Evolution of Flowering Plants by Fungus-to-Host Horizontal Gene Transfer

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ABSTRACT Horizontal gene transfer from parasitic or symbiotic fungi to their angiosperm hosts (without a viral intermediary) is hypothesized. All of the capabilities necessary for successful fungus-to-angiosperm horizontal gene transfer are already known in the plant kingdom. Evidence which would be indicative of a horizontal gene transfer event and organisms where this evidence might be found are suggested. Evolutionary consequences of fungus-to-angiosperm horizontal gene transfer are discussed.

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Introduction

This paper proposes the hypothesis that some of the evolution which has taken place in the angiosperms is attributable to horizontal gene transfer (hgt) from parasitic or symbiotic fungi to their flowering plant hosts. No intervening virus is needed as a DNA carrier. Requirements for successful fungus-to-host hgt are specified, evidence which would support the hypothesis is described, and organisms which might possess this evidence are suggested. The possible ramifications throughout botany, if the hypothesis is true, are mentioned.

This paper is not the first to suggest the possibility of hgt. Hyldig-Nielsen et al. (1982) proposed that an animal globin gene may have been transferred to soybean by a viral intermediary, while Bridges and Salin (1981) postulated the transfer of a gene from a bacterium or alga to eukaryotic vascular plants. In addition, Martin and Fridovich (1981) suggested that hgt took place between a ponyfish and its bacterial symbiont, and Busslinger et al. (1982) hypothesized hgt of a histone gene from one sea urchin species to another. Finally, Krassilov (1973, 1977) advanced the idea that hgt was an important factor in the genesis of the angiosperms, but he did not specify a source for the transferred genes. The present paper is the first to suggest hgt from a fungus to its angiosperm host; a viral intermediary is explicitly not required.

Since, to the best of my knowledge, no experiments have ever been designed and performed to test this hypothesis, the arguments and evidence presented here will be suggestive rather than convincing. The evidence I was able to find refers primarily to the fungus-angiosperm interaction, so that this is the pair of eukaryotes upon which I will focus. There is no reason to think that similar arguments would not apply with other eukaryotic plants acting as the recipients of fungal genes.

The hypothesis

Two different routes lead to the hypothesis of hgt. The first starts with experimental observations of the kind found by the authors of the references mentioned above and ends with hgt as a possible explanation; the general principle, hgt, is induced from the particulars. The second route begins with the general principle of interorganismal DNA transfer and ends with hgt; hgt is deduced as a particular instance of the general principle. Known cases of interorganismal DNA transfer include: transduction, transformation, and conjugation (see, e.g. Hayes, 1976); endosymbiosis (Margulis, 1981), a process whereby an entire prokaryote is transferred to another prokaryote; and prokaryote-to-eukaryote hgt, exemplified by *Agrobacterium tumefaciens*, the crown gall bacterium, which carries a plasmid that is inserted in multiple copies into the DNA of the host plant (Holsters et al., 1982; Chilton, 1982; Schafer et al., 1982). The transfer of genetic material from one eukaryote to another by hgt is a logical next step up on this interorganismal DNA transfer hierarchy.

Three conditions for successful fungus-to-angiosperm hgt

In order for evolutionarily relevant fungus-to-angiosperm hgt to take place the following three conditions must be satisfied: 1) The fungus and the angiosperm must be in intimate contact during those parts of their life cycles during which DNA transfer from the donor fungus to the recipient's reproductive cells is possible. 2) Mechanisms must exist in the fungus and the host which enable physical barriers to DNA transfer to be overcome. 3) The transferred DNA must become incorporated into the genome of the host's reproductive cells. Evidence exists that all three conditions can be satisfied by the fungus-angiosperm interaction.

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Condition 1) can be satisfied if the fungus infects the ovules, pollen, anthers, ovary, zygote, embryo, seed, or the meristematic cells which give rise to these structures. Fungi are known which attack precisely these organs and cells. Smuts attack the floral organs directly (Fischer and Holton, 1957), while a variety of fungal pathogens are seedborne, carried either internally as mycelia or as seed coat contaminants (Mathre, 1978). Some fungi colonize seedlings and eventually pass into the apical meristem whence they move into all the plant parts to which the meristem gives rise (Wheeler, 1968). Others overwinter in the buds of perennial plants or simply colonize the entire host by extensive mycelial growth (Wheeler, 1968). Thus, the first requirement can be satisfied by fungi in a number of ways.

