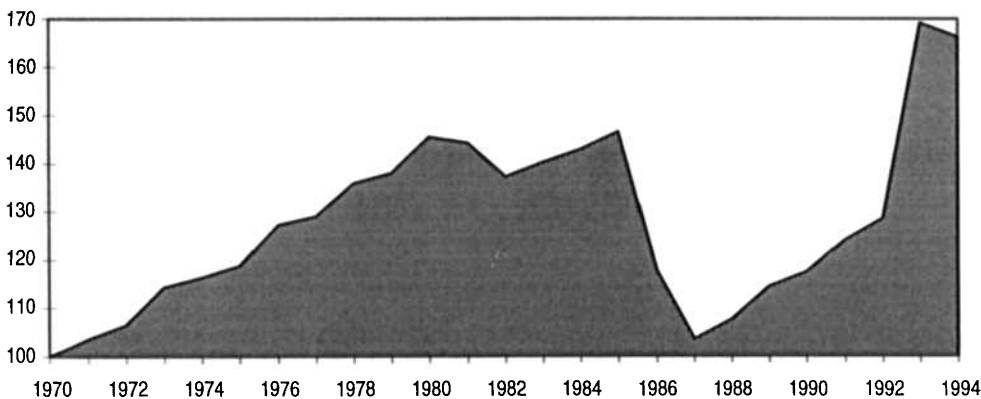
Sources: Goskomstat (1996), Shkolnikov V. and Nemtsov A. (1996), Ryan (1995).

note: (*) The consumption of alcohol for 1994 was obtained by applying the same percentage of unrecorded sales of the past years over the official estimates

Figure 4 indicates that, as mentioned before, according to the unofficial figures, alcohol consumption returned to the previous 1985 levels, while for the official estimates it flattened around 6 litres,

Why did alcohol consumption increase during a period characterised by an extreme reduction in the average purchasing power of households? Several responses can be offered: First, the prices of alcoholic beverages have risen at a significantly slower pace than have prices in general during the transition, so that the relative price of alcohol – a very important explanatory variable of alcohol consumption (Ornestein 1980) – has actually fallen. Moreover, the drop in the relative price of alcohol was greater than the slump in real wages. Thus, recent years have witnessed an improvement in the purchasing power of wages for alcohol (Figure 5).

Figure 5: The alcohol purchasing power of wages in Russia, 1970-94



Source: Author's calculations based on Goskomstat (Narodnoe, various years).

Second, habit (consumption inertia) is a very important feature of alcohol consumption¹⁵. This has meant that, *ceteris paribus*, alcohol consumption has been very rigid respect to fall in real incomes¹⁶.

Third, it has been widely recognised that alcohol is perceived as a powerful stress-reliever. For this reason, psychosocial stress due to painful individual and social adjustment processes might tend to shift consumer preferences towards alcohol consumption. In this case, one proximate determinant is also a function of another proximate determinant, which will force us to establish and assess this relation.

Fourth, as in other Central and Eastern European countries, the lack of policy disincentives on alcohol consumption through limitations on supply, trade controls, bans on advertising, and so on is a striking feature of Russia in recent years.

In an attempt to formalise our model, we could employ the Houthakker-Taylor approach to the study of the evolution of the demand for a specific good which is subject to habit formation or inertia¹⁷. By using a log-log functional form (implying a certain level of saturation), we can formalise our model as follows¹⁸.

$$\ln \text{ALK}(t) = \beta_1 + (\beta_2 \Delta \ln \text{STRESS} + \beta_3 \Delta \ln \text{TCONS} + \beta_4 \Delta \ln \text{ALKP}) + \beta_5 \text{Policy variable} + \beta_6 \ln \text{ALK}(t-1)$$

where alcohol consumption at time (t) is a function of an inertia (stock of habits) term ($\text{ALK}(t-1)$), the extent of which is given by the coefficient β_5 , plus a dynamic short-term effect given by a change in real per capita total consumption (TCONS), alcohol relative price (ALKP) and with psychosocial stress defined as outlined earlier.

There are two limitations on that specification.

