due to some compensatory mechanism) and thus necessitate the determination of all the constants in equations (2) and (4). Such an analysis would require a minimum of 4 determinations, at about 3, 10 or 15, 30 or 40, and 60 or 80 min. In other words, the analysis of BSP curves for abnormals (mild or severe) is much more complicated than that of normal BSP curves if the results are to be equally accurate and clearly definitive. In case the above steps of the curve analysis are omitted or improperly done, the more likely error is the underestimation of the extent of liver impairment.

The present interpretation gives an approximate confirmation of Goodman's conclusions about normal cases, but his results for abnormal cases would sometimes be too high (*3*).

The present theory also applies to the hepatectomized dogs of Cohn, Levine, and Streicher (*1*). In that case $k_3 = r_2 = 0$ and the last term of equation (2) is constant.

This treatment neglects the reabsorption factor described by Lorber and Shay (*4*). That factor should give rise to theoretically low values of k_3 (and higher values of k_1 and k_2), but these would still be correct in regard to over-all physiological significance.

If E is the efficiency with which the liver removes BSP from the blood flowing through it, and if F is the fraction of the total blood volume that flows through the liver each minute, then $k_3 = EF$. Since $0 < E < 1$ it follows that k_3 is an upper limit for liver blood flow, F. However, k_3 itself is the physiologically significant quantity, or measure, of this particular liver function.

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The Influence of Iodoacetate on the Sodium and Potassium Content of *Ulva lactuca* and the Prevention of Its Influence by Light[1]

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The influence of glycolytic inhibitors on cation equilibria and movements in living cells has been investigated by Wildbrandt (*1*), Harris (*2*), and Maizels (*3*) on human erythrocytes, Dean (*4*) on muscle, Dixon (*5*) on brain cortex, and Scott *et al.* (*6*) on baker's yeast. An interpretation of the action of these agents has been based on their inhibitory effect on specific enzyme systems of carbohydrate metabolism which are associated with ion transport and equilibria.

The experiments to be reported represent a study of the influence of one of the glycolytic inhibitors, monoiodoacetate, on the sodium and potassium content of the green alga, *Ulva lactuca*. The study was undertaken to test the applicability of the postulated role of glycolysis in cation regulation in this form.

The cells of this marine organism, like most cells living in a high Na^+ low K^+ medium, accumulate K^+ and partially exclude Na^+. Since the alga consists of large membranous fronds of two layers of cells, it is particularly well suited for investigations involving ion interchange between the cell and its environment.

The *Ulva,* collected from the Eel Pond in Woods Hole, was conditioned before use under incandescent illumination in running sea water. Small uniform samples cut from a single frond were placed in large finger bowls of sea water, containing the inhibitor when present, and maintained in the dark or in the light at the temperature of running sea water (*ca.* 21° C). Samples were removed at various time intervals, rinsed for 1 min in isotonic sucrose (0.6 *M*) to remove adhering salts. The sucrose solution was then removed from the surface by a consistent blotting procedure. A wet weight was determined on the blotted material and, after drying for 12 hr at 110° C, a dry weight was taken. Cell water was calculated by difference. The dried material was ground in a mortar and extracted in 50.0 ml of 10% trichloracetic acid for a few hours. The observation has been made in our laboratory that Na^+ and K^+ are quantitatively extracted from the material by this method as compared with the usual wet ashing techniques. The extracts were analyzed for Na^+ and K^+ by flame photometry, using the Beckman spectrophotometer.

Influence of sodium iodoacetate on cellular potassium in the dark and in the light. The presence of the inhibitor in a concentration of 0.001 *M* results in a marked loss of K^+ from the cells over a period of 24 hr in the dark. Control samples taken at the beginning and end of this period were essentially constant in potassium content (Fig. 1). In the presence of light from a 100-w incandescent lamp placed at a distance of about 1 ft from the alga, the inhibitor is completely ineffective in causing loss of K^+. Rather, the potassium content of the experimentals temporarily increases over that of the controls.

To evaluate further the influence of light on the prevention of the iodoacetate effect, the concentration of the inhibitor was raised to 0.005 *M*. Again the light prevented the loss of K^+ (Fig. 1).

