or no Bohr effect does so in the presence of large excess of IHP or ATP.

We examined the tadpole haemoglobin to determine whether the Bohr effect was exerted within the erythrocyte. Oxygen equilibrium curves were determined as described above as well as with the opal glass method[13]. The tadpole and frog erythrocytes were suspended in 50% sucrose medium and in 10% glucose medium to retain the intracellular ATP level as high as possible. The Bohr effect was absent in the tadpole erythrocyte, however, contrary to the frog erythrocyte, which agreed with our results with the haemolysates, and implied physiological absence of the Bohr effect *in situ*. Manwell[14] found similar results with erythrocytes of *Rana clamitance*

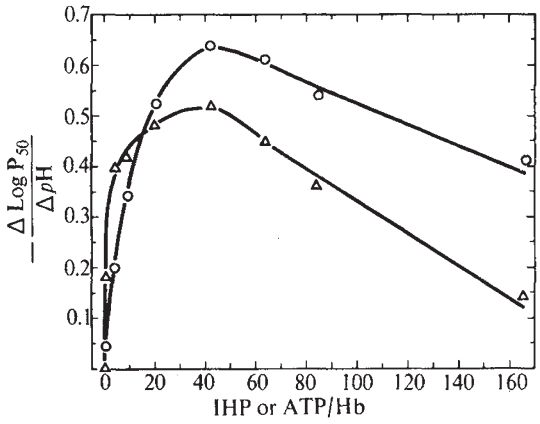

Fig. 3 The Bohr effect of tadpole haemoglobin as a function of the molar ratio of ATP and IHP to haemoglobin, measured between *p*H 7.0 and 7.5 at 24°. ○, Haemoglobin with ATP; △, haemoglobin with IHP.

tadpole. Intracellular level of the organic phosphates reported previously[6] also supports this result; as the molar ratio of ATP and IHP to the haemoglobin was calculated to be 1.2 and 0.09, respectively, the organic phosphate would be too low to decrease oxygen affinity and it was not surprising that there was no Bohr effect even in the erythrocyte. We do not know the physiological meaning of this latent Bohr effect of tadpole haemoglobin but suggest that this may be an intermediate step in haemoglobin evolution before it acquires the apparent Bohr effect.

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1 McCutcheon, F. H., *J. Cell. Comp. Physiol.*, **8**, 63 (1936).
2 Riggs, A., *J. Gen. Physiol.*, **35**, 23 (1951).
3 Hamada, K., Sakai, Y., Shukuya, R., and Kaziro, K., *J. Biochem.*, **55**, 636 (1964).
4 Yoshioka, M., Hamada, K., Okazaki, T., Kajita, A., and Shukuya, R., *J. Biochem.*, **63**, 70 (1968).
5 Frieden, E., in *Metamorphosis* (edit. by Etkin, W., and Gilbert, L. I.), 349 (Appleton-Century-Crofts, New York, 1968).
6 Araki, T., Kajita, A., and Shukuya, R., *Biochem. Biophys. Res. Commun.*, **43**, 1179 (1971).
7 Benesch, R. E., Benesch, R., and Yu, C. I., *Biochemistry*, **8**, 2567 (1969).
8 Tomita, S., and Riggs, A., *J. Biol. Chem.*, **246**, 547 (1971).
9 Gillen, R. G., and Riggs, A., *Comp. Biochem. Physiol.*, **38B**, 585 (1971).
10 Allen, D. W., and Wyman, J., *J. Cell Comp. Physiol.*, **39**, 383 (1952).
11 Antonini, E., Rossi-Fanelli, A., and Caputo, A., *Arch. Biochem. Biophys.*, **97**, 336 (1962).
12 Riggs, A., *Proc. US Nat. Acad. Sci.*, **68**, 2062 (1971).
13 Shibata, K., in *Methods of Biochemical Analysis* (edit. by Glick, D.), **7**, 77 (Interscience Publ., New York, 1959).
14 Manwell, C., *Comp. Biochem. Physiol.*, **17**, 805 (1966).

Erratum

In the article "Transient Virus Infection : Spontaneous Recovery Mechanism of Lymphocytic Choriomeningitis Virus-infected Cells" by J. Hotchin (*Nature New Biology*, **241**, 270; 1973), paragraph 2, line 12, should read ". . . multiplicity of infection was < 1".

A new paragraph should have been inserted after paragraph 3 as follows:

"Monolayers of freshly infected L cells, prepared as above, were seeded onto cover slips which were sampled at intervals of 8 h for several days. The percentage of cells showing specific immunofluorescence rose to about 95% by 48 h and then fell to between 1 and 10% by day 4 or 5 where it remained for many subcultures as a persistent infection. The 90% balance of 'shutdown' non-fluorescent cell monolayers was repeatedly adsorbed with high titre virus seed in attempts to reinfect them. Subsequent 8 hourly sampling and immunofluorescent assay showed that exposure to virus did not increase the percentage of fluorescent cells. It was concluded that for an unknown period after shutdown, the cells were refractory to reinfection with LCM virus. At this time the shutdown cells were fully susceptible to vesicular stomatitis virus (VSV) and supported normal yields of plaques with this virus, but no plaques could be produced with LCM virus. The absence of resistance to VSV eliminates interferon as the cause of the LCM-refractory phase."

The following should also have been added to paragraph 9: "The clones, which had been subcultured into Petri dish monolayers, were identical to the original uninfected L cells in all respects. Virus yield and antigen as judged by titre, plaque size and degree of immunofluorescence developed with the same kinetics as in control L-cell cultures. Control clones, never exposed to the virus, behaved identically after infection."

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