omers whose work laid the foundations for modern progress.

The amateur will thus find not only clear and complete directions for work, but the basic principles which enable him to understand the significance of his results. The professional astronomer will also find the book useful on account of its convenient collection of data for which he had been obliged previously to search through periodicals.

The specialist in astrophysics will naturally find some points capable of clearer statement, and some minor errors such as are apt to creep into first editions. For example, the Zöllner photometer is described on page 118 as used with the historic petroleum lamp, rather than with the modern incandescent lamp. The lack of wave-lengths on the margins of the engravings of spectra is puzzling to one not thoroughly familiar with them, especially as Plate XI. is printed with the violet end to the right, instead of the usual direction. Chapter XII., entitled "Eclipsing Binaries," includes also the "Cepheid Type," though it is not claimed that their changes can be explained by eclipses. On page 229 is the statement that "It was only with the selenium cell that it was possible to determine a change so small as 0.06 magnitude," though as a fact, the extra-focal photographs are capable of determining such changes. The use of *mg.* as an abbreviation for magnitude, is unfortunate, as it usually stands for milligram. Compare the statement in the *Scientific American* that the planet Saturn is 16 inches in diameter, due to the use of the double stroke as a sign of both inches and seconds of arc. This is not the place to give a list of typographical errors, but the statement at the top of page 102, that if star *A* is twice as bright as star *B*, the difference in magnitude is 0.44, might mislead. In the examples of the use of Pogson's rule, in Chapter V., the omission of the problem of finding the combined magnitude of two or more stars, is worth mentioning. In the historical part, the failure to give Mrs. Fleming credit for her part in the creation of the Harvard classification of stellar spectra; also the failure to credit the astronomer royal, Christie, for the "square-root" formula for the reduction of the diameters of stellar images on photographs, to magnitudes, are minor points which might be corrected.

In spite of these minor criticisms the book is a worthy contribution to the series celebrating the semi-centennial of Vassar College.

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THE VITAL EQUILIBRIUM

Following the suggestion of Nernst[1] that varying degrees of permeability of the plasma membrane might be due to a selective solubility of certain of its components, Overton established his lipoid theory. The most serious objection to Overton's theory is that, whereas it accounts most satisfactorily for the permeability of the cell for substances which normally play no part in the cell metabolism, it entirely fails to explain the penetration of sugars, salts and amino-acids, which must constitute an essential part of the cell income. Loeb[2] long ago emphasized the importance of the state of aggregation of the surface colloids as one factor influencing the conditions of permeability. This suggestion was made in connection with his experiments upon the effects of pure solutions of NaCl and combinations of NaCl and polyvalent ions on the eggs of *Fundulus*. Subsequent experiments by Loeb, Höber, Lillie and a host of others, have established beyond a doubt the existence of a physical-chemical relation between the state of aggregation of the cell colloids and the permeability of the cell. A precise and universal statement of the exact nature of this relation has never been made. In the following paper we shall attempt an analysis of the conditions determining the viscosity of cell surfaces and their importance; (1) in producing changes in permeability and (2) in "antagonisms." It appears that the metabolic

[1] Nernst, W., '90, *Zeitschr. f. physikal. Chem.*, 6, 37.

[2] Loeb, J., '01, *Pflügers Arch.*, 88, 68; '02, *Amer. Jour. of Physiol.*, 6, 411.

activities of cells, in so far as they involve an interchange of material through the surface layer, depends upon the shifting of a *surface-solution equilibrium.* Since an interchange of material eventually becomes essential for the continuation of any living system, we have called this solution equilibrium the "Vital Equilibrium."