First, unfortunately, we can add only one policy variable, namely, a dummy variable for the years of the anti-alcohol campaign. We have only scattered information on the number of authorised outlets for alcohol (supply) and nothing on changes in trade regulations and other similar restrictions on alcohol consumption or on public expenditures on anti-alcohol advertising.

Second, A preliminary analysis clearly show a strong correlation between TCONS and both ALKP and STRESS¹⁹. A way for bypassing this problem is to use the variable WALKPP (wage alcohol purchasing power) which represent the potential demand of alcohol in terms of wages.

¹⁵ This feature of alcohol consumption is widely accepted. Farrel (1952) included beer and spirits consumption amongst the category of goods with irreversible consumption function. Indeed, the first order autocorrelation coefficient is 0.765

¹⁶ In the Intriligator, Sweitzer and Salehi (1983) alcohol consumption estimate, they utilized the prevalence of alcohol consumption as a proxy for habit formation.

¹⁷ Actually they adapted the stock-adjustment structure to habit formation by redefining the stock variable as a "psychological stock of habits" (Phlips 1982)

¹⁸ It is unclear whether alcohol consumption follows a random walk process. The ADF test rejects the H0 of unit root at 10% but not at 5%. Actually the result is the following: $\tau = 2.8350$ vs 5% threshold of 3.01

¹⁹ Respectively: 0.738 and 0.7211

The calculation of our model produces the following results.

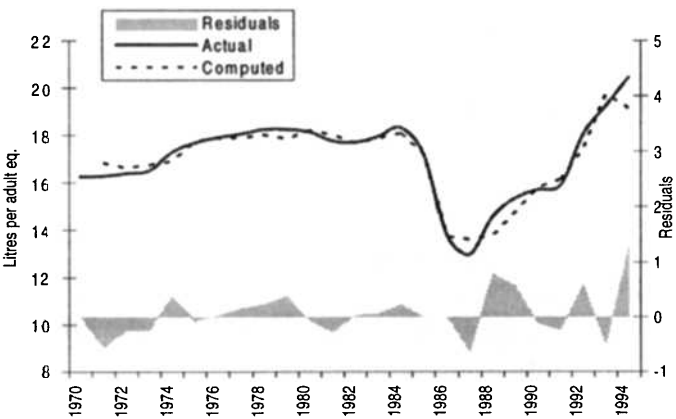
Estimation by Least Squares with Robust
Standard Deviation

Dependent Variable:	<i>ln</i> (ALK)
Annual Data From:	71:01 To 94:01
R-Bar =	0.92818
Bera-Jarque test (Normality)	2.060
Godfrey-Breush-Pagan (AR 1)	1.241
Breush-Pagan test (Homoscedasticity)	7.043

	<i>Variable</i>	<i>Coefficient</i>	<i>Std Error</i>	<i>T-Stat</i>	<i>Significance</i>
1.	Constant	1.80918	0.62711	2.8849	0.00948
2.	$\Delta \ln \text{STRESS}$	0.07968	0.02879	2.7672	0.00565
3.	$\Delta \ln \text{WALKPP}$	0.22384	0.08096	2.7645	0.00570
4.	$\ln \text{ALK}\{1\}$	0.84301	0.18490	2.9589	0.00923
5.	<i>Dummy</i> 85	-0.06675	0.01490	-3.6159	0.00653
6.	<i>Dummy</i> 86	-0.19206	0.03892	-5.7340	0.00095

Clearly, the estimate partly confirms the hypotheses expressed in the formulation of the model. The contribution of both the policy dummy and wage alcohol purchasing power are significant. As expected habit formation (inertia) accounts for an elasticity extremely high (84%). Low is the elasticity of Psychosocial stress even if highly significant. Misspecification tests do not signal problems and an analysis of predicted vs. actual alcohol consumption show that the model is robust for the transition period and it catches almost all turning points, apart from, of course, the years with dummies.

