TABLE I.
Relation of Size of Litter to the AP Lactogen Content of Nursing Rabbits.

Group	No. of rabbits	Body wt avg, g	Pituitary wt avg, mg	Units per pituitary		Units per mg pituitary tissue		Units per 100 g body wt	
				R.-T.*	I.U.*	R.-T.	I.U.	R.-T.	I.U.
Control	11	3360	42.20	30.05	1.35	.71	.032	.89	.040
Exp.	10	3617	41.03	27.10	1.22	.66	.030	.75	.034

*1 International Unit = 22.2 Reece-Turner units (Meites, J., Bergman, A. J., and Turner, C. W., *Endocrinology*, 1941, **28**).

It will be seen that there is an average of 30.05 Reece-Turner units or 1.35 I.U. of lactogen per pituitary in the control rabbits which were allowed to keep their entire litter. Holst and Turner,[6] using a similar group of 4 lactating rabbits with entire litters, found an average of 26.63 Reece-Turner units or 1.20 I.U. per pituitary. In the experimental group of rabbits, with litters uniformly reduced to 2 young, there was an average of 27.10 Reece-Turner units or 1.22 I.U. per pituitary. Since the difference between these 2 groups is too small to be significant, it is concluded that the size of the nursing litter does not influence the lactogen content of the pituitaries of nursing rabbits.

Summary. The lactogen content of the AP was quantitatively determined in 21 lactating rabbits killed on the 20th day postpartum, No significant difference was found in hormone content between the 11 does which were permitted to keep their entire litters of 5 to 11 young, and the 10 does whose litters were uniformly reduced to 2 on the 5th day postpartum.

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Effect of 1(—)-Tyrosine on Liver Glycogen of the Normal Rat.*

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In an earlier publication[1] the effect of feeding *dl*-phenylalanine and *dl*-tyrosine on the liver glycogen of rats was described. The former

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1 Butts, J. S., Dunn, M. S., and Hallman, L. F., *J. Biol. Chem.*, 1938, **123**, 711.

compound gave rise to an appreciable amount of glycogen in the liver, whereas the hydroxy acid failed to elicit a significant response. In this communication the effect of feeding the naturally occurring isomer of tyrosine on liver glycogen formation is reported.

Female rats ranging from 130-180 g in weight were used in this study. They were subjected to a 48-hour fast preceding the feeding of the tyrosine. Filter paper was placed in the cages as it was found that this would be eaten and cause bulk, thereby decreasing the chances of diarrhea. As demonstrated earlier[2] this ingested paper had no effect on glycogen formation.

The insolubility of the tyrosine precluded the possibility of feeding it as the free acid. Since neither the hydrochloride nor the sodium salt is well tolerated in fairly large amounts, a suspension of the 1(—)-tyrosine was prepared as follows: the tyrosine was ground to a fine powder, suspended in a 1.75% aqueous gum tragacanth medium in a proportion of 10 g of the acid to 100 cc of the medium, giving a uniform suspension readily administered by stomach tube. To avoid a large excess of the tyrosine at any one time, the animals were fed approximately 200 mg every 2 hours. The control animals were similarly fed 2 cc of the gum tragacanth suspension containing 5 g sodium chloride per 100 cc of the suspension.

At the end of 3, 6, 9, and 12 hours, groups of animals were sacrificed, under amytal anesthesia, the livers removed and analyzed for glycogen according to the method of Good, Kramer and Somogyi.[3]

To prove that an excess of tyrosine had been fed the gastrointestinal tracts were removed, minced, and extracted three times with dilute hydrochloric acid. The washings from each sample were pooled and 10 cc of a 20% trichloracetic acid solution was added. After standing 24 hours the samples were made up to a known volume and filtered. They were then analyzed for amino nitrogen by the Van Slyke procedure. Although the method used did not quantitatively recover the amino acid present, as judged by the recovery of known amounts of tyrosine, it was sufficiently accurate to establish the presence of an excess of the amino acid. The average recovery was 82%, with known amounts of tyrosine. Therefore, it seemed certain that an excess of tyrosine was present and that maximum absorption was occurring at all times.

Table I gives the results of 1(—)-tyrosine feeding on liver glycogen deposition.

[2] Butts, J. S., Blunden, H. D., and Dunn, M. S., *J. Biol. Chem.*, 1937, **119**, 247.

[3] Good, C. A., Kramer, H., and Somogyi, M., *J. Biol. Chem.*, 1933, **100**, 485.

TABLE I.
Glycogen Content of Livers of Female Rats Receiving 1(—)-Tyrosine.

Control				1(—)-Tyrosine			
No. of animals	Elapsed time, hr	%	Stand. Dev. of mean	No. of animals	Mg Tyrosine fed	%	Stand. Dev. of mean
15	3	.09	± .015	8	300	.26	± .045
15	6	.09	± .010	8	600	.39	± .016
14	9	.06	± .001	12	800	.67	± .078
16	12	.04	± .006	9	800	.69	± .104

Discussion. In view of the earlier findings that *dl*-tyrosine when fed to a starving rat did not cause an appeciable deposition of liver glycogen it is rather surprising that under comparable conditions l(—)-tyrosine does cause an increase. One is therefore forced to the conclusion that under similar experimental regimes, the l(—)-tyrosine quantitatively effects liver glycogen formation in a very different manner from the racemic mixture.

It was noticed that when urine from a rat fed l(—)-tyrosine was treated with ammonium phosphomolybdate, as in the Briggs[4] method for homogentisic acid, a copious reduction occurred. The animals were fed 4 mg per g of body weight per day. The feeding was continued for 6 days with no other food available during this time. Although less intense reduction occurred when the amount of l(—)-tyrosine was lowered to 2 mg per g per day a distinct color did develop.

It is not claimed that homogentisic acid alone was responsible for this effect, as it is known that other di-hydroxy compounds cause a similar reduction. Actually there is no proof that homogentisic acid was present, as we were unable to isolate a derivative of this compound. Under similar conditions when *dl*-tyrosine was fed no detectable reduction occurred.

Conclusion. l(—)-Tyrosine when fed to rats starved 48 hours produces an increase in liver glycogen. This is in marked contrast to the result obtained when *dl*-tyrosine is fed. The urine from rats fed l(—)-tyrosine caused a reduction of ammonium phosphomolybdate. Under similar experimental conditions *dl*-tyrosine caused no such reduction.

[4] Briggs, A. P., *J. Biol. Chem.*, 1922, **51**, 453.