The Electrochemical Mechanism of Trap Closure in *Dionaea Muscipula*

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Dionaea muscipula, the most spectacular of all carnivorous plants, has drawn an amazing amount of attention since its first description by John Ellis in 1770. It is also the uncontested favorite of our visitors who can't resist putting their probing fingers into the traps. Little do they know how damaging this may be to the plant as the following story relates.

The general mechanisms of *Dionaea* closure were studied by Charles Darwin in 1875 and Burdon-Sanderson more than a century ago (Williams, S.E., 1973), and simply described many times (Pickard, 1973). Despite advances in electrophysiology over the past 30 years, detailed knowledge of the trap closure is largely hypothetical.

The first requirement for an effective trap is speed; the speed to detect the prey's presence, the speed to inform the "motor tissues" and the speed to close the trap. However, the transmission of messages in plants is notoriously slow. In a number of species, some cells acquired a structure which is well known from animal cells, namely, the *excitable membrane* (Guyton, 1966, p.58; Vander *et al.,* 1970, p. 134). All cells, whether plant or animal, are surrounded by a membrane which carries enzymes and other proteins responsible for the control of secretions, mutual recognition, permeability, etc. The main difference between regular and excitable membranes is that the latter are capable of functioning in two very different modes depending upon the electrical charge they carry. They possess, as it were, two settings. Other cell membranes are also electrically charged but cannot be excited (Guyton, 1966, p. 58).

How does a membrane become charged? Membranes have the property of gaining or losing certain chemical ions, namely the positively charged potassium ions (K+) and to a lesser degree the negative chloride ion (Cl—). As the K+ is much more abundant inside the cell than outside; it will tend to leak out and the cell will lose positive charges and become negative. After a time, the cell negativity becomes so strong that it prevents further K+ from leaving (the negative cell attracts the positive ions). An equilibrium will be reached with the inside of the cell becoming negative. This is called the *resting potential* (Guyton, 1966, p. 59; Boriss & Libbert, 1984, p. 310), of about —80 mV in a sensory cell (Benolken & Jacobson, 1970 cited CPN 1:9 (1972). Any leak of negative ions will be balanced by more K+ ions leaving and thus the resting potential remains steady.

The driving force for K+ ion movements is the difference in the intracellular and extracellular concentrations which together with the permeability of the ion, defines the actual value of this resting potential (Jacobson & Stuart, 1974 cited in CPN 4:20 1975). The membrane is now *excitable* and perceives changes in its environment so that any physical or chemical stimulation can switch the membrane to the activated mode.

The *epidermal cells* of the trap all possess excitable membranes and repeated stroking of the external epidermis will cause an electrical response which makes the trap more sensitive when sensory hairs are stimulated (Sibaoka, 1966; Di Palma *et al.,* 1966). Earlier writers denied this reactivity of the abaxial epidermis (Lloyd, 1942, p. 188). Although rough handling is necessary to observe a reaction, the slightest touch to a sensory hair will provoke closure and is considered to be the true trigger for trap closure.

It is the special structure of the sensory hair that is responsible for the low sensitivity threshold. The distal part is a long lever which amplifies any minor force such as a tiny insect brushing against it. Below the lever portion are the thick-walled sensory and podium cells with a thin portion indented around the rosette of sensory cells. When the lever is moved, the thick walls will hardly budge, but all the forces will be concentrated on the thin portion (Haberlandt, republished 1982), and the underlying excitable membrane of the sensory cell will switch to its activated state (Benolken & Jacobson, 1970).

A very light stimulus will have only minimal effects on the membrane: the permeability to K+, although decreased, will always remain greater than the permeability to Cl—. The cell will lose some of its resting potential, but as soon as the potassium permeability has returned to normal, a massive outflow of K+ will restore normal cell negativity. Such a transient and partial loss of negativity, which is proportional to the stimulus, is called a *graded response* or *local potential* since it was not propagated to neighboring cells. The importance of the graded potential is that it represents the electrical translation of a mechanical or chemical stimulation and is capable of switching the membrane to the activated state.

