EDITORIAL REVIEW

Nutritional effects on thyroid and catecholamine metabolism

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Introduction
Adaptation to a change in energy intake is fundamental to survival and in prolonged semi-starvation, energy expenditure can be halved (Keys, Brozek, Henschel, Mickelson & Taylor, 1950). Similarly, overfeeding normal young men induces a marked alteration in metabolism, a weight gain of only 10–15% being accompanied by a 70% increase in energy expenditure (Sims, Danforth, Horton, Bray, Glennon & Salans, 1973). Thus man has adaptive mechanisms which generate large changes in energy output, and this seems to involve two major thermogenic hormones, thyroid and catecholamines. The thyroidal changes in response to starvation will be considered first.

Thyroidal changes in starvation

Effects on thyroxine
The total concentration of free and bound thyroxine (T₄) usually remains unchanged (Portnay, O’Brian, Bush, Vagenakis, Azizi, Arky, Ingbar & Braverman, 1974; Grant, Edwards, Howard, Challand, Wraight & Mills, 1978) but if it does fall (Rothenbuchner, Loos, Kiessling, Birk & Pfeffier, 1973; Carlson, Drenick, Chopra & Herschman, 1977; Pamblad, Levi, Burger, Melander, Westgren, Van Schenck & Skude, 1977) then recovery occurs as the fast continues (Carlson et al., 1977). Circulating concentrations of T₄ will be affected by release from the thyroid and peripheral metabolism. Whereas some authors (Vagenakis, Burger, Portnay, Rudolph, O’Brian, Azizi, Arky, Nicod, Ingbar & Braverman, 1975) have found no change in the T₄ production rate, Croxson, Hall, Kletzky, Jaramillo & Nicoloff (1977) have observed a fall in thyroidal iodine release which ‘escaped’ on the fifth day of the fast; this appears unrelated to iodine intake (Carlson et al., 1977). Any fall in T₄ production will be balanced by a reduction in T₄ catabolism to tri-iodothyronine (T₃) (Grant et al., 1978), so that only small changes in T₄ concentrations have been observed during fasting or semi-starvation. A rise in free T₄ has been observed during starvation (Portnay et al., 1974; Pamblad et al., 1977; Azizi, 1978), but it is unlikely to be due to the recognized fall in thyroid-binding globulin and thyroid-binding pre-albumin (Schatz, Sheppard, Palter & Jaffri, 1967; Shetty, Watrasiewicz, Jung & James, 1979b) since only about 30% of the former and less than 2% of the latter protein are normally saturated with T₄. Since plasma free fatty acids in physiological concentrations can displace T₄ from the plasma proteins (Hollander, Scott, Burgess, Rabinowitz, Merimee & Oppenheimer, 1976), the increase in free fatty acids on starvation could be expected at least to double the free T₄ concentration.

Sites and control of T₄ metabolism
In normal euthyroid man, the thyroid secretes about 24% of the T₄ produced in the body and only 2.5% of the reverse T₄ (rT₄) (Chopra, 1976). Just over 80% of the T₄ secreted is deiodinated to either T₃ or rT₃, the rest being excreted into the intestine or metabolized to tetraiodothyroacetic acid. The daily production rates of T₃ and rT₃ are similar but, as the thyroid secretes more T₃ and rT₃, this implies that more T₄ is deiodinated peripherally to rT₃ (Chopra, 1976). Peripheral deiodination is more important than thyroidal output in determining the plasma concentrations of T₃ and rT₃.
Liver and kidney, both of which undergo profound metabolic changes on starvation (Cahill, 1976), appear to be the most active peripheral sites for T₃ production (Chopra, 1977), the liver, on an organ basis, contributing twice as much T₃ as the kidney. Muscle, despite its large mass, contributes very little and heart, spleen, brain and intestine are relatively inactive, although the foetal brain shows greater activity. It is unlikely that each cell controls its own deiodination of T₄ to T₃ in order to meet its own needs, since metabolically active tissues, such as the adult brain, have relatively poor deiodination activity.

**Tri-iodothyronine**

All investigators have observed a rapid decline in T₁ on starvation (Vagenakis et al., 1975; Carlson et al., 1977; Chopra, 1977; Azizi, 1978) but the delay in the response varies from 1 to 4 days and may depend on the carbohydrate content of the pre-fasting diet (Azizi, 1978). The fall in T₃ is less in obese women than obese men, perhaps in response to the stimulating effect of oestradiol on the peripheral conversion of T₄ into T₃ (Azizi, 1978).