Influence of sodium iodoacetate on cellular sodium in the dark and in the light. Concomitant with the K^+ loss caused by the 0.001 *M* NaIAA in the dark the

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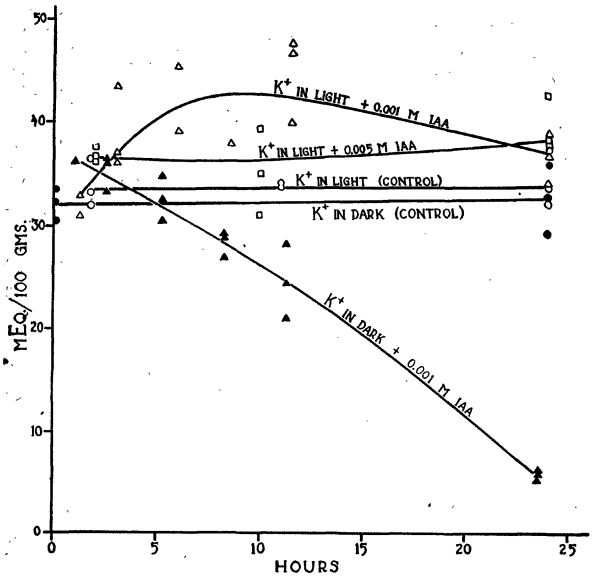


FIG. 1. The influence of monoiodoacetate on the potassium content of *Ulva lactuca* in the light and in the dark. Potassium is expressed in terms of cell water.

cellular sodium increases over that of the controls, although the condition of darkness alone is sufficient to cause some sodium increase. Illumination again prevents this action of the inhibitor, and the sodium is actually somewhat reduced compared to the controls (Fig. 2). Light also prevents an increase in sodium when the inhibitor concentration is raised to 0.005 *M*.

An examination of a large body of unpublished data indicates a close correlation between potassium content and calculated cell water in *Ulva lactuca,* while sodium content is most consistent on a dry weight basis. For these reasons, the potassium and sodium data are expressed as indicated, although essentially the same results appear when the data for each ion are expressed on either basis. These observations may reflect a largely ionized cellular potassium and a partially bound sodium. Confirmatory evidence for the ionized state of potassium has been recently obtained in our laboratory by experiments with K^{42} which indicate that all the potassium in the cell is readily exchangeable (*7*).

The normal variation in potassium content encountered in samples from different fronds or in different samples from the same frond, probably a reflection of varying physiological conditions within the cells, is usually accompanied by a reciprocal sodium variation.

Iodoacetate, in low concentrations, has been shown by experiments *in vitro* to inhibit selectively glyceraldehyde dehydrogenase, one of the glycolytic enzymes (*8*). It has been proposed, since iodoacetate caused a loss of K^+ from erythrocytes, muscle, brain cortex, and yeast cells, that the normal accumulation of this cation is dependent on the intact glycolytic system (*3–6*). The additional observation by Maizels (*3*) of a gain of Na^+ by human erythrocytes in the presence of the inhibitor suggests an active sodium extrusion process dependent on glycolytic energy. The interpretation of the action of iodoacetate in the dark in causing a loss of K^+ and a gain of Na^+ in *Ulva lactuca* is consistent with this hypothesis.

The prevention of these ion shifts by light lends further support to this hypothesis, since phosphoglyceric acid, the compound which is formed by the action of glyceraldehyde dehydrogenase, has been shown by Calvin and Benson (*9*) and confirmed by Fager and Rosenberg (*10*) to be the first stable product formed in the photosynthetic reduction of carbon dioxide. Hence, in the light, even in the presence of the inhibitor, which prevents the glycolytic formation of phosphoglyceric acid, this intermediate is made available to cellular metabolism by photosynthesis. The above evidence suggests, therefore, that the normal Na^+ and K^+ distribution in this organism depends on the utilization, in some way, of phosphoglyceric acid. The degradation of this compound could provide energy for synthetic processes maintaining the normal constituents of the plasma membrane, thus affecting the permeability per se of the cell for cations. This phase of cation regulation has been particularly emphasized by Parpart and Green (*11*) in relation to factors affecting the Na^+ and K^+ balance in the erythrocyte. Since iodoacetate inhibits glycolytic synthesis of adenosinetriphosphate, another interpretation for these results might be that ATP ions, which according to a novel hypothesis proposed by Ling (*12*), "when absorbed on proteins could supply a strategically indispensable part of the fixed charges which would selectively attract K^+ and displace Na^+ from the cell."

