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Recovery of vertical resistance and conversion of breakdown by potato plants suffering from root -knot nematodes

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INTRODUCTION

Most people think that deciduous trees shed their leaves in order to avoid a winter, or a tropical dry season. And so they do. But this is not the only reason. They also shed their leaves to achieve a break in their parasitism, and to resuscitate their biochemical locks. This additional function of leaf-shedding explains several conundrums that baffled botanists for years. For example, it explains why a temporary resistance should evolve in a tree that lives for centuries. It also explains why a tree such as rubber (Hevea brasiliensis) should be deciduous, and have vertical resistance to a disease called leaf blight (Microcyclus ulei), even though it occurs wild in the Amazon valley, which is continuously warm and wet. And it explains why the members of the Mendelian school could not find any single-gene resistances in various important crops derived from wild plants that have continuous epidemics, such as sugarcane, citrus, and olives.

Plant hosts cannot re-lock their biochemical locks, but they solve this problem in another way. They regularly destroy all tissue that has a biochemical lock, and that has probably been matched

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by the end of a discontinuous epidemic. The only host tissue that has a lock is seasonal tissue, and it is discarded at the end of each season. All the locks that have been unlocked by the parasite are destroyed by leaf-fall in a deciduous tree, or the death of all tissues, except the seed, in an annual herb. Come the end of the season, the parasite is out in the cold, and on its own.

The biochemical locks are not re-locked but, in the new season, they are replaced with new tissues that are both parasite-free, and have locks that are unmatched and functioning. This is the importance of discontinuity. In each new epidemic, there has to be a successful infection of each host individual, if the epidemic is to develop fully. That successful infection must be an allo-infection. And it must be a matching infection. At the beginning of each new season, the system of locking is fully functional again.

MATERIAL AND METHOD

The loss of seasonal tissue represents the "recovery" of vertical resistance, and is the converse of the "breakdown." In the course of one complete seasonal cycle, the state of the vertical resistance can change from being unmatched and functioning, to being matched and broken down, to being unmatched and recovered. This corresponds to a system of both unlocking and re-locking. And the system of locking can endure indefinitely.

For example, the system of locking continues to function as young deciduous trees replace old deciduous trees in a forest that might endure for millions of years. The only criterion is that the diversity of locks and keys must be maintained, and there are various genetic mechanisms that can ensure this. The system of locking will also endure indefinitely in an ecosystem of annual plants, as new unmatched plants replace the dead, matched plants of the previous season.

It seems that discontinuous epidemics are always caused by r-strategist parasites. They have to be r-strategists, if they are to exploit a food supply that appears very suddenly at the beginning of a favourable season, and then disappears, equally suddenly, a few weeks later, at the end of that

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season. Small organisms, such as microscopic parasites, and tiny insects, can take full advantage of such an abundant, but short-lived, food supply only if they have a population explosion.

However, there is a serious problem with population explosions. Like chemical explosions, they are tricky things. They are thoroughly unreliable, and they can very easily get completely out of hand. They are difficult to stop, once they have started, and they equally difficult to curb and restrain. And they can do a great deal of damage if they are not restrained. In an abnormal season that favored the parasite, there could be a population explosion so vast that the very survival of the host population was seriously threatened. And, if the survival of the host is threatened, the survival of the parasite is threatened with it.

This, then, suggests the function of the system of locking conferred by vertical resistance. It is to slow down the population explosion of an r-strategist parasite. It is to stabilize an otherwise unstable, unreliable, unpredictable, and thoroughly dangerous situation. The host population simply cannot afford to be periodically devastated by a parasite population explosion. And the parasite simply cannot afford to devastate its host population because, to do so, would threaten its own survival. So, the two species have evolved an incredibly elegant system of locks and keys that prevents damaging population explosions and, at the same time, ensures the survival of the parasite without excessive damage to the host.

