which provided additional substrate for PGE₂ synthesis would also provide additional substrate for the synthesis of prostacyclin, thromboxane, and the leukotrienes. The authors provide no indication as to the effect of increased linoleate intake on the balance of eicosanoids, or of the effects of changes in this balance on the course of peptic ulcer disease.

The proposed hypothesis relies heavily on the epidemiological evidence linking the decline in peptic ulcer disease with the rise in essential fatty acid intake. Their data shows that between 1909/1913 and 1980, there has been a 300% increase in the intake of linoleic acid while during the same period the intake of saturated fatty acids has increased by only 10%. Does this mean that the rise in the incidence of coronary heart disease is not after all associated with a rise in the intake of saturated fats but with a rise in the intake of the polyunsaturated essential fatty acids? This dubious epidemiological evidence must be supported by other data for tentative acceptance of this hypothesis. One would need to know whatever is reportedly seen for the USA as a whole, is also evident with different age-groups, different regions of the USA, for men and women, etc. Equally one would require international comparisons of diet and peptic ulcer disease.

Science proceeds through the integrated processes of conjecture and refutation. In proposing a hypothesis, vagueness should be avoided and precision encouraged. Just as there are rules in refutation there are rules in conjecture, otherwise we may just as well take up astrology.

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Reply

Sir,—We appreciate the opportunity to respond to Dr Gibney’s letter. Dr Gibney questions the concept that long term increase in dietary linoleic acid ingestion could influence cell membrane content of prostaglandin precursors and the generation of prostaglandins.

When linoleic acid is ingested (as a triglyceride) it is absorbed in the jejunum and converted to dihomo-gamma-linolenic acid and arachidonic acid which are then stored as membrane phospholipids in many organs including the stomach and duodenum. When the tissues are stimulated, phospholipases are activated, mobilising the fatty acids and initiating their conversion to prostaglandins.

The literature is replete with reports showing that supplemental feeding of linoleic acid to man or animals does indeed result in increased membrane prostaglandin precursors.¹⁻⁸ A recent report of chronic high linoleic acid feeding in the rat showed enhanced endogenous PGE₂ production, decreased basal acid secretion, and protection against stress ulcerations.⁹ Thus, Dr Gibney’s points expressed throughout the first three paragraphs of his letter are unsubstantiated by published scientific reports and runs counter to published research data.¹⁻⁹

Dr. Gibney’s fourth paragraph raises a point which is important but is not clearly understood. The feeding of linoleic or arachidonic acids on a chronic basis results in storage of the fatty acids in cell membrane phospholipids. The specific type of cells in the tissues—for example, macrophages—can determine the type of prostanoid formed. Thus, linoleic acid feeding does not necessarily result in elaboration of thromboxanes in the gastroduodenal mucosa. Instead, in our experiments we find that arachidonic acid administration induces predominantly the synthesis of PGE₂¹⁰ while linoleic acid administration results predominantly in the synthesis and release of PGE₁¹¹.

In his fifth paragraph, Dr Gibney tries to fault our hypothesis because it does not explain ‘the rise in the incidence of coronary heart disease’. The thrust of this argument is impertinent to our hypothesis. Our paper is about ulcer disease, not coronary heart disease. Furthermore, published scientific data show that the incidence of coronary disease has been decreasing in the USA and that the feeding of linoleic acid lowers serum cholesterol decreases platelet thrombosis, and decreases cardiovascular mortality in human trials.¹² ¹³

The strength of our hypothesis¹⁴ rests on three separate factors. Well established epidemiological data, enumerated in our publication, clearly show that the incidence and the virulence of peptic ulcer disease have decreased over the past few decades. Concomitantly, the dietary ingestion of linoleic acid by the general population has increased dramatically. Because our published experiments have clearly shown that linoleic and arachidonic acids can be converted to prostaglandins which can protect the gastroduodenal mucosa against injury,¹⁰ ¹¹ we proposed that the increase in dietary intake of linoleic acid could account for the decreasing incidence and virulence of peptic disease. Thus, our hypothesis rests on firm biochemical, experimental, and epidemiological finding and is amply supported by well established reports in the literature. Dr Gibney accused us of having an astrological leaning. Our careful reading of his letter leads us to wonder...
Correspondence

whether Dr Gibney does not gaze more at the stars than at the available scientific literature!

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References


Collagenous colitis

sir,—The cause for collagen deposit in the sub-

epithelial basement membrane of the intestinal mucosa is unknown. It does, however, occur in more than one third of untreated patients with adult coeliac disease.1, 2 The increased cell turnover in coeliac disease would therefore argue against ‘the most popular concept’ that ‘cell turnover is reduced, allowing fibrocytes to spend longer in the mature phase, hence producing more collagen and a thicker collagen plate’.3 Furthermore, removal of gluten from the diet results in the disappearance of the collagens deposit in coeliac disease so that the possibility that a dietary or other ingested factor might play a role in collagenous colitis should not be excluded.

The clinical history of many of the reported patients with collagenous colitis3-10 appears identical to those patients with chronic diarrhoea, incapacitating at times, and essentially normal laboratory, radiological and physical findings save a small but significant increase of plasma cells in their jejunal mucosa. They showed a dramatic response to gluten withdrawal from their diets.11 Unfortunately, biopsies were not taken from their normally appearing mucosa on sigmoidoscopy.

Consequently, I am puzzled why, in the reports readily available to me,3-10 no consideration has been given, either in discussion or treatment, of the possibility that a dietary component might be an important factor in these interesting patients.

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References