Carbon cycle

Surprise from the shallows

from M. Whitfield

A RARE new insight into the workings of the carbon dioxide cycle is offered by the paper of Byrne *et al.* on page 321 of this issue and its companion, from the same group, which is published in this week's *Science* (226, 1074; 1984). In suggesting, from observations in the North Pacific, that the flux of calcium carbonate shells from the surface layers into the deep oceans has been grossly underestimated, these papers imply a significant shift in our understanding of the oceanic carbon dioxide system.

From an analysis of the particles that settled in large conical traps, deployed at various depths for only a day or so, Byrne and his colleagues were able to show for the first time that pteropod shells, which are made of aragonite — the most soluble form of oceanic calcium carbonate — contribute a flux of calcium carbonate to the deep ocean which is at least as great as that provided by the calcite shells of the foraminifera and coccolithophorids. Furthermore they show that, unlike calcite shells which fall into the sediment and then redissolve only slowly, most of the pteropod shells dissolve in the water column. Earlier measurements had been inconclusive; tow-net sampling had allowed no estimate to be made of the settling rate of pteropods, and long-term sediment traps had inadequately registered the presence of pteropods because their shells had dissolved in the weeks or months before the traps were recovered.

As the shells settle out of the surface layers into deeper water, they resist progressively more corrosive conditions until reaching their lysocline, where they begin to dissolve. Typical lysocline depths for the North Atlantic are 3.2 km for aragonite and 4.6 km for calcite. For the older and more acidic deep waters of the North Pacific, the lysocline depths are typically 0.6 km for aragonite and 0.8 km for calcite. Although calcium carbonate dissolves at and below the lysocline it can still be found in sediments down to the compensation depth, where rates of settling and dissolution just balance.

For each mole of calcium carbonate dissolved, one mole of carbon dioxide is converted into bicarbonate (resulting in an increase in oceanic alkalinity). The new findings make it possible to establish a realistic alkalinity balance for the oceans. Moreover they suggest that the oceans might act as a more rapid and effective sink for fossil-fuel carbon dioxide than had been thought possible when attention was focussed on the dissolution of calcite from the ocean sediments. In that case the response time is measured in centuries or millenia because of the slow circulation of

the carbon dioxide-rich waters down to the compensation depth. To dissolve a significant amount of calcium carbonate, these acidic waters must penetrate the sediments, still further slowing down the process.

The new observations indicate that 5-20 per cent of the smaller pteropod shells will have dissolved by the time they have reached a depth of 1,000 m. This dissolution provides a ready explanation for the mid-water alkalinity maximum that is observed in North Pacific waters substantially supersaturated with respect to calcite, which has previously been ascribed to the possible, but rather unlikely, long-distance transport of high alkalinity water from the edges of the ocean basin.

To close the circle, it is also necessary to consider the enhancement of shell dissolution by the increasing oceanic concentration of carbon dioxide that is resulting from the burning of fossil fuel. The new observations make it clear that this carbon dioxide has only to penetrate to the aragonite lysocline for it to have an effect. Model calculations suggest that it has

already reached a depth of 300 m — close to the aragonite lysocline in the North Pacific — as the result of the slow downward mixing of surface waters. If the dissolution of pteropod shells in relatively shallow waters is enhanced in this way, there will be a corresponding reduction in the alkalinity of the deeper water. In turn, this will accelerate the dissolution of the calcite shells and the larger aragonite shells in the sediment. Furthermore, as the falling shells begin to dissolve, their rate of fall will be reduced and hence their dissolution will become progressively more complete.

On a global basis, this process could accommodate up to one fifth of the current flux of fossil-fuel carbon dioxide and would therefore be much more important than the dissolution of shallow-water, magnesium-rich calcites that has previously been considered the best candidate to account for any rapid response. As oceanic carbon-cycle models become sufficiently sophisticated to incorporate biological processes in a realistic fashion, it will be instructive to see to what extent the oceanic uptake of fossil-fuel carbon dioxide will be influenced by the shift in the alkalinity balance implied by the flux of pteropod shells to the deep ocean and by their tendency to dissolve in transit. □

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Cell biology

A spectrum of spectrins

from A. J. Baines

SOME two years ago I alluded in these columns to the discovery of analogues of the red cell membrane skeleton protein, spectrin, in unexpected places¹. Since then they have been looked for and found — at least by the somewhat rudimentary technique of immunological cross-reaction — in almost every kind of cell that the imagination of cell biologists can run to.

The outsider, trying to follow the trail of discovery, is apt to be put off the scent by a multiplicity of terminologies. Not only are several names (tissue spectrin, caldesmon, and fodrin) still in use for the spectrin-like proteins of non-erythroid cells, but we now have multiple names for their subunits too. For instance, the smaller subunit of brain spectrin, because it binds to the membrane-associated retaining protein, ankyrin, and hybridizes with the larger, alpha subunit of either brain or red cell spectrin, is sometimes termed beta by analogy with the erythrocyte beta chain (for example ref. 2). On the other hand, its electrophoretic mobility in SDS differs from that of erythrocyte beta chain and peptide maps of the two are quite distinct, so what seems to be the same peptide turns up elsewhere under the designation gamma (for example, ref. 3). Ultimately, the relationship between these polypeptides will need to be resolved by

direct comparison of sequences, using that of the greater part of the red cell spectrin molecule⁴ as the standard.

So what does spectrin do in different cells? In parallel with its tissue distribution, some indications of the cellular functions in which it may be involved have accrued. Originally it was supposed that spectrin and its associated proteins, which together make up the red cell membrane skeleton, would perform much the same function in, say, a brain cell as they would in a red cell. Thus it was conceived that a plasma membrane needed a supporting cytoskeletal apparatus to prevent its disintegration, although, to be sure, no brain cell has to withstand the rigours endured by the red cell in circulation.

Complicating this attractively simple model has been the discovery that tissue spectrins are able to enter into associations with proteins not present in the mature red cell, as well as being able to bind protein 4.1, actin, ankyrin and calmodulin. They can bundle microtubules⁵ and bind to at least one of the microtubule-associated proteins (tau)⁶, activities which may be reflected in the antigenic cross-reactivity of spectrin with MAP II, another microtubule-associated protein⁷. Brain ankyrin also binds to microtubules⁸, although in