rapid spike of depolarization followed by a long plateau. The spike, but not the plateau, overshot the zero potential level. In this example the membrane repolarized within about 12 seconds, but some action potentials lasted up to 40 seconds. Often, a small prepotential was seen.

Although the shape of the action potential resembles somewhat that of ventricular myocardium, the two types of muscle differ in other respects. The resting potential and the action potential are higher in heart than in arterial muscle. A single contraction lasts much longer in arterial (30 to 200 sec) than in ventricular muscle (1 to 3 sec). In heart muscle the cell membrane remains depolarized practically throughout the contraction, whereas in arterial muscle the membrane usually repolarizes before maximum tension is reached. The relatively shorter refractory period in the artery permitted summation of contractions when the rate of firing was increased, resulting in steadily maintained contractions. As in cardiac muscle an increase in the frequency of the action potentials decreased their duration.

A different pattern was seen in the veins. Some spontaneous contractions occurred, but they were less rhythmic and more variable in strength than the arterial contractions. Each contraction was preceded by one or more action potentials (Fig. 1). These consisted of a spike of depolarization. However, repolarization was much more rapid than in the arteries, so that the plateau following the spike was often barely perceptible. Action potentials were often followed by a period of hyperpolarization. When trains of spikes followed one another closely, the contractions summated to produce a fairly steady contraction.

The differences between the action potentials seen in the artery and the vein emphasize the danger of considering the configuration in one type of vessel as representative of that in all vascular smooth muscle. However, spikelike action potentials were recorded in the inferior vena cava of the guinea pig, and long action potentials similar to those in turtle arteries were recorded with difficulty in the aorta of the frog (*2*).

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2. This work was conducted while one of us (I.C.R.) held a Harkness fellowship of the Commonwealth Fund and was supported in part by a grant (HTS 5147) from the National Heart Institute, U.S. Public Health Service. We thank J. W. Woodbury for his advice and criticism.
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Repression of Ornithine Transcarbamylase Protein Formation by Arginine

Abstract. Arginine-repressed cells of *Escherichia coli* W do not form a protein immunologically related to ornithine transcarbamylase. This was determined by lack of cross-reactivity of repressed cell extracts with rabbit antisera prepared against purified ornithine transcarbamylase. The results indicate that arginine acts by blocking the synthesis of the entire enzyme-protein.

Escherichia coli W cells grown in the presence of arginine contain only negligible amounts of ornithine transcarbamylase (OTC), an enzyme in the biosynthetic pathway leading to arginine formation. It has been shown (*1*) that arginine acts as a repressor of the synthesis of OTC activity in these cells, and recent evidence (*2*) suggests that perhaps ornithine competes with arginine for the site of repressor action. When arginine is removed from the growth medium, a 100-fold increase in OTC activity occurs in *E. coli* W; this increase in activity has been correlated with new protein synthesis (*3*).

In order to investigate the function of arginine in the regulation of ornithine transcarbamylase synthesis, it was first necessary to determine whether arginine blocks the synthesis of the entire OTC-protein or, alternatively, changes the configuration of the active site or prevents synthesis of a small portion of the molecule. If these cells form an enzymatically inactive OTC-like protein in the presence of arginine, this altered protein should be detectable by immunological techniques. With the use of specific antisera to the inducible enzyme, β-galactosidase, it has been shown (*4*) that uninduced cells contain no antigenically active protein related to this enzyme. Experiments described in the present report suggest that in like manner arginine represses the formation of the entire OTC-protein.