The fractional catabolic rate expressed as a percentage of the T₃ pool remains unchanged during the fasting period (Balsam, Ingbar & Sexton, 1978), but the slower rate of T₃ production must be balanced by a fall in the absolute rate of T₃ catabolism for serum T₃ to stabilize at a lower level. The production rate of T₃ seems to be the important controlling factor and in association with fasting the free T₃ falls as well as the total serum T₃ (Carlson et al., 1977; Azizi, 1978). This response in free T₃ differs therefore from that of free T₄.

**Reverse T₃**

Most investigators have found a progressive increase in rT₃ on starvation, beginning much later than the fall in serum T₃ (Carlson et al., 1977; Pamblad et al., 1977; Azizi, 1978). After 2–3 weeks of fasting or semi-starvation the rT₃ returns to normal (Carlson et al., 1977) at a time when T₃ concentrations are being maintained at a low level. Changes in T₄ and rT₃ are therefore dissociated and the changes in rT₃ depend mainly on alterations in rT₃ catabolism, unlike the primary change in T₃ production (Vagenakis et al., 1975; Eisenstein, Haggis, Vagenakis, Fang, Ransil, Burger, Balsam, Braverman & Ingbar, 1978). The catabolic rate of rT₃ is much higher (Chopra, 1977) than the rate of T₃ breakdown, thus explaining the much smaller circulating concentrations of rT₃ despite its higher production rate. These nutritional studies, together with drug experiments (Chopra, Williams, Orgiazi & Solomon, 1975; Chopra, 1977), therefore support the concept of a dual T₄–deiodinating enzyme system, which also accounts for the differences in T₃ and rT₃ production rates in semi-starvation (Chopra, 1976).

**Dietary control of T₃ and rT₃ metabolism**

Although the conversion rate of T₄ into T₃ falls on fasting or semi-starvation, it is becoming apparent that this enzymatic step is not affected solely by alterations in energy supply. Spaulding, Chopra, Sherwin & Lyall (1976) fed three obese patients with three types of diets containing 3347 kJ (800 kcal); the first consisted only of carbohydrate, the second 20% protein, 80% fat, and the third was 25% carbohydrate (50 g), 20% protein and 55% fat. Studies were undertaken between periods of weight maintenance on an unspecified regimen. T₄ remained unchanged on all three test diets, but T₃ fell on the carbohydrate-free diet only, the fall being equivalent to that seen on total fasting. This suggested that dietary carbohydrate was the key to the preservation of T₃ concentrations and that only 50 g was sufficient to maintain serum T₃. However, two of the three subjects on the carbohydrate-only diet began the test diet with low T₃ concentrations so the evidence is inconclusive. We have shown in 11 obese women that an energy intake of about 2510 kJ (600 kcal), which included 85 g of carbohydrate, failed to maintain serum T₃ (Jung, Shetty, Barrand, Callingham & James, 1978). Nevertheless, these studies did involve a substantial reduction in carbohydrate intake from 510 g daily.

The specificity of carbohydrate has recently been confirmed by Danforth, Horton, Sims, Burger, Vagenakis, Braverman & Ingbar (1979), who initially equilibrated three subjects on a known-weight maintenance diet and then fed an isocaloric diet in which the carbohydrate was completely replaced by fat. This resulted in a marked fall in circulating T₃ levels equivalent to that seen in normal subjects fasted for 8 days. It remains unclear, therefore, how much the carbohydrate intake needs to be reduced before alterations in thyroid function occur. After a 4-day fast, refeeding obese patients on a 3347 kJ (800 kcal) diet with 70 g of carbohydrate resulted in a return of the T₃ concentration to normal within 4 days (Azizi, 1978). Thus the combination of our studies with 85 g of
carbohydrate and those of Azizi (1978) with 70 g of carbohydrate suggest that T₃ production is sensitive to the change in carbohydrate intake rather than to the absolute intake of carbohydrate.

