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Avoidable death: tobacco, alcohol, and road accidents in France

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While deaths caused by bovine spongiform encephalopathy can be counted on one hand in France, about a hundred thousand deaths are caused each year by tobacco (around 60,000 deaths in 1995), alcohol (45,000) and road accidents (8,000). A look at the most frequent causes of avoidable death in France shows that the consumption of alcohol is on the decline as are diseases related to alcohol, and that deaths due to road accidents are also decreasing, despite a regular increase in the kilometres travelled. The evolution in the damage caused by tobacco is more complex due to the delay between the consumption and the evolution of the pathologies: it is increasing significantly for women, while it seems to be stabilizing for men.

Tobacco consumption and cases of lung cancer on the increase

It is difficult to evaluate the number of deaths caused by tobacco and alcohol as these causes interact with other causes, which must be taken into consideration (see box 1). This is also true for road accidents. The intensity and the duration of exposure to the different risk factors in the genesis of chronic diseases produced by the consumption of tobacco and alcohol must also be considered at the same time. Thus, cancer of the larynx can occur after thirty years of smoking, and its mortality rate will depend on the average number of cigarettes smoked daily, the number of years of smoking, the age at which smoking started, and also on the development of medical treatment.

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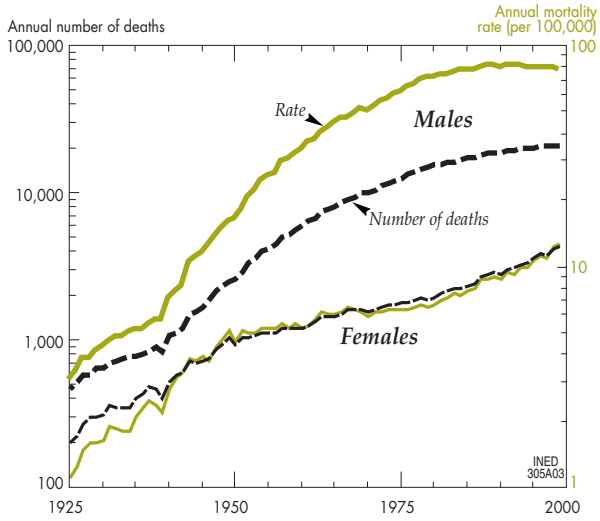
Figure 1 – Evolution of mortality from lung cancer in France since 1925



Note: The scale of the Y-axis is geometrical in order to facilitate the comparison between males and females.
Sources: CEPIDC-INSERM – Vallin and Meslé [2].

The evolution in the death rate from the pathologies induced mainly by these products is a good indicator of trends [1]. Following a high increase, male mortality due to lung cancer reached a plateau in the mid 1980's and the rate has declined slightly since 1989 (Figure 1). For women on the other hand, although the rate is much lower than for men, it is increasing regularly. These trends are in accordance with tobacco consumption, with a gap due to the delayed effects of

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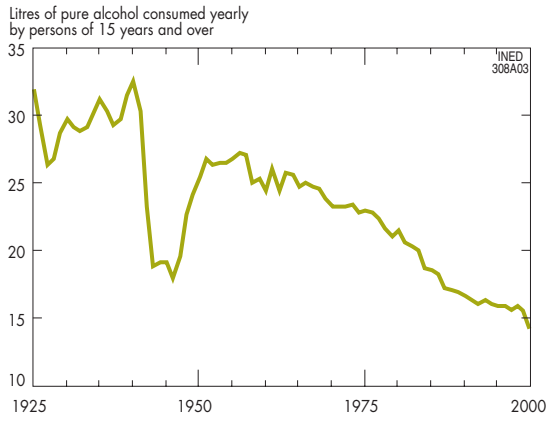
smoking. (A recent fall in tobacco consumption, linked to increasingly heavy taxation, will be reflected in future mortality.)

Decreasing alcohol consumption and cases of alcoholic cirrhosis

The scarcity of alcohol during the 1939-1945 war caused a sudden decline in mortality from alcoholic cirrhosis of the liver (Figures 2a and 2b). At the end of the war and for twenty years afterwards, the upward trend continued. Then it reversed, and for the last forty years we have been witnessing a regular decrease in mortality from alcoholic cirrhosis which follows a reduction in the average amount of alcohol consumed per inhabitant.

