fibers would be at a severe disadvantage in the event of a circulatory disturbance. The delivery of oxygen and other blood borne materials to intracellular locations may be further impaired by hypertrophy of injured fibers. A disturbance in the microcirculation in human patients with Duchenne muscular dystrophy has been suggested by Demos (4) as a result of observed abnormalities in the peripheral circulation time.

The biochemical effects of high oxygen tension, on dystrophic chicken muscle, are not known. It is possible that oxygen therapy is effective due to an alteration in the microcirculation. We have not yet determined how long these beneficial effects of therapy can be maintained. It may be concluded, however, from this study and from functional data previously reported (2), that continuous high oxygen therapy does retard the progress of hereditary muscular dystrophy of the chicken during early stages of the myopathy.

Summary. Fertile eggs from normal and genetically-dystrophic chickens were placed in an environment chamber on day 19 of development. The atmosphere contained 45% oxygen until the chicks hatched, at which time it was raised to 70%. The chickens were maintained in the high oxygen atmosphere until 6 weeks of age when they were sacrificed for histological examination. The breast muscles of oxygen-treated dystrophic chickens underwent only minimal cytological changes when compared to both untreated normal and dystrophic controls. Breast muscles of oxygen-treated normal chickens showed no changes. It is concluded that continuous treatment in a high oxygen environment retards the progress of the hereditary myopathy.

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Rat Tissue DPNH–Cytochrome *c* Reductase Activity in Altered Thyroid States* (32954)

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Changes in the activities of many enzymes have been studied in a variety of tissues after alterations in thyroid states. The effect of thyroidectomy on smooth muscle, however, has not been extensively investigated. Barker (1) found the succinoxidase activity of the uterus to be quite variable after thyroidectomy and after thyroxine treatment. Since enzyme complex DPNH–cytochrome *c* reductase studies have been limited to liver with respect to thyroid function (2), this present investigation is concerned with effects of thyroidectomy and thyroxine treatment on liver, heart, bladder, and uterus with respect to the activity of this enzyme. Effects of thyroidectomy on the activity of DPNH–cytochrome *c* reductase and DNA content in the estrogen-stimulated uterus are also reported.

Materials and Methods. Female albino Sprague-Dawley rats weighing between 150–200 gm were ovariectomized and divided into five groups: sham thyroidectomized, thyroidectomized, thyroidectomized given daily injections of L-thyroxine (100 μg/kg of body wt.) for 2 weeks, sham thyroidectomized and thyroidectomized given daily injections of estradiol 17β (2.1 μg/kg of body wt.) for 3 days. Animals were fed *ad libitum* and were given 1% $CaCl_2$ as drinking water for 1 month during the development of the hypothyroid state and before thyroxine and estrogens were

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administered. Basal oxygen consumptions were determined using a multichanneled closed-circuit metabolic apparatus 2 weeks after the surgical removal of the thyroid gland and after 2 weeks of the prescribed thyroxine treatment. A decreased oxygen consumption of 25%, was observed in the thyroidectomized rats when compared with the control group. Thyroxine treatment of the thyroidectomized group resulted in an increased BMR of 64%. Radioactive ^{131}I uptakes (0.5 μC/rat) were utilized to verify the completeness of the thyroidectomies. Rats were fasted for 24 hours prior to sacrifice by decapitation.

DPNH–cytochrome *c* reductase was assayed according to a modification of the method of Bever (3). The DNA was prepared according to the procedure of Volkin and Cohn (4) and analyzed using the colorimetric procedure of Dische.

Results. The DPNH–cytochrome *c* reductase values are shown in Table I. There was a decrease in enzyme activity in liver and heart of the thyroxine-treated animals and in heart in the thyroidectomized animals when compared with controls. There was no change in enzyme activity in liver after thyroidectomy. Bladder and uterus showed no change either after thyroidectomy or thyroxine treatment. The thyroidectomized estrogen-stimulated uteri were no different from the nonthyroidectomized estrogen-stimulated uteri, although both of these groups had values that were significantly higher than the nonestrogen-stimulated thyroidectomized and nonthyroidectomized groups.