If we examine a series of two-phase systems beginning with a coarse suspension and extending through fine suspensions, colloidal suspensions, colloidal solutions, hydrophilous colloidal the "molecular disperse" systems of Ostwald and ionically disperse systems such as dilute solutions of electrolytes like NaCl, we observe two striking changes: first, an increased subdivision of the disperse phase and, second, an increased intimacy of relation between disperse phase and solvent, a necessary result of the enormously developed surface in the former.[3] We find, furthermore, that in any of these systems there always exists an equilibrium between disperse phase and solvent. The opposing forces are in the direction of an increased aggregation and dispersion, respectively; we may therefore speak of an aggregation equilibrium. This equilibrium is shifted by the addition of solutions of any substance, organic or inorganic, by heat, ultra-violet light, etc. For example, if we add $CaCl_2$ to the negative suspension colloid As_2S_3, a precipitation occurs, *i. e.,* there is an increased aggregation of the disperse phase. Reciprocally, small quantities of 0.1 N $CaCl_2$ will clear an opaque colloidal solution of egg-white. There is an increased dispersion and the system becomes more like a true solution. Now the limits of the above series are total insolubility and complete solubility. Any change in the direction of increased dispersion means a change in the direction of a true solution, *i. e.,* an increased solubility. No sharp limits occur between true solutions and colloidal solutions. A solution of cane sugar, for example, though a molecular disperse system, certainly represents a lesser degree of dispersion than any solution of an electrolyte. Again, when two salt solutions having a common ion are combined, there appears the familiar phenomenon of association or decreased dispersion, an equilibrium shift in the direction of greater aggregation, in this case from ionic to molecular dispersion. We may therefore legitimately dispense with the term "aggregation equilibrium" and, even though we are dealing with colloidal systems, substitute the more familiar "solution equilibrium."

The hydrophilous colloids which are of particular interest to physiologists are peculiarly susceptible to slight changes in hydrogen ion concentration. Here the changes in aggregation are reversible to a far greater degree than in the colloids lower in the scale. The limits of reversibility of the solution equilibrium may be said to include a far greater range of aggregation states than in the colloids of the lower classes.

An examination of the experimental data[4] shows that for a number of different hydrophilous colloids the following anion order of dispersion obtains:

$$SO_4 < \text{Tartrate} < \text{Citrate} < \text{Acetate} < Cl < ClO_3 < NO_3 < Br < I < SCN.$$

The most indifferent region lies between acetate and chloride; SO_4 has the greatest tendency towards aggregation, while SCN produces maximum dispersion.[5] In many cases, especially in precipitation experiments, the addition of the electrolyte may have no visible effect upon the colloid. When this occurs the changed equilibrium may be detected by a vis-

[3] Höber, R., '14, *Physikal. Chemie d. Zelle u. d. Gewebe.* 4 Aufl., Kap. 7, p. 305 ff.

[4] Hofmeister, F., '91, *Arch. exper. Pathol. u. Pharmakol.,* 28, 210; Höber, R., '07, *Hofmeisters Beitr.,* 11, 35; Porges u. Neubauer, '07, *Biochem. Zeitschr.,* 7, 152; Hardy, '05, *Jour. of Physiol.,* 33, 251.

[5] By a sufficient increase in the hydrogen ion concentration the anion order may be *completely inverted.* Thus the effect of an alkali salt upon the state of aggregation of any hydrophilous colloid depends directly upon the hydrogen ion concentration. Posternak, '01, *Ann. Inst. Pasteur,* 15, 85; Pauli, W., '03, *Hofmeisters Beitr.,* 5, 27; Höber, R., '07, *ibid.,* 11, 35.

cosity determination, one of the most accessible methods for tracing changes in the state of colloidal aggregation.

The viscosity of colloids or its reciprocal, fluidity, shows peculiar variations with different degrees of dispersion. When the dispersion is greatest, *i. e.*, when the disperse phase is in "solution," we find that the fluidity is also at a maximum. An increased aggregation means a decreased fluidity which, however, continues only to the point at which the disperse phase begins to separate out from the dispersion medium as a suspension colloid or suspension. When this point is attained, the fluidity is suddenly reversed and approaches more and more that of the pure dispersion medium. Now whether the precipitation of the disperse phase is brought about by the action of electrolytes or, for example, by elevated temperature (heat coagulation) the physical-chemical effect upon the fluidity is the same (Fig. 1, *B*).

Since all substances have an influence one way or the other upon the solution equilibrium of a colloidal system, we may, theoretically, divide them into two groups; (1) those favoring solubility of the disperse phase (increased dispersion, increased fluidity) and (2) those favoring insolubility (aggregation, precipitation, coagulation, initial increased viscosity).