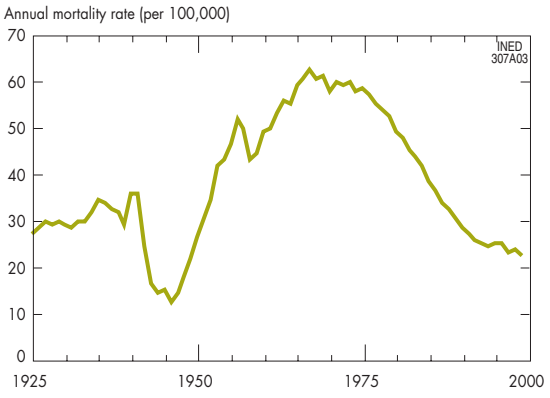
For certain pathologies, the evolution is less easy to explain. Primary liver cancer caused 4,010 deaths in 1979, and 6,900 in 1999. In the past, it generally developed from an alcoholic cirrhosis. Its current level is due to the development of viral chronic hepatitis and progress in the treatment of cirrhosis which allows a greater number to evolve and develop into cancers, particularly when high alcohol consumption is associated. Having said this, mortality from cirrhosis is observed at the same ages as previously (Figure 3), but at lower rates for all ages. Multiple substances pass through the liver and its cancers can be caused by substances other than alcohol or chronic hepatitis.

Figure 2a – Evolution in alcohol consumption in France since 1925



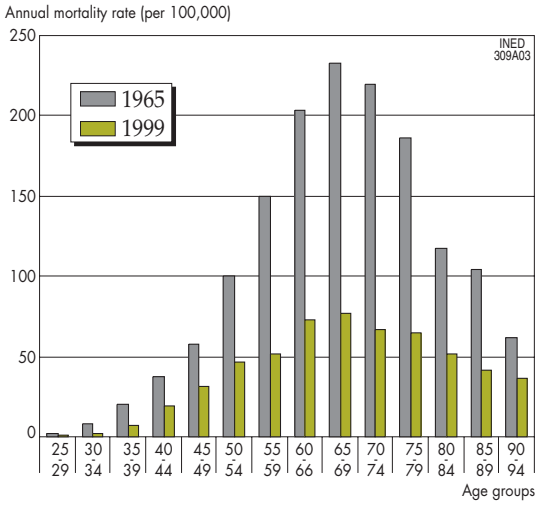
Sources: Ledermann [3], Nizard and INSEE.

Figure 2b – Evolution of male mortality from alcoholic cirrhosis of the liver in France since 1925



Sources: CEPIDC-INSERM – Vallin and Meslé [2].

Figure 3 – Male mortality rate from alcoholic cirrhosis according to age and year in France



Sources: CEPIDC-INSERM – Vallin and Meslé [2].

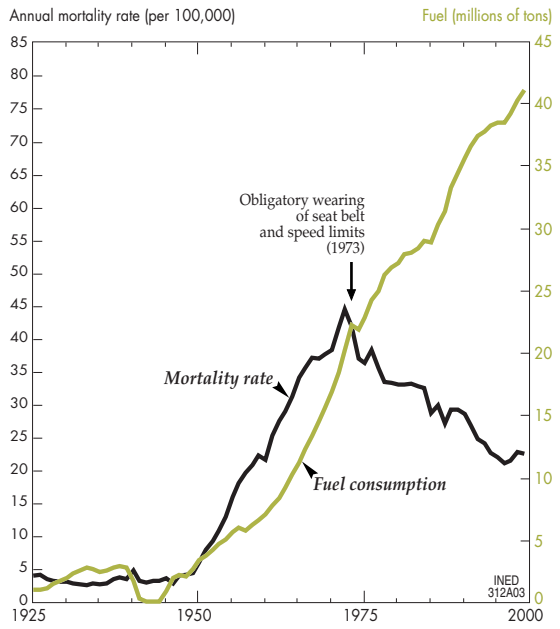
Mortality due to road accidents: an arrested increase

Mortality from road accidents seems easier to identify than mortality attributable to alcohol and tobacco, even if one must go beyond the idea of an accident in order to evaluate the fraction of traffic mortality due, for example, to excess speed, alcohol, inexperience, or the loss of physical or psychological abilities which are the real causes of the accident.

