Muscle, Erythrocyte and Plasma Electrolytes and Some Other Muscle Constituents of Rabbits with Nutritional Muscular Dystrophy.* (28117)

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Residual muscle from patients with hereditary muscular dystrophy contains more sodium and less potassium than does normal muscle even though the concentration of these cations in plasma is not abnormal(1). Since it was first produced by Goettsch and Pappenheimer(2), nutritional muscular dystrophy in animals given a diet deficient in Vit. E has been the subject of numerous metabolic and pathologic studies(3). The only report on electrolyte content of residual muscle in nutritional muscular dystrophy of which we are aware is that of Fenn and Goettsch(4) reporting increased sodium and decreased potassium and magnesium. Since laboratory technics have improved in the 25 years that have elapsed since Fenn and Goettsch's report, we undertook to measure muscle, erythrocyte and plasma electrolytes, as well as some other constituents of muscle and plasma aldolase and lactic dehydrogenase in rabbits with nutritional muscular dystrophy.

Materials and methods. Four male yearling New Zealand white rabbits weighing approximately 1 kg were fed a diet consisting of 13.5% vitamin-free casein, 42% sucrose, 5% lard, 13.5% brewers' yeast, 3% cod liver oil, 3% salt mixture and 20% non-nutritive fiber(5,6). A control group of 4 rabbits was fed the same diet supplemented with 50 mg α-tocopherol per kg diet mixture. After 25 days on the diet, the animals were sacrificed and sodium, potassium, calcium, magnesium and chloride in muscle, erythrocytes and plasma were measured, as well as muscle noncollagenous nitrogen (NCN), adenosine triphosphate (ATP), phosphoryl creatine (PC) and free creatine; and plasma aldolase and lactic dehydrogenase by methods already described(7).

Results and discussion. When sacrificed, the Vit. E deprived rabbits were markedly weak and showed the other stigmata of nutritional muscular dystrophy(2) while control animals were unaffected. The analytical results are summarized in Table I. Muscle from Vit. E deprived animals showed statistically significantly lower levels of muscle potassium, magnesium, ATP and PC, while levels of sodium, calcium and chloride in muscle of dystrophic rabbits were significantly higher than the levels in controls. No significant differences in erythrocyte and plasma electrolytes were observed between Vit. E deprived and control animals. Plasma levels of aldolase and lactic dehydrogenase were elevated in the Vit. E deprived animals.

In patients with hereditary muscular dystrophy, increased sodium and low potassium levels in residual muscle were noted by Horvath *et al.*(1), low muscle ATP and PC was found by Ronzoni *et al.*(8), while Dreyfus *et al.*(9) found high serum levels of aldolase and other glycolytic enzymes in dystrophic patients. Similar abnormalities have been observed in mice with hereditary muscular dystrophy(7.10). The observation of similar changes in nutritional muscular dystrophy in these studies is particularly noteworthy because nutritional muscular dystrophy is not hereditary. Plasma and tissue levels of Vit. E in hereditary muscular dystrophy are normal and the hereditary disease does not respond to administered Vit. E(11). indicating that the primary defects in the 2 diseases are probably different.

The changes observed in the Vit. E deprived animals may result from a secondary loss of cell membrane integrity. In dystrophic mice, Zierler(10) found increased loss of aldolase and potassium from excised muscle. Similar permeability studies have not been carried out in Vit. E deprived animals.

Summary. In rabbits with nutritional muscular dystrophy as a result of feeding 3% cod liver oil together with dietary Vit. E depriva-

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TABLE I. Muscle Electrolytes, ATP and PC; Erythrocyte Electrolytes; and Plasma Electrolytes, Aldolase and Lactic Dehydrogenase in Normal and Avitaminosis-E Rabbits.

	Muscle					Erythrocytes			Plasma			
	Normal		Vit. E deprived			Normal	Vit. E deprived		Normal		Vit. E deprived	
Sodium	115	± 7.8	214	± 17.7 *	meq/kg DFFM	8.03 ± 1.15	7.73 ± .57	meq/kg	144	± 8.9	138	± 1.9 meq/l
Potassium	444	± 15.9	413	± 10.8 *	"	117.3 ± 13.2	97.9 ± 1.31	"	5.67	± .91	5.02	± .51 "
Calcium	22.5	± 3.9	67.8	± 18.4 *	"	<.6	<.6	"	6.33	± .16	7.05	± .27 "
Magnesium	81.0	± 10.0	55.3	± 15.2 *	"	16.4 ± 2.6	12.1 ± 1.0	"	3.13	± .64	2.51	± .13 "
Chloride	49.3	± 6.0	174.3	± 22.3 *	"				98.8	± 3.8	97.3	± 1.7 "
NCN	138	± 1.3	128	± 1.8 *	g/kg DFFM							
Wet wt/DFFM	5.43	± .22	6.23	± .17*				Aldolase	105.5		532.5	U/ml
ATP	5.59	± .71	2.88	± .33*	μM/g			LDH	221		307	"
Creatine	319	± 45	172	± 19	* g/kg							
PC (as creatine)	176	± 26	142	± 16	"							

* Statistically significant differences ($P < .05$) in values of "dystrophic" rabbits compared to controls.

Each value is the mean ± S.D. of 4 animals.

NCN is non-collagenous nitrogen; DFFM is dry, fat-free muscle; ATP is adenosine triphosphate; PC is phosphorylcreatine and LDH is lactic dehydrogenase.

tion, residual muscle was found to have lower potassium, magnesium, adenosine triphosphate and phosphorylcreatine levels than controls while levels of sodium, calcium and chloride were increased. Erythrocyte and plasma electrolytes did not differ significantly from controls. The dystrophic animals also had elevated levels of plasma aldolase and lactic dehydrogenase.

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