Turning now to the conditions of the colloids especially at the surfaces of cells, we find, in some cases, sharply differentiated membranes. In many animal cells such membranes are not demonstrable, but for our present discussion this is of little moment, since we are concerned with a colloidal boundary which must exist at the surface of every cell. In experimental studies upon single cells as, for example, animal eggs, variations in the constitution of the environmental medium produce profound changes in the cell. Whatever changes may occur within the cell as a result of such variations, it is certain that these changes are secondary to an initial or primary effect at the cell surface. Liquefying agents are believed to produce an increase in cell permeability.[6] *Arbacia* eggs, for example, when treated with solutions of sodium or potassium thiocyanate, begin to lose their pigment after two or three minutes. This is to be regarded as an expression of an increase in the normal permeability of the cell surface.

It has been shown for a number of physiological objects[7] that the deleterious action (liquefying action) of neutral alkalin salts decreases from SCN to Cl in an order corresponding satisfactorily with the Hofmeister series. In these experiments, when the solutions of the salts are brought into contact with the cell surface, the degree of dispersion of the surface colloids must be increased. The dispersion is greatest in solutions of thiocyanates and least in chlorides. A physico-chemical expression of this increased dispersion is the increase in the fluidity of the cell surface. Now, since the speed of diffusion of ions and molecules through any fluid medium depends upon the viscosity of that medium,[8] it is clear that an increased fluidity of the cell surface involves a facilitation of diffusion of soluble substances from either side of the cell surface. In other words, by a liquefying action at the surface, the permeability of the cell is increased and diffusion in both directions across the surface is facilitated.

Since there is this very definite correlation between liquefaction (dispersion) and increased permeability, it is obvious that, in the normal condition of the cell, we must have a greater aggregation of the surface colloids than during liquefaction. Bearing in mind the fact that pure solutions of all substances affect the solution equilibrium of colloids one way or the other, we should expect, *a priori*, to find certain agents producing an increased aggregation of the surface colloids of the cell; we should expect to find true solutions of

[6] Lillie, R. S., '13, *Jour. of Morphol.*, 22, 695.

[7] Schwarz, C., *Pflügers Arch.*, 117, 161; Lillie, R. S., '10, *Amer. Jour. of Physiol.*, 26, 106; Spaeth, R. A., '13, *Jour. of Exper. Zool.*, 15, 527.

[8] In the case of water-swollen gels, the speed of diffusion of crystalloids is approximately the same as in pure water, but it diminishes rapidly when the water content falls below a certain value. Bechhold u. Ziegler, '06, *Zeitschr. f. physik. Chemie*, 56, 105.

electrolytes or non-electrolytes which, when brought into contact with the cell surface, would reduce the degree of dispersion of the surface colloids. This process is not, however, a simple reciprocal of liquefaction. A *slight* increase in the aggregation of the surface colloids would involve a rise in the viscosity of the cell surface and, if an equilibrium were established, the speed of diffusion of ions and molecules across the cell surface would thus be reduced. As we noted above, the speed of diffusion of any substance across the cell surface is one index of the degree of permeability of the cell for that substance. Hence we may say that with an increase of viscosity at the surface, the permeability of the cell would be decreased. If now the concentration of the agent that is responsible for the increased aggregation at the cell surface were still further increased, there would be an additional increase in the viscosity of the surface. But, as we have already stated, the viscosity of colloids is sharply limited by the state of aggregation of the disperse phase. When the precipitating disperse phase begins to separate out from the dispersion medium, the viscosity suddenly decreases. Similarly, when at the surface of the cell the disperse phase begins to separate from the dispersion medium, *the fluidity of the cell surface* must rise abruptly. The sharp increase in fluidity would obviously involve a sudden removal of the barrier to diffusion for ions and molecules and they would pass the cell surface more rapidly.[9]

From the above considerations it seems, therefore, that the permeability of a cell may be increased either (1) by bringing into contact with the surface a solution of some liquefying agent like a thiocyanate, *i. e.,* some agent that increases the solubility (degree of dispersion) of the surface colloids and the fluidity of the surface, or (2) by bringing into contact with the surface a solution containing an excess of some deliquefying or precipitating agent like $CaCl_2$ which, by increasing the state of aggregation of the surface colloids eventually separates disperse phases from solvent, the fluidity of the cell surface approaching that of the pure dispersion medium.

