Stomatal Opening: Role of Potassium Uptake

Fischer (*1*) proposes that his results "strongly suggest that the basic mechanism of stomatal opening, in response to light plus CO_2-free air, is stimulation of the uptake of potassium." I believe that his evidence supports the opposite conclusion (*2*) that "in order to account for stomatal opening, the guard cells would have to absorb 13 times as rapidly as the most rapidly absorbing cells." Fischer's treatment with KCl did not actually induce opening, but merely doubled the size of the aperture. This increase in size of aperture required 3 to 4 hours, or 6 to 25 times longer than normal opening requires, namely, 10 to 30 minutes. Yet he performed the experiments under conditions that must lead to a far more rapid potassium effect than that in nature, for the following reasons. (i) His KCl solution bathed the whole undersurface of the guard cells, but in nature only about one-fourth of the cell surface is in contact with liquid (from adjacent cells). Bathing the whole undersurface would increase the rate of uptake of both solute and water at least two to three times. (ii) Fischer eliminated the turgor pressure effect of other epidermal cells when he killed them. Stomatal opening would therefore occur at about one-half the cell-sap concentration required in the guard cells under natural conditions. (iii) He eliminated competition for K^+ by the mesophyll cells, which are normally also in contact with the epidermal cells, and whose active K^+ absorption from them would also be stimulated by light, since, as Fischer points out, this is a general characteristic of cells with chloroplasts.

Therefore, the opening described by Fischer as being due to K^+ absorption is 10 to 100 times too slow to account for the normal process. Although Fischer's conclusion cannot apply to normal whole leaves, perhaps it is applicable in the case of epidermal strips in general. However, the opening of stomata in epidermal strips floating on distilled water can occur in the complete absence of K^+ (*2*). Fischer proved this when he obtained opening to half the full width in the complete absence of K^+. The addition of K^+ merely accelerated the opening to full width, which normally occurs in distilled water after a longer time.

Fischer's experiments support the classical concept that stomatal opening is due to an increase in turgor pressure of the guard cells, and that this is initiated by an increase in their cell-sap concentration. Under artificial conditions, any solute absorbed, whether K^+ or anything else [for example, glycerol (*2*)] will add to the total cell-sap concentration and will, therefore, contribute to the opening. But Fisher's conclusion that this artificially added solute is the primary cause of stomatal opening and that increases in other solutes are of only secondary importance is in complete opposition to the facts.

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References

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Levitt (*1*) considers that the stomatal opening in epidermal strips of *Vicia faba* which I have described (*2*) was abnormal, especially since the opening was so slow. Actually 3 hours' exposure to light was given to open stomata because this had previously been shown to be the time necessary for maximum stomatal aperture in floated leaf discs of *Vicia faba* (and not 10 to 30 minutes as suggested by Levitt). Similarly there is no reason to suggest that the K^+ supply to the stomata of a floated epidermal strip was better than it normally would be to stomata of a leaf; under both conditions, solutes probably have equal access to the guard-cell limiting membrane through the relatively porous cell wall system. Although Levitt claims that the stomatal opening I recorded would have occurred "at about one-half the cell-sap concentration required . . . under natural conditions," this is in direct opposition to the data of my Table 1. Indeed a considerable part of my paper was devoted to establishing that the responses to light plus CO_2-free air, of stomatal aperture, guard cell solute potential cell-sap concentration), and guard cell starch were the same in epidermal strips supplied with KCl, as in leaf discs floating on water.

Passing to the role I attribute to K^+ in stomatal opening (that of the major solute accumulating in the guard cell with opening), Levitt (*3*) has already concluded that the rates of solute uptake involved would be too great. However, in repeating his conviction (*1*), he makes no reference to the quantitative data on K^+ uptake which I presented (*2*). In making comparisons of rates of uptake one must consider, as well, the proportion of nonabsorbing cells in the tissues, the surface area of absorbing cells, and the concentrations of external solute used. Thus, my rates of K^+ uptake in the light in 10 m*M* KCl, which were equivalent to about 300 μmole per gram of guard cells per 3 hours, may not have been unexpectedly greater than the maximum rates, measured by Rains (*4*), of 15 μmole per gram (fresh weight) per 3 hours for corn leaf slices, in the light in 0.1 m*M* KCl.

Finally, I do not exclude the possibility of other important solutes, as mentioned by Levitt (*1*), in the guard cells in other situations. I simply state that in the *Vicia faba* system used, it was unnecessary to invoke other solutes (than K^+ and a counter ion) to explain what seemed, in all respects, normal stomatal opening.

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