with either vit. B_{12} or folic acid administration. In all cases, the values for the N^1-methylnicotinamide tended to increase on the third day of nicotinamide administration. Hence there is no apparent trend toward exhaustion of "methyl donator stores."

The values for the 24-hour excretion of N^1-methylnicotinamide previous to nicotinamide supplementation as recorded in Table I are all within the range of 3 to 17 mg reported by Huff and Perlzweig(10) for 67 normal male medical students. The values of methylated compound excreted are somewhat higher in terms of percent of nicotinamide administered than other reports(9) in the literature, but the nicotinamide dose in these experiments was also greater.

There is no indication that under the conditions here employed the methylation of nicotinamide can be used as a measure of the nutritional adequacy of vit. B_{12} or folic acid. Apparently the state of vit. B_{12} and folic acid deficiency existing in pernicious anemia does not influence the patient's ability to methylate nicotinamide.

It is possible that the administered doses of nicotinamide are not large enough to produce a condition of metabolic stress on the "methyl donators." It is also considered possible that in humans, vit. B_{12} and folic acid are not concerned in the enzyme systems governing either the transfer or formation of the methyl group of N^1-methylnicotinamide.

Summary. Administration of nicotinamide to four patients with pernicious anemia results in the urinary excretion of approximately 20% of the compound as the methylated derivative. The levels of N^1-methylnicotinamide are similar before and after therapy. The methylation of nicotinamide, under the conditions here employed, cannot be used as an index of the nutritional adequacy of vit. B_{12} or folic acid.

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Influence of Desoxycorticosterone Acetate and Cortisone Acetate on Body Weight of Chick Embryos.* (19031)

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A retardation in growth of chick embryos without increasing the incidence of developmental defects was noted by Landauer(1) following the injection of adrenal cortical extract into the yolk sac. Recently, Karnofsky and his associates(2,3) have studied cortisone acetate and noted the effects of this steroid on chick embryo development. A definite growth inhibiting action was demonstrated for cortisone acetate and the "cortisone-effect" also involved characteristic developmental modifications. It appeared of interest to estimate the effect of an adrenal steroid not oxygenated at C-11 and for this purpose desoxycorticosterone acetate (DCA) was chosen for comparison with cortisone acetate at the same dosages.

Varying amounts of DCA (Percorten, Ciba)† and cortisone acetate (Merck) were injected into the yolk sac of White Leghorn chick embryos on the 6th day of incubation. The embryos were sacrificed on day 18,

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1. Landauer, W., *Endocrinology,* 1947, v41, 489.

2. Karnofsky, D. A., Stock, C. C., and Rhoads, C. P., *Fed. Proc.,* 1950, v9, 290.

3. Karnofsky, D. A., Ridgway, L. P., and Patterson, P. A., *Endocrinology,* 1951, v48, 596.

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TABLE I. Influence of Adrenal Steroids on Body Weight of Chick Embryos Injected on Day 6 and Body Weight Taken on Day 18 of Incubation.

No. embryos	Treatment	Avg body wt, g
17	.25 mg desoxycorticosterone acetate	17.7 ± 1.2*
17	Uninjected controls	24.5 ± .6
30	.50 mg desoxycorticosterone acetate	15.6 ± .9
24	.1 ml sesame oil	20.9 ± .4
30	Uninjected controls	20.9 ± .5
14	.25 mg cortisone acetate	24.5 ± .8
28	.50	20.6 ± .7
14	1.00	20.6 ± 1.1
21	Uninjected controls	24.9 ± .5

$$^{*}\,E = \sqrt{\frac{\Sigma d^2}{n(n-1)}}.$$

weighed and examined for morphological abnormalities. Non-injected or vehicle injected eggs were incubated with each experimental group as some variation in body weight was noted among the control embryos.

The data are presented in Table I. It is evident that DCA when administered as one injection of 0.25 mg effectively retarded body weight increase and that a dosage of 0.5 mg was no more effective. Both dosages, however, significantly retarded body weight increase without inducing abnormalities whereas sesame oil alone had no effect. In comparison, cortisone acetate did not influence body weight increase at the 0.25 mg dosage in contrast to DCA. However, 0.5 mg significantly depressed body weight gain but again a doubling of the steroid dose was no more effective. The dosages of cortisone acetate which were effective on body weight did not induce morphological defects. These data are in essential agreement with Karnofsky, Ridgway, and Patterson(3) who consider the minimum effective dose of cortisone acetate for an 8-day embryo to be 1.5 mg, the LD_{50} being 2.0 mg. These investigators(3) established a minimum dosage effect at a level which caused morphological changes but did find that smaller amounts of cortisone acetate had some effect on body weight only. Karnofsky, Ridgway, and Stock(4) mention that DCA is active at 0.9 mg per egg. In contrast, a significant growth depressing effect was obtained in our studies with 0.25 mg of DCA, a dosage which proved ineffective with cortisone acetate. The mechanism of this steroid action is not apparent.

Summary. A retardation of body weight increase in White Leghorn chick embryos was obtained by injecting either desoxycorticosterone acetate or cortisone acetate into the yolk sac at 6 days of incubation and observing the effects on day 18. Desoxycorticosterone acetate depressed body weight increase at a dosage level of 0.25 mg per egg, whereas 0.5 mg of cortisone acetate was required to induce the same effect.

4. Karnofsky, D. A., Ridgway, L. P., and Stock, C. C., *Fed. Proc.*, 1951, v10, 204.

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