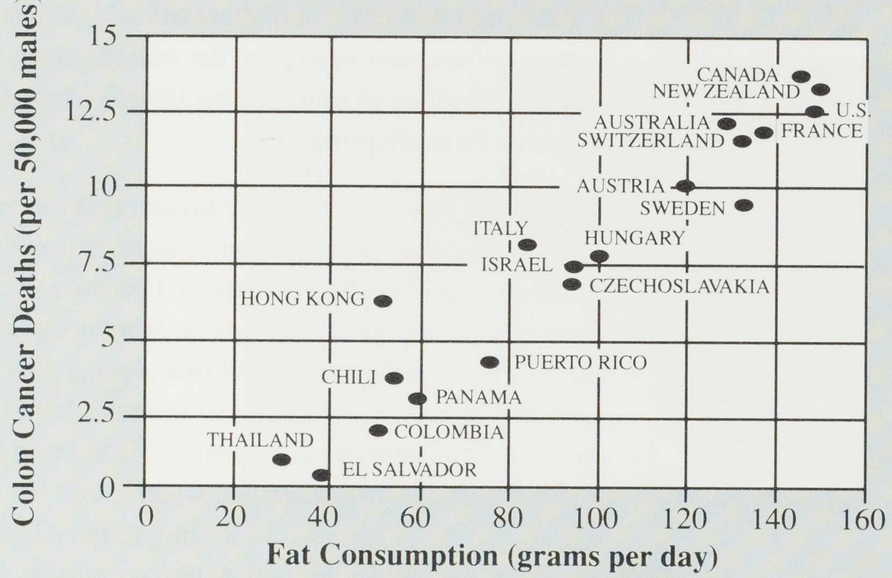
"there is little evidence that arsenic compounds are carcinogenic in animals." Finally, in 1987, scientists managed to produce cancer in animals. This was 180 years after arsenic was first suggested as a human carcinogen and over 70 years since the first attempt to induce the disease in animals.



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▲ ***Diet and Disease:*** *Human population studies have identified the link between the high fat (Western) diet and cancer of the colon.*

Animal experiments have also given contradictory results in studies of diet and cancer. Epidemiological research has shown that too much fat in the diet can cause cancer of the colon with *saturated* fat the chief culprit. However, according to animal tests, it is the *unsaturated* fats that are the most dangerous.[38] With regard to dietary fiber, animal research is again confusing with some experiments showing a reduced risk of cancer and others an increased risk.[39] The human evidence, which is all that matters, suggests that fiber can protect against cancer of the colon. But the absurdity of animal experiments is especially highlighted by tests carried out on the natural substances present in fruit and vegetables.[40] These chemicals have been evolved by the plant as a defense against predators and parasites. When tested on rats and mice at high doses, many of these substances were shown to cause cancer. Yet it is well known from human experience that diets rich in fruit and vegetables actually *reduce* the risk of cancer!

Nevertheless, proponents of animal tests argue that they do have validity since nearly all human carcinogens have (eventually) been shown to cause cancer in some species of animal. But this is misleading: if substances like asbestos, tobacco, arsenic, benzene, alcohol, naphthylamine and soot were not *already known to be human carcinogens,* scientists would not have persisted with attempts to induce the disease in animals. Reliance would have been placed on one or two routine feeding or inhalation tests of the type to which new chemicals are now submitted. As a result, many of the most dangerous human carcinogens would have been deemed safe. For new substances, where epidemiology has had no opportunity to assess risks, researchers should be calling for more reliable test systems rather than a continuation of the biologically flawed approach with rats and mice.

It is not only in cancer research where animal data is preferred to epidemiology. There is direct epidemiological and clinical evidence to show that a proportion of the population reacts adversely to food additives such as colors, preservatives, antioxidants and flavor enhancers. Symptoms include hyperactivity, asthma and eczema. But failure to produce corresponding symptoms in animals has been cited as grounds for doubting the human data.[32]

In another case, reliance on animal experiments rather than epidemiology delayed a full realization that lack of food early in life can harm the brain.[41] During the first quarter of the 20th century, there was considerable interest in the possibility that lack of food during childhood might interfere with the proper development of the brain and therefore affect the later achievement of the individual. Unfortunately, almost all the research was carried out on animals and showed that starving baby or adult rats had no effect on the brain. Not surprisingly, the topic was abandoned and was only resumed in the late 1950s when children with histories of undernutrition were persistently found to underachieve both in school and in formal tests.