Support for this conclusion comes from the vertical resistance to Hessian fly (Mayetiola destructor,) which is a stem borer of wheat. This resistance is exceptional in that it is quantitative vertical resistance. Although its inheritance is qualitative (i.e., Mendelian), its effects are quantitative. That is, it confers incomplete resistance to non-matching strains of the insect, and no protection whatever against matching strains. This means that a non-matching strain of the fly can allo-infect a wheat stem, and survive within it.

With quantitative vertical resistance, a non-matching infection does not kill the parasite. It merely slows the growth of the parasite, and prevents it from reaching maturity. At first sight, this is ludicrous because this kind of resistance does not control the parasitism. Quantitative vertical

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resistance appears to have no evolutionary survival value. And, if it has no evolutionary survival value, why should it evolve at all?

The answer appears to be that quantitative vertical resistance did not evolve to prevent allo-infection, or even to prevent parasitism. It evolved to prevent damaging population explosions, and it does this by controlling the reproduction of the parasite. And this is probably the ultimate function of all vertical resistances. A few infections, and a little damage to the host population, are quite unimportant compared with the disaster of an uncontrolled population explosion in the parasite.

We have seen that vertical resistances appears to reduce parasitism by reducing the frequency of matching allo-infection. And, at first sight, this reduction of parasitism appears to be the obvious function of vertical resistance. In fact, the ultimate function of vertical resistance is probably to reduce reproduction in the parasite and, hence, the control of population explosions in the parasite. Most vertical resistances achieve this by the simple expedient of controlling allo-infection. A few do it by allowing allo-infection, allowing some parasitism, and some growth of the parasite, but by either preventing, or greatly reducing, parasite reproduction.

But this is a digression. Let us return to the two kinds of epidemic. In practice, this difference between continuous and discontinuous epidemics is crucial to the functioning of vertical resistance. Consider the epidemics of a leaf parasite of a hypothetical tree. If the tree is deciduous, the epidemic is discontinuous, and the vertical resistance will function at the start of every new epidemic. If the tree lives for, say, five hundred summers, its vertical resistance will protect it through five hundred epidemics. By chance, in a few of these epidemics, the tree will be matched quite early in the season, and it will suffer accordingly. However, every tree can tolerate an occasional bad epidemic. Equally, in a few of these epidemics, the tree will be matched so late in the season that it suffers no parasitism at all. On average, it will be matched sufficiently late for the parasite to do only very minor damage in each season.

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Now consider an evergreen tree which has a continuous epidemic. Its first infection must be an allo-infection but, after that, it can remain parasitised by auto-infection for the rest of its life, and all auto-infection is matching infection. Vertical resistance would protect this evergreen tree only until the first matching allo-infection occurred, probably when the tree was still a very young seedling. The vertical resistance would then be useless for nearly five hundred subsequent summers. A gene-for-gene relationship cannot function in a continuous epidemic and, consequently, its evolutionary survival advantage is negligible. For this reason, a gene-for-gene relationship never evolves in host-parasite systems that have continuous epidemics.

This, then, was the bane of Mendelian breeding for resistance. If a crop is derived from a wild plant that is an evergreen perennial, it will have horizontal resistance but no vertical resistance. Conversely, if the wild progenitor of a crop is an annual herb, or a deciduous tree or shrub, that crop will have both horizontal and vertical resistances. The evolutionary survival value of a gene-for-gene relationship in a discontinuous epidemic is remarkable and, for this reason, it will often, but not necessarily, evolve in annual herbs, and against the leaf parasites of deciduous trees and shrubs. A Mendelian breeder, looking for a genetic source of qualitative, vertical resistance, will not find it in evergreen perennials. He may find it in crops with discontinuous epidemics, but he will not necessarily do so. A biometrician, on the other hand, looking for quantitative, horizontal resistance, will invariably find it, in any crop, and against any parasite of that crop.

It was remembered that a Mendelian breeder needs a genetic source of resistance. If he cannot find it, the resistance breeding cannot even begin. A biometrician, on the other hand, does not need a genetic source of resistance. He needs merely to increase an existing level of quantitative resistance by changing gene frequencies in a mixed population. He can thus breed any crop for resistance to any parasite, and he can do so without first finding a source of resistance.