Figure 6. Actual and Predicted Alcohol Consumption



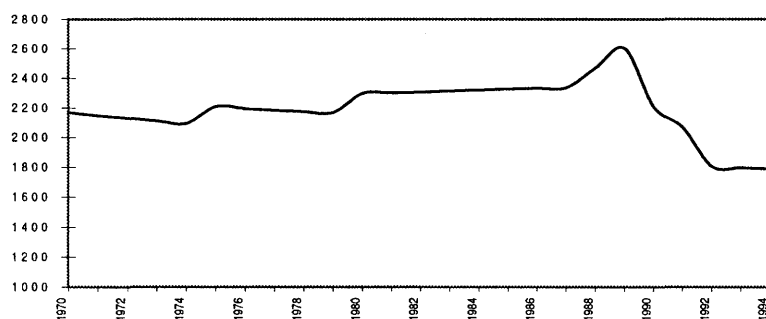
4.3 Smoking

Smoking is a principal risk-factor in terms of premature deaths due to various types of cancers and, to a lesser extent, to all types of cardiovascular diseases. It exhibits a very strong habit formation probably more than alcohol, and, like this, its prevalence is higher among the Russian population than it is among the populations of Western countries. The prevalence in Russia and in these countries was almost the same during the 1960s, but, like the prevalence of alcohol consumption, it started to increase in Russia during the 70s, reaching a peak before the transition.

Some authors are convinced that almost 20 per cent of the excess adult male mortality and 3 per cent of female mortality can be attributed to long exposure to smoking²⁰. The impact of smoking in terms of lung cancer and CVD mortality requires protracted exposure for a significant number of years. The inclusion of this variable in our model would not demonstrate the expected and well-established dangerous effect, given the very complex structure of distributed lags, which are also well beyond the range of the available sample.

Nor is it possible to examine a consumption function for smoking. There are two reasons: the first one is related to the strong random walk behaviour of the series²¹, the second one, in case we would modelize the short term variations, is the limitation on the explanatory variables, indeed although data on cigarette consumption are available, they do not provide enough information on prices for the entire sample period. Nonetheless, cigarette consumption does appear to have slightly declined throughout the region over 1989-94, as incomes dropped and relative prices, particularly for imported cigarettes, soared (which differs from the situation for alcohol consumption) in 1992, though these data do not include information on the consumption of smuggled cigarettes (WHO-HFA 1997). After the sharp increase in 1992, relative prices diminished and consumption flattened at about 5 cigarettes per day per adult.

Figure 7. Annual number of cigarettes smoked per adult equivalent



source: Narodnoe(various years); Russian Goskomstat; WHO-Euro

4.4 Diet

A drop in food consumption, especially in animal proteins, occurred during the transition as a consequence of the reduction in real incomes. However, in view of the high calorie and fat consumption of the past and the substitution of expensive sources of nutrients with cheap ones, the drop in food expenditure was not so significant except among the poorest segment of the population (Paniccià 1997, Cornia 1994).

²⁰ See for instance, Peto R, Lopez AD, Boreham J, Thun M, Heath CJ. (1992) Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet* n. 339: 1268-1278.

²¹ An Augmented Dickey-Fuller test has been performed on that series. The results clearly show the presence of a random walk.
ADF t-test (with 1 lag): -1.2839 (5% threshold = -2.63)

The level of undernutrition in Russia may be considered mild, and the limited extent of income-nutrient elasticity and the previous levels of the income/consumption ratio and, so, of the health stock (the ‘threshold effect’) have cushioned the average effect of the impoverishment process. The main impact has been felt in the distribution of food intake, with a widening nutritional gap among people in various income classes.

Table 3: Food consumption in selected deciles in Russia (monthly kilograms), 1995

	Households in the lowest income decile	Households in the ninth income decile	Ratio ninth/lowest decile
Bread/bread products	7.3	9.5	1.30
Potatoes	8.4	10.4	1.24
Vegetables	4.8	8.6	1.79
Sugar/sugar products	1.6	2.8	1.75
Meat/meat products	2.6	5.7	2.19
Milk/milk products	14.3	25.6	1.79
Eggs (units)	11.1	20.0	1.80

Source: Goskomstat (1996).

