Mechanisms Of Trap Movement 1: Rapid Growth in *Drosera, Dionaea* and Scientific Notions Of How Venus's Flytraps Close[1]

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Plant and fungal movements often result from differential curvatures or the relative change in size of the cells on each side of a structure. When the term differential curvature is used it usually brings to mind processes in which different relative growth rates on each side of a structure result in its bending. However, it can be seen in figs. 1-3 that differential growth is only one mechanism by which such changes could occur. Others involve a relative loss or gain of turgor of the cells on each side of the structure.

There are three genera of the family *Droseraceae* with movements involved in their trapping mechanisms. In each of these genera it has long been clear that the movements were the result of differential curvatures. By 1934 major papers on the mechanisms of movements in each of the three genera proposed different mechanisms by which these curvatures would be brought about. *Drosera* tentacles were thought to move by a growth mechanism involving a relatively rapid increase in wall plasticity on the abaxial (back) of the tentacles (Hooker, 1916), *Dionaea* traps were thought to move by a rapid gain in turgor by the abaxial (back or outer) side of the trap lobes[2] (Brown, 1916). *Aldrovanda* trap lobes were thought to move by a rapid loss of turgor in the abaxial (inner) side of the trap lobes (Ashida 1934; Figs. 1-3).

Of course by 1979 any well trained plant physiologist believed that slow plant movements, such as those of phototropism, were growth movements and rapid nastic movements were all due to the rapid loss of turgor. Certainly the mechanism of *Mimosa* was well known (Sibaoka, 1980) and we assumed it could be generalized to all rapid movements in plants. This assumption was not just implicit it was often openly stated. Most of us just assumed that *Dionaea* moved by a turgor mechanism. Few read 1916 papers on *Dionaea* and those who did thought that Brown had to be mistaken since a rapid turgor gain seemed unlikely. His data, showing an irreversible increase in cell size of the outer lobes of the trap on closure, which he referred to as "growth" even though he invoked a turgor mechanism to accomplish it, had to be in error since it did not fit with our preconceptions. Ashida's (1934) model for *Aldrovanda* was much more in keeping with our ideas and so I considered that to be the mechanism of *Dionaea* trap lobes as well (Williams,1973; Williams,1976). I certainly received no arguments from my colleagues for such orthodox ideas however inconsistent with the data they might be.

Barbara Pickard and I, when writing a review paper for a July 1979 Symposium in Madison, WI on Plant Movements (Williams and Pickard, 1980)[3], decided to present the paper as a comparison of the *Drosera* mechanisms with *Dionaea* mechanisms since it had become apparent that there were many parallels in these two genera (Williams, 1976). When the data was lined up side by side it became apparent that all evidence for both plants was parallel. Despite this, irreversible changes in cell length in *Drosera* tentacles were said to be due to growth by Hooker (1916) and irreversible changes in cell volume in *Dionaea* were ascribed to a mechanism involving a rapid turgor gain by

Brown (1916). Brown's hypothesis lacked appeal because a rapid gain in turgor seemed unlikely and because it was clear that the trap opened by a growth mechanism and got successively larger each time it moved. Robert Clealand had introduced the concept of acid growth (Clealand, 1980)[4], a process which could be quite rapid, and Barbara Pickard and I (Williams and Pickard, 1972) had demonstrated that the "slow" growth response of *Drosera* tentacles could occur within 10 to 15 sec. It was not unthinkable that the rapid response of the *Dionaea* trap was due to acid growth.

Alan Bennett (then a graduate student at Cornell, now a professor at the University of California at Davis) and I began a series of experiments in Roger Spanswick's laboratory at Cornell and at Lebanon Valley College in Pennsylvania which confirmed the accuracy of Brown's measurements. These also demonstrated that the trap would not close if wall acidification was prevented by neutral buffers and that trap closure would occur if wall acidification was artificially caused with acid buffers (Williams and Bennett, 1982). The only thing that bothered us was that the gradient of hydrogen ions and electric potential in plant cells is such that hydrogen ions should passively flow into the cell instead of out!