This sensitivity of T₃ production to specific changes in the diet has been confirmed by experiments in vitro with liver slices from animals either fasted or fed on various amounts of glucose (Balsam et al., 1978). The rate of production of T₃ depended on the amount of glucose provided in the drinking water. As direct addition of glucose to the incubated liver slices failed to affect T₃ production, glucose by itself does not appear to be the key factor responsible for altering T₄ deiodination. T₃ production in rat microsomal fractions is enhanced more by cytosol from glucose-fed than from starved animals, although the activity of the cytosol from the fasted rats can be restored to normal fed conditions by enrichment with either nicotinamide–adenine dinucleotide hydrogen phosphate (NADPH) or glutathione (GSH) (Balsam, Ingbar & Sexton, 1979). GSH acts directly on the enzyme in the microsomal fraction, whereas NADPH acts within the cytosol, possibly by increasing the availability of GSH (Balsam et al., 1979). The stimulatory effect of NADPH suggests that the activity of the pentose phosphate pathway may influence T₃. The addition of insulin and glucagon to liver homogenates does not affect T₃ production (Chopra, 1977), so these hormones are unlikely to initiate directly the changes in T₃ production observed on fasting.

Overfeeding can also increase serum T₃. Danforth et al. (1979) overfed subjects for 3 months with either an excess of carbohydrates or fat, but found a rise in serum T₃ only in those overfed with carbohydrates. However, Davidson & Chopra (1979) have suggested that an excess of non-carbohydrate calories might modulate T₃ production when a normal amount (~200 g) of carbohydrate is fed.

Spaulding et al. (1976) have suggested that changes in rT₃ depend on energy intake rather than dietary carbohydrate since they found that rT₃ increased by 58% on fasting but not on any of their three 3347 kJ diets. However, Danforth et al. (1979) found that a weight-maintenance isocaloric but carbohydrate-free diet led to an increase in serum rT₃ similar to that found on fasting. Dietary protein may also affect rT₃ metabolism, since semi-starvation on a 837 kJ (200 kcal) diet with 37.5 g of protein possibly leads to a greater increase in rT₃ than is seen on a similar energy intake with only 3 g of protein (Byfield, Durrant, Bird, Land, Royston, Himsworth & Garrow, 1978). As catabolism is the dominant factor affecting rT₃ concentrations, it is not surprising that the dietary factors altering rT₃ concentrations are different from those affecting T₃ levels. The dietary induction of altered rT₃ levels are probably not physiologically important since rT₃ appears to be metabolically inert.

Other mechanisms for the control of T₃ production

Since a large number of hormonal changes occur on starvation, the peripheral T₄ deiodination step could be affected by signals other than those relating directly to hepatic carbohydrate metabolism. Steroidal hormones could be involved since pharmacological doses of dexamethasone result in a fall in T₃ and an increase in rT₃ (Chopra et al., 1978). An increase in plasma cortisol has been reported on fasting (Galvao-Teles, Graves, Burke, Fotherby & Fraser, 1976; Pambld et al., 1977), although Croxson et al. (1977) found no alteration in either the diurnal pattern of plasma cortisol or in urinary output of cortisol or 17-hydroxycorticosteroids. When Jensen, Noland & Jubbi (1978) gave prednisone daily to hypothyroid patients recovering on T₄ supplements, they found no effect on peripheral T₄ deiodination. In this study the steroid intakes were closer to physiological levels and since work in vitro with liver homogenates suggested that the effect of steroids on peripheral deiodination is dose-dependent (Chopra, 1977; Balsam et al., 1978), the experiments with dexamethasone were probably not physiologically or nutritionally relevant.

TSH does not affect the rate of T₃ deiodination (Chopra, 1977) when tested in vitro. It is also unlikely that the inhibitory action of rT₃ on T₄ deiodination to T₃ in vitro is physiologically important as Woebber & Maddux (1978) have noted clear inhibition of T₃ production only with a rT₃:T₄ ratio of 1:10, whereas the normal ratio in man increases from 1:240 in the fed state to only 1:160 on starvation. Adrenaline enhanced T₃ into T₃ conversion in animal tissues (Galton, 1965), whereas amiodarone, an adrenergic blocking agent, decreased the generation of T₃ (Burger, Dinickert, Nicod, Jenny, Le Marchand-Beraud & Vallotin, 1976; Balsam et al., 1978). These effects can be reproduced in vivo. Thus propranolol produces a fall in serum T₃ by reducing its production, and induces a rise in serum rT₃ in hyperthyroid, euthyroid and hypothyroid patients on T₄ supplements (Theilade, Hansen, Skovsted, Faber, Kirkegård, Nutrition, thyroid and catecholamines 185
the evidence on the effect of nutritional changes on catecholamine metabolism.