There is substantial evidence suggesting that the diets of the poor are deteriorating and that they increasingly consist of more fatty and unhealthy food items. Indeed, there are some indications that, while reducing the average overall per capita consumption of meat and sausages, the substitution effect triggered by the fall in household incomes is pushing the rate of consumption of cheaper, fatter and lower quality cuts of meat upward. The gap in food consumption between the lowest income decile and the second lowest income decile is more pronounced for vegetables, milk and meat (Table 3). Furthermore, nutritional surveys (for instance, the RLMS (1995)) clearly show that the amount of protein derived from fat that is consumed has risen especially among the lowest deciles.

After almost 30 years during which the consumption of k-calories and fat climbed, the situation began to change in 1989, so that the average consumption of k-calories approached the minimum requirement, and the consumption of fat declined sharply (Figures 6a and 6b).

Figure 8a: Daily k-calorie intake per capita in Russia, 1960-94

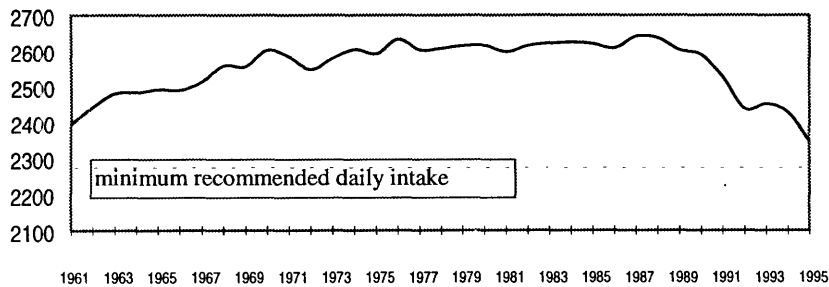
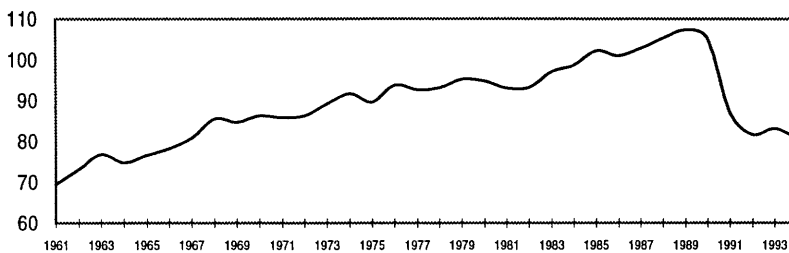


Figure 8b: Daily intake of fat per capita in Russia, 1960-94 (in grams)



Source: FAOSTAT.

As for smoking, the daily fat intake followed a random walk process with drift and deterministic trend up to 1989²², since that date a structural break occurred producing a stabilisation around 83 grams of intake since 1992, while the intake of k-calories and proteins continued to fall.

However, apart from the limitations in the availability of data, the data do not take into account the sharp and sudden increase in the differences in diets between the various strata of the population. The amount of fat in the diets of the poor has climbed as fatty food items have become relatively less expensive.

As for smoking, the inclusion of fat intake in an estimate which permits few lags can generate inaccurate results, although it is quite well established that prolonged exposure to a fatty diet is one of the main causes of CVD mortality.

5. The process of accumulation of health assets

As noted before a key role in determining mortality is played by the health assets stock and its process of accumulation. We can start our analysis from the first step of the epidemiological transition. It took place in Russia from the late 30s up to the 50s with a large decrease of the IPD mortality especially amongst young age-groups. This accelerated after the W.W.II by the contribution of the improving standard of living, equality, nutrition and housing. The transition was undoubtedly helped by vaccination campaigns and the extensive adoption of cheap technologies needed to fight against IPD. This provoked, amongst other things, the ageing of population as resulted from the contraction of IPD mortality causing a structural increase of the exposition of more population to typical middle-age/elderly diseases like CVD. The result, as shown before, was an increase of such as morbidity, which turned out also in the increasing mortality by those causes. While the western countries tackled this problem by entering in their second step of epidemiological transition (reduction of adult mortality) this did not take place in Russia. It seems that the process of accumulation of health assets almost ended during the 70s and 80s.

