

# The Effect on Virulence of *Trichinella Spiralis* from Passage Through Rabbits, Guinea Pigs and Rats\*

An Abstract

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**V**IRULENCE is an innate physiological potentiality which varies rapidly and significantly in any given strain of micro-organisms. These variations which occur under certain circumstances are a common observation in almost all strains of parasitic forms.

Variations in the virulence of many organisms are often brought about by changes in the environment. In bacteria a change from a high to a low grade of virulence is commonly seen as the result of the use of an unsuitable culture media, or as the result of the prolonged artificial growth of the organism. The change from a low to a high grade virulence has been accomplished by altering the environment in which the organism grows. Passages of culture through blood serum has been found effective as a method of raising virulence.

One of the earliest known observations on the changes in virulence of an organism which occurs as a result of animal passage is Pasteur's work on rabies. Pasteur showed that the virus of hydrophobia when successively passed through rabbits gained in virulence, while with only three passages through monkeys the virulence was reduced almost to extinction. The same relationship between host and parasite also exists with other micro-organisms. The virulence of attenuated pneumococci is increased by successive passage through mice.

With parasitic helminths no work has been done with the intention of studying the change in virulence of parasites in relation to animal passage, but with indirect evidence the same conditions as with bacteria are known to occur. Boyd, working with *Plasmodium praecox* in canaries, observed that the virulence of the parasite increased by rapid passage from one bird to another.

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In this study of the effect on the virulence of *Trichinella spiralis* from passages through rats, rabbits and guinea pigs, it has been demonstrated that the parasite suffers a gradual diminution in virulence as the infection is passed from rabbit to rabbit and from guinea pig to guinea pig. The loss in virulence of the strains was manifested by a fall in the ratio of the total number of larvae obtained from the muscles to the number of larvae fed, and by the absence of symptoms after the administration of large doses of infective larvae to normal rats. With the rabbits these changes begin to show after the fourth passage of the strain through these animals. With the guinea pigs the decrease in virulence starts with the second passage. The parasite apparently suffers damage caused by some unknown factor in these hosts.

In the rabbit muscle, the trichina apparently are injured by some factor in the rabbit tissue. This is shown by the fact that the longer the infection stays in the rabbit, the greater the damage to the parasite. This assumption is made on the findings in one of a pair of rabbits which was killed 360 days after infection, and its larvae recovered by digesting the muscles in an artificial pepsin and hydrochloric acid mixture. In this rabbit, 20 per cent of the recovered larvae were dead and 40 per cent were still encapsulated, suggesting that the capsule had been very resistant to the action of the digestive mixture. The other rabbit of the same pair, treated similarly to the first rabbit (except that it was digested 35 days after infection) had only 0.04 per cent of its larvae in a dead stage and no capsules. The glycogen content of the rabbit muscle may have some bearing on this problem. Lewis observed that a depletion of glycogen in the muscles is associated with an increased invasion of the muscle with trichina, but the effect of the amount of glycogen in the muscle after the parasite has been encysted between the muscle fibers is not known. It seems that the larvae obtained from the successively fed rabbits and guinea pigs and administered in fatal doses to normal rats were weakened so that they did not seem to develop naturally in the intestines of the rats. The number of larvae obtained from the muscles of these rats was not large enough to answer for the great infective doses administered to them. Once within the rat muscle, full recovery seemed to have been gained by the larvae. This is proved by the fact that no dead larvae were found after digesting these rats, and that doses from these rats proved fatal to normal rats.

With the successive passages through rats it is clearly seen that the parasite has been well adapted to the offensive and defensive powers of the rat. The parasite uses this adaptation to such an extent that when it enters a new rat it proceeds on its invasive course with a speed and violence greater than that manifested in the previous host. If a parasite can adapt itself to the resistant mechanism of an animal, and if it is passed successively through hosts of the same species, the exposure to the offensive conditions of the succeeding hosts will be considerably less. Thus the parasite will achieve more success in its growth and development, and will cause more damage as it is passed from animal to animal.

In most of the experimental work done to obtain variations in the virulence of organisms by passage through suitable or unsuitable animals, the factors within the host which cause such variations have not been widely studied. The host should be considered as a culture media which might or might not offer a proper environment for the growth of an organism.