Appendix A

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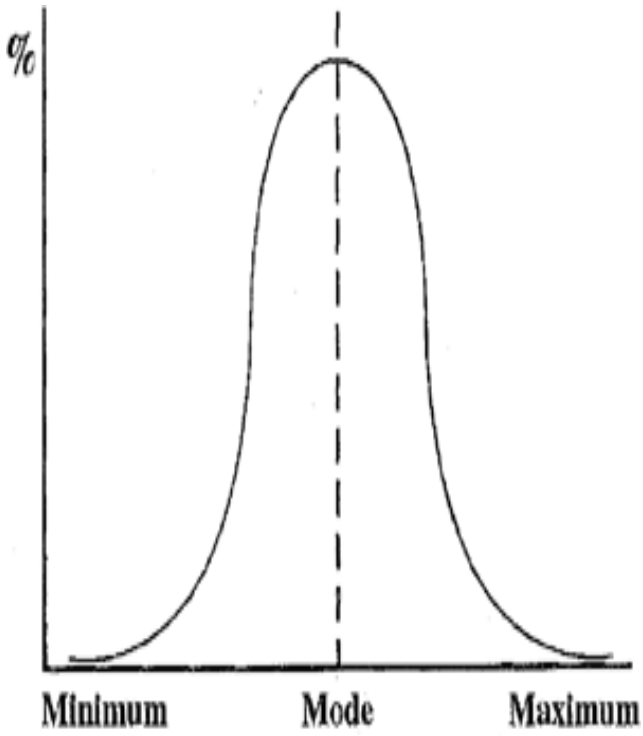

This figure shows a graph that is known as the "bell-shaped" or Gaussian curve. It represents a "normal" distribution of a quantitatively variable character, which might concern height in men, or the level of resistance in plants. This variable is represented on the horizontal axis of the graph, and it ranges from the minimum to the maximum. The percentage of the population, which possesses a particular value of that variable, is represented on the vertical axis of the graph, with the highest percentage at the top. The dotted line is the mode. In a symmetrical curve, the mode is also the mean, or the average, and it is exactly halfway between the minimum and the maximum.

Appendix B

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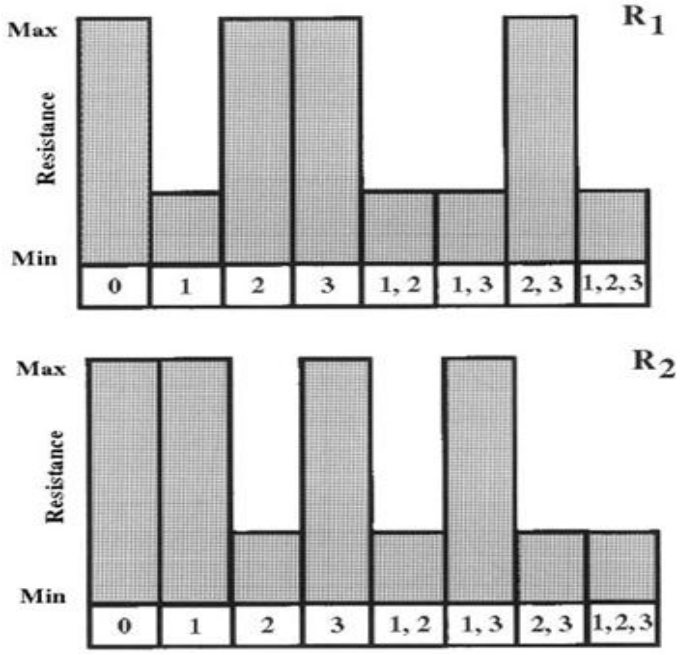

Each of these two diagrams represents a potato clone and its reactions to eight races of the potato blight fungus Phytophthora infestans. The top diagram represents a clone with the resistance gene R1 and it is susceptible to any blight race that possesses the matching gene for parasitic ability. It is also resistant to any race that lacks this gene. The bottom diagram represents a clone with the resistance gene R2 and it is susceptible to any blight race that possesses the matching gene. It is also resistant to any race that lacks this gene. Differences in this kind of resistance are parallel to the vertical axis of the diagram and it is accordingly called vertical resistance. Vertical resistance is thus qualitative resistance in its inheritance and its effects. It is due to a gene-for-gene relationship. (After Vanderplank, 1963).