The normal influences at the cell boundary must have a considerable aggregating effect upon the surface colloids since we do not normally find the cell pigments or other constituents diffusing outwards. Osterhout,[10] furthermore, has shown conclusively that certain electrolytes increase the electrical resistance of a cylinder of *Laminaria* discs. This is to be considered an expression of decreased permeability. These electrolytes ($MgCl_2$, $CaCl_2$, HCl, $La_2(NO_3)_6$) all have, in certain concentrations, a distinct coagulative or dehydrating effect upon a variety of colloids.[11] The effect of $CaCl_2$ is of particular interest since at first it increases the resistance. After a time, however, the resistance again decreases, finally falling below the initial value. This is precisely what we should expect if the effect of the $CaCl_2$ were upon the viscosity of the surface colloids. The theoretical correlation between increased dispersion and increased permeability, as well as that between increased aggregation and initial decreased permeability, is thus actually substantiated by experiment.

Permeability studies upon living cells have brought out one very striking and at first sight anomalous, fact, viz., for many substances which are not concerned with the normal metabolic processes of the cell, the cell surface is readily permeable, whereas for sugars, salts, amino acids, etc., which must constitute a large proportion of its nutritive material, it is nearly or quite impermeable. The latter substances normally occur, however, within the cell, which forces us to assume that at some previous period the surface must have permitted their passage to the cell interior. This passage to the interior of the cell could have been accomplished only under conditions of increased permeability. Now the cell content is obviously not to be regarded as permanent and fixed and we must account for a mechanism of metabolic interchange. Such a mechanism

[9] Ostwald, Wolfg., '11, *Grundriss d. Kolloidchemie,* 2 Aufl., p. 307.

[10] Osterhout, W. J. V., '15, SCIENCE, 41, 255 for a summary of results.

[11] Mines, G., '10, *Jour. of Physiol.,* 40, 327; '11, *ibid.,* 42, 309; Höber, R., u. Spaeth, R. A., '14, *Pflügers Arch.,* 159, 433.

is to be sought in some type of physical-chemical equilibrium that permits the permeability above and below a norm to vary reversibly within definite limits. We have pointed out above that there is in every colloidal system an aggregation or solution equilibrium between disperse phase and dispersion medium. The colloids at the surface of the cell are no exception to this rule. The continued action of a liquefying agent at the cell surface produces a marked increase in permeability and eventually death by irreversible liquefaction. On the other hand, a coagulating agent, *i. e.*, an agent that increases the aggregation of the disperse phases of the surface colloids, produces *at first* a decrease in permeability, but, if the action be sufficiently prolonged, the disperse phases separate out from the dispersion medium and death follows as a result of surface coagulation. Once the disperse phases have begun to separate from the dispersion medium, the fluidity of the cell surface approaches that of the pure dispersion medium which obviously involves a tremendous increase in permeability. Thus cell death, whether by irreversible surface liquefaction or by irreversible surface coagulation, invariably involves an increase in the permeability of the cell. The term "cytolysis" has been loosely applied to cover both cases, though from a physical-chemical standpoint we are dealing with antithetical processes.

The degree of aggregation of the surface colloids, *i. e.*, the degree of intimacy of relation between disperse phases and solvent appears, upon last analysis, to be the critical condition upon which depends the continuation of the cell as a living system. The degree of aggregation of the disperse phases at the surface of the cell is directly dependent upon their solubility in the dispersion medium. This solubility is determined (1) by the concentration, nature and number of electrolytes or organic substances occurring in the liquid phase, and (2) by the temperature of the whole system. We may, therefore, say: (*A*) at the surface of every cell there is a solution equilibrium, a *vital equilibrium,* between disperse phases and solvent; (*B*) the permeability of the cell is determined by the maintenance or shifting of the vital equilibrium.