There are two sources for data on traffic mortality. The police and the military police register all accidents that occur on public highways, including deaths occurring up to 6 days after the incident. It is estimated that nearly 6% of deaths which occur later, up to 30 days after the accident [4], should be added. Medical death certificates, that include deaths that occur later still, should consistently produce higher results. Yet the opposite is observed. In 1979, 12,480 deaths were counted in police statistics and 11,016 in statistics using death certificates produced by Inserm (National Institute for Medical Research). In 1989, the values were 10,528 and 10,268 respectively and in 1999, they were 8,029 and 7,918. The difference between both sets of numbers has decreased from 12% to 1.4%, a sign of improvement in the certification of deaths by doctors. The evolution of traffic mortality is one of the best

examples of the efficiency of preventive action against avoidable deaths. Between the end of the Second World War and 1973, the increase was practically linear, parallel to the increase in traffic. Following the maximum of 16,617 deaths in 1972, the decline began suddenly in the middle of 1973, with the application of a general speed limit and the requirement of wearing seat belts (Figure 4). In recent years, after stagnating at around 8,000 deaths (7,720 in 2001), the decline has resumed in 2002, with 7,242 deaths. The future trend will depend on the government's ability to implement the many decisions adopted during the last few months. These decisions have already had a major psychological effect, now credibility of action must be ensured on the ground.

Figure 4 – Male mortality from road accidents and trends in fuel consumption in France since 1925



Note: The consumption of petrol and diesel is not representative of traffic for the period 1940-1944 because of the use of replacement fuel (producer gas and coal gas).

Sources : Mortality rates: CEPIDC-INSERM - Vallin and Meslé [2] • Fuel consumption: Committee of Petroleum Product Distributors

From identifying causes to sharing responsibility

In the battle against avoidable mortality, progression from a statistical link to causality can be made by a set of arguments but they first need to be accepted. The tobacco industry has made every effort to discredit epidemiological proof by considering unknown associated factors, mainly genetic, as if their existence eliminates the causal role of tobacco in the occurrence of fatal lesions. While the debate is practically at an end concerning tobacco and alcohol, it has reopened for road accidents. The production of cars whose maximum speeds are incompatible with the legal limits was long ago confirmed by insurance companies as a causal factor in road accidents. In these circumstances, each one tends to place the responsibility for risk control in the other's area. The division of roles between the individual who acts and the state which educates, informs, regulates and possibly forbids, does not depend on simple criteria. Behind the distribution of causality is that of responsibility.

The recent trend in mortality due to road accidents proves that a significant number of avoidable deaths can be influenced by sensible measures that encourage rather than impede on human liberty. Dependency, handicap, and the loss of human life represent forms of human misfortune and we must put behind us the simplistic debate between the choice of dying young after having lived well, and that of living. Developing an aptitude for pleasure and for life are not incompatible objectives. In order to better understand how to avoid the trap of becoming conditioned to risk, the evolution of avoidable deaths must be carefully followed, pathology by pathology. Interest in epidemiology is still too low in our country, but the situation is changing, the quality of death certificates is improving and a growing number of researchers are moving towards a discipline that governs the quality of public health policy.

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Box 1

How to measure mortality attributable to alcohol and tobacco

Death certificates prioritise the "disease" that caused death, they cannot list all the factors that contributed to the occurrence of the disease. Different methods help to quantify the fraction attributable to each of them.

◆ Global methods

These include interviewing a sample of people about their consumption of alcohol for example, and later recording their deaths. The curves expressing mortality according to consumption are usually J shaped, with excess mortality of non-drinkers compared to those with a low alcoholic consumption. The minimum is located at a level of one or two glasses a day (10 to 15 grams of pure alcohol). Those who used to drink heavily and have stopped completely but who may have retained an increased risk of death, albeit minimized, must be excluded from the survey. Once this bias has been removed, the favourable effects of low consumption are retained in the majority of published studies, with nevertheless some exceptions, and differences according to type of alcohol (wine has a protective effect against all causes of death, beer and other alcohols only have a protective effect against cardio-vascular mortality, according to some studies).

