Thermogenic response to temperature, exercise and food stimuli in lean and obese women, studied by 24 h direct calorimetry

BY SANDRA BLAZA* AND J. S. GARROW†

Nutrition Research Group, Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ

(*Received 10 June 1982 – Accepted 7 September 1982*)

1. Total heat loss was measured by 24 h direct calorimetry in five obese and five lean women who were maintained throughout the study on a diet supplying 3·3 MJ/d. Each subject was measured five times to assess the effect of temperature, exercise and food on energy expenditure. Within each weight group a Latin-square design was used to balance sequence effects on the thermogenic responses to temperature, exercise and food.

2. Compared with the control day, on which no thermogenic stimulus was given, the increase in 24 h heat production by the lean and obese women caused by 30 min exercise on a bicycle ergometer against a load of 20 N was 10·1 and 10·3 W for obese and lean groups respectively. There was no evidence in either group of a measurable long-term increase in metabolism which would increase the energy cost of the exercise above that predicted from indirect calorimetry during the exercise.

3. The increase in heat production associated with ingesting an extra 4·4 MJ (obese group) or 4·0 MJ (lean group) was 3·4 and 3·0 W respectively. This response was similar to that predicted from indirect calorimetry for a few hours after the meal.

4. The obese and lean groups differed in metabolic response to calorimetry at the upper or lower limits of the thermal comfort zone, which was determined individually for each subject. The difference from control values in the obese group was an increase of 3·8 W on the 'warm' run, and a decrease of 2·0 W on the 'cool' run. Among lean subjects the change was an increase of 0·4 W on the 'warm' run, and an increase of 4·8 W on the 'cool' run. The differences between the groups did not achieve statistical significance. The lower and upper temperature limits were similar in the two groups: 23·2–26·4° for the obese group, and 23·3–26·2° for the lean group.

5. The most striking difference between lean and obese subjects in the present study was the much higher resting metabolic rate, and total energy expenditure, of the obese group. During the control run the obese group had a mean energy expenditure of 96·1 W, compared with 61·7 W in the lean group. There was no overlap: the lowest energy expenditure for an obese subject was 81·4 W and the highest for a lean subject was 76·1 W. In comparison to this large difference in baseline the magnitude of the thermogenic responses was small.

6. Under the conditions of this study there was nothing to support the view that a failure of thermogenic response is an important factor in the causation of human obesity. To support that view it would be necessary to show differences in thermogenesis in lean and obese subjects which were at least an order of magnitude greater than those which we have observed.

There is no doubt that the excessive fat stores in obese people represent an imbalance between energy intake and energy expenditure. However, attempts to show that obese people habitually eat more than lean people have usually failed (Garrow, 1978), nor has it ever been shown that obese people generally have a lower resting metabolic rate, or more efficient absorption of energy from food, than lean people. This paradox might be resolved if it could be shown that obese people have a thermogenic defect: if, in response to stimuli such as cold, warmth, exercise and the ingestion of food, they showed a smaller elevation of resting metabolic rate than lean people.

Work with genetically obese rodents has shown that in these animals at least, a thermogenic defect is likely to be the cause of obesity (Davis & Mayer, 1954; Kaplan & Leveille, 1974; Trayhurn *et al.* 1976). This defect is demonstrable as an inability to increase heat production in cool environments and a consequent drop in body temperature.

* Present address: Animal Studies Centre, Freeby Lane, Waltham-on-the-Wolds, Melton Mowbray, Leicestershire LE14 4RT.

† For reprints.

Similarly, obese mice show only 50% of the response of their lean litter-mates to noradrenaline injection (Trayhurn & James, 1978).