Once the reproductive cells of the host are reached, six obstacles to the transfer of fungal DNA remain. In moving from the nucleus of the fungus to the nucleus of the angiosperm, the fungal DNA must pass through the fungal nuclear membrane, fungal plasmalemma, and fungal cell wall, as well as these same structures in the host. These are the barriers to DNA transfer of condition 2), and they are formidable. Nevertheless, mechanisms are known, some in the fungus, others in the host, whereby these barriers can be overcome.

Fungi have many ways of surmounting the obstructions presented by the host cell wall, plasmalemma, and nuclear membrane. The fungal symbionts in some crustose lichens, for example, have the capability of forming intercellular haustoria which can penetrate the protoplast of the algal symbiont without killing it (Hale, 1974). Other fungi produce pectinases which attack the cell wall and disrupt the permiability of the plasmalemma, nuclear membrane, and other cell membranes (Williams, 1979; Misaghi, 1982; Griffin, 1981; Wheeler, 1978). Many plant pathogens produce cellulases, hemicellulases, and lignin-degrading enzymes (Mount, 1978; Griffin, 1981; Misaghi, 1982). Others produce lipases which attack the host cell membranes (Griffin, 1981). It is known also that during pathogenesis host organelles, cells, and tissues are lysed by fungal enzymes and membrane permiability is altered (Aist, 1981). Thus the barriers posed by the cell wall, plasmalemma, and nuclear membrane of the host can readily be breached by the fungus using its enzyme arsenal.

There may also exist a less destructive method of moving fungal DNA into the host. It is known that short pieces of viral DNA are capable of moving from one plant cell to another via plasmodesmata (Misaghi, 1982), and short pieces of fungal DNA might do the same (if, perhaps, their chromosomal proteins have been removed?). In addition, viral DNA is capable of entering subcellular organelles, such as nuclei and chloroplasts (Misaghi, 1982), and short pieces of fungal DNA might have this capacity also.

Just as fungal enzymes overcome obstacles to DNA passage in the host, host enzymes overcome obstacles to DNA passage in the pathogen or symbiont. In infection by endotrophic mycorrhiza, for instance, much of the contents of the fungus is known to pass into the host cell (Harley, 1968), indicating that the walls and membranes which constrain the fungal cell contents are damaged in some way by the host. In symbiotic fungi of orchids, the hyphal coils in some parts of the host are digested leaving behind an unorganized mass, and it is believed that generally hyphae within the host tissue lose substances to the host both during this digestion and also possibly directly across the membranes of living filaments (Harley, 1969). Glucanases and chitinases have been found in host plants and presumably play a role in host plant resistance to infection by degrading the glucan and chitin components of the fungal pathogen (Misaghi, 1982), and phytoalexins in higher plants are known to cause damage to the mycelia of fungal parasites (Misaghi, 1982). All this evidence suggests that under certain conditions both fungal and host barriers to DNA transfer might be overcome, so that the fungus-angiosperm interaction would satisfy condition 2).

Condition 3), the assimilation of the transferred fungal DNA into the genome of the angiosperm host, is, in my view, the most difficult requirement to fulfill. Even so, circumstantial evidence is available which shows that even this last hurdle can be jumped. For example, transposable DNA elements in maize are inserted and excised more or less anywhere in the maize genome (Fincham and Sastry, 1974), and the yeast, *Saccharomyces cerevisiae*, likewise contains a family of such transposable elements (Finnegan et al., 1982). As mentioned above, the Ti-plasmid carried by *Agrobacterium tumefaciens* becomes integrated into moderately repetitive sequences of a variety of host plants (Schafer et al., 1982). Moreover, some of the dispersed DNA repeats in cereal genomes are probably transposable elements (Flavell, 1982) which are thought to be excised, replicated, and then reintegrated into the plant's own chromosomes. These examples provide evidence that mechanisms for the assimilation of foreign or additional DNA into a eukaryotic plant genome already exist, and so could perhaps play an important role in the integration of fungal DNA into the host DNA.

