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MEER OOR VETSUG

'n Onlangse redaksionele artikel[1] het sommige van die moderne idees aangaande vetsug besigtig, veral met die oog op Mayer se oorsig.[2] Ons hervat hierdie besigtiging.

Genetiese faktore by die ontwikkeling van vetsug in die mens is baie minder duidelik as by eksperimentele diere. Dit is soveel moeiliker om die uitwerking van die omgewing uit te sluit. Die gelokaliseerde vetsug van die Hottentotsvrou se vetboudigheid word ongetwyfeld deur oorerwing bepaal. Van nog meer belang is die studies van identiese en nie-identiese tweelinge. Twee verskillende verslae[3,4] het 'n noue gewigsverband tussen identiese tweelinge aangedui en self gesuggereer dat die genetiese faktore van meer belang as dié van die omgewing is. Dit moet egter besef word dat hierdie studies oor gewig *per se* gegaan het en nie oor vetheid nie. Verskeie ander verslae het getoon hoe dikwels ouer/kind-vetsug voorkom en hoe seldsaam vetsug tot 'n enkele geslag beperk is. Dit is egter geen goeie bewys van die genetiese oorerwing van vetsug nie, net so min soos dit 'n goeie bewys is van, byvoorbeeld, tuberkulose.

Chemiese toksiene wat vetsug veroorsaak, is nie by die mens bekend nie, maar goud-tioglukose sal dit in muise veroorsaak. Mayer en sy medewerkers het getoon dat daar fundamentele verskille tussen hierdie tipe van vetsug en die oorerflike vetsug-hiperglisemie-sindroom bestaan, wat hulle stelling versterk dat vetsug van veelvoudige oorsprong is.

Dit is onmoontlik om te bepaal watter rol endokrienfaktore miskien in die produksie van vetsug speel. In Cushing se sindroom, met oormatige sirkulerende hidrokortisoon, word baie van die liggaamsproteïene omgesit in vet en dit, tesame met 'n verhoogde aptyt en verminderde fisiese aktiwiteit, is voldoende om die vetsug te verklaar—die aanwesigheid waarvan deur die kifose van vertebrale ineenstorting beklemtoon mag word. Die enigste ander endokrientoestand wat ongetwyfeld in verband met sommige gevalle van vetsug staan, is hiperinsulisme, waar die aptyt definitief toeneem. Aangesien ‚endokrien'-vetsug geen uitsondering op die reël van die oorsaaklike verband van 'n kalorieopname bó die van 'n kalorieverbruik is nie—sal dit nodig wees, indien hormoon-faktore meer algemeen daarby betrokke sou wees, om uit te vind op watter wyse hulle aptyt of voedselopname-reëling affekteer.

Swangerskap en die menopouse word dikwels met vetsug geassosieer, tog het sekshormone, gonadektomie, ens., eksperimenteel baie inkonsekwente en onbeduidende uitwerking op gewig gehad. Ewemin is skildklier- of harsingslymkliersiektes by eksperi-

EDITORIAL

MORE ABOUT OBESITY

A recent editorial article[1] partially surveyed some modern ideas concerning obesity, particularly in the light of Mayer's review.[2] We continue the survey.

Genetic factors in the development of obesity in man are far less clear cut than in experimental animals. It is so much more difficult to exclude the effects of environment. Certainly the localized obesity of the Hottentot woman's steatopygia is genetically determined. More significant than this are the studies of identical and non-identical twins. Two different reports[3,4] have indicated a very close correlation for weight between identical twins, even suggesting that genetic factors are more important than environmental ones. However, it must be realized that these studies concerned weight *per se*, and not fatness. Several other surveys have shown the frequency of parent-child obesity and the infrequency of obesity confined to a single generation. This, however, is not good evidence for the genetic inheritance of obesity, any more that it would be for, say, tuberculosis.

Chemical toxins which produce obesity are unknown in man, but gold thioglucose will do so in mice. Mayer and his associates have shown fundamental differences to exist between this type of obesity and the hereditary obese-hyperglycaemia syndrome, which strengthens their argument that obesity is of multiple origin.