In human subjects the evidence is less clear-cut. Jung *et al.* (1979) showed that obese women had a smaller thermogenic response to infused noradrenaline than lean controls, but Danforth *et al.* (1981) failed to observe this defect in obese Pima Indians. Pittet *et al.* (1976) found a smaller thermic response to an oral load of 50 g glucose in obese women than in lean women, and Kaplan & Leveille (1976) reported a barely significant decrease in the thermogenic response to a protein meal in obese women. Shetty *et al.* (1981) also found a reduced thermogenic response to a mixed meal in obese women, but noted that dietary thermogenesis alone could not provide a satisfactory explanation for obesity, since the difference between the lean and obese subjects was not sufficient to compensate for the higher resting metabolic rate in the obese subjects. They postulated that additional thermogenic defects must exist for familial obesity to be explained entirely on a metabolic basis.

In part these conflicting results can be explained by the technical difficulties in measuring a thermogenic response by indirect calorimetry. Typically the increase in metabolic rate when a test meal is given to a fasting subject is approximately 10–15% of baseline at the maximum effect approximately 1 h after the meal, and this effect decreases to unmeasurable levels by approximately 4 h after the meal. Ideally, measurements should be continued until baseline levels are reached, but this is rarely done, so the reported 'response' depends on the duration of measurement. The baseline from which the response is measured is also liable to variation of approximately 5% from day-to-day in the same subject (Garrow & Hawes, 1972), and is influenced by the previous diet of the subject. In the reports of Pittet *et al.* (1976) and Kaplan & Leveille (1976) the previous diets of the lean and obese subjects were not controlled, and Shetty *et al.* (1981) provided different diets for their obese, post-obese and lean subjects. None of these publications provides information about the response of subjects to more than one thermogenic stimulus; thus we do not know if those who respond well to infused noradrenaline, for example, also show large responses to other stimuli such as food, cold or exercise.

To overcome these technical difficulties we report our findings on 24 h heat losses, measured by direct calorimetry, in obese and lean women who were maintained on an identical diet and exposed to a series of thermogenic stimuli such as might be encountered in everyday life.

METHODS

Subjects

The physical characteristics of the five obese and five lean subjects are shown in Table 1. The obese subjects were patients referred to J. S. Garrow, and the lean subjects were volunteers recruited from staff or their friends. Throughout the experiment subjects were confined to a metabolic unit (Garrow *et al.* 1978) and supplied with a diet designed to provide 3·4 MJ/d. The actual energy intake and composition of the diet taken by each subject is shown in Table 2.

Measurement of body composition

Body-weight was measured daily before breakfast, with an empty bladder, on a beam balance capable of weighing to within 50 g. Fat-free body mass was measured by two methods: total body water was measured by dilution of a tracer dose of deuterium oxide (Halliday & Miller, 1977), and total body potassium by counting the radiation from ^{40}K in a whole-body counter (Smith *et al.* 1979). From these values fat-free body mass was

Table 1. *Subject characteristics*

Group and subject	Age (years)	Height (H) (m)	Weight (W) (kg)	W/H^2 (kg/m^2)	Fat (%)
Obese					
NM	20	1·61	91·7	35·2	44·7
BS	32	1·57	90·6	36·9	39·0
VA	37	1·69	114·7	40·3	48·2
JH	46	1·68	93·0	33·0	49·2
CC	41	1·63	91·7	34·6	41·3
Mean	35	1·64	96·3	36·0	44·5
Lean					
JR	21	1·73	59·3	19·8	10·5
KL	22	1·78	67·0	21·2	13·2
SR	32	1·58	47·2	18·9	16·8
FC	21	1·75	64·9	21·3	24·1
DW	21	1·60	53·4	21·0	14·8
Mean	23	1·69	58·4	20·4	15·9
Lean:obese differences	$P < 0·05$	—	$P < 0·001$	$P < 0·001$	$P < 0·001$

Table 2. *Mean daily energy intake, and contribution of protein, fat and carbohydrate (CHO) to energy intake, during the experimental period and the composition of additional intake on the 'food' test day*

