readily outlined, and will be described elsewhere.

The efficiency of the redox pump. Franck and Mayer (*11*) in their theoretical development of the "osmotic diffusion pump" found that under the most favorable conditions there would be about a 30% efficiency, and a concentrating power of about 1.3 times, higher concentration ratios being possible with a layered series.

For the active secretion of H ions by the redox pump it will be seen from the above treatment that the immediate efficiency can approach 100%. Insofar as the electrons must be carried to some final acceptor, free energy may be lost in this process, and the overall efficiency much reduced. At the same time, the energy change involved in the further passage of the electrons could be negligible or utilized in another but quite different system.

Apart from its possible very high efficiency, the most attractive feature of the "redox pump" is the fact that the active carrier and the energy source are one and the same system.

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Effect of Aureomycin on the Respiration of Normal Rat Liver Homogenates

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Recently Loomis (*1*) published a note to the effect that aureomycin specifically depresses phosphorylation without inhibiting respiration of normal mitochondria. Concurrently a study of the effect of aureomycin on enzyme systems of whole rat liver homogenates was in progress in this laboratory. In the course of this study it was found that the addition or omission of certain components in the basal medium of the system profoundly influenced the oxygen consumption in the presence of aureomycin. The basal medium used was essentially that of Pardee and Potter (*2*), with minor changes.

The most marked effect was caused by the omission of citrate from the medium (Fig. 1). Without citrate

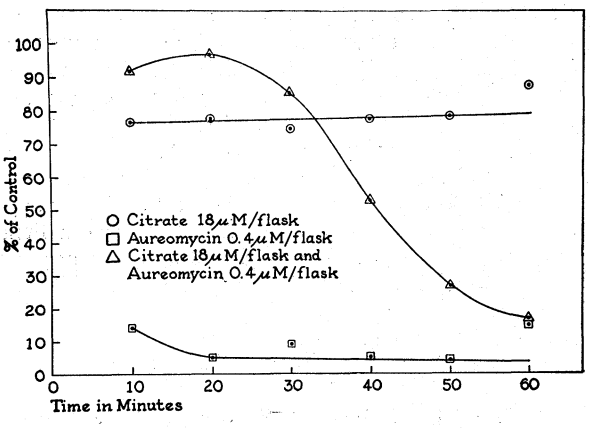

FIG. 1. Inhibition of respiration of rat liver homogenates by aureomycin.

in the presence of aureomycin, respiration is virtually brought to a halt within the first 10 min of the incubation. By contrast in the presence of citrate, the rate of oxygen consumption does not start to decline until after 30 or more min of incubation. An additional 30 min must elapse before the oxygen uptake approaches the level of the citrate-free aureomycin preparation.

This suggests that a possible mode of action of aureomycin may be through blocking some part of the Krebs cycle. Studies to determine the probable sites of action are in progress in this laboratory.

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A Synthesis of 2-Desoxy-D-Ribose[1]

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Recently we synthesized the 2-desoxy-D-ribose by the following route: Glucose → calcium gluconate → D-arabinose → D-acetobromoarabinose → D-diacetylarabinal → D-arabinal → 2-desoxy-D-ribose.

2-Desoxy-D-ribose.—D-arabinal was prepared according to Karrer and Becker (*1*), mp 81°.

$$[\alpha]_D^{22^\circ} = \frac{+3.92^\circ \times 100}{1 \times 2} = +196^\circ \text{ (in water)}.$$

1.1 g of crystalline D-arabinal was dissolved in 18.3 ml of ice-cold 1.0 *N* sulfuric acid, and the solution was allowed to stand at 0°. It gradually became faintly yellow, and after 2½ hr a faint turbidity occurred, accompanied by a flocculent precipitate. At this time the solution was soon neutralized with barium hydroxide and finally with barium carbonate. After removing the precipitate and barium carbonate, the clear and less colored filtrate was concentrated to a thick syrup under reduced pressure without heating.

[1] Aided by a grant from the Scientific Research Fund of the Department of Education.

Then we took up the syrup with absolute alcohol and treated it with about twice its volume of dry ether. The resulting precipitate was filtered and the filtrate was evaporated in a vacuum desiccator over phosphorus pentoxide and calcium chloride.

From the thick syrup after several days the 2-desoxy-D-ribose was obtained in crystalline form. This was washed with a small amount of cold *n*-propyl alcohol, mp 80°. The yield of the sugar was a little more than 0.3 g. The sugar gave a strongly positive Dische and Kiliani test. Its specific rotation was as follows:

$$[\alpha]_D^{22^\circ} = \frac{-1.13^\circ \times 100}{1 \times 2} = -56.5^\circ \text{ (in water)}.$$

Benzylphenylhydrazone.—0.3 g of 2-desoxy-D-ribose was dissolved in 1.5 ml of *n*-propyl alcohol, and 0.39 g of freshly distilled benzylphenylhydrazine was then added. After 3 hr in a desiccator over calcium chloride, the mixture had changed to a crystalline mass. It was washed with ether and recrystallized from *n*-propyl alcohol. Yield, 0.4 g. The substance melted at 129° (uncorrected).

