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Myxomatosis: its decline as a killer

C. D. Gooding

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ALTHOUGH nearly every farmer in Western Australia is familiar with myxomatosis, very few landowners really understand the factor causing the disease to spread or, just as importantly, why the severity of the disease is declining.

Although the disease is spread naturally, it is spread by what is known as a mechanical process. Any one of a number of agents can be responsible. In the confined spaces of a warren, with rabbits huddled closely together, droplet infection occurs to a limited extent. In fact, one of the earlier methods used to spread the disease was to blow a fine mist of myxomatosis virus solution into the burrows. This was expensive, but some inoculation did occur this way.

Insect vectors (the carrying agents) are chiefly responsible for the spread of the disease in the field. The carriage of myxomatosis virus is purely mechanical, unlike malaria where part of the life cycle of the organism takes place within the mosquito. Virus particles adhere to the mouth parts of an insect when it bites a diseased part of an infected rabbit, and are deposited in or under the skins of the rabbits it subsequently bites. In this way living virus is transferred from a sick rabbit into a healthy one.

Any insect which bites rabbits will spread the disease. In early experiments, an ordinary household pin was used to transmit the disease under laboratory conditions. It is clearly obvious that the spread of the disease in the field is primarily determined by the number of vectors available. Other factors, such as a ready source of virus (in the form of sick rabbits) are also important if a spread of the disease is to take place. It is now generally agreed that every wild rabbit in Australia will come in contact with the disease during its lifetime, and that most will survive.

**Myxomatosis versus the Rabbit**

In 1951 the myxomatosis virus was first released into the rabbit population of Western Australia. Immediately this was done, there was set in motion a train of events to which there can only be one end result—"the peaceful co-existence of the two opposing forces." The rabbits in Australia came from parent English stock introduced into Victoria in 1859. They had never come into contact with the Myxoma virus of South America. In 1951 a highly virulent disease was suddenly let
During the recent summer, myxomatosis was widespread throughout most of the wetter parts of Western Australia. It was noticeable that it was predominantly the younger rabbits which were affected. There is a very good reason why the disease strikes hardest at the young rabbits. The disease has now lost a lot of its original killing power but is still capable of making a useful contribution to rabbit control.

loose amongst a completely susceptible rabbit population. The result was dramatic, as those who witnessed the struggle between virus and rabbit will recall. Only two out of every 1,000 rabbits infected with the disease, survived. Clearly, round one went to myxomatosis. The initial blow, although devastating, was not a knockout, and as always happens in nature, “the rabbits rallied and began to fight back.”

There are two basic factors which determine the severity of a disease. These are (i) the virulence of the organism myxomatosis, and (ii) the resistance of the host animal. This resistance can be in two forms—acquired (immunity) and natural (genetic resistance).

Changes in the Virulence of the Organism (Attenuation)

In the constant battle which has raged between myxomatosis and the rabbit since 1951, certain very important alterations have occurred in both opposing forces. It is not in the best interests of the virus to kill off all the rabbits, since it must then surely die out itself. In fact, a good rabbit population is necessary for the disease to carry on its own existence.

Nature, obviously quick to realise that the killing power of the original disease was not favourable to the continuance of the virus (998 out of every 1,000 died) quickly brought about a change in the virulence of the disease which resulted in a greater survival rate. Most of the changes which occurred resulted in the appearance of “field strains” of lower virulence. These field strains resulted from what are known as mutations (changes in the genetic make-up of the virus). The appearance many years ago of a red Granny Smith apple was one example of a mutation. A mutation (or change) occurred in one bud cell on a normal green Granny Smith tree and the limb which grew from this produced red instead of green apples. Propagation of buds from this one original limb has perpetuated the red Granny Smith. There are many other examples of this from the horticultural field. Field strains of myxomatosis of lesser virulence occurred in exactly the same way, and have been passed on from rabbit to rabbit in much the same way as the red Granny Smith has been propagated.

Nature in its bid to safeguard these weaker strains, has provided a very valuable tool to help them to replace the more virulent strains with which they occurred, side by side in the field. The spread of the disease is dependent upon two things—an abundance of vectors (usually mosquitoes)—and an available source of the disease (infected rabbits). The weaker strains of the disease which have lower mortality rates are also active for much longer in each rabbit before death or recovery occurs. For example, the original strain from which two out of every 1,000 rabbits survived, kills the rabbit in approximately 12 days. This means that each rabbit can only pass on the infection during a twelve day period, whereas the strains where 300 to 700 survive out of every 1,000 rabbits infected, can pass on the disease for about 35 days. In the random distribution of myxomatosis by mosquitoes, the chances of spreading the disease are loaded three to one in favour of the weaker strains. (Some more virulent strains are also produced by mutations, but these are of even shorter duration and die out very rapidly.)
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The hundreds of field strains which now exist have been classified into a series of grades (I-V with Grade I, the original highly virulent form). These grades, with their killing rate and duration for infection (called the "infective period") are given here. These gradings are based on the effect of each strain upon susceptible rabbits. As we shall see later, not all rabbits are susceptible, because of acquired immunity and genetic resistance.