We may now summarize the foregoing conclusions as follows:

1. In the limiting colloidal system of every cell, whether in the form of a differentiated membrane or not, there exists an equilibrium between disperse phases and dispersion medium.

2. A shifting of this equilibrium in the direction of greater dispersion causes an increased permeability of the cell surface, since the fluidity of the system is increased, the viscosity of the surface is lowered, and a more rapid diffusion occurs across the surface both into and out of the cell.

3. A slight shift of this equilibrium in the direction of increased aggregation involves a solidifying action at the surface, an increased viscosity, a slower rate of diffusion across the surface and a consequent decrease in permeability.

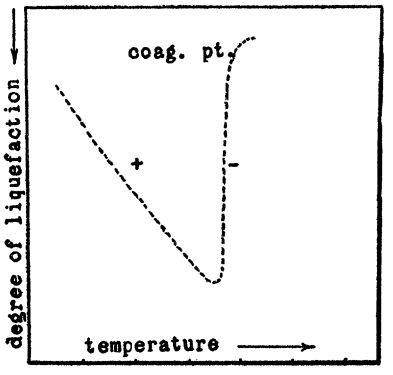
4. A considerable shift of this surface equilibrium in the direction of increased aggregation (insolubility of the surface colloids) involves a decrease in the degree of intimacy between disperse phases and solvent; the fluidity is suddenly increased and diffusion across the surface is correspondingly facilitated.

5. The critical condition of any cell surface, upon which eventually depends the continuation of the cell as a living system, is the state of aggregation of its surface colloids, *i. e.*, the relation of disperse phases to dispersion medium. We may, therefore, speak of a solution equilibrium, a *vital equilibrium* at every cell surface, reversible within definite limits, the overstepping of which produces death by surface liquefaction, on the one hand, or by surface coagulation, on the other.

We have thus far considered the cases involving the effects of single electrolytes upon the surface colloids of cells. We shall now briefly consider the physiological effects (1) of combinations of electrolytes and (2) of elevation of temperature.

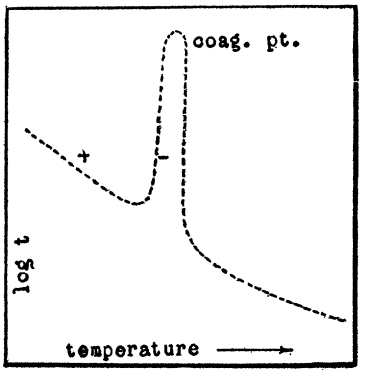
In any combination of electrolytes it is clear that if it were possible exactly to compensate the dispersion effect of one constituent or

group of constituents by the aggregation effect of another, the solution equilibrium of the surface colloids of a cell exposed to such a combination would remain unchanged. Stated in physiological terms, if we could compensate coagulative and liquefactive forces at the cell surface, the vital equilibrium would remain normal and we should obtain no injurious effect. Compensating effects of this sort are actually realized in solutions like those of Ringer and Locke, or in sea water. We have here a number of combined chemical stimuli which, when acting singly, produce distinct liquefactive or coagulative effects upon living cells, but which, in combination, are relatively harmless. According to the conception of a solution equilibrium at the cell surface, the non-injurious effects of compensated solutions of two or more constituents are to be referred to the failure of these solutions markedly to increase or decrease the solubility of the colloidal disperse phases. Such phenomena of physiological compensation have been collectively termed "antagonisms."



A.

FIG. 1, *A*. An empirical curve representing the liquefying action of atropine upon the melanophores of *Fundulus* at different temperatures. The points are relative. Thus at 22° C. there is more liquefaction than at 10° C., but less than at 36° C. At 37° C. there is again less liquefaction than at 22° C., but more than at 5° C. Up to 36° C. the temperature coefficient of liquefaction is therefore positive, while beyond 36° C. for a few degrees, it becomes negative.



B.

FIG. 1, *B*. Ostwald's curve showing the effect of elevated temperature upon the viscosity of egg-white. Here the temperature coefficient of liquefaction is positive up to about 57.5° C., while beyond this point to about 60° C. it becomes negative.

A comparison of the two curves shows that above and below a critical point in each system (36° C. and 57.5° C.) the temperature effects are antithetical.