	Mean daily intake				Supplement on 'food' day			
Group and subject	Energy (MJ)	Protein (%)	Fat (%)	CHO (%)	Energy (MJ)	Protein (%)	Fat (%)	CHO (%)
Obese								
NM	3·31	21·5	32·0	46·5	4·42	6·1	40·2	53·7
BS	3·22	20·2	21·0	58·8	4·28	12·0	52·5	35·5
VA	3·29	21·1	25·6	53·4	4·06	10·1	29·5	60·5
JH	3·30	19·3	21·5	59·2	4·78	13·6	57·4	29·0
CC	3·28	19·8	21·8	58·5	4·58	12·0	39·1	48·8
Mean	3·28	20·4	24·4	55·3	4·42	10·8	43·7	45·5
Lean								
JR	3·24	23·7	25·2	51·1	3·80	15·0	42·2	42·8
KL	3·28	24·5	18·0	57·5	4·67	13·2	67·7	18·9
SR	3·38	20·5	17·4	62·1	4·33	19·0	55·7	25·3
FC	3·26	24·9	12·1	63·0	3·86	29·9	64·2	5·8
DW	3·43	40·2	16·4	43·4	3·52	25·2	72·8	2·0
Mean	3·32	26·8	17·8	55·4	4·04	20·5	60·5	19·0

estimated on the assumption that fat-free tissue contained 730 g water/kg and 60 mmol K/kg (Garrow, 1981), and fat mass was calculated by subtracting fat-free mass from body-weight.

Calorimetry

Resting metabolic rate was measured by indirect calorimetry in a ventilated hood apparatus (Garrow & Hawes, 1972) before breakfast on the first and second morning after admission to the ward, and on each test day immediately before entering the calorimeter chamber.

Total 24 h energy expenditure was measured by a direct calorimeter (Garrow *et al.* 1977).

Subjects wore light cotton trouser-suits, and were given only one sheet with which to cover themselves at night. Subjects were not permitted to smoke in the calorimeter.

Measurement of thermal comfort zone

On the second day after admission to the metabolic ward the subjects spent from 10.00 to 17.00 hours in the calorimeter chamber. This period was used partly to familiarize the subjects with the calorimeter, and also to determine the zone of thermal comfort for each subject. The chamber was at 25° when the subject entered, but the temperature was then increased or decreased at a rate of 2°/h until the subject signalled that the temperature was uncomfortably high or low. This was done by turning a knob marked 'temperature control' inside the calorimeter towards a warmer or cooler setting, which signalled to the operator to reverse the direction of temperature change. In the course of the 7 h run the temperature would complete two or three cycles, depending on the range of tolerance of the subject. The comfort zone was taken to be from the highest temperature which the subject signalled as too cold to the lowest temperature which was signalled as too hot.

Hormone assays on urine: cortisol and catecholamines

Urine samples were collected immediately and frozen at −70° for assay of cortisol using the assay kit from Miles Laboratories, Slough, Berks, and [1,2,3,6,7-^{3}H]cortisol from Amersham International, Amersham, Bucks. Another portion was preserved by adding 100 ml 2 M sulphuric acid/l and assayed for catecholamines by the method of von Euler & Lishajko (1961) as modified by Crout (1961).

Subject protocol

On each test day the subject was weighed at 08.30 hours. Resting, fasting metabolic rate was measured by indirect calorimetry between 08.30 and 09.15 hours, then breakfast was given, body temperature was measured and the subject entered the calorimeter chamber by 10.00 hours. At 12.00 hours the subject emptied her bladder, and the calorimetry period began and ended at 12.00 hours on the following day. The sequence of tests (control, warm, cool, exercise, food) was rotated according to a Latin-square design for the five obese and five lean subjects, so each test came first, second, etc. for one obese and one lean subject.

The 'warm' and 'cool' runs were conducted with the calorimeter at the upper or lower limit of the comfort zone for that subject. All other tests were conducted with the calorimeter at the mid-point of the comfort zone for that subject. On the 'exercise' test day the subject rode a bicycle ergometer (Monark, Varberg, Sweden) for 30 min at a speed such that the periphery of the flywheel travelled at a speed of 4·5 m/s against an external work load of 20 N. On the 'food' test day the subject ate a food supplement in addition to the normal food allowance; the composition of this supplement is shown in Table 2. The exercise of the 'exercise' day, and the supplement on the 'food' day, were taken during the afternoon of the first day, so the thermogenic response would be completed before the end of the calorimetry period at 12.00 hours on the second day. The subject's body temperature was measured at the beginning and end of the calorimetry period.