If the intensity of the stimulus is increased, the local potential will reach a threshold where the properties of the membrane change completely. The permeability of K+ drops sharply, and the enzymes pumping Cl— out of the cell are activated. The cell loses sufficient negative charges for the membrane potential to change from the classical —80 mV to +80 mV (Shanos, 1986), the inside of the cell is now positive. This is called the *action potential* (Boriss & Libbert, 1984, p. 19), a very important phenomenon which commands many functions in animals and in plants (Pickard, 1973). Soon after the onset of the action potential, the permeability of K+ is restored and the active transport of chloride stops. Large amounts of potassium now leave the cell following the chemical and electrical gradients until the resting potential of —80 mV is reached once again. The K+ and Cl— lost during the action potential will then slowly be taken back by the cell (Boriss & Libbert, 1984, p. 20). For a study into the ionic movements in *Dionaea;* see Lichtner & Spanswick, 1977 cited in CPN 6:74 1977.

It is worth noting that the cell walls of the sensory and neighboring cells are reinforced with water-repellent (hydrophobic) cutin granules (Buchen *et al.,* 1983, p. 463). These structures are well known from *Drosera* and *Drosophyllum* tentacles (Haberlandt, republished 1982, p. 68 and 73; Diels, L., 1906) and *Dionaea* glands (Robins & Juniper, 1980, p. 280), where they prevent uncontrolled movements of water and solutes in the extracellular space. Are these structures purely vestigial here or are they of some use? The sensory hairs derive from the ancestral tentacles (Juniper, 1987). They may prevent the ions and accompanying water molecules lost during the action potential from flowing away, which would make their re-absorption more difficult.

Now, I would like to discuss some insight into the biochemical mechanisms underlying the action potential.

There was a note of surprise when it was shown that the closed traps of *Dionaea* contain a substance well known from primitive animal muscles: *lysophosphatidic acid.* This is an activator of the enzyme phospholipase D, which breaks up (hydrolyses) some phospholipid components of the membrane (Lea, 1976). One of the hydrolysis products is thought to diffuse into the cytoplasm and triggers a massive release of calcium from the *endoplasmic reticulum* (Wibo, 1987). Now the sensory cells of the hairs contain at both their basal and apical poles an extensive arrangement of concentric smooth endoplasmic reticulum surrounding from one to four vacuoles containing polyphenols (Buchen *et al.,* 1983). Both structures are known storage places for calcium (Buchen *et al.,* 1983, p. 465-466; Boriss & Libbert, 1984, p. 89).

Calcium and magnesium play a major role in the control of enzyme activity, and it was shown that both ions determine the amplitude of the action potential in *Dionaea* (Jacobson & Stuart, 1974 Cit. CPN 4:20 1975). Calcium may be needed for the opening and closing of the channels through which chloride ions are pumped out of the cell (Guyton, 1966, p. 62). Magnesium is probably necessary for the pump enzyme to bind to the fuel which allows it to function: adenosine triphosphate (ATP) (Boriss & Libbert, 1984, p. 303).

The action potential in plants is a rather sluggish affair that is temperature dependent in the refractory period and lasts about one or two seconds (Lloyd, 1942, p. 186-187; Pickard, 1973; Williams, 1980). In comparison, animal nerves have a duration of 1-2 milliseconds (Guyton, 1966, p. 66; Vander *et al.,* 1970, p. 140).

If the cells of *Dionaea* have not restored the normal resting permeabilities, then an action potential cannot be produced. This is called the *refractory period* and it has interesting consequences (Vander *et al.,* 1970, p. 145). If a sensitive hair is touched twice in rapid succession, then the second stimulus will not trigger an action potential (Macfarlane, 1902),

for the sensory cells will still be in their refractory period. This may be of some use to the plant in decreasing the risk of unnecessary closure by the single stroke of a windblown particle or a raindrop, although the latter appears to be a frequent cause of closure (Williams, 1980). If there are two well-separated stimuli, it's likely that prey is moving inside the trap.