It was at this point I remembered Mark Jaffe's (1973) experiments on ATP changes in the midrib during trap closure. This paper, which was my inspiration when I wrote *Why a flytrap is not a Bear Trap* (Williams, 1973), had never made sense to me. The midrib is not where the movement takes place, the units used in the paper did not make sense and I, still believing in Ashida's mechanism, saw no logical reason to look for ATP changes. Suddenly there was a reason to expect ATP changes. Maybe every cell in the excitable zone of the trap, or at least the trap epidermis, responded in the same way. Maybe Jaffe's ratios would hold even if his units did not make sense. Alan Bennett and I froze trap lobes in the open and closed condition and found that Jaffe's ratios did hold and that about 30% of the trap ATP disappeared during closure (Williams and Bennett, 1982; Fig. 3). When we spoke to Roger Spanswick, who was on sabbatical in California, on the phone that evening Alan and I were saying we had a "proton cannon" (the mechanism that uses ATP to move hydrogen ions across cell membranes is often called a "proton pump"). It appeared the power to quickly move hydrogen ions across the membrane was there.

Within the Droseraceae *Dionaea* and *Drosera* have the same mechanism causing their rapid, action potential initiated movements. Although taxonomically it might have been expected and evidence had been sitting around since 1916 it still came as something of a surprise. *Aldrovanda* the other genus in Droseraceae with an active trap is still reported to move by Ashida's rapid turgor loss mechanism (Ashida, 1934; Iijima and Sibaoka, 1983). This may be so but the evidence is not yet firm. If the *Dionaea* and *Aldrovanda* mechanisms differ we are in for another surprise.

Literature Cited

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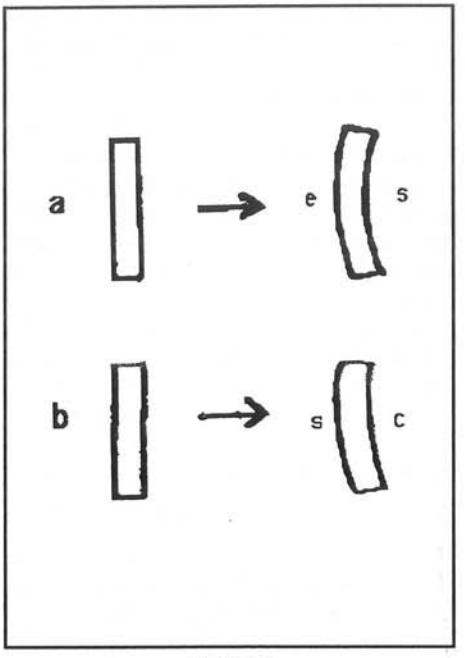

