Cosmopolitan underwater fauna

SIR—In their interesting letter published in Scientific Correspondence, Smith *et al.*[1] reported their discovery of a chemosynthetic community on a whale carcass, and suggested that the whale's skeleton provided a source of sulphides to fuel their metabolism. But this speculative idea that this chemoautotrophic fauna is similar to hydrothermal vent faunae is not supported by other observations.

The *Beggiatoa* spp. mats described by Smith *et al.*, for example, are ubiquitous on sulphide-rich environments such as intertidal mudflats, anoxic basins and fish farms[2-4], and are not restricted to vents and seeps. Further, of 219 published species of vent fauna, only 11 have been found in other habitats, including carcasses (V.T., unpublished data). The biogeographical patterns of true vent fauna are more likely to be related to seafloor spreading than by a fortuitous mechanism such as via carcasses of large animals.

In any event, the species described by Smith *et al.* are not all found in vents. The described molluscs exploit sulphide availability and are found in many such habitats. A few, like *C. pacifica*, may have the adaptations to enter vents and seeps. The truly endemic vent animals are not found on this carcass. But we do not believe that a few shared features between vent and whale carcass habitats provide evidence for an important dispersal mechanism.

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How secretion is inhibited

SIR—It has been suggested[1] that glucose inhibition of glucagon secretion is mediated by GABA activation of chloride channels in the pancreatic α_2-cells, that the GABA is co-secreted with insulin from the pancreatic β-cells and that any cirumstances in which insulin secretion is stimulated will lead to suppression of glucagon release. This suggestion is an interesting possibility which, however, can hardly explain inhibition by glucose.

My scepticism originates from the physiology of glucose homeostasis. Whereas secretion of blood glucose-lowering insulin begins above 4–5 mM of the sugar, the release of blood glucose-raising glucagon is half-maximally inhibited by such concentrations[2]. The postulated glucose-induced co-secretion of GABA with insulin would consequently start in a concentration range where glucagon secretion is only marginally affected by the sugar.

An alternative explanation for glucose inhibition of glucagon release is a sugar-induced lowering of cytoplasmic Ca^{2+} caused by intracellular sequestration and outward transport[3,4]. Although the glucose dependence of this phenomenon has yet to be elucidated, it is noteworthy that a similar glucose-induced lowering of cytoplasmic Ca^{2+}, which is a component in the action of the sugar on the pancreatic β-cell, is half-maximal at 6 mM glucose[5].

A final comment concerns the citation of previous work. The original detection of GABA within the pancreatic islets was not made by Okada *et al.*[6], although the authors probably believed this at the time. But the discovery was actually made four years earlier in two independent laboratories[7,8].

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Diacylglycerol as messenger

SIR—Smith *et al.*[1] recently reported that the secretagogue glucose decreases the voltage threshold for activation of L-type Ca^{2+} channels in insulin-secreting cells. This finding nicely confirms, in normal rat pancreatic β-cells, results obtained with the carbohydrate secretagogue glyceraldehyde on the rat insulinoma cell line RINm5F (ref. 2). But it provides no information on the nature of the metabolic product supposed to stimulate the channels.

It is well established that Ca^{2+} channel function can be modulated by cyclic AMP-dependent phosphorylation[3], but carbohydrate stimuli have little or no effect on cyclic AMP levels in insulin-secreting cells[4]. On the other hand, it is known that glucose and glyceraldehyde evoke rapid *de novo* synthesis of diacylglycerol in normal pancreatic β-cells[5] and insulinoma cells[6].

Since the cell-permeable diacylglycerol analogue didecanoylglycerol has been shown to mimic the effect of carbohydrate secretagogues on gating of single L-type Ca^{2+} channels in insulinoma cells[7], diacylglycerol may fulfil the required messenger role. Diacylglycerol could work by protein kinase C-mediated phosphorylation, or by an as yet unknown mechanism[8].

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Copper-bottomed earwax

SIR—The unexplained liking of several cats for human ear wax (*Nature* **343**, 220; 1990) is a reminder that pica, a desire for unusual food, can sometimes be traced to dietary deficiency, such as a lack of iron[1]. Cerumen (ear wax) has a high copper content[2], which may relate to its bactericidal and mycocidal properties[3]. Copper occurs in most tissues of the animal body, and minute amounts are necessary for the incorporation of iron into haemoglobin[4]. In the cytochrome system, the function of copper is similar to that of iron. Copper requirements vary among species, and are influenced by the intake of other mineral elements such as iron and molybdenum[5]. If there is a copper deficiency, anaemia is a common sequel[4].

It may be that some domestic animals experience a copper-deficient diet, leading them to seek supplementary sources of this mineral.

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