This communication suggests that various Western diseases are caused by a relative deficiency of essential fatty acids (EFAs), which means a low ratio of EFA to the antagonistic long-chain saturated and trans fatty acids. It is suggested that arachidonic acid, formed from dietary linoleic acid with the aid of pyridoxine and protected by tocopherols, is the most relevant EFA. [The SC® indicates that this paper has been cited in over 260 publications since 1956.]

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There was a reason for publishing this speculative communication in April 1956. It was exactly 10 years after Oxford University's medical advisers had reported that a generous offer from the recently established Wellcome Trust through its chairman, Sir Henry Dale, to establish a Wellcome Institute of Human Nutrition should be refused because "in 10 years' time there will be no human nutritional problems to work on." I told the vice-chancellor I would work for 10 years to try to show that many noninfective Western diseases were caused by a relative deficiency of essential fatty acids (EFAs). But having set up my university department largely at my own expense and twice paid the salaries of the staff myself, I accepted a professorship in the US and published in this communication the speculations I had wanted to investigate, amplifying them the following year in an oration for World Health Day.1 However, the Earl of Woulton, British Wartime Minister of Food, asked me to remain in England to start a central Institute for Human Nutrition, and this we now have.

As a medical student, my possible career as an experimental scientist was ruined because I noticed what I believe is the most challenging fact in medicine: the expectation of life of a middle-aged man in the world has hardly altered since 1841 (or, in the US, since 1900). Yet at that time most middle-aged men died of tuberculosis or pneumonia. The chronic noninfective diseases such as heart disease and cancer were rare, as they tend now to be in Third-World countries. I thought the increase in such diseases must be related to nutrition, and the most likely dietary change was in the type of fats that were eaten.

Immediately after medical qualification in 1937, I went to the US to see the work being done on the recently discovered EFA. During World War II, I was involved with a nutrition organization for the Ministry of Health, but we occasionally found time to estimate EFA and pyridoxine. At the end of the war, I was busy relieving the Dutch famine and then had five teams in Germany. In 1949, in the primitive university department I set up, the brilliant Indian scientist Rama- lingaswami came to work as a postgraduate student on the pathology of EFA deficiency. Later, I had the assistance of Basnayake from Sri Lanka, and we studied deposition of cholesterol in EFA deficiency. Ancel Keys joined us for two years in 1951, at the time when he considered that all fats raised plasma cholesterol equally.

In 1944 I had worked with Eskimos, who had the highest intake of fat in the world but very rich EFA, low plasma cholesterol, and no noninfective Western diseases. So after the closure of the department, I went into the scientific wilderness. Ten years ago, I went back to the Eskimos with Bang and Dyerberg. Then, for 100 days, I lived on an Eskimo diet (only seal, fish, and water), recently described2 as "heroic and foolhardy": people all around the world wanted bits of me to analyse.

I believe that the 1956 paper has been highly cited because it was the first real suggestion that various Western diseases are caused by a relative deficiency of EFA. I have recently reviewed the functions of EFA3 and again speculated about the wide variety of Western diseases that might be caused by a relative deficiency of EFA.4