**Pituitary–thyroid interrelationships**

With the fall in circulating $T_3$ concentrations one would normally expect the pituitary to respond by increasing TSH output as the $T_3$ inhibition of both TRH secretion from the hypothalamus and TSH output from the pituitary was removed. Yet circulating levels of plasma TSH either remain the same (Carlson et al., 1977; Pamblad et al., 1977) or even fall (Croxson et al., 1977; Azizi, 1978). Croxson et al. (1977) observed an immediate loss of the diurnal rhythm of TSH secretion on fasting, which suggests that the inflow of food was the primary signal for the circadian cycle in TSH release. The signal presumably acts in the hypothalamus, which is both involved in the regulation of food intake and the control of the pituitary. The effect appears to be specific since the clinical variation in plasma cortisol, which is dependent on pituitary ACTH output, is preserved during fasting (Croxson et al., 1977).

A blunted TSH response to exogenous TRH can be shown within 35 h of fasting and the effect may be more generalized since prolactin secretion may also be reduced (Vinik, Kalk, McLaren & Paul, 1974; Vinik, Kalk, McLaren, Hendricks & Pimstone, 1975). The pituitary remains sensitive to circulating thyroidal hormones since maintaining the serum $T_3$ level during fasting with exogenous $T_3$ leads to a decrease in TSH concentrations and a fall in the pituitary response to TRH. Reducing plasma $T_4$ by pretreating with potassium iodide also induces an enhanced TSH response (Gardner, Kaplan, Stanley & Utiger, 1979).

Since the pituitary remains responsive to $T_4$ and $T_3$ during fasting, the decline in TSH suggests either a diminished output of TRH or the role of an inhibitory factor acting at the level of the pituitary. The change in circulating $T_3$ cannot be responsible for inducing this change since the use of propylthiouracil to reduce serum $T_3$ leads to the expected increase in basal TSH and an increased response to TRH (Geffner, Azukizawa & Hershman, 1975). Glucocorticoids (Wilber & Utiger, 1969) and growth hormone (Root, Snyder, Rezvani, DiGeorge & Utiger, 1973) can both reduce TSH responsiveness, but neither are markedly increased in fasting (Carlson et al., 1977) and metyrapone, given to block adrenal steroid genesis, does not alter the TRH response during fasting (Croxson et al., 1977). Furthermore, pituitary adaptation does not appear to be an intrinsic response since pituitary slices from starved animals, studied in vitro, preserve their response to $T_4$ and $T_3$ (Chopra, Carlson & Solomon, 1978).

Pituitary inhibitory factors are recognized and include dopaminergic neurones. L-Dopa reduces the TRH responsiveness of the pituitary (Refetoff, Fang, Rapoport & Friesen, 1974) and dopamine inhibits TSH release in hypothyroidism (Scanlon, Weightman, Mora, Heath, Shale, Snow & Hall, 1977). Further evidence for the dopaminergic regulation of TSH is outlined in a previous Editorial Review (Scanlon, Rees Smith & Hall, 1978). Whether fasting induces changes in the pituitary-linked dopaminergic system is uncertain, but might be inferred from the changes in pituitary reactivity during fasting and is suggested by the reported reduction of a raised prolactin level in a patient with hypothyroidism during starvation (Croxson et al., 1977).

Adjustments in pituitary responsiveness also occur on overfeeding. Despite an increase in $T_3$ in those individuals overfed with carbohydrate, there is no suppression in either the basal concentrations of TSH or the peak TSH response to TRH. The rate of decline of TSH after TRH stimulation does accelerate on overfeeding, but this acceleration may not depend on altered pituitary function (Danforth et al., 1979). Overfeeding with fat did not produce an increase in $T_3$ nor any change in TSH. Thus the alterations in thyroid metabolism are, as in underfeeding studies, dependent mainly on the carbohydrate intake. The response to overfeeding with carbohydrate differs from that in hyperthyroidism since on overfeeding there is a selective response in $T_3$ turnover compared with the increase in both $T_4$ and $T_3$ turnover found in hyperthyroidism.

