Letters to the Editor

Is the Mediterranean diet relevant to myocardial infarction? Olive oil consumption versus geographical latitude

From PIET HEIN JONGBLOET

Sirs—The discussion between Bertuzzi et al.1 and Fernández Jarne et al.2 concerning non-fatal myocardial infarction (AMI) and olive oil consumption in Italy and Spain highlighted the controversy. In three of the four Mediterranean case-control studies, two in Italy and one in Greece, any support for a strong inverse relation between olive oil and coronary heart disease (CHD) was lacking; in contrast, an inverse association was reported in Spain.2 This inverse association has been put in the public domain and both research groups agree that this association remains open to discussion.

People who eat large quantities of saturated fat, in the form of butter, cheese, and other milk products, often have one of the lowest rates of cardiovascular disease and too many disturbing paradoxes, such as the so-called French, Italian and Albanian paradoxes, remain vexatious and provocative.3 Life in Mediterranean countries might be protective, but the time has come for a major paradigm shift of ecological differences in health outcomes: not ‘the olive’, i.e. the lifestyle and behaviour might be essential in the causation of cardiovascular heart diseases (CVD), but the geographical location, i.e. ‘where the olive grows’.4 A geographical South-to-North gradient in the prevalence of CHD within countries demands attention, e.g. in Great Britain5 and France,6 and on a still more intricate scale, among countries at different latitudes, such as Italy, The Netherlands and Finland,7 or France versus Northern Ireland.8

A similar geographical gradient has been established in many constitutional diseases and very different countries, e.g. schizophrenia,9 suicide,10 and prostate,11 breast,12 and other cancers.13 This latitude effect in incidence increases away from the Equator, not only in the Northern hemisphere, but also in the Southern hemisphere, as for example, in fatal neural tube defects14 and diabetes type 1.15 All these conditions are characterized not only by this latitude effect, but also by seasonality of birth.16,17 This connection reveals an interesting causal relationship between these conditions and the ovulatory pattern, and in particular to pathological conceptions.

The consistent and predictable relations between the South-to-North gradient and timing of mating seasons in cats,18 non-human19 and human primate populations20,21 indicates seasonality of the ovulatory rate. The so-called seasonally pre-ovulatory hyperpenis gynopathy (SPrOOH)-hypothesis states that optimal or high-quality oocytes coincide with the peaks of this ovulatory pattern, the less-optimal and poor quality ones appearing in the transitional stages between the ovulatory seasons.16,17 The (patho-)physiological processes of oocyte maturation in animals22,23 and the circumstantial evidence of similar phenomena in humans16,17,24 explain these seasonally bound coincidences. The further from the Equator, the stronger the seasonality of the ovulatory pattern and the higher the rate of non-optimal conceptions. Geographical latitude and its intricate relation to seasonality of birth reveals new mechanisms and disease pathways and, in addition, the ability to explain social, geographical, and temporal patterns of disease distribution.16,17

References

Can dietary fatty acids affect colon cancer risk? Reply to Leitzmann and Giovannucci

From ANDRE NKONDJOCK,1,4 BRYNA SHATENSTEIN,2 PATRICK MAISONNEUVE3 and PARVIZ GHADIRIAN4

Sirs—In their commentary entitled ‘Can dietary fatty acids affect colon cancer risk?’ Drs Leitzmann and Giovannucci have critically evaluated our recently published paper on the association between specific fatty acids and the risk of colorectal cancer, and raised some important points. They have clearly justified that diet is not the only source of many fatty acids, so that isolating diet-related fatty acid effects on colon cancer is complex. We agree. In fact, very few nutrients are provided exclusively by diet. Some essential nutrients are synthesized endogenously to a certain extent, in addition to being consumed in foods or supplements.

Drs Leitzmann and Giovannucci have suggested that other factors in dairy products, such as calcium, rather than medium chain fatty acids may have accounted for the inverse associations observed between these fatty acids and colorectal cancer risk. Based on our study, the main sources of medium chain fatty acids among French-Canadians were dairy products, and there is evidence that dietary calcium intake is associated with reduced colorectal cancer risk. It has been hypothesized that the protective effect of calcium could be due to the formation of calcium soaps in the colon, which neutralize the bowel-irritating effect of bile acids.3 It is indeed possible that calcium may have contributed to this effect since a significant inverse association of dietary calcium with colon carcinoma, with approximately 30% risk reduction, was found in this population.4

Another point made by Drs Leitzmann and Giovannucci was that the increased colorectal cancer risk associated with arachidonic acid, which we observed in our study, may have reflected a correlated component of meat intake. There is strong evidence that high consumption of red meat, especially when well done and highly cooked, increases the risk of colorectal cancer, particularly in individuals who also smoke and are genetically susceptible.5 We disagree with their hypothesis, however, since in our study we found that the main arachidonic acid sources were poultry products (white meat), which have not been consistently associated with an increase in colorectal cancer risk.

Drs Leitzmann and Giovannucci have also suggested an alternative explanation for the gender-specific variations observed in our study. Since a higher proportion of ever smokers were likely to be male, they proposed that smoking-related colon cancers among men might have diluted the relative risks associated with specific fatty acid intakes—if these cancers develop through a pathway different from that of fatty acid intake. We addressed this point by adjusting for smoking, which did not substantially change our findings. In addition, we investigated the effect modification by smoking status in both genders. We noted important changes in females, while our findings were unaltered in males, ruling out their proposition. For example, alpha-linolenic acid was associated with a 22% reduction in colorectal cancer risk. Among women who never smoked, this inverse association became stronger and the trend was highly significant (OR = 0.31; 95% CI: 0.13, 0.77; P = 0.002), while no association was observed among those who ever smoked (OR = 1.45; 95% CI: 0.69, 3.04; P = 0.486).

Finally, underlying mechanisms regarding the possible role of specific fatty acids in the aetiology of colorectal cancer have been proposed,6 so that the hypothesis generated by our data, and stressed by Drs Leitzmann and Giovannucci, that diet-related individual fatty acids could affect colorectal cancer is promising and deserves further evaluation.

References