Further hints that DNA transfer and incorporation can and have occurred are supplied by symbionts and endosymbionts. It is known that the globin portion of leghemoglobin in soybean nodules is synthesized and coded for in the host while the heme portion is synthesized and coded for in the symbiotic bacterioid (Bergersen, 1980; Nadler and Avitar, 1977), and that some mitochondrial and some chloroplast factors are coded for by nuclear genes (see, e.g. Grun, 1975). This means that gene transfer has successfully taken place between symbionts or endosymbionts and their hosts (or vice versa) sometime in the past.

Host cells contain, of course, the full complement of genes for the enzymes which carry out DNA replication, transcription, and repair. It seems possible that these cells might integrate fungal DNA into their own genomes by mistake, especially if the structure of their DNA or the process of replication is interfered with. And the signs are that fungal pathogens do interfere with DNA and its replication. It is reported that the structure of nuclei and nucleoproteins of pea cells infected with *Fusarium solani* are changed (Misaghi, 1982), and that modifications in the structure and function of chromatin have been observed in rust-infected wheat leaves (Misaghi, 1982). Furthermore, in the early stages of pathogenesis, protein and nucleic acid metabolism are known to be altered (Wheeler, 1978). Perhaps RNA host polymerases are modified by a fungal parasite or symbiont in the same way that host metabolism is altered by bacteriophages and plant viruses (Samborski et al., 1978; Misaghi, 1982). Moreover, fungi produce many plant hormones: auxin, gibberellins, cytokinins, and ethylene (Williams, 1979; Misaghi, 1982), some of which are capable of influencing cell division. In light of all this evidence, it seems to me conceivable that once the fungal DNA is transferred to the reproductive cells of the host, it could enter the host nucleus and become integrated into the host cell genome, satisfying the third requirement for fungus-to-angiosperm hgt.

Fungus-to-angiosperm hgt viewed as a fungus-host interaction

The range of possible host-fungus interactions may be viewed as a continuum running from fungi which are harmless to their hosts, through those which exist in a symbiotic equilibrium with their hosts, on to those which are able to parasitize their hosts to such an extent that host death is the result. One can imagine that any particular angiosperm-fungus pair whose interaction did not lie near the extremes of the continuum could potentially take part in an hgt event, for any time an infection of host reproductive cells occurs, and the host survives long enough to reproduce, evolutionarily relevant hgt might occur.

A fungus-to-angiosperm hgt scenario

I envision the following scenario as one leading to fungus-to-angiosperm hgt. A fungus infects the reproductive cells of the angiosperm host, destroying cell walls and damaging host cell membranes. The host attempts to repel the invader and marshals its own complement of enzymes which destroy fungal cell walls, damage fungal membranes, and break up fungal DNA. As the host attempts to repair its damage, including the harm done to its DNA, fungal enzymes interfere with the specificity of the host DNA and RNA polymerases, and fungal hormones interfere with or induce replication and cell division in the host. After cell and nuclear division are complete, one or both of the daughter cells contain genes from the fungus integrated by mistake into their own chromosomes.

How often might fungus-to-angiosperm hgt occur?

It is impossible to give an estimate of the frequency of occurrence of this kind of hgt, but what can be done is to mention the sorts of factors which would need to be considered in making such an estimate. One factor has already been mentioned above, namely, that many fungus-angiosperm pairs are candidates for hgt, and among all the infections found in these pairs any which allowed the host to reproduce might represent an hgt event. Since a single host is prey for a number of different pathogens, and since a single pathogen may attack many different hosts, the possibilities seem endless. Thus, if hgt occurs with any regularity, one would expect at least one example of it to have been found; yet, none are known. Still, this lack of examples is perhaps not so surprising even if hgt does occur, since, as we shall see, evidence which would require fungus-to-angiosperm hgt to explain it is not easy to obtain and is certainly not likely to be stumbled across during experiments which are not designed specifically to detect it. Perhaps fungus-to-angiosperm hgt has not been observed simply because no one has thought to look for it!