By use of a 100-fold purified OTC preparation from *E. coli* W (*5*), an antiserum was prepared from rabbits according to the method of M. Cohn (*6*). One milligram of purified OTC emulsified with Freund's adjuvant containing mycobacterium was injected into the rabbits intramuscularly under the scapula. The injection was repeated weekly for 30 days, and after two additional weeks the rabbits were bled. The serum obtained from rabbit blood was clarified by centrifugation, and the globulin fraction was collected by precipitation with ammonium sulfate at 33 percent saturation. The precipitation of a 100-fold purified OTC antigen with the prepared antisera is shown in Fig. 1. At the equivalence point about 0.24 mg or 212 units of OTC are precipitated per milligram of antisera protein in the precipitate. It was possible to demonstrate 30 percent of the original OTC activity in the washed precipitate by running it gently through a thin-nosed pipette. However, it was not possible to release the enzyme

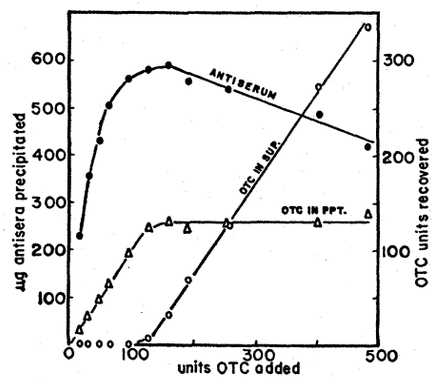

Fig. 1. Relationship between amount of rabbit antiserum precipitated and amount of purified ornithine transcarbamylase added. Precipitation reactions were carried out as follows: 0.1 ml of rabbit antiserum prepared as explained in the text; increasing amounts of purified OTC (specific activity = 873 μmole of citrulline produced per minute per milligram of protein) were added; final volume of each tube to 1.7 ml with 0.15*M* NaCl; incubated at 3°C for 3 days; centrifuged; precipitates washed three times in 0.15*M* NaCl. Curve •: micrograms of antiserum protein precipitated per tube calculated by subtracting the antigen precipitated from the total protein precipitated. At the point of maximum antiserum precipitation (590 μg), 129 units of OTC were precipitated. Curve o: units of OTC remaining in the supernatant determined as reported previously (*3*). Curve △: units of OTC in the precipitate calculated by subtraction of curve o from total OTC added. OTC activity was also demonstrated in the precipitates as discussed in the text.

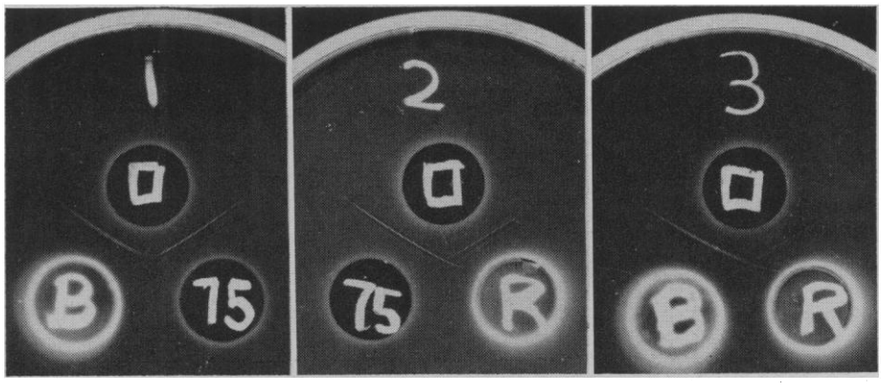

Fig. 2. Ouchterlony agar diffusion plates showing the reaction of anti-OTC with purified OTC, and extracts from arginine repressed and derepressed *E. coli* W cells. Well □: 0.18 mg of anti-OTC serum protein; Well 75: 28 μg of OTC containing 1040 units per milligram of protein [one unit of OTC activity equals 1 μmole of citrulline produced per minute at 37°C under the conditions of assay previously reported (*3*)]; Well B: 0.3 ml of crude extract of derepressed *E. coli* W cells containing 45.6 units of OTC and 3.1 mg of protein; Well R: 0.3 ml of a crude extract of repressed *E. coli* W cells containing 0.8 units of OTC and 3.1 mg of protein. The distance between wells was 14 mm. Plates were incubated for 6 days at 27°C, and contact prints were made. Duplicate plates were run in all experiments.

from the antibody by treatment with acetic acid at *p*H 5 or by ammonium sulfate fractionation.