It is impossible to estimate the part which endocrine factors may play in the production of obesity. In Cushing's syndrome, with excessive circulating hydrocortisone, much of the body protein is turned into fat and this, together with an increased appetite and diminished physical activity, is sufficient to account for the obesity, the appearance of which may be accentuated by the kyphosis of vertebral collapse. The only other endocrine state clearly related to obesity in some cases is hyperinsulinism, where the appetite is plainly increased. Since 'endocrine' obesity is no exception to the rule of causation by a calorie intake in excess of output, if hormonal factors are to be more generally implicated, it will be necessary to discover in what way they affect appetite or the regulation of food intake.

Pregnancy and the menopause are frequently associ-

mentele of menslike vetsug betrokke. Daar bestaan nogtans geen twyfel oor die noue verband tussen vetsug en sekere veranderings in endokrienstofwisselingverwantskappe nie. 'n Aanduiding hiervan is dat suikersiekte so dikwels by vetsugtiges voorkom terwyl dit tipies is dat die dulding vir koolhidrate gedurende die ‚aktiewe' fase van vetsug (solank soos gewig nog opgetel word), groter word.

Die hipotalamus, met sy ‚aptytsentrums' waarna alreeds verwys is,[1] is meer gewis aan vetsug verbonde. Letsel van hierdie gebied in die mens, asook in eksperimentele diere, kan vetsug veroorsaak, gepaard met 'n duidelike verhoging in die voedselopname. As die harsingslymklier aangetas is, is dit slegs weens die omvang van die primêre beskadigende letsel en nie weens enige verwantskap tussen hierdie klier en die aptyt *per se* nie. Hipotalamiese vetsug by die mens is uiters seldsaam; Fröhlich se sindroom waar pituitêre skade ook aangetref word, bestaan so te sê nie. Die werklike meganisme waardeur voedselopname deur die hipotalamus beheer word, is nie bekend nie.

Die introduksie van frontale lobotomie het getoon dat die voorhoofslobbe ook help om voedselopname te beheer, aangesien die meerderheid van pasiënte ná die operasie vetsug met verhoogde aptyt ontwikkel.

Die belangrikheid van aktiwiteit op gewig kom vervolgens in aanmerking. Mayer beweer dat hierdie faktor stelselmatig deur klinici en gesondheidsopvoeders, en 'n mens mag byvoeg ook deur fisioloë, verkleineer word. Hy siteer die onjuiste mening (1) dat oefening betreklik min kalorieverbruik vereis en dus min verband met die vraagstuk van vetsug het, en (2) dat 'n toename van fisiese aktiwiteit outomaties gevolg word deur 'n toename van aptyt en dus self-verydelend as 'n gewigsreëlaar is.

Aangaande die eerste wanopvatting wat die kalorieverbruik deur oefening minimiseer, behoort 'n mens die offisiële tabelle van daaglikse kalorieverbruik in verskillende beroepe vlugtig deur te kyk. 'n Sittende beroep mag dus 'n kalorieopname van 2,400 kalorieë vereis, terwyl 'n arbeider meer as 6,000 kalorieë nodig mag hê. Heel klein toevoegings tot die kalorievereistes (bv. 'n potjie muurbal per dag) sal sowat 30 lb. liggaamsvet per jaar verteenwoordig. Hoogtepunte van fisiese aktiwiteit mag amper 'n addisionele kalorieverbruik van 1,300 per uur vir 'n ‚gemiddelde' persoon met 'n liggaamsoppervlakte van 1·77 vierkante meters bereik. Aangesien die kalorieverbruik van oefening naasteby eweredig met liggaamsgewig is ('n 200-lb.-man sal twee maal soveel kalorieë as 'n 100-lb.-man by die klim van 1,000 voet, verbruik), volg dit dat 'n vetsugtige persoon meer liggaamsvet as 'n normale persoon, vir dieselfde hoeveelheid oefening, sal verbrand.

Dit kan dus gesien word dat soos 'n persoon se gewig vermeerder, so vermeerder sy kalorieverbruik ook—wat 'n té groot voedselopname was om 'n liggaamsgewig van 150 lb. in stand te hou, mag net presies genoeg wees om 200 lb. te onderhou. Op hierdie wyse dus is vetsug self-beperkend en, gelukkig, as die oortollige voedselopname gerig is, neem die gewig nie steeds toe nie tensy die opname ook progressief toeneem. Dit is egter ook duidelik dat 'n fisies aktiewe persoon wat ooreet, baie gou 'n selfreëlende toename in gewig

ated with obesity, yet sex hormones, gonadectomy etc. have had very inconsistent and minor effects on weight experimentally. Neither has thyroid or pituitary disease been implicated in experimental or human obesity. There is, nevertheless, no doubt of the close association between obesity and certain altered endocrine/metabolic relationships. This is indicated by the so frequent appearance of diabetes in the obese person, whereas during the 'active' phase of obesity (while weight is still being gained), carbohydrate tolerance is typically increased.