**Nutritional influences on catecholamine metabolism**

Catecholamine metabolism is markedly affected by both under- and over-nutrition. When obese adults are switched from a 10-9 MJ (2600 kcal) to 2-5 MJ (600 kcal) diet, we have found a 40% decrease in
the urinary excretion of 4-hydroxy-3-methoxy-mandelic acid within 48 h of energy restriction (Jung, Shetty, Barrand, Callingham & James, 1979b). Within the first 24 h of dieting there is an appreciable decrease in the urinary output of 4-hydroxy-3-methoxymandelic acid, which, given the time taken to clear pre-existing metabolites from body water, implies that there is a rapid fall in catecholamine metabolism when the food supply is interrupted. Since these studies were conducted on standardized protein intakes, a fall in tyrosine or phenylalanine input cannot be considered responsible.

Carbohydrate supply or a change in energy intake as such appears to be responsible as energy intake was reduced by restricting the carbohydrate intake alone. The fall in 4-hydroxy-3-methoxy-mandelic acid intake was reduced by restricting the carbohydrate cholamine metabolism when the food supply is interrupted. Since these studies were conducted on standardized protein intakes, a fall in tyrosine or phenylalanine input cannot be considered responsible.

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4.8 to a normal value of 17.1 mg/l and semi-starved subjects (Kolanowski, Jeanjean & Lambert, 1975) consistent with a reduction in sympathetic activity. These nutritional changes in noradrenaline turnover are not seen in mice with lesions of the ventromedial nucleus (Young & Landsberg, 1978a), suggesting that they reflect an active suppression of the sympathetic system at the hypothalamic level, a concept in keeping with the suggestion made above that the pituitary may also be in part inhibited during fasting.

Salt restriction alone tends to increase plasma noradrenaline and urinary free noradrenaline (Kelsh, Light, Luciano, Oliver & Vardnay, 1971). This may reflect the overflow of noradrenaline from an increase in peripheral neuronal vasomotor activity in an attempt to maintain plasma volume rather than indicating a true effect of salt restriction on catecholamine metabolism. The reported increase in cardiac noradrenaline turnover in salt-restricted animals (Young & Landsberg, 1978b) may also reflect a selective change in cardiovascular function on salt restriction.

Concomitant salt restriction may explain the reported rise in urinary 4-hydroxy-3-methoxy-mandelic acid on a 3 day fast in both normal-weight and obese subjects (Januszewicz, Sznajderman-Ciswicka & Wocial, 1967; Misbin, Edgar & Lockwood, 1970). However, this is not a consistent finding for others have found no increase (Pinter & Pattee, 1968; Drenick, Alvarez, Tamasi & Brickman, 1972), and Brodows, Campbell, Al-Azy & Pi-Sunyer (1976) actually observed a fall in urinary catecholamine output during a 48 h fast. If sodium input is maintained then urinary noradrenaline and 4-hydroxy-3-methoxymandelic acid consistently fall in fasting and semi-starved subjects (Kolanowski, Jeanjean & Lambert, 1975; Jung et al., 1979b).

In malnourished children, 4-hydroxy-3-methoxy-mandelic acid output is high when expressed either on an absolute basis or in terms of body-surface area (Ramirez, Fletes, Mizrahi & Parra, 1978), but whether these children were infected (a frequent complication) or were already being treated with a high energy diet is unclear. The importance of concomitant stress or infection in over-riding the effect of food restriction is borne out by an analysis of the catecholamine output of malnourished children with and without infection. Graham & Placko (1975) found elevated catecholamine concentrations in infected malnourished children, but their data show that, in five uninfected marasmic infants, the free catecholamine excretion rose from 9.8 ± 4.8 to a normal value of 17.1 ± 3.2 μg day⁻¹ m⁻² surface area during partial recovery. Similar reductions in catecholamine output were found in uninfected children with kwashiorkor (Hoeldtke & Wurtman, 1973) and increased during rehabilitation (Parra, Klish, Cuellar, Serrano, Garcia, Argote, Canseco & Nichols, 1975).