Extracts from derepressed *E. coli* W cells with a high level of OTC and extracts from arginine-repressed *E. coli* W cells with low levels of OTC were compared on the basis of the precipitation reaction with the specific antiserum. Since the preparations of repressed cells demonstrated a small precipitation reaction which could not be accounted for on the basis of observed OTC activity alone, the Ouchterlony agar diffusion method was employed (*7*). With a 200-fold purified preparation of OTC, formation of a large distinct precipitation line was demonstrated along with two minor lines by this method.

Diffusion agar plates were then prepared with crude extracts of repressed and derepressed *E. coli* W, and purified OTC was opposed to the rabbit anti-OTC serum. Figure 2 illustrates contact prints of three diffusion plates obtained. On diffusion plate 1 (Fig. 2), the purified enzyme (Well 75) formed a definite reaction of identity with the crude extract of derepressed cells (Well B), both in the major heavy precipitation line (OTC) and in the minor lines due to contaminating proteins. On plate 2 the same amount of protein from a crude extract of arginine-repressed cells (Well R) formed no reaction of identity with respect to the major heavy line of purified OTC reacting to anti-OTC serum. The contact print of plate 3 shows the lack of identity of protein from repressed cells (with respect to the major line) when allowed to diffuse simultaneously with the extract from derepressed cells against the anti-OTC serum.

The evidence above strongly favors the hypothesis that arginine, or a repressor formed from arginine, blocks in some yet unknown way the formation of the entire OTC-protein molecule at the site of synthesis (presumably the ribosomes). However, it should be pointed out that the lack of a precipitation reaction or the absence of a displacement reaction as shown by Perrin *et al.* (*4*) does not eliminate the possibility of synthesis of at least part of the OTC molecule or β-galactosidase molecule, as the case may be, at the enzyme-forming site during repression (*8*).

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Plumage in Lal Munia (Amandava amandava)

Abstract. An Indian ploceid finch, *Amandava amandava*, develops brilliant nuptial plumage in males during the breeding season. The females and males outside of the breeding season have modest brown hen plumages. The fact that castrates, whether originally of male or of female sex, assume nuptial plumages during the breeding season of the species indicates that plumage character is under the control of hypophyseal hormones and that hypophyses are passing through an activity cycle which is independent of presence or absence of gonads.

Plumage of African weaver finches is under hypophyseal control (*1*), specifically under luteinizing hormone (*2*), and this fact has been made use of in developing an assay for luteinizing and chorionic gonadotropic hormones (*3*). These finches are natives of Africa and are imported at great cost in the United States, where many laboratories are now maintaining them. As quite a few species of finches occur naturally in India (*4*, *5*), it was, therefore, decided to investigate their plumage control.

We have investigated some species of Indian finches and one of these, lal munia (*Amandava amandava*), which is a very beautiful bird (family, Ploceidae), has yielded encouraging results. Males are brilliantly colored and have vermillion red bills during the nuptial phase. The head and upper plumage are crimson colored. The tail is black and the outer feathers are tipped with white. From chin to breast it is deep crimson, sparsely spotted with white. Females are dull brownish with wing coverts and inner secondaries tipped white and upper tail coverts crimson with small terminal white specks. The tail is brownish black, the chin yellowish white, while the throat and upper breast are grey. Abdomen and under tail coverts are light brownish white (*5*). Males in eclipse resemble females.

A number of birds of both sexes were castrated during the fall, and their feathers were subsequently examined every month. During March and April, males, castrated males, and castrated females regenerated male nuptial plumage. This coincides with the progressive phase of their sexual cycle (*6*). Intact females maintain hen plumage all year round.

The appearance of nuptial plumages in normal males and castrates of both sexes suggests that cock plumage is gonad independent in this species and that the ovarian hormone suppresses