The new epidemiological pattern provoked by the first epidemiological transition found the soviet health system completely not prepared. The main task of the health system in such a case was to shift the provision of the health system from an extensive-base to an intensive-base with a high technological progress embedded, but unfortunately this did not take place.

²² The ADF test for the joint H0 Unit Root and deterministic trend is $\tau = .8574$ vs. a 5% threshold of 5.18

According to C.Davis (1996) the main reason relies on very bad planning made by the soviet authorities in which the health care system received a low priority²³ in the allocation of resource during the plan formulation. The main consequences during the plan implementation were: no commitment to overfulfilment the output plans, hard budget constraints no commitment to fulfil the supply plans, limited extra-productive capacity and high shortage intensity, plus high tolerance in achieving the plan-targets and a relative low labour remuneration. What did imply in terms of health assets flows?

First, in terms of self-produced technological progress this meant a wide gap with Western Europe.. Indeed both the medical and pharmaceutical industry lagged well behind the western one, the same happened for the R&D medical sector not directly linked with defence apparatus. The low priority and the hard type of shortage opened a big technological gap between the russian and western health and pharmaceutical industry East- and West. As C.Davis (1997) pointed out:

“Medical facilities pharmaceutical factories and biomedical research institutes lagged the West in most important production technologies, maintained old and technologically obsolete machinery in service, did not possess new technologies (e.g. CTA, magnetic resonance) and had a very low level of capital productivity. All facilities experienced disruptions of production and forced substitution of inputs due to shortage economy”

Second, the physical stock suffered from that impoverished supply to the medical system but also by tighter budget constraints. As mentioned before not only the pharmaceutical industry but also the medical system received a low priority by the planners. Despite a steady and substantial increase in GDP (almost 3.5-5% per year during the 70s) the amount of resource to the production of health services were very low (4% during the same period) especially if compared with the most advanced OECD countries (8%). Again, the share of total state budget health expenditure dropped from 6.6 in 1965 to 5 in 1980 following a steady decline. This turned out in a quantitative an qualitative deficiency of medical equipment and supply of medical commodities, worsening, inter alias, the situation of safety of the treatment by increasing the morbidity due to infections under medical treatment. The scarcity of resources devoted to investments provoked a net contraction of the accumulation of physical capital (mainly hospital) and a lack of maintenance of those existing. The same for the medical machinery.

Third, the health system was characterised by a “quantity drive approach”, which allowed to expand the volume of basic medical services which in turn assured to the health status to not-worsening especially in terms of IPD, but without the effectiveness needed to tackle this new epidemiological pattern significantly.

As many other services the medical provision was almost detached from the demand of consumers and mostly buyers oriented. Another specific production factor, the organisation of the service, was poor. Quoting C.Davis (1997), it could be easily defined the health system how an intersection of competencies amongst different ministries with duplications and unnecessary overlaps. The same took place for the pharmaceutical industry, provoking in the all system a plethora of bottlenecks.

²³ This is not the case of another socialist country like Cuba, where Health Care has received a very high priority and where the achievement in terms of Health Production are widely recognized as the best in the world, given the low economic performance of that country

By summing up, it could expect that at least during the 70s and the early 80s not only the health assets did not increased but their depreciation was not fully replaced by the new flows of investments and technological progress.

The situation slightly improved with the onset of perestroika, with the introduction of partial reforms of the health system, but the situation carried on its deterioration quite soon after 1989 with the incoming problems caused by the economic situation. The onset of transition blew up the existing problems. with a significative difference: if during the soviet years at least the health system could guarantee a significant worsening of the health situation, during the transition the economic recession was utilised for imposing higher budget constraints and for diminishing the already low priority of the health care provision both in terms of inputs and outputs.

This provoked a dramatic collapse of the health care and a sharp decrease of the health assets. First, the production of the domestic pharmaceutical industry dropped in by 40% from 1990 to 1994. To be noted that this production was only partially replaced by drugs imported²⁴ given the international high relative prices and the above mentioned lack of financial resources. The remuneration of factors, namely labour, fell also relatively to other occupations by almost 50% provoking flights to other jobs (especially amongst paramedical personnel) and a further drop in the already low job-motivation. Physical stock is affected by a steady process of deterioration, given the chronic lack of maintenance. The resources devoted to R&D suffered from the first budget cuts and now are very low. In waiting for a health system reform which could raise the priority of this sectors without dismantling it by using westernised ugly theories the health assets are sharply deaccumulating.

