

Can Salt Hypothesis Explain the Trends of Mortality from Stroke and Stomach Cancer in Western Europe?

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The salt hypothesis was revived in 1904 by Ambard and Beaujard^[1] and since then, many researches around the world have been studying this aspect.^[2] The salt hypothesis states that salt is necessary for the genesis of essential hypertension.^[3]

Moreover, the mortality rates of stomach cancer and stroke were found to decrease in a similar way over a given time in different countries.^[4] Based on an observation in 1965—that stomach cancer mortality and stroke mortality were strongly correlated—the hypothesis was presented that salt could be involved in the etiology of both the diseases, although by different mechanisms.^[5] While salt intake could have an influence on blood pressure and thus increasing the risk of stroke mortality,^[4] it can also have a caustic influence on the stomach mucosa, stimulating atrophic gastritis leading to stomach cancer.^[6]

We performed a time series analysis in which special attention was paid to cohort patterns.^[7,8] We studied the trends in stroke and stomach cancer in relation to infant mortality rate in birth cohorts born between 1860 and 1939 in 7 low-mortality European countries, observing a general cohort-wise decline in mortality from stroke and stomach

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cancer in all countries and in both sexes.^[7,8] We also projected mortality trends from ischemic heart diseases, stroke, and stomach cancer in 7 European countries up to 2030.^[9-12] A potential question was the possibility of explanation of the observed mortality trends from stroke and stomach cancer with salt hypothesis.

With regard to stroke, it is obvious that salt is important, through its effects on hypertension, which in turn is widely accepted as a major risk factor for stroke mortality; therefore, a salty food can have a substantial impact on stroke mortality, that is, indirectly through hypertension. However, the observed decline in stroke mortality since 1970s is consistent with the start of hypertension control programs in Western Europe rather than changing pattern of salty foods in the population. In fact, the salt hypothesis is not consistent with our results for stroke.

For stomach cancer, although infection with *Helicobacter pylori* is a key risk factor, it is not a sufficient cause for the development of stomach cancer.^[13] Thus, it is important to consider the role of salted food intake in the causal link between *H. pylori* infection and stomach cancer. A review in 2005 suggested that the majority of the geographic

variation in gastric cancer mortality worldwide and in Japan can be explained at the population level by the daily salt intake level.^[14] Therefore, the effect of salt intake is very important and stomach cancer trends can be explained by salt intake levels and its changes.

Although the decline in stomach cancer mortality in Europe suggests a general reduction of salt intake, this alone was not sufficient to result in a decline in stroke mortality. The stroke–salt intake association might then have been modified by other factors, such as increased fat intake and obesity, causing high blood pressure. Further studies are therefore needed to clarify the role of salt intake as a linking factor in stomach cancer and stroke.

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