Hypolactasia and protection against Plasmodium spp infection in Homo sapiens

EDITOR,—Anderson and Vullo’s hypothesis (Gut 1994; 35: 1487–9) is based on the contention that hypolactasia is causally associated with riboflavin deficiency (consequent upon the low levels of flavin ‘nucleosides’ which it is thought to have cumulatively produced a survival advantage against human Plasmodium spp infection. Available evidence (quoted by the authors) indicates, however, that such a deficiency is not necessarily disadvantageous on the metabolism of the malarial parasite.

The overwhelming consensus of current opinion is that the hypolactasia phenotype represents the normal state for Homo sapiens (and all other mammals), and that persistence of the enzyme into adult life in certain groups before ‘artificial’ mass population movements (for example, northern Europeans to the USA and Australia) from a significant survival advantage; by involving malaria as the major ‘driving force’, Anderson and Vullo are thus interpreting the scenario in ‘reverse’ fashion.

Plasmodium spp are extremely ancient organisms; before they infected the earliest mammals, they utilised primitive organisms, including reptiles, birds, etc, as reservoir hosts. The Anderson and Vullo hypothesis therefore implies that they had evolved previously the hypolactasia phenotype in the animal kingdom developed as an exceedingly early protective mechanism against Plasmodium spp, and persistence of the enzyme into adult life only developed very much later in areas where Plasmodium spp was not a major environmental hazard affecting survival. The Hamitic tribes of east, west, and central Africa (most adults possess persistence of the enzyme into their adult life) presumably migrated into northern Africa comparatively recently (within the last few thousand years) — and were hence ‘unprotected’ against Plasmodium spp. How then, did the Hamitic ‘primitives’ survive Plasmodium falciparum infection? I know of no evidence that Hamites are, at the present day, more prone to P falciparum infection compared with Bantu with the exception that they lack certain major genetic disease(s)5,6 (see later!) P vivax infection was certainly a problem in northern Europe until recent times; however, it never caused the widespread acute mortality associated with human P falciparum infection — which probably originated in Africa also comparatively recently.

The approximate parallel outlined in the hypothesis between the geographical areas incorporating the hypolactasia phenotype and those in which major haemoglobinopathies (which clearly possess a selective advantage against P falciparum infection) are comparatively common, is of interest. 5,6 But even here the degree of protection is only partial; riboflavin deficiency has probably exerted a comparatively minimal, and certainly a lesser, effect in terms of selective Darwinian pressure.

Therefore, although this hypothesis seems superficially attractive, I have grave doubts that it explains the human lactase polymorphism more satisfactorily than several of the previous theories.1

G C COOK Hospital for Tropical Diseases, St Pancras Way, London NW1 0BE

3 Cook GC, Kajubi SK. Tribal incidence of lactase deficiency in Uganda. Lancet 1966; i: 725–30.;

In vitro dissolution of cholesterol biliary stones

EDITOR,—We read with great interest the article by Bergman et al (Gut 1994; 35: 1653–8) who reported on in vitro cholesterol gall stone dissolution with methyl-tert-buty1 ether (MTBE) and ethyl propionate in the presence of bile with or without dimethyl-sulphoxide (DMSO). We would like to comment further on the important role of DMSO on stone dissolution.

We have experience with the clinical use of different solvents containing DMSO. Based on previous studies by Montet’s group in which it was shown that the addition of DMSO to MTBE improved the dissolution rate of cholesterol1 or biliary calculi2 we have recently studied the bide duct stone dissolving capacity of multi component solvents in humans.3 In these mixtures, DMSO was present both in solvent 1 (aqueous glycine-NaOH buffer solution of EDTA, sodium deoxycholate and 30% DMSO) and in solvent 2 (DMSO/MTBE 70/30). Solvent mixtures were infused continuously and alternately for 16–24 hours through a naso-biliary catheter placed into the common bile duct over the stone(s). In 20 of 22 patients with pigment or mixed biliary duct stones(s), or both, which were too large to be removed after endoscopic sphincterotomy, a stone free outcome of 22 or good dissolution of stones (11 of 22) was achieved with these solvents. Only mild toxic side effects were seen during treatment. These mixtures containing DMSO proved effective enough to dissolve not only cholesterol but also pigment or mixed stones, or all three.

Our recent in vitro investigation in the field of contact dissolution therapy of bile duct stones provides further evidence on the beneficial effect of DMSO.4

T TAKACS
First Department of Medicine, Albert Szent-Gyorgy Medical University, Szeged, Hungary

3 Takacs T, Hajnal F, Nagy I, Montet JC, Lonovics J. Effective dissolution therapy of bile duct stones with a new multicomponent solvent. European Journal of Gastroenterology and Hepatology 1993; 35: 867–70.;
In vitro dissolution of cholesterol biliary stones.

T Takacs and J C Montet

Gut 1995 37: 157-158
doi: 10.1136/gut.37.1.157-b

Updated information and services can be found at:
http://gut.bmj.com/content/37/1/157.3.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/