6. The estimate

In view of the above discussion on proximate determinants and long-term factors, the following specification has been calculated.

$$\Delta \ln CVD_{(t)} = \exp (\alpha_{(t)} + \beta \ 1/\Delta ALK_{(t)} + \sum \gamma_i \ 1/\Delta SMOKE_{(t-i)} + \sum \delta_i \ 1/\Delta FAT_{(t-i)} + \beta_i \ 1/\Delta STRESS_{(t)})$$

As shown in the discussion on alcohol consumption, there is a clear relationship between ALK and psychosocial stress. Therefore a reduced model reduced model without alcohol, but with the explanatory variables of it (including stress) has been estimated

²⁴ Which was almost stable during 1992 1993 and 1994 desoite the above mentioned fall in the domestic production

1. The estimate for males produces the following result (last year result).

Estimation by Kalman Filter

Dependent Variable:	$\ln(\Delta\text{CVDM})$
Annual Data From:	71:01 to 94:01
R-Bar =	0.67814
Durbin-Watson Statistic =	1.7651

	Variable	Coeff	Standard Error	T-Stat	Signif
1.	Constant	0.12062	0.01769	6.81866	0.00001
2.	1/ ΔWALKPP	-0.00087	0.00028	-3.11520	0.00820
4.	1/ ΔSTRESS	-0.01107	0.00464	-2.49559	0.01355
6.	1/ ΔSMOKE	-0.25046	0.15185	-1.64939	0.12301
8.	1/ ΔFAT	-0.00004	0.00003	0.98486	0.34140
10.	<i>dummy85</i>	-0.01199	0.05590	-0.21442	0.83355
11.	<i>dummy86</i>	-0.13204	0.04851	-2.72208	0.01744
3.	1/ $\Delta\text{WALKPP}\{1\}$	-0.00046	0.00037	-1.24193	0.23620
5.	1/ $\Delta\text{STRESS}\{1\}$	-0.00103	0.00421	-0.24513	0.40510
7.	1/ $\Delta\text{SMOKE}\{1\}$	-0.07970	0.17660	-0.45130	0.65921
9.	1/ $\Delta\text{FAT}\{1\}$	0.00004	0.00004	0.97278	0.34741

The estimate partly confirms the hypothesis of proximate determinants, and the long-term time-varying factor which constrains the variation in mortality seems to fit in. The percentage of variance explained by the model is not so high but the significance of variables seems to confirm, partly our hypothesis. More precisely, both alcohol and stress seem to exert an effect on CVD mortality. Especially the latter is important in determining variations in CVD mortality directly and by means of alcohol consumption. By using the incremental contribution methodology (Theil 1984), we can see that stress accounts for almost one third of the total variance explained (R-square bar), net of collinearity (Table 4) which accounts for 21%. However, we do not know how much of this explained variance is mediated through alcohol. Nonetheless, we can assume, given the low percentage explained by a key variable in the alcohol model, namely, WALKPP, and given the estimated alcohol consumption function, that it accounts for a very high proportion. Alcohol directly, despite it is significant, indeed does not seem to affect in a strong way, in the short term, CVD mortality, confirming the low impact on the bulk of CVD mortality, namely Ischemic heart disease. It is important to note that amongst the antialcohol campaign dummies only 1986 (the peak of that campaign) is significant.