$C_{18}H_{22}O_3N_2$ (314.2) Calc, N 8.92; found, N 8.94.

Its specific rotation was

$$[\alpha]_D^{22^\circ} = \frac{-0.33^\circ \times 100}{1 \times 2} = -16.5^\circ \text{ (in pyridine)}.$$

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The Effect of Sensitization and X-Radiation on the Metabolism of I^{131} Labeled Proteins[1]

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Various methods of tracing antigens in animal tissues have been employed since Metchnikoff's first titrations (1897) of tetanus toxin localized in mouse tissues (*1*). Among other nonradioactive agents that have been used as tracers are arsenic-proteins (*2*), iodinated serum (*3*), colored antigenic proteins prepared by azo conjugation (*4–7*), and fluorescent antibodies prepared by isocyanate conjugation (*8*). Since the introduction of radioactive isotopes into medical research, several such compounds have been so employed. Tobacco mosaic virus tagged with P^{32} has been traced in mice (*9*), I^{131}-tagged antibody has been traced in rats and mice (*10*), and I^{131}-tagged proteins have been traced in guinea pigs (*11*). The radioactive isotopes lend themselves extremely well to quantitative work and to studies of tissue localization by radioautography.

[1] This work was supported by grants from the Atomic Energy Commission and from the U. S. Public Health Service.
[2] The authors wish to acknowledge the capable technical assistance of Mary C. Johnson and Maria P. Deichmiller.

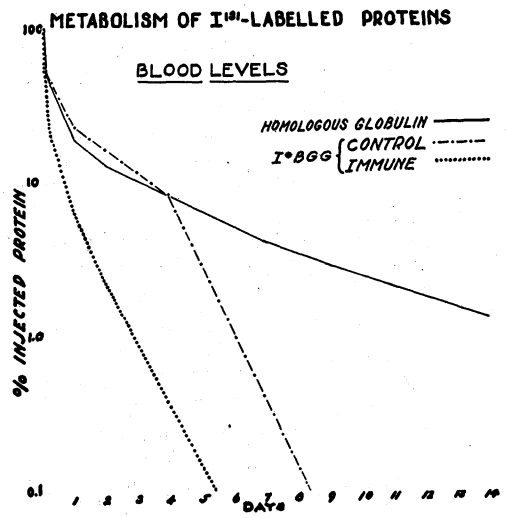

FIG. 1. Elimination curves for I*BGG in normal control and immune rabbits and homologous globulin (I*RGG) in normal control rabbits.

In particular the I^{131} label is desirable because:

1. It can be attached to proteins in traceable amounts without measurably altering their immunologic specificity.
2. Available evidence indicates that the I*[3] protein bond is a stable chemical link which *in vitro* resists wide changes in temperature and pH, salt exchange dialysis, prolonged storage and enzymatic action (*11, 12*). *In vivo* it appears that the iodine remains attached to the protein as long as the latter is immunologically detectable.
3. Prompt excretion of the I* label liberated by antigen metabolism can be accomplished by iodine prefeeding to saturate the iodine-utilizing tissues.
4. The I* liberated by protein metabolism is not appreciably incorporated either by synthesis or interchange into the rabbit's own proteins as determined by activity measurements of plasma protein fractions after administration of I* labeled homologous globulin. Furthermore, I* injected as inorganic iodide into iodine-prefed animals is rapidly excreted unchanged in the form of iodide. In contrast, the injection of I* attached as a protein label is followed by the excretion of nonprotein organic combinations of I*, probably diiodotyrosine and perhaps other amino acid forms, as well as iodide, suggesting actual protein degradation prior to liberation of the iodine label. This difference in excretion forms was determined by paper partition autoradiograms (*13*).

Labeled proteins were injected intravenously into 2-kg rabbits as follows: Labeled homologous globulin (I* RGG) to normal rabbits; labeled bovine γ globulin (I* BGG) to normal rabbits in amounts of 500, 75, or 1 mg; 75 mg I* BGG to rabbits immunized 20 days earlier by an intramuscular injection of 325 mg bovine γ globulin adsorbed on aluminum hyroxide; 75 mg I* BGG to rabbits, 48 hr following their exposure to 500–600r of 200 kv whole-body x-ray; 10 mg I* BGG to normal mice; and 90 mg I* BGG coupled with diazotized *p*-aminobenzoic acid (P-AP*) to normal rabbits.

All tracer studies were done by β-counts of dried

[3] I* hereinafter refers to I^{131}.