died. They were flaccid, of an ocherous brown color and were filled with the introduced nematodes. One infected grub and the sixteen controls pupated and transformed. This experiment was repeated towards the last of June with approximately identical results.

During the course of the first experiments some of the worms derived from the May 29 and 31 field collections were fixed and sent, on June 4, to Dr. G. Steiner, of the U. S. Department of Agriculture, for identification. Dr. Steiner informed us that the nematode was undoubtedly a parasite and not a saprozoic free-living form. It belongs to the family *Oxyuridae,* and has been described by Dr. Steiner as a new genus and species under the name of *Neoaplectana glaseri.*[1] Whether *Neoaplectana* accompanied the Japanese beetle to this country or is a usual parasite of some native insect, from which it has reached *Popillia japonica,* is not yet clear.

During the summer and early fall frequent examinations of living and dead grubs were made. Some of these were obtained from the original locality as well as from other sources in New Jersey and Pennsylvania. However, *Neoaplectana* was found in only one place, namely Tavistock, near Haddonfield. On June 15, one pupa from this station was found parasitized with *Neoaplectana.* On June 21, a reexamination of the small plot of ground where the nematodes were first found yielded three Japanese beetle grubs, two pupae and two adults, all parasitized. The adults seemed to have been in the process of emerging from the soil when stricken. The June 21 collection was the last *Neoaplectana*-parasitized material obtained. After this date the adults emerged and the next generation of grubs were too small and scarce for satisfactory work until about the last of August.

One dead second-stage larva obtained from Pitman, New Jersey, August 24, and three from Haddonfield September 17, were heavily infested with several species of nematodes that appeared smaller than the spring parasite. These nematodes were undoubtedly saprophytes which multiplied within the bodies of the grubs after they succumbed from other causes. We were able to cultivate several generations of these worms on hay infusion agar to which a small amount of coagulated horse serum had been added. The females of these worms laid eggs very freely on the medium. Healthy Japanese beetle grubs infected with these nematodes did not die, although they acquired the worms, probably per os, and maintained from one to six of them within their alimentary tracts for about two weeks. The saprophytic species were the only ones found throughout the early autumn, and they also apparently disappeared from the field after the middle of October.

[1] *J. Wash. Acad. Sci.,* 19: 436, 1929.

Neoaplectana appears to be ideally adapted as a parasite for the Japanese beetle, for the reason that the host spends most of its long life cycle in the ground, which is undoubtedly also the natural environment of the nematode during at least a portion of its existence. According to present indications the parasite still occurs locally. However, it possesses great reproductive and lethal capacities, and therefore might prove a valuable adjunct if distributed throughout the territory infested with the Japanese beetle.

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REVERSAL OF THE STIMULATING ACTION OF HYDROSTATIC PRESSURE ON STRIATED MUSCLE

THE application of hydrostatic pressure to striated muscle commonly results in a marked augmentation of the tension developed during a single twitch.[1] Pressures of the order of 1,000 pounds per square inch in our previous study caused an increase of about 30 per cent. in tension and heat production. Most of our work, however, has been carried out on cardiac muscle in which tissue the effects of pressure are greater and the results may be predicted with considerable confidence.[2] Certain observations made in the interim on striated muscle failed to show as great a stimulation as was reported earlier, and occasional preparations were entirely uninfluenced by pressure. A further study has therefore been undertaken on striated muscle in which the effects of various environmental factors have been investigated in relation to the magnitude of the response under pressure. The results, a preliminary report of which is here published, are of interest in relation to the theories of pressure action and to the general problem of muscular contraction.

All observations have been made on sciatic-gastrocnemius preparations from the frog by a technique previously described.[1]

When the muscle was immersed in an oxygen free Ringer's solution or in one in which the acidity had been increased to about a pH 6.8 by the addition of hydrochloric acid, a characteristic change was noted. At first pressure resulted in the usual augmentation

[1] McKeen Cattell and D. J. Edwards, *Am. Jour. Physiol.,* 86: 371, 1928.
[2] D. J. Edwards and McKeen Cattell, *Am. Jour. Physiol.,* 84: 472, 1928.

in the response of the muscle, but after successive applications of pressure there was no increase in tension above the control value. It was further observed that if a muscle, when immersed in an oxygenated normal Ringer's solution, was fatigued by repeated stimulation, its response under pressure was less than the control, *i.e.*, the action of pressure on the tension developed by the muscle in response to a single stimulus is opposite in direction to its action in a fresh preparation. It seems probable that oxygen lack and acid solutions produce their effects by promoting fatigue. The following points have been established regarding the reversal of the pressure effect in the fatigued muscle: (1) The degree of depressed is roughly proportional to the pressure employed. (2) The tension developed by the fatigued muscle is commonly reduced to about one half by a pressure of 1,600 pounds per square inch. (3) The response very quickly, possibly instantaneously, returns to the control value when the pressure is released.

In the case of cardiac muscle the stimulating action of pressure on the tension can no longer be obtained when the temperature is reduced to 5° C., and we have secured some evidence of a reversal occurring at that point. (In press, *American Journal of Physiology.*) Studies now in progress on striated muscle indicate that there is a critical point at approximately 13° C. at which the stimulating action of pressure passes into depression. At a few degrees below this temperature the depressive action becomes marked and becomes greater as the pressure is increased. A few preparations have shown, in the vicinity of the critical temperature, a stimulation under the lower pressures (1,000 pounds) and depression at higher pressures (2,100 pounds). As the temperature is lowered pressure causes a further reduction in the response until at about 1° C. high pressure (2,100 pounds) almost completely suppresses the development of tension. At room temperature the magnitude of the response of the muscle continues to increase with increasing pressure up to at least 2,100 pounds per square inch, which is the greatest pressure we have employed.

When pressure is released the muscle again responds with the development of tension, the record differing in no way from the control taken before the application of pressure to the cold muscle. When such a muscle is warmed to room temperature, pressure once more causes an increase in the development of tension corresponding closely with the result obtained before the muscle was chilled.

Measurements of the duration of the phases of contraction and relaxation of cardiac muscle show that moderate pressures result in only a slight prolongation of these periods, and the same appears to hold for striated muscle at room temperature. On the other hand, at the lower temperatures pressure causes, along with the depression in tension development, a striking prolongation in the duration of the phases of contraction and relaxation which, for the higher pressures, may reach 100 per cent.

Certain interesting differences of behavior between cardiac and striated muscle are indicated by these results. In the first place, it appears impossible to fatigue cardiac muscle in such a manner as to produce a reversal of the pressure effect such as occurs in striated muscle, and in the second place the critical temperature, in which reversal of the pressure effect occurs, is about 8° C. lower in cardiac muscle.

The significance of these results in relation to the mechanism of muscular contraction will be discussed at a later date in connection with the publication of the complete data.

SUMMARY

The usual augmenting action of pressure on the tension developed by striated muscle in response to a single stimulus changes to depression on fatiguing the muscle. On release of the pressure the tension of the single twitch again reaches the control value.

A similar reversal may be brought about by cooling the muscle, the change usually occurring at about 13° C. Below this temperature the depression is roughly proportional to the pressure. At about 1° C. the tension development may be completely inhibited by high pressure. The muscle responds normally upon release of the pressure, and upon warming pressure again causes augmentation of contraction.

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