The protocol was approved by the Northwick Park Hospital Ethical Committee.

RESULTS

The initial resting metabolic rate for each subject, and the lower and upper limits of their comfort zones, are set out in Table 3. The resting metabolic rate of the obese group was higher than that of the lean group ($P < 0{\cdot}01$) but there were no significant differences between the groups in the upper or lower limits of the comfort zone, or the range between these limits. Since obese people have greater thermal insulation than lean people they might

Table 3. *Initial resting metabolic rate (RMR), and minimum and maximum limits of thermal comfort zone, of obese and lean subjects*

Group and subject	Initial RMR (ml oxygen/min)	Thermal comfort zone (°)	
		Minimum	Maximum
Obese			
NM	283	22·0	26·5
BS	308	24·3	25·5
VA	335	22·0	26·0
JH	271	23·0	26·0
CC	257	24·5	28·0
Mean	291	23·2	26·4
Lean			
JR	240	23·0	26·0
KL	214	24·0	27·0
SR	161	23·0	25·0
FC	237	23·0	26·0
DW	210	23·6	27·0
Mean	212	23·3	26·2

have been expected to indicate a lower, or narrower, zone of thermal comfort. No such effect was observed in our subjects, nor has such an effect been reported by others (Fanger, 1972). It might have been expected that subjects with a higher metabolic rate would choose a lower temperature for thermal comfort, since hypothyroid patients with depressed metabolism are intolerant of cold, and hyperthyroid patients dislike warm conditions, but this expectation is not supported by the results in Table 3, whether comparisons are made between or within subject groups.

The results of direct calorimetry in each subject for the control run, and for the four types of thermogenic stimulus, are set out in Table 4. Since all subjects were maintained throughout the study on a low energy intake (Table 2) metabolic rate tended to decrease with time. Thus if the nature of the thermogenic stimulus is ignored, and the mean heat losses for the first to fifth calorimetry run are calculated, for the obese group the values are 106·6, 103·9, 94·9, 97·5 and 92·6 W, and for the lean group the average heat losses for the first to fifth run are 71·9, 66·0, 62·9, 62·2 and 64·1 W. The Latin-square design ensures that when group mean values are calculated for each thermogenic stimulus, and for the resting metabolic rate measured immediately before each calorimetry run, these sequential effects are cancelled out. There were no significant differences between groups in the mean resting metabolic rate before each type of calorimetry run.

The thermogenic response of each group to each stimulus may be expressed as the mean heat loss for the group with that stimulus minus the mean heat loss for the group in the control run (Table 4). No correction has been applied for changes in heat stored in the body, since temperature changes during the calorimetry runs did not exceed 0·1° in any group; this would affect the estimated heat production rate over 24 h by less than 0·5 W.

The obese group showed an increase of 3·8 W on the 'warm' run, and a decrease or 2·0 W on the 'cool' run, whereas the lean group showed an increase of 0·4 and 4·8 W for the 'warm' and 'cool' runs respectively. In view of the lack of thermogenic response to cold in genetically obese animals (Trayhurn *et al.* 1976) these differences between the lean and obese human subjects are interesting, but since the differences do not achieve statistical significance this result may be due to chance.

