more than four-fold by the sickling of Hb SS red cells. In contrast, deoxygenation had no effect on calcium leak into normal red cells. Control studies on samples from patients with elevated (>7%) reticulocyte counts revealed normal calcium flux rates (data not shown). Most of the uptake of $^{45}Ca^{2+}$ by sickle red cells probably represents exchange of unlabelled intracellular calcium for labelled extracellular calcium but three samples of deoxygenated Hb SS red cells, incubated for 1.5 h at 37° C, were found to accumulate an average of 50 μM Ca^{2+} l^{-1} of packed cells. No net accumulation was detectable in three control samples from normal individuals. This calcium accumulation in Hb SS erythrocytes was equivalent to a 25% increase in total cellular calcium.

We have found that Hb SS erythrocytes contain approximately eight times as much calcium as do erythrocytes from normal individuals, that ISC appear to contain approximately twice as much calcium as do Hb SS cells of normal morphology and that Hb SS red cells display markedly increased permeability to calcium, which is further enhanced by deoxygenation and sickling of these erythrocytes. This evidence indicates that the leakiness of Hb SS red cells leads to an abnormal accumulation of intracellular calcium. This, perhaps combined with progressive metabolic depletion, could explain the diminished filterability of oxygenated Hb SS red cells which has been reported by several investigators[13–16], and is likely to be critically involved in the shortened survival of Hb SS red cells. LaCelle *et al.*[17] have proposed that increased calcium and decreased ATP concentrations cause the stiffening of the erythrocyte membrane which characterizes normal senescent red cells. Membrane stiffening, in turn, would lead to sequestration and destruction of erythrocytes in reticuloendothelial organs such as the spleen and liver. A similar, though much more rapid, series of events seems to underlie the greatly accelerated red cell destruction in haemoglobin SS disease. In addition, marked calcium accumulation may be involved in the formation of ISC *in vivo*.

We suggest, as have others[8], that calcium alters the sol-gel characteristics of the red blood cell membrane, favouring the latter. In Hb SS erythrocytes, an excessive accumulation of membrane calcium, occurring during sickling, probably "fixes" the membrane in the sickled shape, thereby producing the ISC.

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Erratum

IN the article "Accumulation of Fossil CO_2 in the Atmosphere and the Sea" by A. W. Fairhall (*Nature*, **245**, 20; 1973), Figs 1 and 2 of the original manuscript were substituted for the corresponding figures of the revised version. As far as Fig. 2 is concerned, the differences are small, but Fig. 1 (revised) contained data which were essential to the discussion. The correct figure appears below. Also line 2 of page 22 should read, "$[n_a^{t'} - n_a]/[N_a + n_a(t')]$, expressed as a percentage where $t' =$".

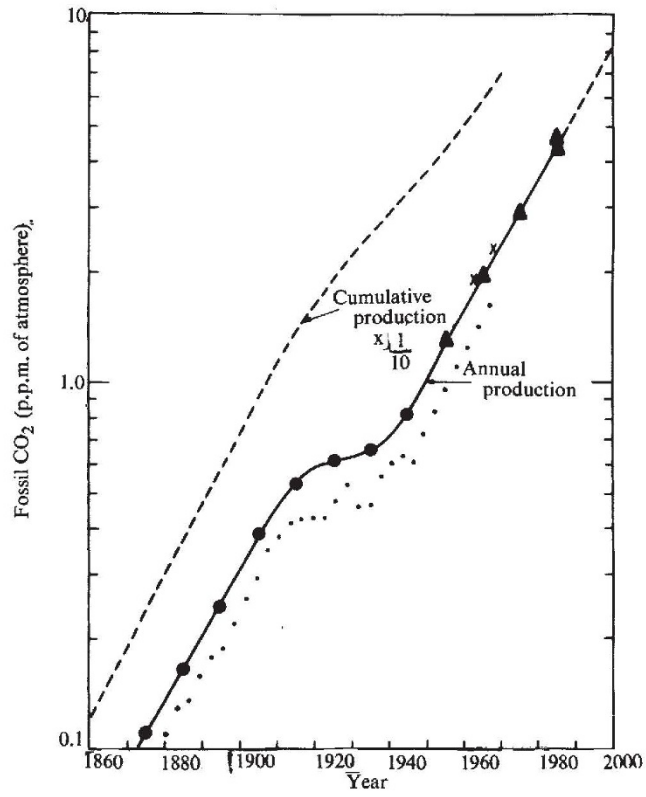

Fig. 1 Production of fossil CO_2 since the 1870s: the solid circles are the estimates of Revelle and Suess[10] extended to 1890 by estimates of OECD, which along with the data from the 1960s are from ref. 11. The dots are estimates of Keeling[12]. The dashed line is the cumulative production to the present day based on the Revelle–Suess production curve.

The author also wishes to take this opportunity to add the following information:

Using a simple box model for the atmosphere, biosphere, mixed layer of the sea, and the deep sea, and a logistic-curve representation of future fossil CO_2 emissions, Zimen and Altenhein[1,2] have reached substantially the same conclusions as myself. In their analysis the calcite undersaturation of the sea would be reached before the turn of the century.

1 Zimen, K. E., and Altenhein, F. K., *Naturwissenschaft*, **4**, 198 (1973).
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