Increased Uptake of Iodine-131 by the Thyroid Gland after Administration of Hesperidine Methyl Chalcone. (19985)

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Scott(1) has shown that the rat's thyroid takes up 50 to 200% more radioiodine under the influence of hesperidine methyl chalcone (H.M.C.). It was therefore decided to try this drug in human beings in order to determine its effect upon thyroidal accumulation of radioiodine. This drug is known to suppress the output of I^{131} through the kidney; probably I^{131} stays in the body in circulation a long enough time to favor increased uptake by the gland.

Method. Nineteen patients having the diagnoses shown in Table I were studied. All of these were considered to be euthyroid except K.E. and O.C. who were judged to be hyper- and hypothyroid respectively. Two periods of tests were done on each patient. These were the first or the patient's control period before the administration of H.M.C. and the second or his testing period following the administration of H.M.C. At the start of the control period, 20 μc I^{131} was given orally. Thyroid gland uptake measurements were done at 24-hour intervals for 96 hours. After a rest period of three days, the same patient's thyroid uptake was again measured for residual I^{131}, then 1.0 g of H.M.C. mixture in water was ingested. After an hour lapse to allow for effective absorption of the drug the patient ingested 80 μc I^{131}. At 24-hour intervals, for 96 hours, his thyroid gland uptake was measured and due correction for decay and effective half-life was made for the residual I^{131} uptake from the control period. The tracer dose of 80 μc I^{131} was large enough to be readily measurable in the presence of residual I^{131} and thus also to minimize statistical differences between the measurements of the two periods.

Results. Studies on the nineteen patients

TABLE I. Percentage Increase of Thyroid Uptake over Normal Following Oral Administration of Hesperidine Methyl Chalcone. I^{131} was administered orally to label iodide pool of body.

Patient	Hr after I^{131} and H.M.C. 24	48	72	96	Avg increase	Diagnosis
B.B.	16	0	16	19	13.7	Hypertension
M.W.	31		70	34	45	Peptic ulcer
W.S.	41	16	16	21	24	Diabetes mellitus
V.J.	36	20	20	36	28	Inf. hepatitis
K.E.	14	19	28	12	18.3	Hyperthyroidism
O.C.	75	60	18	67	55	Gun shot wound—chest
W.J.	17	8	0	8	8.3	Prob. tbc.
L.J.	18	5			11.5	Gastric ulcer
H.M.	25	29	17	30	25.2	Hepatitis
L.E.	15	24	11	5	13.8	Epilepsy
C.J.	14	30	18		20.7	Hypertension
C.E.		30		5	17.5	Esophagitis
T.G.	12	30			21	Ca lung
Mc G.B.	60	44	32	35	42.8	Rheum. arthritis
U.W.	25	10	24	30	22.3	Hemiplegia
C.D.	9	13	77	50	37.3	Gastric ulcer
W.H.	0	8	0	17	6.3	Arteriosclerosis with myocarditis
G.J.	17	27	41	50	33.7	Diabetes mellitus
O.P.	32	0	4	0	9	Encephalitis
	25.4 ±2.75*	20.7 ±3.6	24.5 ±5.45	26.1 ±4.67		

* Mean stand. error $= \pm\sqrt{\frac{\Sigma \text{ dev}^2}{n(n-1)}}$.

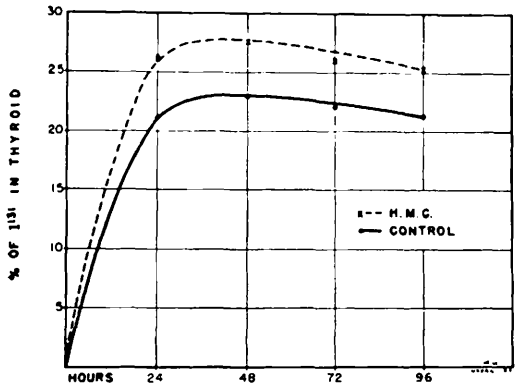

FIG. 1. Average thyroid uptakes on 19 patients before and after hesperidine methylchalcone (H.M.C.) administration using I^{131} as a tracer.

summarized in Table I show that the oral administration of one gram of H.M.C. significantly increases the thyroidal accumulation of I^{131}. This increase averaged between 20.7 and 26.1% at all observed time periods. H.M.C. was more effective in increasing thyroid uptake in some patients than in others. In this study the range was from 6.3% to 55% greater than pre-H.M.C. period when the uptakes for the four time periods were averaged. The shape of the composite uptake curves when H.M.C. was administered is similar to that obtained before the administration of H.M.C. (Fig. 1). Although it was not possible to obtain hourly or 24-hour urine samples on all patients, they were not significantly different when H.M.C. was administered.

Discussion. These data suggest that the bioflavone derivative, H.M.C., is capable of increasing the thyroidal accumulation of I^{131} in man. These results are consistent with those obtained in rats. In all of the subjects studied, an increase in thyroid uptake was observed. If this drug is used in conjunction with I^{131}, smaller doses might be required to obtain comparable results in thyroid therapy. Such possible usefulness should be evaluated.

1. Scott, Kenneth G., PROC. SOC. EXP. BIOL. AND MED., in press.

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Participation of Adrenal Cortex in Alterations in Carbohydrate Metabolism Produced by Epinephrine.* (19986)

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The subcutaneous injection of epinephrine (0.02 mg per 100 g body weight) into fasted normal rats is followed in a period of 4 hours by a depletion of muscle glycogen and an accumulation of glycogen in the liver(1). Since similar injections of epinephrine also cause a release of adrenal cortical steroids by reason of their capacity to cause ACTH secretion(2), the question arises as to the possible participation of the adrenal steroids in the alterations in carbohydrate metabolism that follow epinephrine injection.

This has been tested by comparing the effects of epinephrine injection in fasted normal and adrenalectomized rats. In addition, since marked differences in the response of these animals was observed, the influence of adrenal cortical hormones in correcting this difference was also estimated.

Methods. Male Wistar rats weighing 180-280 g were used. All animals were kept in a constant temperature room and on a uniform and constant diet. All were fasted for 24 hours before the experiment. Adrenalectomized animals received 1% NaCl as drinking fluid after operation and were used 7-14 days later. Glycogen in liver and muscle was determined by the usual methods, and blood glucose and lactate on samples obtained from

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