That there is some physical-chemical principle behind all "antagonisms" is strongly suggested (1) by the appearance of the compensation phenomenon between such widely unrelated chemical substances[12] and (2) by the compensation that appears between liquefying agents and elevated temperature. This last case seems of such importance as to warrant a detailed consideration.

If a colloidal solution of egg-white be gradually heated to 35°–40° C. it becomes slightly less translucent, *i. e.*, there is an increased dispersion. A viscosity measurement[13] shows that an increase in fluidity continues uniformly up to about 57.5° C. At this point there is a sharp reversal of the reaction and the viscosity rises rapidly to about 60.0° C.,

[12] For example "antagonisms" have appeared between various alkaloids such as atropine and eserine, nicotine and curare, between alkaloids and salts as atropine and $CaCl_2$ and $MgCl_2$ and between such salts as KCl and cobalt hexamine chloride (Höber u. Spaeth, *loc. cit.*).

[13] Ostwald, Wo., '13, *Koll. Zeitschr.*, 12, 213.

the coagulation point (Fig. 1, B). Owing to this peculiar property of coagulation, which is, physically, an increase in the state of aggregation, whatever may be the nature of the chemical processes involved, we have here opposite effects produced by a slight and considerable increase in temperature, respectively; the effect of heat may be either liquefactive[14] or coagulative. We should expect, *a priori*, that by adding a powerful liquefying agent to an hydrophilous colloid, the coagulative effect of heat might be overcome wholly or in part, since this would introduce a dispersion factor into the equilibrium. This actually occurs as Pauli[15] and Pauli and Handovsky have shown. Pauli found that upon adding neutral thiocyanates, which are powerful liquefying agents, to egg-albumin, it could not be coagulated even at the boiling point of the mixture.

Four years ago I observed that the liquefying effect of atropine or atropine sulphate upon the melanophores of *Fundulus* could be distinctly reduced by sufficiently elevating the temperature of the solution. Recently[16] I have found that for temperatures up to approximately 36° C., atropine shows a normal positive temperature coefficient, *i. e.*, the liquefying effect increases with a rise in temperature. If, however, we expose contracted melanophores to identical solutions of atropine at 22° C. and at 37° C. for a period of five minutes, the cells from the warm solution show distinctly less liquefaction than those at room temperature. That the cell colloids are not coagulated by the higher temperature is shown by the activity of the cell upon being returned to NaCl or KCl solutions. Thus in this case, for a few degrees, between 36° C. and the elevated coagulation point of the cell protoplasm ($< 43°$ C.) the temperature coefficient of liquefaction for atropine becomes negative (Fig. 1, A). From the foregoing considerations we should expect an elevation in temperature to increase the solubility of the disperse phases at the surface of the melanophore.[17] We have, in addition, the liquefying effect of the atropine. Hence we have here the combined liquefying effect of atropine and elevated temperature, *i. e.*, two forces tending to drive the disperse phases of the cell surface into solution and to increase their degree of dispersion. Beyond 36° C., however, further increase in temperature tends to initiate the first steps in the process of heat coagulation involving a decrease in the state of aggregation of the surface colloids. The inhibition of the atropine effect above 36° C. is, therefore, to be interpreted as due to an elevation in the viscosity of the surface colloids which retards the diffusion of the alkaloid into the cell. Bearing in mind these antithetical physical effects of low and high temperatures, it appears that the experimental data both in the case of colloidal solutions of egg-white and in that of living cells (melanophores) comply well with the theory.[18] A liquefying agent in proper concentration may prevent heat coagulation and, reciprocally, a sufficient elevation in temperature may protect the system against liquefaction.

These physical-chemical relations may offer an explanation of the extraordinary habit of certain blue-green algæ which normally thrive at a temperature of 68° C.[19] The water in which these algæ live contains numerous salts in solution and we suspect at once that among these salts there is a powerful liquefying agent which prevents coagulation by the abnormally high temperature, as in Pauli's experiments upon egg-white. We should expect that a reduction in temperature would prove fatal to such algæ since, under these altered circum-

[14] Lillie, R. S., '15, *Biol. Bulletin*, 28, 260.