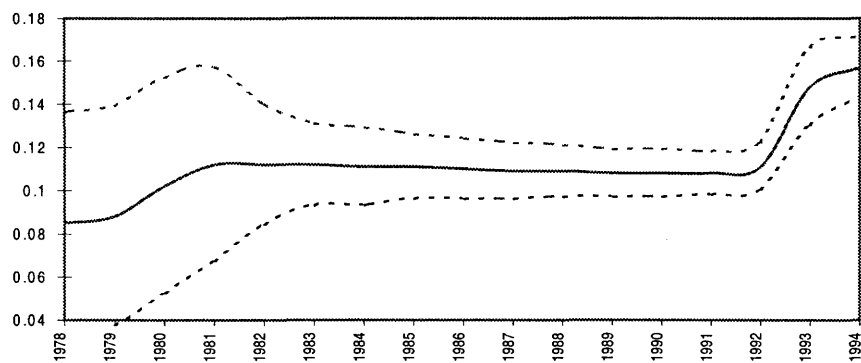
Table 4: The average incremental percentage contribution to the average explained variance in the male CVD mortality model

	Variables	
1.	Constant	0.411
2.	ΔALKPP	0.123
	$\Delta\text{ALKPP}\{1\}$	0.012
3.	ΔSTRESS	0.325
4.	$\Delta\text{STRESS}\{1\}$	0.010
5.	ΔSMOKE	0.024
6.	$\Delta\text{SMOKE}\{1\}$	0.002
7.	ΔFAT	0.001
8.	$\Delta\text{FAT}\{1\}$	0.000
9.	<i>dummy86</i>	0.091

For the other two proximate determinants (fat and smoke), the regression does not provide good results. In current and lagged values, neither of them is significantly different from zero. This does not mean that they do not contribute to CVD mortality. Epidemiological evidence demonstrates quite clearly that there is a substantial relationship between these two variables and CVD mortality. The only problem is that, as implied by the epidemiological pathway, the exposure over time to such risk-factors is a complex lagged process which cannot be tackled within a ‘short’ sample.

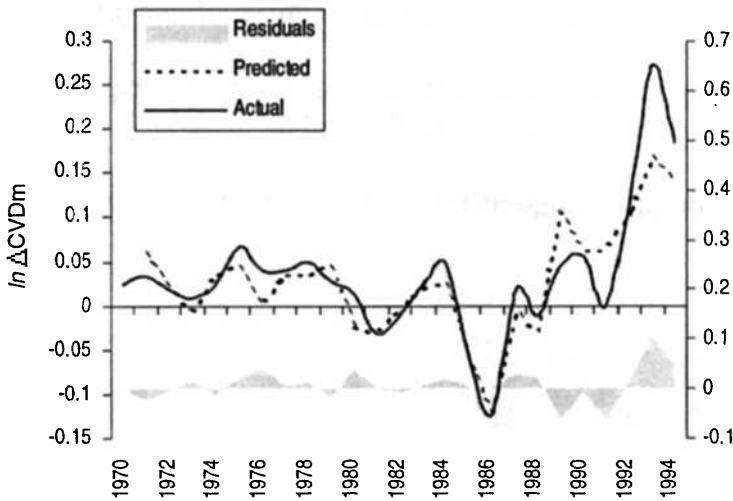
It is important to determine the path of the long-term factor, which contributes an average of almost 40 per cent to R-squared. A Western pattern would typically involve a steady decrease over the sample period, but, as one might expect, observations of the evolution of the parameter for Russia (Figure 7) show that it was nearly stable for the entire period under investigation, slightly decreasing only during the 1980s.

Figure 9: The time-varying asymptote in the male CVD mortality estimate



The onset of transition produced a significant increase, probably due to a sharp deterioration in health assets that had been occurring earlier. But an analysis of the goodness of fit of the model makes evident the weakest aspect of this specification, that is, the behaviour long-term factor in the latest short-term deterioration. Actually as shown in Figure 10. the model is able to predict only partly the variance in the last years of the transition

Figure 10. Actual vs. Predicted Rate of Change in CVD Male Mortality Among 15-60 Age Group



and this is mainly due to the relative low value of the threshold²⁵ which, probably, partly underestimates previous health assets deterioration.