The concept of a reduced catecholaminergic drive during semi-starvation or fasting, developed by Young & Landsberg (1977a), seems counter to many of the accepted ideas on the control of plasma substrate concentrations during fasting. Traditionally the increase in lipolysis, the maintenance of adequate plasma glucose concentration, and the higher glucagon output on fasting have all been taken as indicative of an enhanced catecholamine drive. Several approaches have now combined to show that these responses are not dependent on increased catecholamine activity. Adrenalectomy in animals does not prevent the increase in free fatty acid output on fasting (Levy & Ramey, 1958; Goodman & Knobil, 1959; Edmondson & Goodman, 1962; Stern & Maickel, 1963; Åkerblom, Martin & Cingolani, 1969) and replacing adrenal cortical function with a constant...
dose of steroids in these animals allows a normal free fatty acid response to fasting. Misbin et al. (1970) fasted men for 68 h and tested the effect of total adrenergic blockade with phentolamine and propranolol. Adrenergic blockade reduced but did not suppress the increase in fatty acids. If adrenergic blocking agents are given after 58–62 h of fasting, then they have little effect on the elevated concentrations of free fatty acid (Pinter & Pattee, 1968). These observations are in keeping with a rapid decline upon energy restriction in the adrenergic component of lipolysis, with lipolysis under the dominant control of the declining plasma insulin concentrations.

If the adrenergic stimulus is rapidly suppressed during fasting at a hypothalamic level, then one would expect the denervation of fat depots or transections of the cord might limit the mobilization of fat only in the early stages of fasting; after longer fasts denervation would have little effect. Animal experiments do show that unilateral denervation tends partially to protect the denervated tissue from fat depletion (Cantu & Goodman, 1967). However, denervation also leads to a marked fall in blood flow to the tissue (Cantu & Goodman, 1967) and this may account for the difference in fat depletion. If men with a complete transection of the cervical cord are fasted, then a normal rise in plasma free fatty acids occurs, there being no increase in adrenergic blocking agents are given after 58-62 h of fasting, then they have little effect on the elevated concentrations of free fatty acid (Pinter & Pattee, 1968). These observations are in keeping with a rapid decline upon energy restriction in the adrenergic component of lipolysis, with lipolysis under the dominant control of the declining plasma insulin concentrations.

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Interactions of thyroidal and catecholamine responses to dietary changes

Studies with propranolol confirm that catecholamines do affect thyroidal metabolism but it is not clear whether nutritional changes in catecholamines can physiologically alter T3 production. Thus propranolol has an equivalent effect on circulating T3 and rT3 when obese women are on either a weight-maintenance or a slimming diet (Jung et al., 1979a). If catecholamines regulated T3 production, then propranolol during slimming should have been less effective in reducing an already suppressed catecholamine and T3 production rate. Similarly, feeding with l-dopa, as a precursor of catecholamines, failed to alter serum T3 during semi-starvation despite maintaining the metabolic rate (Shetty, Jung & James, 1979a), although l-dopa did prevent the rise in rT3 (Shetty, Jung, Barrand, Callingham & James, 1978).

Although in prolonged starvation serum T3 recovers to near pre-fasting levels (Carlson et al., 1977) with a normal TSH response to TRH (Portnay et al., 1974; Grant et al., 1978), the resting metabolic rate and overall energy expenditure remain depressed. It appears that, with prolonged energy restriction, nuclear T3 receptors decrease (Schussler & Orlando, 1978) and this may not necessarily be a direct consequence of the fall in serum T3 (Bernal, Coleoni & De Groot, 1978). Thus peripheral adaptation may limit energy expenditure and allow a resetting of the hypothalamo-pituitary axis. It is known that T3 influences the apparent activity of adrenergic receptors so an interaction at the receptor level is also possible (Kunos, Vermes-Kunos & Nickerson, 1974; Williams, Lefkowitz, Watanabe, Hathaway & Besch, 1977).

Catecholamines are also known to stimulate the secretion of thyroxine from the thyroid, but this effect is normally over-ridden by the control exerted by TSH (Melander, 1977). Of potentially greater importance are the synergistic effects of catecholamines and T3 on peripheral tissues, particularly well seen in the metabolic response to cold acclimatization and the generation of heat in non-shivering thermogenesis (Horowitz, 1978; Le Blanc, 1978). Both of these processes depend on the continued activity of brown fat in animals and this tissue may also be responsive in adult man (Foster & Frydman, 1978; Jung, Shetty, James, Barrand & Callingham, 1979c). The interaction of catecholamines and thyroid hormones may also be clinically significant in determining the metabolic basis for obesity (Jung et al., 1979c).

We conclude that the regulation of catecholamine and thyroid metabolism by nutritional factors is an important mechanism for altering energy expenditure in both animals and man. The hormonal systems appear to interact and thus should be considered as part of the regulatory system governing energy balance.

References

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