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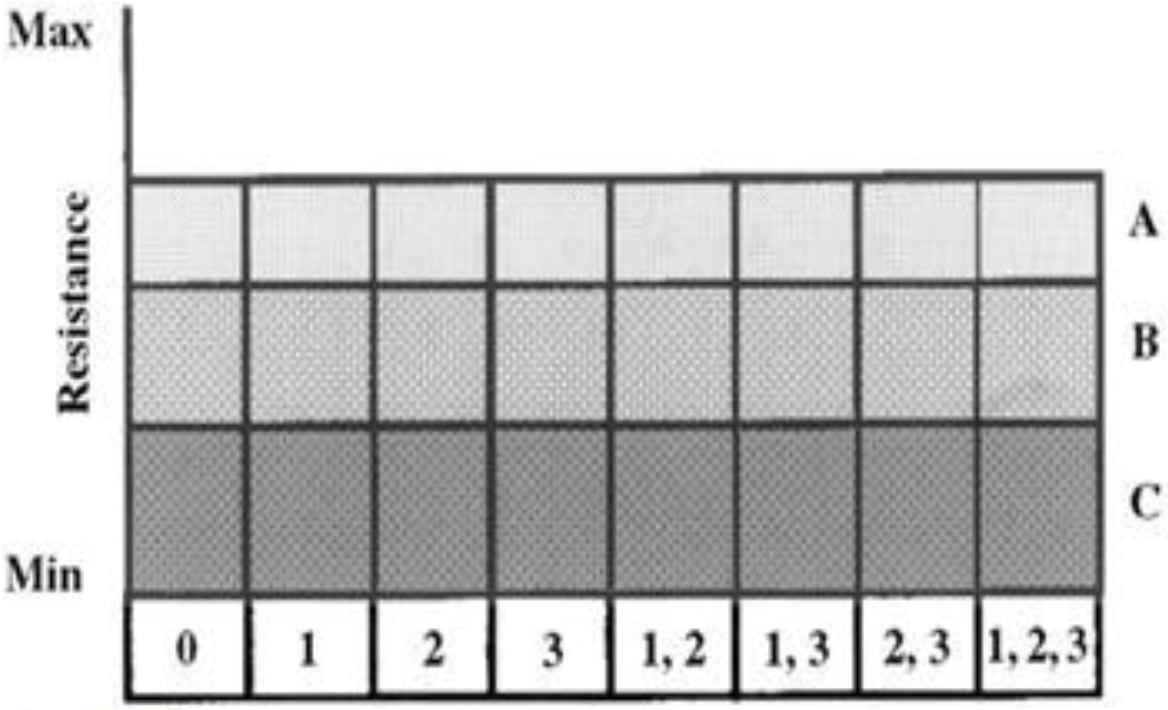

This diagram represents three potato clones which have no vertical resistance genes at all. Each clone is accordingly susceptible to every one of the blight races. However, clone C is more susceptible or, conversely, less resistant, than Clone B, and Clone B is more susceptible than Clone A. Differences of this kind of resistance are parallel to the horizontal axis of the diagram and it is accordingly called horizontal resistance. Horizontal resistance is not due to a gene-for-gene relationship, and it is normally inherited polygenically. It is consequently quantitative in both its inheritance and its effects. (After Vanderplank, 1963).

CONCLUSION

We should note also that most of the crop species in temperate countries have discontinuous epidemics, and vertical resistances, because they evolved in a region that has winters. And most of the research in crop science has been done in temperate regions, and on temperate crops, grown in the wealthy, industrial nations. Conversely, many tropical crops have continuous epidemics, and they lack vertical resistance. But relatively little research has been done on these tropical crops, grown in impoverished, non-industrial countries.

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These differences of climate and research have done much to exaggerate the importance of vertical resistance, and to disguise the importance of horizontal resistance.

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