sues of hypothyroid, hyperthyroid and euthyroid rats to a high concentration of epinephrine was essentially the same. It is suggested that the magnitude of the lipolytic response to physiologic doses of epinephrine may be related to the basal lipolytic activity of the adipose tissue.

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Enzyme Activities in the Developing Muscle and Liver of Normal and Genetically Dystrophic Chick Embryos. (29804)

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Asmundson and Julian(1) reported muscular dystrophy as a genetic anomaly in a strain of New Hampshire chicks. They attributed this condition to a recessive gene which also affects the body conformation. The age at which the characteristic symptoms of avian muscular dystrophy could be detected varied from 4 to 12 weeks. Cornelius *et al*(2), in a study using the same strain of chicks, reported that 4 weeks to 18 months old dystrophic birds exhibited higher activities of serum aldolase and glutamic-oxaloacetic transaminase (GOT) in serum and tissues when compared to normal birds. Zalkin and Tappel(3) reported that oxidation of succinate is impaired in tissue from vit. E-deficient rabbits. Allen *et al*(4) found that activity of cytochrome *c* reductase was elevated in muscles from vit. E-deficient rabbits while cytochrome *c* oxidase was decreased during the first week of the deficiency and then approached normal level. The purpose of this investigation was to determine the activities of the succinic dehydrogenase, GOT and cytochrome *c* oxidase in embryos and day-old chicks from the genetically dystrophic and normal strains of New Hampshire chicks in an effort to elucidate the effect of the dystrophic gene on the activity of these enzymes.

Procedure. Eggs were collected daily from hens maintained on a practical type diet. The muscular dystrophy strain of birds was from a stock originally obtained from Asmundson and Julian in 1958 and maintained at this station. A normal strain of New

TABLE I. Enzyme Activities of Tissues from Embryos and Day-Old Chicks of Normal and Dystrophic Strains of Birds.

	Days of Incubation										
	7		10		14		18		21		1
	N†	H‡	N	H	N	H	N	H	N	H	N
Day embryo homogenate											
Succinic dehydrogenase	8.64§	1.93	—	—	—	—	—	—	—	—	—
	(3.84)**	(.21)	—	—	—	—	—	—	—	—	—
Cytochrome *c* oxidase	9.85‖	1.42	—	—	—	—	—	—	—	—	—
	(3.62)	(.97)	—	—	—	—	—	—	—	—	—
GOT	11.32¶	1.88	—	—	—	—	—	—	—	—	—
	(4.01)	(.80)	—	—	—	—	—	—	—	—	—
east muscle											
Succinic dehydrogenase	—	—	3.07	4.24	9.12	3.68	6.62	2.15	10.34	.88	3.18
	—	—	(.51)	(1.19)	(6.14)	(.56)	(.29)	(.89)	(6.00)	(.45)	(.59)
Cytochrome *c* oxidase	—	—	2.69	1.85	8.13	3.22	5.99	1.99	10.12	.66	3.64
	—	—	(1.74)	(.98)	(3.87)	(1.42)	(1.70)	(1.18)	(5.90)	(.51)	(.58)
GOT	—	—	2.48	2.76	3.52	2.45	4.15	1.55	8.15	1.68	2.91
	—	—	(.67)	(1.35)	(2.27)	(.99)	(1.70)	(.49)	(5.11)	(.25)	(.50)
g muscle											
Succinic dehydrogenase	—	—	3.43	2.98	5.01	3.15	5.19	4.07	9.02	2.04	3.25
	—	—	(4.23)	(.58)	(.90)	(.88)	(1.59)	(1.53)	(2.17)	(1.23)	(.67)
Cytochrome *c* oxidase	—	—	2.07	.29	3.33	2.24	4.03	1.70	9.59	1.28	3.58
	—	—	(1.72)	(.64)	(.36)	(.42)	(1.91)	(.79)	(3.04)	(.43)	(.58)
GOT	—	—	1.26	1.62	1.66	1.74	3.69	4.24	9.58	4.79	4.31
	—	—	(.79)	(.24)	(.76)	(.42)	(1.38)	(1.70)	(2.11)	(.83)	(1.34)
ver											
Succinic dehydrogenase	—	—	13.70	6.07	7.27	3.18	1.73	.81	2.30	.49	2.30
	—	—	(7.38)	(.32)	(1.12)	(.56)	(.28)	(.47)	(.63)	(.32)	(.46)
Cytochrome *c* oxidase	—	—	16.16	1.60	9.51	2.93	2.73	.98	4.94	.45	6.55
	—	—	(11.29)	(.98)	(2.08)	(.44)	(.44)	(.18)	(1.30)	(.21)	(1.69)
GOT	—	—	17.61	10.30	9.66	7.28	3.07	4.18	3.83	4.82	3.53
	—	—	(8.55)	(1.00)	(1.20)	(.96)	(.60)	(.35)	(.83)	(.91)	(.94)