The hypothalamus is more certainly connected with obesity, with its 'appetite centres' mentioned before.[1] Lesion of this area in man, as well as in experimental animals, may cause obesity, with marked hyperphagia. If the pituitary gland is also affected, this is merely because of the extent of the primary damaging lesion, and not because of any relation between this gland and the appetite *per se*. Hypothalamic obesity in man is extremely rare; Fröhlich's syndrome, in which pituitary damage also occurs, is, to all intents and purposes, non-existent. The actual mechanism through which the hypothalamus controls food intake is unknown.

The introduction of frontal lobotomy has indicated that the frontal lobes also help to control food intake, since the majority of subjects develop obesity with increased appetite after the operation.

The importance of activity on weight comes next for consideration. Mayer claims that this factor has been systematically minimized by clinicians and health educators and, one may add, by physiologists also. He quotes the erroneous belief (1) that exercise requires relatively little calorie expenditure and is therefore of little relevance to the problem of obesity and (2) that an increase in physical activity is automatically followed by an increase in appetite and is therefore self-defeating as a weight regulator.

Regarding the first misconception, which minimizes the calorie expenditure due to exercise, one should glance at the official tables of daily calorie requirements in different occupations. Thus a sedentary job may require an intake of 2,400 calories, while a labourer may need above 6,000 calories. Quite small additions to the calorie requirement (e.g. a game of squash a day) would represent some 30 lb. of body fat in a year. Peaks of physical activity may approach an additional calorie usage of 1,300 per hour for a 'mean' man with a body surface of 1·77 sq. metres. Now, since the calorie cost of exercise is roughly proportional to body weight (a 200-lb. man will consume twice as many calories as a 100-lb. man in climbing 1,000 feet), it follows that an obese subject will burn up more body fat than a normal subject for the same amount of exercise.

Thus it may be seen that as a person's weight increases, so does his calorie expenditure, so that what was too large a food intake to maintain a body-weight of 150 lb. may be just exactly what is needed to maintain 200 lb. In this way, then, obesity is self-limiting and, luckily, if the excess food-intake is small, the weight does not continue to advance unless the intake is progressively increased also. It is also evident, however,

sal bereik weens die energie wat hy verloor deur die ekstra gewig wat hy moet dra. 'n Sittende persoon daarenteen, sal minder energie verbruik om die ekstra gewig te dra en gevolglik sal sy gewigstoename groter wees.

Die belangrikheid van fisiese aktiwiteit by eksperimentele diere word getoon deur die kruising van die vetsugtige-hiperglisemiese-muise met dié wat die *,waltzing*'-geen dra. So 'n kruising produseer 'n muis in voortdurende beweging en hulle gewig is slegs sowat 30% meer as dié van die nie-vetsugtige muise, in plaas van die 200-300% oortollige gewig van die sittende vetsugtige muise.

1. Van die Redaksie (1956): S. Afr. T. Geneesk., **30**, 907.
2. Mayer, J. (1953): Physiol. Rev., **33**, 472.
3. Newman, H. H., Freeman, F. N. en Holzinger, J. J., Gesiteer deur Mayer, J., *loc. cit.*[2]
4. Von Verschuer, gesiteer deur Mayer, J., *loc. cit.*[2]

that a physically active person who overeats will rapidly reach a self-regulating gain in weight, because of the energy lost in moving that extra weight. A sedentary person, on the other hand, will expend less energy in moving the extra weight and hence his weight gain will be greater.

In experimental animals the importance of physical activity is shown by crossing the obese-hyperglycaemic mice with those which carry the 'waltzing' gene. Such a cross produces a mouse in constant motion, whose weight is only some 30% over that of the non-obese mouse, instead of the 200-300% excess weight of the sedentary obese mouse.

1. Editorial (1956): S. Afr. Med. J., **30**, 907.
2. Mayer, J. (1953): Physiol. Rev., **33**, 472.
3. Newman, H. H., Freeman, F. N. and Holzinger, J. J. Quoted by Mayer, J., *loc. cit.*[2]
4. Von Verschuer. Quoted by Mayer, J., *loc. cit.*[2]