Figure 1

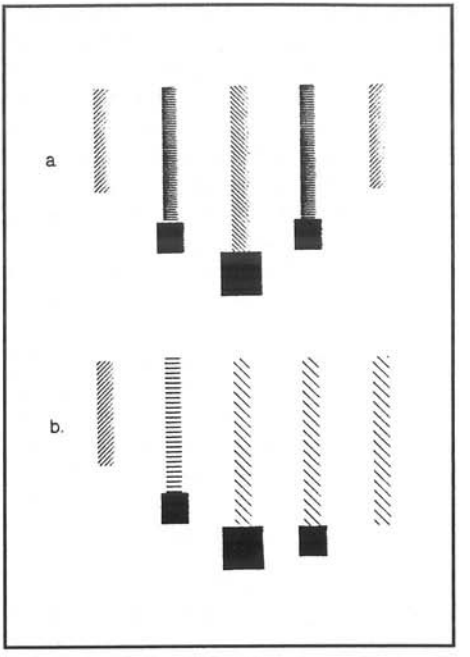

Figure 2

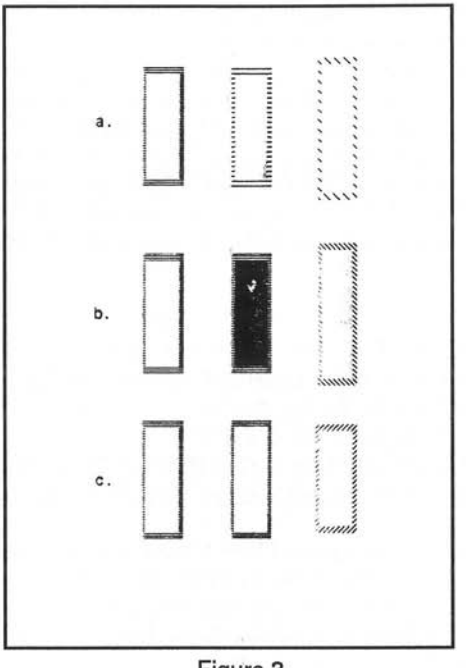

Figure 3

Figures

Figure 1. Movements of plant structures often occur as a result of curvatures caused by differential explanation and/or contraction on the two sides of the structure. **a.** Differential explanation (in this instance explanation on side "E" with no change on side "S") results in the curvature illustrated. **b.** Differential contraction (in this instance contraction on side "C" with no change on side "S") results in the curvature illustrated.

Figure 2. Elastic structures, such as rubber bands, return to their original shape when tension on them is relaxed. Plastic structures, such as modeling clay, remain distorted after tension on them is released. Many things, such as a child's balloon, have some of each property and do not totally return to their original shape when first stretched then allowed to spring back. **a.** A perfectly elastic cell wall being stretched by a weight and returning exactly to its former state just as a perfect spring would. **b.** A perfectly plastic cell wall which remains completely distorted when stretched by a weight. Real cell walls would vary between these two extremes. The early phases of plant growth have long been known to involve an increase in plasticity (decrease in elasticity) of the cell walls. Hooker (1916) was expressing prevailing ideas about plant growth when he invoked this mechanism to account for the growth of *Drosera* tentacles.

Figure 3. a. Cell expansion can result from a loosening and an increase in the plasticity of cell walls that allows them to be extended by the pressure that is in plant cells (turgor pressure). Even though the turgor pressure is necessary for such an expansion it is the change in the wall properties that allows it to occur. Such an event is involved in the early phases of growth and movements caused by such a mechanism would be called a growth movement. **b.** Expansion can also occur by the cell producing or taking up additional dissolved substances such as sugars or potassium ions which increase the osmotic pressure and draw water into the cell resulting in an increase in turgor pressure. Here again it is the turgor pressure that expands the cells but this time the movement is initiated by an increase in the concentration of dissolved substance and the walls merely act as springs being stretched by the pressure created by the water that is taken up. Movements caused by such a change would be called turgor movements. **c.** Reduction of the size of the cell can also occur by a turgor change. Loss of dissolved sugars or ions from the cell would decrease the osmotic pressure and result in the loss of water from the cell. As a result the cell would shrink in size. Here again the cell wall need only act in an elastic way as a spring. Thus turgor movements can occur due either to expansion from a turgor gain or shrinkage from a turgor loss. Combinations of the above mechanisms are also possible although more complex.

Footnotes

[1] This is part one of a projected three article series by this author on the subject.

[2] Brown (1916) referred to these movements as growth movements, probably because he had determined that they involved an irreversible enlargement of cells, but his hypothesized mechanism for the movements was a turgor gain that caused a plastic expansion of the walls of the outer epidermis during the movement.

[3] The symposium was attended by Takao Sibaoka who gave the paper before ours in the session. He reported the first intracellular recordings of action potentials in *Aldrovanda* (1980). I had a pleasant conversation with Dr. Sibaoka afterwards at the banquet.

[4] At the same conference in another session Cleland (1980) presented a review of his then relatively new theory on "acid growth" as the mechanism of auxin action. He may have attended our session.