In a similar manner we have derived the following equation for the ratio ($C^{14}O_2$ from 6-C^{14}-glucose/$C^{14}O_2$ from 1-C^{14}-glucose), which we shall designate W

$$W = \frac{(E/6R)}{(E/6R) + (1 - E)}, \text{ whence}$$

$$E = \frac{6RW}{1 + W(6R - 1)} \quad (2)$$

Again, using the experimental values of Bloom, *et al.*,[2,3] for R and W, namely, 2 and 0.3, respectively, we calculate that 84% of the CO_2 derived from glucose was formed glycolytically, a value in good agreement with that obtained from our equation (1).

In our experiments with rat liver slices, the observed ratio $U = a/b$ was higher than that reported by Bloom, *et al.*,[2] and varied between 1 and 1.8. However, these workers added lactate, acetate and gluconate, in high concentrations, to their media. Under such conditions, as shown in Table I, $C^{14}O_2$ formation from glucose is depressed, and the relative importance of the hexose monophosphate shunt is increased.

TABLE I

250-mg. rat liver slices incubated with 2.5 ml. of Krebs–Ringer bicarbonate buffer containing 55 μmoles of labeled glucose. The addition was 50 mm. each of lactate, acetate and gluconate. Gas phase 95% O_2 + 5% CO_2; incubated for 3 hours at 37°.

Label in glucose	Addition		% of C^{14} in $C^{14}O_2$	$U = a/b$	$E^a \times 100\%$
even-C^{14}	None	a	2.8		
				1.2	94
1-C^{14}	None	b	2.3		
even-C^{14}	+	a	0.8		
				0.6	79
1-C^{14}	+	b	1.4		

[a] These values of E are calculated from our equation (1) using the value of $R = 2$ determined experimentally by Bloom, *et al.*[2]

Thus, in rat liver slices, over 90% of the CO_2 is derived from glucose *via* glycolysis, and even under the special conditions of Bloom, *et al.*,[2,3] about 80% of the CO_2 is formed glycolytically.

DEPARTMENT OF PHYSIOLOGY J. KATZ
UNIVERSITY OF CALIFORNIA S. ABRAHAM
SCHOOL OF MEDICINE R. HILL
BERKELEY, CALIFORNIA I. L. CHAIKOFF

RECEIVED JANUARY 25, 1954

IDENTIFICATION OF A FOURTH ABNORMAL HUMAN HEMOGLOBIN

Sir:

In addition to normal adult (A) and fetal (F) hemoglobins three abnormal forms (S, C and D) of human hemoglobin have been described.[1,2] We wish to report the identification of a fourth abnormal form in the erythrocytes of a child (M. M.) with an atypical anemia. Filter paper electrophoresis of hemoglobin from this individual in 0.01 M sodium barbital, pH 9.2, at room temperature revealed two components, one with the mobility of hemoglobin F and the other with a mobility very nearly that of hemoglobin C. The same result was obtained by moving boundary electrophoresis in 0.01 M Na_2HPO_4, pH 8.8, at 1.4°. Moving boundary electrophoresis in cacodylate buffer of ionic strength 0.1 and pH 6.5 at 1.4° showed a component with the mobility of hemoglobin F and a component with a mobility greater than that of hemoglobin A but slightly less than that of sickle cell (S) hemoglobin. The mobilities of hemoglobins A, S and C in this buffer are, respectively, 2.4, 2.9 and 3.2 × 10^{-5} cm.^{2} sec.$^{-1}$ volt^{-1}.[1] The component having the mobility of hemoglobin F comprised 41% of the total by electrophoretic analysis. This component was isolated from the ascending limb of a moving boundary experiment in 0.01 M Na_2HPO_4; its ultraviolet absorption spectrum was found to be that of hemoglobin F. The ultraviolet spectrum of the original specimen did not differ significantly from that of a comparable mixture of hemoglobins A and F.[3] When the original specimen was partially denatured with dilute sodium hydroxide, the amount of alkali-resistant hemoglobin recovered corresponded to the amount of the fetal electrophoretic component in the specimen. The alkali-resistant component was found to have the ultraviolet spectrum of hemoglobin F. The non-fetal component, isolated from the ascending limb of the pH 6.5 moving boundary experiment, had the color and visible spectrum of hemoglobin. The solubility of the original specimen as amorphous ferrohemoglobin in 2.58 M phosphate buffer of pH 6.8 at 25° was 1.94 g. per liter, a value similar to those of mixtures of hemoglobins A and F under the same conditions.[4] We conclude from these results that the hemoglobin specimen examined consisted of a mixture of hemoglobin F and a hitherto undescribed abnormal human hemoglobin, which we shall call hemoglobin E. It differs from all of the previously described forms in its electrophoretic behavior. Its absorption spectrum, solubility and lability to alkali denaturation are similar to those of normal adult hemoglobin. A detailed account of this work will be published later.

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(4) H. A. Itano, *Arch. Biochem. Biophys.*, **47**, 148 (1953).

GATES AND CRELLIN LABORATORIES OF CHEMISTRY
(CONTRIBUTION NO. 1889)
CALIFORNIA INSTITUTE OF TECHNOLOGY
PASADENA 4, CALIFORNIA
CHILDREN'S HOSPITAL, AND THE HARVEY A. ITANO
DEPARTMENTS OF BIOCHEMISTRY AND OF PEDIATRICS
UNIVERSITY OF SOUTHERN CALIFORNIA W. R. BERGREN
LOS ANGELES, CALIFORNIA PHILLIP STURGEON

RECEIVED MARCH 22, 1954

REACTION OF BIS-(CYCLOPENTADIENYL)-TITANIUM DICHLORIDE WITH ARYLLITHIUM COMPOUNDS[1]

Sir:

Bis-(cyclopentadienyl)-iron was first described[2] in 1951. Since then, analogous complexes of a number of other transition elements have been prepared.[3] The structure of the iron complex has

(1) This work was carried out under Contract Nonr-582(00) with the Office of Naval Research.
(2) T. J. Kealy and P. L. Pauson, *Nature*, **168**, 1039 (1951).
(3) (a) G. Wilkinson, THIS JOURNAL, **74**, 6146, 6148 (1952); **76**, 209 (1954); (b) G. Wilkinson, P. L. Pauson, J. M. Birmingham and F. A. Cotton, *ibid.*, **75**, 1011 (1953).