The action potential has another interesting property. Not only does it allow the sensory cells to react to the presence of prey, the activated state of their membranes will be *transmitted* from cell to cell until it reaches the tissue responsible for trap closure. When the extracellular fluid becomes negative during the action potential, electrical currents appear which tend to depolarize the fluid surrounding neighboring resting cells. But electrophysiological calculations show that because of the thickness of intervening walls, the cells are too far apart for this depolarization to trigger an action potential. This transmission presumably occurs through small cytoplasmic tubes connecting the cells, called *plasmodesmata* (Mackie, 1970 cited in CPN 1:13 1972). At this level, the membrane of the cell is continuous with that of its neighbor. Plasmodesmata are especially abundant between the sensory and podium cells of the sensitive heir (Buchen *et al.*, 1983, p. 463 and 467), and presumably also link together all the epidermal cells. The different densities of plasmodesmata over the surface may explain why diffusion of action potentials is more rapid in the direction perpendicular to the midrib, and faster also in the inferior, abaxial epidermis (Pickard, 1973 citing Burdon-Sanderson).

Each time a sensitive hair is stimulated, the action potential will diffuse over the trap surface like a ripple on a pool with a velocity of 6-17 cm/sec (Luttge, 1985) or 10-20 cm/sec (Williams, 1980, p. 75). It will spread over the area of a typical trap in 0.25 seconds (Williams, 1979 in abstract of Takao Sibaoka).

How does the action potential get from the hair and superior epidermis to the lower side of the trap? It probably cannot go through the parenchyma, which is too loose and does not seem to have sufficient plasmodesmata. The depolarization probably goes around the rim of the trap. Already we saw that rubbing the marginal teeth may produce closure, so the epidermis on the edge of the trap must indeed be electrically connected to the abaxial epidermis. There's an intriguing remark in one of Darwin's letters about his being able to block the spreading action potential to the opposite lobe by inflicting a small incision (Godbout, 1978). This would imply that the signal can't cross the abaxial side of the midrib and the parenchyma of the trap.

Finally, it should be noted that the action potential gives rise to a response that stays the same regardless of the strength of the stimulus. This is known as the *all-or-none response.* Once we acquired an understanding of how *Dionaea* detects the presence of prey in the trap, we now can turn our attention to the next act: the closure of the trap itself.

Basically, the trap closes because of very fast growth of the epidermis. What does this imply at the level of the cell walls? Water molecules are constantly moving due to random thermal agitation and a certain fraction of these will cross the cell membrane. Now the cell contains many chemical substances dissolved in its water so that the inside of the cell contains fewer water molecules in a given volume than in the extracellular space. The result is that there is more water moving into the cell than leaving it, and if nothing is done, the cell will swell up and burst. To control this *osmotic flow,* plant cells surround themselves with a resilient cell wall built up of macromolecules (Vander *et al.*, 1970, p. 53). These are linked together by strong covalent and weaker hydrogen bonds. An example of the latter are calcium-bridges between carboxyl groups of protopectin molecules (Boriss & Libbert, 1984, p. 354). When the osmotic inflow of water distends the cell wall, there will be a build up of pressure which will ultimately prevent excess water from entering the cell. When an individual cell needs to grow to cause closure of a VFT, the wall itself must be allowed to soften and expand first. Experimental evidence suggests that acidification of the wall plays a major role here. Indeed, flooding a trap with acid buffer causes closure (Williams & Bennett, 1982). Although the exact mechanism is as yet unknown, the concept itself was recently criticized (Dorffling, 1986).

It is thought that the acidity activates wall enzymes which cut covalent bonds between the protopectin and elastin of the matrix and maybe even bonds involving cellulose (Progress in Botany 44: 188 1982). It is also surmised that enzymes assume their active condition when an excess of hydrogen ions displaces calcium ions which formed bridges keeping the enzyme molecules folded up. It was shown that EDTA, a calcium-binding substance and chelator mimicks acid growth (*ibidem;* Hock, 1984). Another theory suggests that calcium-bridges of the cell wall itself may be broken by the acid pH (Progress in Botany 44: 188 1982). However, acidification of both epidermis surfaces would then cause overall growth of the trap rather than closure (Williams & Bennett, 1982).Closure is caused when only the outer (lower) epidermis expands (due to cell growth) while the inner surface remains relatively rigid. Re-opening of the trap requires reversal of this process.

When the enzymes break up the crosslinks between macromolecules, the wall becomes visco-elastic. This means that it can stretch, but it does not return to its original shape (Boriss & Libbert, 1984, p. 142). The molecules slide past each other and new crosslinks are formed between chemical groups facing one another. One will guess by now that the action potential will cause the membranes to pump acid into the wall which loosens it and allows it to lengthen (Williams & Bennett, 1982, p. 1120). Just as the pumping out of chloride ions provokes the inversion of membrane polarity during the action potential, the secretion of acid consumes a large amount of cell fuel, namely ATP. It was shown that 29% of the total leaf ATP is used up in 3 seconds during closure. Since most of the trap tissues are inactive, the proportion of ATP burned up in the external epidermis may be close to 100% (*ibidem,* p. 1121).

