adsorb the colicins they produce but are immune to them.

The most intriguing property of PBSX is the way it incorporates host DNA. Approximately 30–40 per cent of the entire *B. subtilis* genome is incorporated into PBSX particles, and it seems that the host DNA is broken down into pieces of unique size, 22*S*, rather than being fragmented randomly. This process can occur in the absence of host DNA synthesis and therefore in the absence of replication of the presumed PBSX prophage genome. The appearance of 22*S* DNA is, however, inhibited by chloramphenicol so fragmentation of the host genome must depend on the synthesis of some protein after PBSX induction with mitomycin C.

In many respects this fragmentation of the host chromosome is analogous to the way in which concatenate T_4 DNA is parcelled into T_4 phage, the *B. subtilis* chromosome being the equivalent of a T_4 concatenate. The size of the DNA is probably determined by the phage head proteins such that the *B. subtilis* DNA is split to produce PBSX "head full" units.

Muscular Dystrophy

from our Medical Biochemistry Correspondent

MUSCULAR dystrophy in humans is an inherited condition for which there is no satisfactory treatment. Several biochemical changes have been noted in affected humans and in animals with similar conditions, but the fundamental causes of the muscular wasting have not yet been determined. Some laboratory animals suffer a condition resembling muscular dystrophy when they are deprived of vitamin E (α-tocopherol), but human muscular dystrophy is not improved by giving patients vitamin E. These animals, dystrophic through lack of vitamin E, were much improved by giving hexahydrocoenzyme Q_4 and it was later shown that hexahydrocoenzyme Q_4 improved the condition of a strain of mice with inherited muscular degeneration very similar to human muscular dystrophy.

Because coenzyme *Q* or ubiquinone is a normal component of the electron transport chain, there may be some abnormality in the concentration or functioning of ubiquinone in dystrophic mice. Nilsson, Farley, Schiller and Folkers (*Arch. Bioch. Biophys.*, **123**, 422; 1968) have now shown that the organs of mice suffering from advanced dystrophy contain only about half the ubiquinone concentration as do organs from normal litter-mates. Ubiquinone was measured in the hearts, livers and kidneys of the animals, for the wasted muscles provided very little tissue. Young weanling mice at 1·5 months of age showed almost the same coenzyme *Q* concentrations, whether they were destined to become dystrophic (103 μg/g) or to remain healthy (120 μg/g). At about 6 months of age, the concentration in the healthy mice had risen to 209 μg/g but the very ill dystrophic mice had only about half this concentration (112 μg/g). The synthesis of ubiquinone from *p*-hydroxybenzoic acid was much the same in all the animals, suggesting that if the low concentration in dystrophic animals is the result of decreased synthesis of ubiquinone, the defect is in the conversion of phenylalanine or tyrosine to *p*-hydroxybenzoic acid.

The marked decrease in ubiquinone concentration in dystrophic mice may explain the beneficial effect of hexahydrocoenzyme Q_4 in these mice. Because ubiquinone is a component of the respiratory chain, it is not unreasonable to look for changes in respiration or oxidative phosphorylation in dystrophic animals. With dystrophic hamsters, it has been known for some time that there is a decrease of respiratory rate and oxidative phosphorylation of heart homogenates, but these changes seem to be absent in isolated mitochondria from genetically dystrophic mice. According to Wrogemann and Blanchaer (*Can. J. Biochem.*, **46**, 323; 1968), there seems to be no difference between the respiration and oxidative phosphorylation of isolated mitochondria from normal Syrian hamsters and the myopathic strain BIO 14–6. The dystrophic hamsters were not obviously ill at 97–124 days of age, but histological examination and serum creatine phosphokinase levels showed that the process of degeneration had started. There was no significant difference between isolated heart and muscle mitochondria from the healthy and dystrophic hamsters in rate of oxygen uptake, ADP/O ratio and respiratory control ratio using pyruvate-malate as substrate. The oxidation of L-α-glycerophosphate was the same in both sets of animals but the oxidation of reduced nicotinamide adenine dinucleotide (NADH) was 35 per cent less in the mitochondria from dystrophic animals (this is more likely to be due to a reduction in the mitochondrial permeability to NADH than any respiratory chain defect). There was no difference in the number of mitochondria per cell, so it seems that at least in the early stages of muscular dystrophy respiration and phosphorylation can proceed as well in dystrophic animals as in normal animals.

RNA Terminal Sequences

from our Cell Biology Correspondent

THE 3′ terminal sequences of RNA from bacteriophages are of particular interest because the complementary strands are made in the 5′→3′ direction during phage replication—Spiegelman and his collaborators have retracted the heretical suggestion that synthesis can occur in the 3′→5′ direction—and so the 3′ terminal sequence of the plus strand is template for the 5′ terminal sequence of the minus strand. Moreover the RNA synthetases which catalyse this minus strand synthesis have specific template requirements. The Qβ enzyme, for example, only replicates homologous Qβ RNA and the basis of this specificity may reside in the 3′ base sequence of the template.

De Wachter and Fiers and Weith and Gilham have previously reported that the 3′ sequence of the closely related phages f2 and MS2 is GpUpUpApCpCpApCpCp CpA (see *Nature*, **217**, 311; 1968). Weith, Asteriadis and Gilham (*Science*, **160**, 1452; 1968) now report that the 3′ terminal sequence of the serologically unrelated phage Qβ differs from those of f2 and MS2. Like f2 and MS2, however, the last two bases in Qβ are CpA. The terminal base A was identified after alkaline hydrolysis as the unique nucleoside among all the nucleotides which are liberated from internal positions. A fourteen base fragment liberated by ribonuclease T_1 had the composition 9Cp; 4UpCp. It lacked the terminal adenosine, which was removed during the isolation procedure, and the 3′ terminal sequence of Qβ RNA must therefore be Gp(9Cp; 4Up)CpA—a sequence which is quite distinct from that of f2 or MS2.