Although the dose-effect link is clear in these studies, they are by definition limited by the diversity of factors that affect mortality and the fact that they are often correlated. For example, the lack of concern that the so-called "happy heavy drinkers" have for their health can result in harmful behaviour in many areas, notably a low or inadequate recourse to systems of prevention or care. Taking into account all the identified and quantified risk factors (alcohol consumption, smoking, weight, sedentary lifestyle, blood pressure, biological factors of arteriosclerosis, etc.) we are still able to explain hardly more than 50% of the mortality variations. The different factors are better separated by studies that specifically analyse each pathology involved.

◆ Analytical methods

These try to specify the role played by a risk factor in the production of a disease, by observing whole populations or a representative sample that includes two groups differing only by the risk factor under scrutiny. The relative risk is calculated directly, since the occurrences of the pathologies are available for both groups, but the method is slow if the risk factor has delayed effects. By calculating the standardized mortality rate of lung cancer in smokers and non-smokers, Doll and Hill [5] showed in 1956 that the incidence was of 0.07 new cases annually among non-smokers, and 0.86 among smokers consuming between 15 and 25 grams per day (at the time of the survey, a cigarette contained around 1g of tobacco) and 1.66 for smokers consuming over 25 g per day. The ratio of 12.3 between 0.86 and 0.07 is a relative risk.

How do we achieve a result in a short time and at a low cost? The best method consists of comparing the characteristics of a group displaying the pathology in question and those of a group not affected (case-control study). It is therefore not possible to obtain the incidence and directly calculate the relative risk as we are not comparing an exposed group to an unexposed group, but a group of unwell people to a group of (currently) healthy people. The result is an approximate relative risk that is called an "odds ratio". Doll and Hill paired a control to each case of pulmonary cancer treated in twenty hospitals in London between 1948 and 1952 and they measured smoking in both groups. They were then able to evaluate the proportion of non-smokers and smokers that could have been observed in the unwell population, in the absence of a statistical link between the occurrence of such a cancer and a certain level of smoking. The ratio between the expected value and the observed value is the odds ratio.

Catherine Hill calculated the number of deaths attributable to alcohol and tobacco in France by using studies of specific pathologies where a dose-effect link had been shown and by taking into account the estimated levels of consumption among the population of France [6] [7]. The possibility of "double counting" caused by the frequent co-existence of excessive alcohol consumption and smoking should be noted, particularly in the case of cancers of the upper aero-digestive tract.

The results of this type of study can be compared from one country to another. While deaths from alcoholic cirrhosis in the strict sense, that is identifiable as such by codes 571-0 to 571-3 of the International Classification of Diseases (ICD, 9th revision), were 5,961 in France for the year 1985, Catherine Hill attributed 45,000 deaths to alcohol for the same year, by taking other pathologies associated with alcohol into account. The ratio between the global mortality attributed to alcohol and that attributed to alcoholic cirrhosis is 7.9. In an American study [8] which uses comparable methods, the ratio is 9.0, and 6.3 in a Canadian study [9]. These values are not very different, despite the imprecision of the coefficients used to calculate the fraction of deaths attributable to alcohol and some differences in the pathologies included in the studies. The difficulty is maximal when the risk factors simultaneously affect an organ like the larynx and the oesophagus, in the case of alcohol and tobacco, for example. Calculating "crude" odds ratios and deducting the attributable fractions without making adjustments for the large numbers of drinkers of alcohol who are also smokers, leads to the attribution of the same cancers to alcohol and tobacco. The second difficulty in this situation is evaluating the level of a possible synergy between the combined effects. In this case, the risk is not only multiplicative, it becomes superior to the product of the relative risks induced by these factors taken in isolation. This point is especially debated for cancers of the larynx and the oesophagus when the effects of tobacco and alcohol are combined, and also for pulmonary cancers occurring in smokers exposed to other carcinogens like asbestos, for example.

Box 2

Information: Excess mortality due to the heatwave

The excess mortality caused by the August heatwave is a serious issue for demographers. They particularly need to verify whether the victims lost some months or several years of their lives. For the time being, readers can refer to an article written by N. Brouard and A. Désesquelles published in the journal *Population**, which looks at another aspect of the problem: the role of family ties among dependant persons living at home or in an institution.

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* "The Family Networks of People aged 60 and over Living at Home or in an Institution", Population, no.2, Ined, 2003.

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