ay-old chick.
ormal strain.
ereditary dystrophic strain.
ne unit represents amount of enzyme that will oxidize 1×10^{-11} f cytochrome c/sec/mg of tissue protein.

‖ One unit represents amount of enzyme that will redu moles of cytochrome c/sec/mg of tissue protein.

¶ One unit represents amount of enzyme that will oxid moles of NADH/sec/mg of tissue protein.

** Standard deviation.

Hampshires was obtained from a stock known not to have this genetic anomaly. Fifty eggs per strain were placed in a forced-draft incubator for 6 days. On the 6th day, eggs were candled and 30 fertile eggs per strain were selected for the experiment and placed in the incubator. Embryos were taken at 7, 10, 14, 18, and 21 days. Day-old chicks were also included in this study. Five embryos or chicks were used for each age group. Seven-day-old embryos were homogenized in a mortar placed in a crushed ice bath and samples of the whole body homogenate were taken. Embryos 10 days of age and older were relatively larger in size and samples from breast muscle, leg muscle and liver were obtained. Preparation of samples for enzyme assays was carried out as described by Creger *et al* (5). The procedure outlined by these workers for assay of succinic dehydrogenase and cytochrome *c* oxidase activities was used. The activity of GOT was assayed according to the procedure of Karmen and Wroblewski (6). Enzyme activities were expressed per mg of tissue protein. Protein was measured using the biuret method.

Results. Succinic dehydrogenase, cytochrome *c* oxidase and GOT activities were greater in the 7-day whole embryo homogenates from the normal strain than from the corresponding activities in the muscular dystrophy embryos (Table I). At later stages of embryonic development, enzyme activities in breast muscle, leg muscle and liver were generally higher in the normal birds than in the dystrophic strain. Standard deviations are quite large in some instances but differences in enzyme activities, as described, are still apparent.

Discussion. A depression of the activity of succinic dehydrogenase and cytochrome *c* oxidase in the breast muscle, leg muscle and livers of dystrophic embryos and day-old chicks, as compared to the normal strain, was noted. These results suggest a disturbance in the cytochrome system and the succinic dehydrogenase system. Similarly, Allen *et al* (4), using E-deficient rabbit muscles, concluded a possible disruption of the cytochrome system as the site of the metabolic disturbance in the nutritionally induced muscular dystrophy. These workers suggested the cytochrome *c* reductase to be the site of the disruption. The depression of the succinic dehydrogenase system is in agreement with the results of Zalkin and Tappel (3) who found a decrease in the oxidation of succinate in E-deficient rabbit tissues.

The GOT activities in the dystrophic and normal tissues were comparable to each other in most of the determinations. This is in contradiction to the findings of Cornelius *et al*(2) where tissues from 12 to 18 months old dystrophic chickens displayed higher GOT activity than the normal strain. Our results were based on the tissue protein and not on the dry weight of the tissue as reported by Cornelius and coworkers, which may explain the differences in the results.

While gross observations of muscular degeneration have been made in hatched chicks of muscular dystrophy strain of chicks, the metabolic changes reported here may be related to the onset of the muscular dystrophy symptoms in older birds. No previous report was found concerning enzyme activities of embryos from dystrophic strains of chicks.

Summary. Activities of succinic dehydrogenase, cytochrome *c* oxidase and glutamic oxaloacetic transaminase (GOT) have been assayed in embryos and day-old chicks from a genetically dystrophic and normal strain of New Hampshire chicks. Results point out possible defects in the cytochrome *c* oxidase and succinic dehydrogenase systems as the site for the metabolic disturbance in the hereditary muscular dystrophy in chicks.

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