The kind of evidence needed and where one might find it

Ideally, the evidence for hgt should consist of a known ancestral organism, a fungus which infects it, and an organism known to be a descendant of the ancestor and known to possess a gene or genes which are present in the fungus and are absent in the ancestor. (Please note that ancestor and descendant here do not refer exclusively to ancestor-descendant species but may also refer to ancestor-descendant plants, populations, subspecies and so forth.) In addition, one would like evidence showing that it is unlikely that the purportedly fungal genes arose by normal mutational events. This additional evidence might come in the

form of DNA sequences (perhaps untranscribed) which flank both the genes in the descendant and the same genes in the fungus. Flanking sequences are particularly important bits of evidence, since a useful gene might arise by mutation in the descendant, but it is hardly likely that adjacent sequences (especially ones which are untranscribed) would do so also.

Other somewhat less conclusive evidence that hgt had occurred would be the presence in the descendant of more than one fungal gene, since, as the number of such genes increased, the probability that they all arose by normal mutation would rapidly decline. Evidence carrying similar weight would be a fungal gene possessed by an individual whose ancestors were hosts to the fungus, but not possessed by descendants of non-host ancestors.

Even less strong evidence, but still useful in the preliminary stages of an investigation, would be an enzyme or metabolic pathway which was shared by the descendant and the fungus, but was lacked by the ancestor. Of course, evidence of the kind mentioned above would be needed in order to confirm that an hgt event was responsible.

Based on the sort of evidence needed, a number of places come to mind where it might be sought. The first is species pairs for which the ancestor-descendant relationship is known, and in which the descendant has diverged relatively recently. Examples of such species pairs known to me are (listing the ancestor first): *Stephanomeria exigua* and *S. malheurensis* (Gottlieb, 1979), *Clarkia biloba* and *C. lingulata* (Gottlieb, 1974), *Gaura longiflora* and *G. demareei* (Gottlieb and Pilz, 1976) and *Wislizenia refracta* and *Oxystylis lutea* (Iltis, 1957). These organisms have a number of features recommending them. First, if the new species has arisen by hgt, there will not have been much time either for the fungal gene in the descendant to mutate away from the form it had in the fungus, or for the flanking sequences, if present, to become changed by random mutation. Furthermore, any genetic analysis would be simplified, since the genetic background would be nearly identical in both species. I do not mean to imply that the descendant species of a closely related pair of species is any more likely than any other organism to have arisen by hgt; it simply seems to me that if hgt had occurred in the descendant it would be easier to discover it there than in most other situations. Ancestor-descendant angiosperms at other lower taxonomic levels, e.g. subspecies, varieties, populations, and so on could also be examined for hgt, with the same advantages.

Another area which might harbor clues to fungus-to-angiosperm hgt is the alternate hosts of heteroecious fungi, such as *Berberis vulgaris* and the various grasses which are hosts for *Puccinia graminis*, (Wheeler, 1968; Alexopoulos and Mims, 1979), *Vaccinium myrtillis* and *Rhododendron ferrugineum* which are the hosts for *Sclerotina rhododendri* (Wheeler, 1968), and *Vaccinium uliginosum* and *Ledum palustre*, which are the hosts for *Sclerotina heteroica* (Wheeler, 1968). Here one could look for the same fungal genes in both hosts, or for one or more different fungal genes in each host, or even for genes from one host in the other, the fungus having been the intermediary. One would also hope that a fungal gene found in one or both hosts would not be found in closely related non-host organisms; this would indicate that the gene did not arise by normal mutational means, although stronger proof involving DNA sequences would be necessary to cinch the argument.

Or, one might, using a host index (Seymour, 1929; Greene, 1951; Moore, 1959), find the hosts for a single fungus, and examine them for purportedly fungal genes which are lacking in closely related non-host organisms. Cultivated plants, too, could be promising organisms in which to search for hgt because so much is known about their genetics and the genetics of their pathogens. When one considers the large numbers of genetically identical plants that are planted and the huge numbers of infections which take place, this seems to be a situation ripe for hgt. Of course, in order to test for hgt one would need to examine the next year's progeny planted from seed taken from infected previous year's hosts.