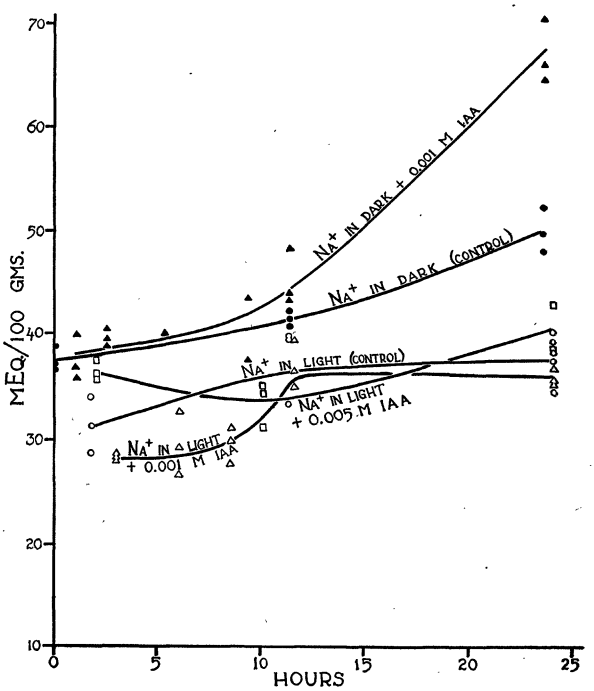
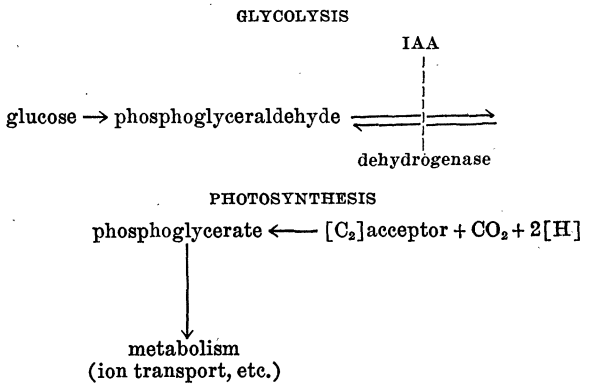


FIG. 2. The influence of monoiodoacetate on the sodium content of *Ulva lactuca* in the light and in the dark. Sodium is expressed in terms of dry weight.

In the opinion of the authors, however, a more probable explanation of the observation here reported, and a view favored by further investigation of the problem in this organism (*13*), is as follows: Ion transport mechanisms, as yet to be elucidated, would be necessary to compensate for the continual flow of these cations with their concentration gradients across the cell surface. Such transports, according to this interpretation, would be energized by the metabolic degradation of phosphoglyceric acid, perhaps through the mediation of high energy phosphate bonds as in ATP.

A further elucidation of the precise mechanisms involved in this problem will pertain to one of the most fundamental activities of living cells: the capacity to maintain within cell boundaries a chemical composition which is characteristically different from that of the external environment, and here particularly the ability to concentrate K^+ and partially exclude Na^+ against their respective concentration gradients.

The postulated reactions may be summarized as follows:



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Association Affairs

Southwestern Division Meeting

Frank E. E. Germann, *Secretary*

Southwestern Division

THE Southwestern Division of the AAAS held its twenty-ninth meeting in Tempe, Arizona, during the week of April 19th on the campus of the Arizona State College. One hundred and eighty persons registered, although the number participating in the meetings was much larger than this. As usual, many students took advantage of the opportunity to attend a regional scientific meeting. The sessions opened with a meeting of the Executive Committee, on which occasion John A. Behnke, Associate Administrative Secretary of the AAAS, represented the Washington office. At this time Wyoming and Montana East of the Continental Divide were officially added to the Division. Mr. Behnke reported briefly on the activities in the Washington office, as well as concerning the annual meetings scheduled for Boston and San Francisco.

In addition to the regular programs of sections, two special symposia were presented.

CONSERVATION SYMPOSIUM

Martin Mortensen, *Presiding*

1. The Nature of Conservancy in Arizona. Leslie N. Goodding, St. David, Arizona.

2. The Status of Conservation Teaching in New Mexico. Howard J. Dittmer, University of New Mexico, Albuquerque.

3. Report on Conservation Education Project Activities in Arizona. Wayne Kessler, Assistant State Conservationist, State Land Department, Arizona.

A summary of activities involving conservation education projects, in the following subdivisions: (1) in public schools, including high schools and grammar schools; (2) in the Arizona Conservation Districts; (3) in connection with cooperative agencies from the federal, state, and local governments.

4. General Discussion by Panel, led by Chairman Mortensen.

DESERT AND ARID ZONE SYMPOSIUM

Peter C. Duisberg, *Presiding*

1. Committee Accomplishment, 1952–53. Victor Schoffelmaier, Glendale, California (Former President, Texas Chemurgic Council).

2. Survey of Desert and Arid Zone Research in Progress in the Southwest. E. J. Workman, President, New Mexico Institute of Mining and Technology, Socorro.