Elimination of Kappa Particles from 'Killer' Strains of *Paramecium aurelia* by Treatment with Chloromycetin

CERTAIN strains of *Paramecium*, known as 'killers', contain in their cytoplasm particles, called 'kappa particles'[1], which are Feulgen-positive, and stain with Giemsa after acid hydrolysis[2]. Killers liberate into the culture medium a substance lethal to *Paramecium* not containing kappa particles, known as 'sensitives'. Kappa particles have many of the properties of cytoplasmic genes[3], but their size (0·2–0·5 μ in length), and their staining reactions, suggest that they may be parasitic micro-organisms[2–5] such as the rickettsiæ, viruses of the psittacosis-lymphogranuloma group, or even small bacteria.

In an attempt to decide between these two possibilities, killer *Paramecium aurelia*, stock *H*, was cultured in baked lettuce infusion which had been inoculated with *Bacterium aerogenes* containing 2 mgm. chloromycetin per c.c. The chloromycetin did not affect the culture density of *Paramecium*, but after nine to fourteen days it was impossible to demonstrate any kappa particles in the *Paramecium*, when stained with Giemsa after acid hydrolysis. These killers, after chloromycetin treatment, had also lost their capacity to kill *P* stock sensitives.

Chloromycetin is an antibiotic active against certain bacteria, rickettsiæ, and viruses of the psittacosis-lymphogranuloma group, and the above result is further evidence for classing kappa particles with these parasitic micro-organisms; but as another antibiotic, streptomycin, has recently been found to eliminate the chromatophores in *Euglena*[6], and in seedlings of higher plants[7,8] the reaction of kappa particles to chloromycetin is not conclusive evidence for their parasitic nature.

Miss R. T. Gilmartin, of the Osborn Zoological Laboratory, Yale University, has treated stock *51* killer *Paramecium* for up to seventy-two hours with concentrations of chloromycetin ranging from 0·02 to 1·25 mgm. per c.c. Under these conditions, the number of kappa particles per animal was reduced, but they were not completely eliminated[9].

Cultures of *Paramecium* for this work were kindly supplied by Dr. J. R. Preer, of the University of Pennsylvania.

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Strength and Fatigue

IT is generally thought that the very large forces which isolated muscles can develop when excited electrically would produce movements so violent as to endanger tendons and bones if they were available in voluntary contractions in normal life. Our strength is kept within bounds by the inability of the higher centres to activate the muscles to the full. In a prolonged effort, this limited capacity to drive anterior horn cells by an effort of will is the part of the neuromuscular apparatus most subject to fatigue, so that the observed falling off in performance is due to an even less complete nervous activation of the muscle fibres rather than to failure of the contractile mechanism itself[1,2].

Against these views stands a simple observation. A subject lays his arm on a table with his hand over its edge and palm upwards. The forearm muscles are exercised by repeatedly flexing the wrist against the resistance of a 10-kgm. weight held by a cord loop in the hand. At first it is easy to make a movement of full range, but after some fifty repetitions the wrist can only be half-flexed. Exercise is then stopped and at the same time the circulation to the forearm is arrested by inflating a blood-pressure cuff on the upper arm. Test movements made every minute show that muscular power does not recover so long as the cuff is kept on (up to five minutes), but returns to normal within one minute when it is released, whatever the interval after imposition. Circulatory arrest alone causes negligible diminution in muscle strength until ischæmic paralysis supervenes in about thirty minutes. The only straightforward interpretation of this experiment is that fatigue in this type of exertion is peripheral. Reid[3], however, avoided this conclusion in a somewhat similar experiment by supposing that inhibitory afferent impulses are set up in the forearm "protective against excessive prolongation of activity and undue development of fatigue processes in muscle", and this was apparently confirmed by observing that good contractions were maintained to electrical stimulation of the median nerve.

Such suggestions can only be refuted by a more elaborate quantitative investigation. For this purpose the adductor pollicis is a better muscle, for (with suitable experimental arrangements to prevent interference) it is the only muscle employed in voluntary adduction of the thumb and it can easily be stimulated electrically through the ulnar nerve without involving other thenar muscles. Voluntary and artificial contractions, therefore, occur in exactly the same muscle and can be validly compared. In this way it has been shown that: (1) the tensions developed by a strong voluntary effort and by a maximal motor nerve tetanus are closely the same; (2) both tensions decline equally during fatigue and remain the same during a subsequent period of circulatory arrest; (3) the muscle action potentials to nerve stimulation do not significantly alter, indicating that neuromuscular block does not develop. (Fatigue was not carried beyond a 50 per cent reduction of muscle strength.) Voluntary effort, then, can call up the full potential power of the muscle; fatigue is due to failure of the contractile mechanism itself and not to nervous factors. Presumably the same metabolic causes in the muscle underlie the fatigue of small voluntary movements, as have long been known from the work of A. V. Hill to limit severe exertions of the body as a whole.

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