<table>
<thead>
<tr>
<th>Grade</th>
<th>No. Surviving (out of each 1,000 Susceptible Rabbits Inoculated)</th>
<th>Infective Period (Survival Time or Time till Recovery)</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>2</td>
<td>12</td>
<td>20</td>
</tr>
<tr>
<td>II</td>
<td>20</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>IIIa</td>
<td>50</td>
<td>20</td>
<td>25</td>
</tr>
<tr>
<td>IIIb</td>
<td>100</td>
<td>25</td>
<td>35</td>
</tr>
<tr>
<td>IV</td>
<td>300-700</td>
<td>35</td>
<td>50</td>
</tr>
<tr>
<td>V</td>
<td>1,000</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

(all survive)

About every four or five years an extensive Australia-wide survey is made of myxomatosis samples collected from the field, and these are all classified (graded) according to the above list. It is significant that with each series of tests, more of the lower grades occur and fewer of the higher grades come to hand. In other words the weaker strains are gradually taking over and swamping the more virulent ones. In the last batch of specimens submitted from this State in 1964, all came into Grades IV and V, with one exception (Grade IIIb), whilst in the 1956 sampling, we had some Grade II, a lot of Grade III and no Grade V specimens.

This process of change resulting in the production of strains with lower virulence is called "attenuation."

Changes in the Resistance of the Host

Immunity

One step taken by nature to safeguard a species of animal against annihilation is to give those individuals which recover from the disease enough antibodies to ensure immunity for the rest of their lives. This is so in the case of a lot of the infectious human childhood diseases such as mumps and measles, which are normally only contracted once during a person's lifetime. (Immunities sometimes—very rarely—do break down and the disease is contracted a second time). In the case of myxomatosis the regular outbreaks of disease amongst the rabbit community will act as booster shots to those rabbits which have recovered and already acquired an immunity.

Immunity also safeguards the rabbit in another way. If the doe has a high antibody level (i.e., a good "solid" immunity) some of these antibodies will be passed to the offspring, probably across the placenta or in the first milk ("colostrum"). This immunity is only temporary, and after about six weeks the antibody level has fallen to a point where, probably, no worthwhile protection is afforded. This same thing applies to human babies, which never contract measles or mumps in the first few weeks of life, provided the mother has a high antibody level herself.

If an outbreak of myxomatosis occurs at a time when most young rabbits are more than six weeks of age (and bearing in mind that a lot of the adults will have recovered hence carry an immunity) then it is principally young rabbits which will be found dead. This is precisely what has happened in the summer just completed. Young rabbits were at the susceptible age at the time when seasonal conditions were right for a build up in mosquito numbers, and so better than usual kills resulted.

Genetic Resistance

So far we have seen two processes which help the rabbit in his survival against the disease. We will now look at what might be termed a direct retaliation by the rabbit.

When the original virus struck into the rabbits of Australia, approximately two out of every 1,000 survived and they were able to survive because they were in some small way different genetically from the bulk of the population. They had some "genetic resistance," which enabled them to withstand the attacks of the disease. This also occurs in humans, e.g., people of European extraction, because of their greater period of contact with the disease, have a much greater natural resistance to tuberculosis than have the aborigines of Australia.

Genetic factors are those which are inherited and passed along from parent
to offspring, and include such things as the white face in Hereford cattle.

The hereditary ability to resist a disease can also be passed on to offspring. So here we have one direct way in which the rabbit is fighting back against the disease. By the process of natural selection (those rabbits without genetic resistance are killed off) this factor of resistance will, over a period of time, safeguard more and more rabbits from the ravages of the virus.

**The Future of Myxomatosis**

The virus used for the original inoculation campaign was one of several myxoma-like viruses found throughout the world. In general terms, resistance or immunity to one also confers immunity to the others. Thus, there is little likelihood that any other similar disease, capable of spectacular results, will be found in nature. Although nowadays the rate of mutation of bacteria and virus can be accelerated under laboratory conditions, the mutant forms produced cannot be predicted. Therefore, the chances of producing another highly virulent form of the disease are very remote. However, landholders can rest assured a close watch is being maintained for a suitable follow-up disease.

At the present time the struggle which began in 1951 and in which the early rounds were so clearly in favour of the virus, has now swung heavily in the rabbit's favour. Unfortunately the end result must inevitably parallel the present position of many human diseases such as the common cold, where the virus causes some discomfort, but has little killing power and virtually no effect (fortunately) upon controlling the population! To reach this stage will no doubt take considerable time.

**Practical Facts about Myxomatosis**

- Myxomatosis is a haphazard and unreliable means of controlling rabbits, as its spread is dependant upon suitable insect vectors, which only occur in great numbers in the high density rabbit areas of W.A. following unusual seasonal conditions.
- Rabbits which have recovered from the disease are immune for life.
- Young rabbits (up to about six weeks) are temporarily immune, and, if they contract the disease during the early weeks of life, they will only suffer a very mild attack and will acquire a permanent immunity.
- Changes in the virus have resulted in field strains of lower virulence. This process of attenuation will continue and result in progressively lower kills. (Rabbits which recover from the weaker strains are also immune for life.)
- Natural selection in the rabbit population is resulting in increased genetic resistance, e.g., a higher percentage of rabbits each year are not affected by the disease because of resistance inherited from their parents.
- It is futile to hold out hope of any other form of the disease being found in the very near future.
- Myxomatosis will continue to play a smaller and smaller part in controlling the rabbits in Australia as time goes on, and, on present knowledge, there is nothing practical that can be done to reverse this trend.
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