Other experiments confirm the importance of ATP. Exogenous ATP fed to the trap enhances closure rates. Illuminated traps produce more ATP through photosynthesis and close faster than traps kept in the dark. Pure oxygen increases the respiratory production of ATP, and the opposite is true for plants grown in pure carbon dioxide (Jaffe, 1973). The significant loss of ATP also explains why plant traps that are too frequently stimulated tend to become sickly and die (Brown, 1916, p. 76). In its native environment the plant sacrifices a significant amount of ATP to obtain scarce elements like nitrogen which it has no other way of getting. Each unnecessary closure wastes a third of the precious cell fuel and weakens the plant. In the abaxial epidermis, the active transport of hydrogen ions into the cell wall seems to replace the loss of K+ which elsewhere reestablishes cell negativity during the refractory phase of the action potential.

All these activities for trap closure are very much temperature dependent. At high temperatures of 40° C and even at 35° C about half of the time the hydrogen ion outflow produced by *one* action potential seems to be sufficient to bring the cell wall acidity down to the enzyme optimum activity. Experimental evidence shows no enzyme activity at pH 5.0, slow and irregular closure at pH 4.5, rapid closure at pH 4.0 which is the optimum, and slower closure at pH 3.0 (Williams & Bennett, 1982). This seems a likely explanation of the results. At usual temperatures, two action potentials are needed to move enough hydrogen ions (Macfarlane, 1902).

The need for two stimuli brings up two interesting consequences. The second stimulus cannot come too quickly after the first because of the refractory period (Williams, 1980, p. 77). If two hairs are touched almost simultaneously, the two fronts of the action potential all cancel where they meet, the opposite cells being in their refractory phase (Williams, 1980, p. 77). If the second stimulus comes within 20 seconds of the first but after the refractory period, the action potential will have a shorter rise and will travel twice as fast as the first (Williams, 1979-review of Sibaoka, 1979). Presumably some of the effects of the preceding depolarization still linger on; maybe the re-uptake of calcium ions by the endoplasmic reticulum was not completed and more chloride and hydrogen ion pumps will be stimulated during the second potential (Hock, 1984, p. 155).

When the interval between stimuli is increased, not only will the re-uptake of calcium be completed, but increasing amounts of hydrogen ions are taken back into the cells. The pH

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drop following the second action potential is not sufficient to reach the optimum pH of the wall loosening enzymes long enough for the growth to take place. The result is partial closure of the trap and a series of stimulations are needed to obtain complete closure (Brown, 1916). There will also be problems with dwindling stores of ATP, probably responsible for the phenomenon known as "fatigue." Each action potential is slower and less efficient than the preceding ones *(ibidem)*. Sickly plants have insufficient ATP and the closure of their traps is sluggish. Since the number of stimulations necessary to close a trap increases with the time interval between them, it seems that at 170 minutes every trace of the preceding action potential has disappeared, and all of the hydrogen ions are taken back with the result that a stimulus at that moment will not contribute to closure (Williams & Pickard, 1979).

The actual driving force for cell lengthening is the osmotic inflow of water into the cells of the external epidermis. This becomes possible due to the cell wall loosening the usual equilibrium between osmotic and elastic pressures. Intracellular pressure drops, allowing the inflow of water which expands the cell wall until a water shortage appears. If cell pressure is increased by firmly holding the trap lobes apart, then closure is very easy to prevent (Schultz, 1965, p. 97). By the end of closure, there will be a localized water shortage. The external epidermis will appear flaccid and the water pressure in the leaf will drop by 0.51 bar in one experiment (Williams & Bennett, 1982).