2. The estimate for females produces the following results.

Estimation by Kalman Filter

Dependent Variable: $\ln(\Delta CVDF)$
 Annual Data From: 71:01 To 94:01
 R-Bar = 0.51409
 Durbin-Watson Statistic = 1.6957

	Variable	Coeff	Std Error	T-Stat	Signif.
1.	Constant	0.10543	0.03855	2.73221	0.01710
2.	1/ Δ WALKPP	-0.00072	0.00035	-2.06574	0.05938
3.	1/ Δ STRESS	-0.00910	0.00455	2.00883	0.06579
5.	1/ Δ SMOKE	-0.30978	0.15929	-1.94482	0.07375
7.	1/ Δ FAT	-0.00003	0.00004	-0.95220	0.35837
	dummy85	0.01445	0.05864	0.24641	0.80921
9.	dummy86	-0.11106	0.05088	-2.18274	0.02400
	1/ Δ WALKPP{1}	-0.00027	0.00039	-0.68853	0.50322
4.	1/ Δ STRESS{1}	-0.00418	0.00397	-1.05309	0.15569
6.	1/ Δ SMOKE{1}	-0.00191	0.18525	-0.01032	0.99192
8.	1/ Δ FAT{1}	0.00005	0.00004	1.19790	0.25234

The estimate confirms the significant difference between the male and the female pattern in CVD mortality. A model which could fit for males loses almost 1/2 of its explanatory role in dealing with female mortality. Even if the main relevant variables for males are still significant for females nonetheless other

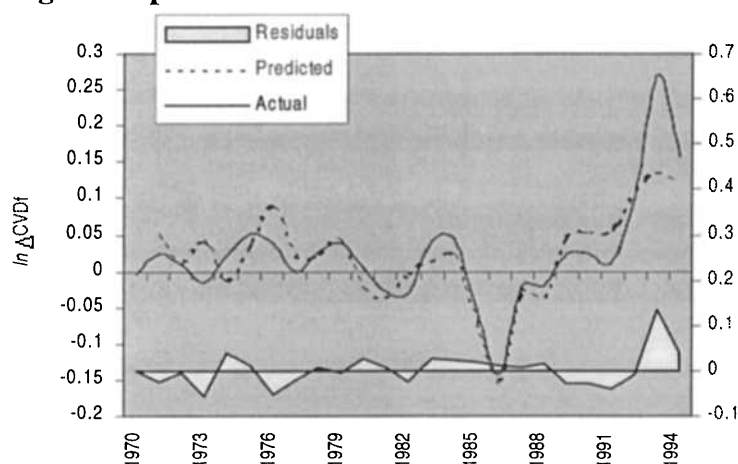
Only the constant is significantly different from zero at 5% level while WALKPP and STRESS are significantly different from zero at the 10% threshold. The percentage of explained variance drops from 70 per cent to almost 50 per cent.

²⁵ In 1993 the percentage increase was about 25% while the threshold allowed only an increase of 16%

This means that, as expected, CVD female mortality is less influenced than is male CVD mortality by short term variations. Not only, amongst the risk factors (apart from the well known problems with fat and smoke) alcohol seems to loose most of the already low contribution to such as mortality.

As for the male mortality even for females the critical year in terms of variance explained is in 1993 when almost 50% of total variation was not caught by the model

Figure 11. Actual vs. Predicted Rate of Change in CVD Female Mortality Amongst 15-60 Age Group



7. Conclusions

In this first attempt to assess econometrically the short-long term determinants of CVD mortality we can draw some unambiguous conclusions.

First, the analysis seems to suggest that both long-term (health assets accumulation) and short term variations of proximate determinants (mostly Psychosocial stress) could constitute significant explanations for the mortality changes (especially the last one)

Particularly PSS seems to play a very important role in the short-term in explaining mortality changes as alcohol has a very secondary importance at least in explaining CVD male mortality changes.

Despite the analysis does not show a significant role of smoke and fat diet it seems that a more accurate specification should be carried on by improving (i) the quality and quantity of data (ii) by specifying the role of such as risk-factors.

Actually the literature is not clear, oscillating between immediate responses in changing diet pattern or smoking prevalence, towards more lagged effects.

However, a further analysis with a better dataset should be carried out, especially in specifying in a more definitive way the two components of the CVD mortality.

Second, the functional form records a weakness about the role of the long-term factor in the last transition years. Clearly the first alternative illustrated in the text could constitutes a better answer for explaining the long and short term contributions to the CVD mortality.

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