The DNA values expressed as μg/gm of tissue wet weight were decreased in estrogen-stimulated uteri in both control and thyroidectomized groups when compared to the nonestrogen-stimulated uteri (Table II).

Discussion. DPNH–cytochrome *c* reductase activity decreased in both liver and heart with thyroxine treatment. Since it has been shown that both cytochrome *c* (5) and succinoxidase activity (1) are increased with thyroxine administration and decreased with thyroidectomy, it seems that there is a shift in pathways from the DPNH-to-oxygen system to the succinate-to-oxygen system. Such a

TABLE I. Tissue DPNH–Cytochrome *c* Reductase Activity in Altered Thyroid States.

	No. of animals	Uterus mean ± SE	No. of animals	Bladder mean ± SE	No. of animals	Heart mean ± SE	No. of animals	Liver mean ± SE
Nonthyroidectomized (control)	8	0.99 ± 0.09	8	6.0 ± 0.28[b]	8	58.9 ± 6.2	8	46.4 ± 4.5
Thyroidectomized	8	0.76 ± 0.13	8	5.9 ± 0.66[b]	8	32.5 ± 2.7[a]	8	52.8 ± 5.6
Thyroidectomized plus thyroxine	11	0.84 ± 0.08	12	5.0 ± 0.58[b]	11	40.3 ± 2.3[a]	9	29.0 ± 2.2[a]
Nonthyroidectomized estrogen stimulated	8	2.54 ± 0.16[a]						
Thyroidectomized estrogen stimulated	8	2.31 ± 0.18[a]						

[a] $p < .05$ when compared to control values.

[b] Average of 4 determinations.

TABLE II. DNA Values in Uteri after Thyroidectomy and Estrogen Stimulation.

	DNA (μg/gm of tissue wet wt.) mean ± SE
Nonthyroidectomized (control)	11,296 ± 1529
Nonthyroidectomized estrogen stimulated	4849 ± 323[a]
Thyroidectomized	10,520 ± 319
Thyroidectomized estrogen stimulated	5007 ± 345[a]

[a] $p < .05$ when compared to appropriate control.

shift results in formation of less ATP's since succinate-to-coenzyme Q does not result in the formation of an ATP. Many investigators find a lowered P/O ratio in hyperthyroidism.

With respect to smooth muscle, no change in enzyme activity or DNA content in bladder or uterus was found with either thyroidectomy or thyroxine injections. Barker (6) found no change in oxygen consumption of gastric smooth muscle after thyroidectomy or thyroxine treatment and Whaley *et al.* (7) found duodenal smooth muscle unresponsive to thyroxine treatment as determined by oxygen consumption measurements. The activity of succinoxidase is unaltered with thyroidectomy in uterine smooth muscle (1). These results support the hypothesis that smooth muscle is not responsive to thyroid hormones.

It appears from the results in Table II that thyroid hormones are not necessary for a response from the uterine muscle tissue to estrogen since activity of the enzyme DPNH–cytochrome *c* reductase increased equally in the thyroidectomized animals as in the nonthyroidectomized animals and the DNA content decreased to the same extent.

Summary. Changes in the activity of DPNH–cytochrome *c* reductase in heart, liver, bladder, and uterus were determined after thyroidectomy and thyroxine treatment. Liver and heart showed decreases in enzyme activity in the hypermetabolic state. Bladder and uterus were unresponsive. Enzyme activity increased and DNA content decreased in uteri after estrogen stimulation irrespective of metabolic status of the animal.

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Barium Stimulation of Esophageal Smooth Muscle* (32955)

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Knowledge of the responses of esophageal smooth muscle to drugs should lead to a better understanding of the mechanisms of esophageal movement in deglutition. The pharmacology of esophageal smooth muscle has received little attention. In intestinal smooth muscle, barium induces contraction either through stimulation of preganglionic nerves, postganglionic nerves or by a direct effect on the smooth muscle cells (3–8). In the dog, whose esophagus is mainly striated muscle, barium *in vivo* induces contractions which are abolished by atropine (9). The

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