In fact, in my opinion, the most worthwhile organism pair in which to look for hgt is a cultivated plant, *Zea mays* subsp. *mays* (corn) and its ancestor, either *Z. mays* subsp. *mexicana* or *Z. mays* subsp. *parviglumis* (both teosintes). Iltis (1983) has shown that *Z. mays* subsp. *mays* almost certainly evolved from *Z. mays* subsp. *mexicana* or *Z. mays* subsp. *parviglumis*, and that this evolution was accompanied by a set of structural and physiological consequences which he calls the 'Catastrophic Sexual Transmutation', whereby the teosinte tassel which terminates each of the primary lateral branches becomes the maize ear by transformation, condensation, and feminization. Iltis (1983) suggests that these changes might be the result of growth-substance-releasing pathogens, but he does not suggest that hgt was involved. Nevertheless, based on Iltis's work, I hypothesize that hgt played a role in the creation of corn with the most likely candidate for the pathogen being *Ustilago maydis* which infects not only *Z. mays* subsp. *mays*, but also *Z. mays* subsp. *mexicana* (Fischer, 1953), and presumably also *Z. mays* subsp. *parviglumis*. Any young meristematic tissue is susceptible to infection (Fischer and Holton, 1957), so that the fungus comes into direct contact with those cells giving rise to the reproductive structures. Furthermore, *Ustilago* sp. are known to cause sex change in both *Z. mays* subsp. *mays*, and in members of the Caryophyllaceae, and *U. avenae* and *U. kolleri* both cause dwarfing and stunting

in oats (Fischer and Holton, 1957). Sex change and dwarfing correspond to the feminization and condensation required by Iltis's theory. I therefore suggest that corn evolved from teosinte by hgt from the fungus *U. maydis* to the host *Z. mays* subsp. *mexicana* or *Z. mays* subsp. *parviglumis*, and that, as Iltis (1983) points out, human selection for the resulting organisms played a necessary part in the ultimate creation of the taxon *Z. mays* subsp. *mays*. Indeed, initially the human selection may have been, not for corn, but for teosinte susceptible to infection by *U. maydis*. Madariaga (1919, cited in Fischer and Holton, 1957), points out that the young smut galls of corn infected with *U. maydis* are used as food in Mexico, and that it is a very old custom throughout Mexico to preserve infected corn in various ways. Perhaps infected teosinte was eaten and preserved in a similar way before corn itself existed, and selection for teosinte strains susceptible to infection hastened corn's eventual arrival.

Implications

Consequences of fungus-to-angiosperm hgt, if it occurs, could be many and far-reaching. Some of the observed convergent evolution in angiosperms (see, e.g. Mooney, 1977) might have been caused by genes which were passed to angiosperms via hgt from a single fungus to a number of different hosts. One of the useful recent methods in systematics, cladistics (see, e.g. Hennig, 1966), relies on shared derived characters (synapomorphies) for creating phylogenetic trees. If some shared derived characters are the result of hgt of the genes for those characters, cladistic analyses based on them would be incorrect. Arguments as to whether a character, such as vessels, were primitive or advanced (in the group being examined) (see e.g. Young, 1981; Carlquist, 1983) would become more complicated if capacity to produce vessels were attributable to genes passed to an organism via hgt. (The differentiation of vessel elements as described in e.g. Esau (1977) sounds to me similar to what occurs during the attack of a plant cell by a fungal pathogen.)

Or hgt might be the cause of those periods of accelerated speciation which figure so prominently in discussions of punctuated equilibrium (see e.g. Stanley, 1979). And the ascendance of the angiosperms might be the result of their ability to take part in hgt. Instances of putative polyphyletic origin of characters, such as angiospermous characters (Krassilov, 1973, 1977) or the C4 and CAM photosynthetic pathways (Teeri, 1982), might be the result of fungus-to-angiosperm hgt, although since fungi are not autotrophic, they may merely have acted as intermediaries of genes for the C4 and CAM pathways. Hgt might supply self-fertile and apomictic organisms a mechanism for generating variability and could account for the surprising amount of variability these organisms sometimes possess. Finally, fungus-to-angiosperm hgt could be a useful tool in eukaryotic plant recombinant DNA work.

Concluding remarks

Fungus-to-angiosperm hgt may not occur at all, or it may occur so infrequently that it is insignificant relative to other means of DNA modification. On the other hand, it may take place so often that it must be considered a significant evolutionary mechanism, worthy of study in its own right. The hypothesis of fungus-to-angiosperm hgt, however, can only be affirmed or denied by experiments specifically designed to detect it.

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