Regulatory mechanisms will come into action. The internal epidermis will adapt to the water shortage by decreasing its osmotic pressure. Sugar molecules will be joined into starch. Amyloplasts will be observed in cells 15 minutes after trap closure (Brown, 1916, p. 79-80). The external epidermis must prevent its cytoplasm from being diluted by water inflow so the excess is pumped into the central vacuole (Boriss & Libbert, 1984, p. 509). After 15 to 20 minutes the water pressure in the leaf has returned to normal and with the crosslinks established, the normal elasticity of the cell walls is restored. At that moment, the experimental suppression of osmotic pressure is unable to cause the trap to re-open as happens if this same experiment is done immediately after closure (Brown, 1916, p. 78).

Everybody knows the famous biology class experiment where a VFT is anesthesized with ether vapours and the trap fails to close after stimulation of the sensory hair. This can be explained by the ether blocking the membrane pumps and thus preventing the action potential. If ether is used on a closed trap, the lobes become floppy and the trap is easy to re-open. I use this trick to observe VFT surfaces under the microscope without them curling up all the time. The ether probably blocks the membrane pumps involved in osmoregulation, preventing the cells of the abaxial epidermis from maintaining the concentrations of solutes within bounds. This is usually done by pumping the excess water into the central vacuole and the uptake of extracellular ions (Boriss & Libbert, 1984, p. 509).

The amount of actual growth varies enormously. The cell lengthening is maximal in the center of each lobe-about 28% (Williams & Bennett, 1982). Other experiments yield rates between 0 and 30% (Brown, 1916, p. 71). A trap may be made to close for a number of times, but each successive time the growth will be less pronounced *(ibidem,* p. 76 with successively 13, 9, and 3.2% per closure). Other factors such as temperature and the health of the plant play a role. Mechanical closure (incomplete? may be obtained 7 to 10 times (Batalin, 1877 cited in Lloyd, 1942, p. 190). If digestion takes place each time, then reopening after the third closure will be slow and the trap usually dies while trying to digest the fourth capture (Schultz, 1965, p. 97). The lesser number of closures may be due to the additional use of ATP for digestion.

One major question remains on why the action potential which reaches both the internal and external epidermis only affects the external epidermis growth. In the experiment with the acid buffer, the trap closes even though the acid enters both tissues. A selective acidification of the cell walls of the abaxial epidermis does not seem to be the explanation. An unstimulated trap does grow, but much more slowly than during closure of course: 3.1% in 18 days; 1.4% and 9.6% in 7 days in three of Brown's experiments (Brown, 1916, p. 74-76).

This shows that cell wall loosening enzymes must be present in both epidermal surfaces because they must both grow, otherwise the trap will close. Measurements after closure show a total absence of growth in the abaxial epidermis *(ibidem,* p. 73). It is concluded that the action potential strongly stimulates growth on the abaxial side of the lobes, while blocking the enzymes on the inside of the trap. Whether the specificity of the epidermis is brought about by the influence of light, of gravity, or by phytohormonal gradients remains to be established.

The trap becomes a small cage immediately after closure. The marginal teeth are interlocked and almost perpendicular to the plane of the lobes. They form the bars of the cage (Lloyd, 1942, p. 193). They often allow small prey to escape so as to avoid wasting the ATP necessary for digestion of low amounts of nitrogen. In cultivated plants, even large prey are sometimes observed getting out of a trap. One may think this is related to the plant's health, but even in nature escapes are common. (40 to 60% of captures in the field study conducted by Williams, 1980, p. 75). The relative water shortage in the external epidermis just after closure and the fact that all the cell wall crosslinks are not restored make this a critical moment for the trap. Many a prey manages to push apart the floppy margins to flee.

Inside the trap the internal epidermis of the two lobes are not in actual contact. The captures tend to move around unceasingly, touching the sensory hairs and causing hundreds of action potentials (Dubosky, 1975; Affolter & Olivo, 1975). These result in a certain amount of slow growth tightening the closed trap (Lichtner & Williams, 1977, p. 884). Eventually, the internal epidermis will come into contact except near the midrib *(ibidem* p. 886). Large prey may be crushed during this narrowing phase which may take from 30 minutes to 12 hours (Lloyd, 1942, p. 189; Schultz, 1965, p. 96; Lloyd, 1942, p. 178). Small captures like ants stay alive in the midrib region. They will only be killed when the secretion of acid digestive fluid begins 5 to 11 hours after closure (Lichtner & Williams, 1977, p. 886). From that time on, there will be no more action potentials and other mechanisms become responsible for maintaining trap closure (Affolter & Olivo, 1975 cited in Lichtner & Williams, 1977, p. 885).

