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 faunas 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Further, of 219 published species of vent fauna, only 11 have been found in other habitats, including carcasses (V.T., unpublished data).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.

VERENA TUNNICLIFFE

Department of Biology,
University of Victoria,
British Columbia, Canada V8W 2Y2

S. KIM JUNIPER

Department of Oceanography,
University of Quebec,
Rimouski, Canada G5L 3A1

1. Smith, C.R. *et al.* *Nature* **341**, 27–28 (1989).
2. Jorgensen, B.B. *Phil. Trans. R. Soc.* **B298**, 543–561 (1982).
3. Juniper, S.K. & Brinkhurst, R.O. *Mar. Ecol. Prog. Ser.* **33**, 41–50 (1986).
4. Grant, J. & Bathman, U.V. *Science* **236**, 1472–1474 (1987).

How secretion is inhibited

SIR—It has been suggested¹ 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 circumstances 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 concentrations². 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 glucose⁵.

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}.

ERIK GYLFE

Department of Medical Cell Biology,
University of Uppsala,
Biomedicum Box 571,
S-751 23 Uppsala, Sweden

1. Rorsman, P. *et al.* *Nature* **341**, 233 (1989).
2. Gerich, J.E. *et al.* *Rev. Physiol.* **38**, 353 (1976).
3. Johansson, H. *et al.* *Biochem. Biophys. Res. Commun.* **147**, 309 (1987).
4. Johansson, H. *et al.* *Cell Calcium* **10**, 205 (1989).
5. Gylfe, E. *J. Biol. Chem.* **263**, 13750 (1988).
6. Okada, Y. *et al.* *Science* **194**, 620 (1976).
7. Briel, G. *et al.* *Acta physiol. Scand.* **84**, 247 (1972).
8. Panten, U. *et al.* *FEBS Lett.* **20**, 225 (1972).

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 phosphorylation³, but carbohydrate stimuli have little or no effect on cyclic AMP levels in insulin-secreting cells⁴. On the other hand, it is known that glucose and glyceraldehyde evoke rapid *de novo* synthesis of diacylglycerol in normal pancreatic β -cells⁵ and insulinoma cells⁶.

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 cells⁷, diacylglycerol may fulfil the required messenger role. Diacylglycerol could work by protein kinase C-mediated phosphorylation, or by an as yet unknown mechanism⁸.

O. H. PETERSEN

MRC Secretary Control Research Group,
Physiological Laboratory,
University of Liverpool,
PO Box 147, Liverpool L69 3BX, UK

C. B. WOLLHEIM

Institut de Biochimie Clinique,
Centre Medical Universitaire,
Universite de Geneve,
1211 Geneve 4, Switzerland

1. Smith, P.A., Rorsman, P. & Ashcroft, F.M. *Nature* **342**, 550–553 (1989).
2. Velasco, J.M., Petersen, J.U.H. & Petersen, O.H. *FEBS Lett.* **231**, 366–370 (1988).
3. Reuter, H. *News physiol. Sci.* **2**, 168–171 (1987).
4. Prentki, M. & Matschinsky, F.M. *Physiol. Rev.* **67**, 1185–1248 (1987).
5. Peter-Riesch, B., Fathi, M., Schlegel, W. & Wollheim, C.B. *J. clin. Invest.* **81**, 1154–1161 (1988).
6. Wollheim, C.B. *et al.* *EMBO J.* **7**, 2443–2449 (1988).
7. Velasco, J.M. & Petersen, O.H. *Q. J. exp. Physiol.* **74**, 367–370 (1989).
8. Kolesnick, R.N. *J. Biol. Chem.* **262**, 16759–16762 (1987).

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 iron¹. Cerumen (ear wax) has a high copper content², which may relate to its bactericidal and mycotoxic properties³. Copper occurs in most tissues of the animal body, and minute amounts are necessary for the incorporation of iron into haemoglobin⁴. 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 molybdenum⁵. If there is a copper deficiency, anaemia is a common sequel⁶.

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

PATRICK M.P. O'CONNOR

Department of Radiology,
Albany Medical Center Hospital,
Albany, New York 12208, USA

1. Reynolds, R.D. *et al.* *Ann. int. Med.* **69**, 435–440 (1968).
2. Yassin, A., Mostafa, M.A. & Moawad, M.K. *J. laryngol. Otol.* **80**, 933–937 (1966).
3. Megarry, S. *et al.* *J. laryngol. Otol.* **102**, 671–672 (1988).
4. Jones, C.J. & Hunt, R.D. in *Veterinary Pathology* 1066–1068 (Lea & Febiger, Philadelphia, 1983).
5. Hays, V.W., Swenson, M.J. in *Dukes' Physiology of Domestic Animals* (ed Swenson, M.J.) 395–412 (Comstock, Ithaca, 1977).