Letters to the Editor.

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Theories of Muscular Contraction.

SINCE Lundsgaard showed that the contractile mechanism can work without production of lactic acid and that this acid, when formed, appears mainly, perhaps entirely, after contraction is over, the one remaining important chemical change, the breakdown of phosphagen (phosphocreatine) is generally supposed to precede and cause contraction. This view, however, scarcely accounts properly for the observed behaviour of muscles poisoned with iodoacetate. In such muscles, when stimulated in nitrogen, exhaustion of the phosphagen supply brings to an end the only available exothermic reaction and the muscle fails to respond ; but it does not fail to contract, *it fails to relax,* for it remains in a state of contracture ('rigor') developing a tension not far short of a maximal twitch. It seems more natural to conclude that the chemical reactions accompanying activity are primarily concerned with relaxation and occur after contraction ; this is to say, they represent stages in a 'recovery process' as that term has been applied to processes in nerve.

When Fletcher and Hopkins had exploded the 'inogen' theory of contraction, it was assumed that the sequence of events must be (1) excitation, (2) an exothermic chemical reaction, (3) contraction. It was therefore supposed that in resting muscle the contractile mechanism (whatever its nature) was at zero energy potential and that the exothermic reaction 'charged up' the system to high potential, thereby producing a structure tending to be of shorter length. Relaxation was the result of dissipating the energy and returning to zero potential. The theory here suggested is the opposite and is a partial return to the 'inogen' theory. In resting muscles the contractile mechanism is supposed to be at a steady high potential, maintained by the resting metabolism. On excitation, the potential falls to zero and the muscle tends to a shorter length. It then relaxes because the contractile mechanism is recharged to its original potential by what is essentially a speeding up of chemical changes occurring the whole time. In quick moving muscles the recharging is begun by the rapid process of phosphagen breakdown ; in slower muscles other slower reactions may perhaps replace the phosphagen mechanism.

The terms 'charge' and 'discharge' are intended to suggest the analogy of an electrical condenser. The contractile mechanism may well consist of longitudinally arranged and electrically polarised surfaces. If these are extensible, the repulsive forces of the electric charges will make them longer when charged than discharged. A mechanism of this sort is similar to that generally supposed to be concerned with the excitation process in nerve. However, the main point is quite independent of any particular type of physical mechanism imagined to exist in the muscle and is a matter of the time relations of the chemical reactions.

The theory here suggested, 'Charged at Rest', seems to fit the facts at least as well as the alternative 'Discharged at Rest', and in some cases better. As already mentioned, it gives a more accurate account of the phenomena of the iodoacetate muscle. It provides a function for the resting metabolism and explains why the metabolism of activity and of rest differ quantitatively rather than qualitatively. Again, it fits in better with the phenomena of the refractory period after excitation. If each excitation discharges the contractile mechanism to zero potential, a second discharge is impossible until recharging has begun, and the second discharge will be smaller than the first until the full potential is reached. On the 'Discharged at Rest' theory there is no reason to expect a refractory phase. Lastly, if, as seems likely, the initial resting state is more unstable the higher the potential, it is easy to see how some muscles can be self-exciting and others not. That is to say, the difference between heart and skeletal muscle is that in the heart the recharging process is slow (long refractory period) but tends to charge the mechanism up beyond the level at which it is stable. In skeletal muscle the recharging is rapid but never exceeds the level at which extra energy has to be provided by an external stimulus to discharge the mechanism.

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    Jan 8.

Inheritance of Milking Capacity.

A STUDY of the milk yields of the progeny of 728 bulls of the Red Danish breed has been made with a view to determine—first, the ability of each bull to transmit the different degrees of milking capacity ; and secondly, having thus obtained an index of this ability, to determine the extent to which it was influenced by the various animals in his pedigree. The average number of daughters to each bull was 18. The figures were taken from the annual reports of the Milk Recording Societies throughout Denmark for 1918–1931. The daughters' yields were mainly calculated on the average of the first two lactations and corrected to the third lactation. The records of the dams and grandams were based on as many lactations as possible, from three to ten, and averaging 5·5. The average of these uncorrected lactations was taken as an indication of the milking capacity of the dam or grandams.

To measure the genetic aspect of the ability of bulls to transmit milking capacity, correlations were made of the average yields of the daughters of these

x	y	N	Total Yield of Milk.			Total Yield of Butter-fat.		
			r	R^x_y	$\sigma y/\sigma x$	r	R^x_y	$\sigma y/\sigma x$
Bulls to Sires .		555	0·255	0·291	0·877	0·324	0·388	0·834
,, to Dams .		725	0·173	0·117	1·47	0·183	0·141	1·30
,, to Paternal	G'sires .	473	0·202	0·263	0·767	0·193	0·267	0·723
,, to Maternal	G'sires .	505	0·194	0·208	0·932	0·258	0·291	0·885
,, to Paternal	G'dams .	721	0·026	0·018	1·45	0·061	0·055	1·10
,, to Maternal	G'dams .	715	0·112	0·077	1·46	0·170	0·134	1·27

The standard error of 'r' ranges from $\pm 0·03$ to $\pm 0·04$.
R = Regression.
Bulls (x) mean total yield of milk = 4403 kgm., σ = 440.
    ,,   ,,   ,, of butter-fat = 179·64 kgm., σ = 20·48.

bulls to the average yields of the daughters of their sires, and to the actual yields of their dams and grandams. Thus the milking capacity of the bulls (as measured by the yields of their daughters) has been correlated to the milking capacity of the sires and grandsires (similarly measured) and to the dams and grandams (as measured by actual production). These correlations are as shown in the accompanying table.