[15] Pauli, W., '99, *Pflügers Arch.*, 78, 35; Pauli u. Handovsky, '08, *Hofmeisters Beitr.*, 11, 415.

[16] Unpublished experiments.

[17] It is impossible to carry out an experiment upon the melanophores of *Fundulus* which is directly comparable to Pauli's experiments on the elevated coagulation point of egg-white. KSCN produces a marked liquefaction upon the melanophores, but only after a relatively long exposure (< 30 minutes). Atropine, on the other hand, brings about an irreversible disintegration at room temperature in concentrations of ca. 0.004 *M* in 0.1 *M* NaCl in about five minutes.

[18] See also Lepeschkin, W. W., '11, *Ber. d. deutsch. bot. Gesellsch.*, 29, 247; '13, *ibid.*, 30, 703.

[19] Setchell, W. A., '03, Science, 17, 943.

stances, the liquefying agent would be free to act. So far as we know no experiments of this sort have ever been performed, though it may be significant that Setchell failed to find any algæ growing at 43°–45° C.

In a recent paper Osterhout[20] advances the hypothesis that substances which increase permeability antagonize those which decrease permeability. He says (p. 256):

> It seems to the writer that the hypothesis offers a rational explanation of antagonism by showing that salts antagonize each other because they produce opposite effects upon the protoplasm.

The nature of these "opposite effects upon the protoplasm" is an increase or decrease of permeability. Osterhout makes no statement as to the meaning of the term "permeability" which, without further qualification, is noncommittal, nor to the *cause* of the permeability changes. With these two fundamental gaps in the theory it seems a far cry to a "rational explanation of antagonism." We have emphasized above that a study of Osterhout's data indicates a direct correlation between decreased permeability and increased surface viscosity. It seems highly probable, however, that all substances producing an initial decrease in permeability will, if allowed to act long enough or in sufficient concentration eventually cause an *increase* in permeability.[21] This conclusion, which we are forced to make from a study of the phenomena of viscosity changes in colloids, complies very well with the experimental data upon permeability changes in both plant and animal cells.

All physical and chemical agents acting upon a colloidal system influence the state of aggregation of the disperse phase, tending either to increase or to decrease the degree of dispersion. Since we have a colloidal system at the surface of every cell, all physical and chemical agents influence the state of aggregation or its equivalent, the solubility of the surface disperse phases in one of two ways, viz., (1) there may be an increase in the degree of dispersion and a corresponding increase in the solubility of the disperse phases and the fluidity of the cell surface, or (2) there may be a decrease in the degree of dispersion or a decreased solubility of the disperse phases which eventually results in a precipitation or coagulation. An "antagonism" is to be considered a physiological compensation of a force favoring dispersion (solubility) by a second force favoring aggregation (insolubility). This relation is reciprocal.

[20] Osterhout, *loc. cit.*

[21] Osterhout calls attention to this fact, but offers no explanation for it.

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SPECIAL ARTICLES

NATURAL CROSS-POLLINATION IN THE TOMATO

EVIDENCE concerning the amount of natural cross-pollination in the tomato has been secured by interplanting two commercial varieties of tomatoes, one a standard and the other a dwarf variety. The difference in habit of growth between these varieties is quite distinct in the early seedling stage. The standard is almost completely dominant over the dwarf type of growth. Any pollen from a standard plant fertilizing a dwarf plant should result in a standard plant in the first generation. To test this point a number of dwarf and standard plants were set three feet apart alternately. They were at least five hundred yards removed from any other dwarf tomatoes. These plants were allowed to set fruit normally and seed was saved from the dwarf plants as the fruit ripened. The dates on which the ripe fruit was gathered correspond approximately to the order in which the flowers were fertilized. Seed from these "open-pollinated" dwarf plants was planted in flats in the greenhouse. The number of standard plants which could be plainly distinguished after six weeks' growth was determined and tabulated.

The approximately two per cent. of crossed plants does not represent all the crossing which might have taken place. Aside from a slightly greater distance, there was an equal chance for the dwarf plants to be fertilized by pollen from other dwarf plants. This crossing would produce only dwarf plants, and hence would not show.