If a trap is closed by mechanical stimulation, there are no action potentials after closure, no chemical stimuli, no narrowing phase, and the trap soon reopens (Lloyd, 1942, p. 193). This shows that chemical stimuli continue to play a major role, even with similar results as action potentials. Actual closure occurs if moist organic matter is deposited on the internal epiderm without triggering the hairs. The movement is nowhere as rapid as after mechanical stimulation, though the narrowing phase will be induced after mechanical closure if adequate chemicals are left inside the trap (Lichtner & Williams, 1977, p. 884-885; Schultz, 1965, p. 96). A trap will remain closed as long as it is perfused with those same substances. In these three experiments above, no action potentials are recorded apart from the ones associated with initial mechanical closure (Affolter & Olivo, 1975 cited in Lichtner & Williams, 1977, p. 885). A three percent solution of saline causes a series of action potentials of abnormal amplitude and duration for several hours sometimes (Balotin & Di Palma, 1963 cited in Lichtner & Williams, 1977; Shanos, 1986). This will be due to osmotic or chemical damage to the cells (Lichtner & Williams, 1977, p. 885). Reports that leaf extracts and insect exudate cause *action potential-mediated* closure must be verified (Balotin & Di Palma, 1963 cited in Shanos, 1986). The exact mechanism of chemically induced closure remains unknown. If a prey is crushed during the narrowing phase, the sodium ions in its haemolymph will be a powerful stimulant. The amino acids glycine and lysine are less important but probably do play a role. Saltfree albumin, glucose and potassium chloride are inefficient as stimulants (Lichtner & Williams, 1977, p. 885). Some insects, like flies, excrete a great deal when caught, and this may act as a stimulus also *(ibidem,* p. 886).

Other captures, like ants, will not produce organic fluid except maybe the formic acid they spray around when frightened until they are being digested *(ibidem,* p. 886). Ammonium ion, a decomposition product, is a potent stimulant, probably relaying the action of the preceding substances as digestion progresses *(ibidem,* p. 885-886). Because the epidermal cell's cuticle is impermeable, the only way chemicals are able to enter the leaf is

through the glands. From there, experiments with radio-labelled substances show them to be distributed throughout the plant. The external epidermis gets twice as much as the adaxial epidermis (Luttge, 1965, p. 336). The external epidermis has to complete its cytoplasmic growth after the longitudinal growth which took place during closure. How the growth of the adaxial epidermis prevents being inhibited by the presence of organic matter is still something of a mystery. It is fairly certain that phytohormones play a role (Lichtner & Williams, 1977, p. 886). Topical use of growth hormones causes trap closure, which lasts for at least 48 hours. The same variations of subepidermial liquid pressure as we mentioned following the action potential are observed here (Kondo & Yaguchi, 1983 cited in CPN 12: 75 (1983).

Auxins stimulate the hydrogen ion pumps like the action potential. This is one of the mechanisms through which they promote growth (Boriss & Libbert p. 509). But the exact way in which the hormonal balance of the internal epidermis is changed so as to alternatively allow and inhibit its growth, is far from being known yet. After digestion is completed and the products are absorbed (usually after ten days: Lloyd, 1942, p. 178; 7 to 10 days depending upon the type of prey: Schwab *et al.*, 1969; sometimes several weeks: Schultz, 1965, p. 96), the internal epidermis starts growing again (Brown, 1916, p. 73-74; Williams & Bennett, 1982, p. 1121) and the trap reopens.

There is one last strange consequence of the closing and reopening mechanisms: Since these involve growth, the trap on re-opening is larger than before.

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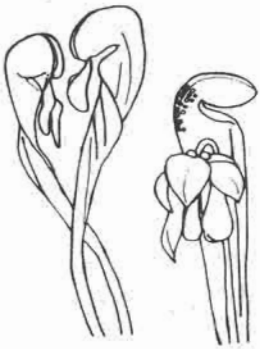
NOTE: Dues for 1989 will be **UNCHANGED**



CARNIVOROUS PLANT NEWSLETTER

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Front cover: *Dionaea muscipula* grown by Peter D'Amato. Photo by Charles Powell II. Please see article beginning on page 80.

Rear cover: *Utricularia sandersonii* grown and photographed by Charles Powell II.

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