The thermogenic response of the two groups of subjects to exercise and food are strikingly

Table 4. *Mean group resting metabolic rate (RMR) immediately preceding the calorimetry runs, and 24 h heat loss for individual obese and lean subjects*

(Numbers in parentheses indicate the sequence of testing)

Group and subject	Control	Warm	Cool	Exercise	Food
Obese					
Initial RMR (ml oxygen/min, mean ± SEM)	257 ± 12	234 ± 12	264 ± 13	254 ± 14	251 ± 9
24 h heat loss (W)					
NM	(4) 101·6	(5) 100·9	(1) 103·4	(2) 126·8	(3) 102·9
BS	(1) 106·4	(2) 105·7	(3) 92·3	(4) 107·5	(5) 97·9
VA	(3) 101·8	(4) 100·5	(5) 95·5	(1) 121·1	(2) 106·2
JH	(5) 83·3	(1) 105·1	(2) 99·6	(3) 90·0	(4) 93·9
CC	(2) 81·4	(3) 87·4	(4) 79·8	(5) 85·5	(1) 96·8
Mean	96·1	99·9	94·1	106·2	99·5
Mean − control		+3·8	−2·0	+10·1	+3·4
Increase as % control		+3·9	−2·1	+10·5	+3·5
Lean					
Initial RMR (ml oxygen/min, mean ± SEM)	193 ± 16	198 ± 14	198 ± 15	203 ± 18	198 ± 15
24 h heat loss (W)					
JR	(3) 76·1	(4) 76·3	(5) 81·4	(1) 98·9	(2) 83·2
KL	(4) 65·4	(5) 63·1	(1) 78·8	(2) 74·9	(3) 64·7
SR	(2) 47·5	(3) 47·8	(4) 47·9	(5) 55·3	(1) 55·0
FC	(1) 67·8	(2) 64·4	(3) 64·6	(4) 69·6	(5) 68·8
DW	(5) 51·8	(1) 59·0	(2) 60·0	(3) 61·2	(4) 51·7
Mean	61·7	62·1	66·5	72·0	64·7
Mean − control		+0·4	+4·8	+10·3	+3·0
Increase as % control		+0·6	+7·8	+16·7	+4·9

similar: 10·1 and 3·4 W for the obese group and 10·3 and 3·0 W for the lean group. Furthermore, these responses agree with theoretical predictions. The bicycle exercise involved a work rate of approximately 500 W for 30 min; this energy dissipated over 24 h would give an increase in mean heat loss of approximately 10 W. Similarly the food supplement provided approximately 4 MJ, and approximately 10% would be expected to be lost as a result of dietary thermogenesis. On this assumption the expected increase in average heat loss over 24 h would be 4·6 W; the observed value suggests that only 6·5% rather than 10% of the energy in the supplement was lost as a result of dietary thermogenesis.

Since the thermogenic responses observed in this study were quite small, it was important to ensure that differences in the level of anxiety, or of physical activity, between the lean and obese groups did not cause spurious results. We have shown (Blaza & Garrow, 1980) that anxiety causes measurable increases in metabolic rate, and also in urinary cortisol and catecholamine excretion. However, in the study reported here the excretion of these stress hormones during calorimetry runs was similar to that observed on days when the subjects were resting in the ward, except in the case of one lean subject who produced a high cortisol excretion on the day of an 'exercise' calorimetry run. We therefore do not believe that the subjects were unduly anxious or stressed during the calorimetry runs.

The level of physical activity during calorimetry runs was monitored in a semi-quantitative manner by means of an ultrasonic movement detector (C. F. Palmer, London) inside the calorimeter chamber. During the 'exercise' runs the ultrasonic reading was approximately 100 times greater than during the other four types of run. However, there was no suggestion

Table 5. *Thermogenic responses to four stimuli among obese and lean individuals*

(Observed heat losses over 24 h (see Table 4) have been corrected for sequence effects. Response (W) is corrected heat loss minus control heat loss for that subject)

Group and subject	Warm	Cool	Exercise	Food
Obese				
NM	+2·4	−7·9	+18·9	−1·8
BS	+2·6	−7·5	+11·0	+4·7
VA	+1·9	0·0	+13·0	+1·2
JH	+11·5	+8·6	+1·5	+8·0
CC	+8·5	+3·4	+11·7	+12·9
Lean				
JR	+2·3	+9·4	+18·7	+5·0
KL	−0·5	+8·1	+6·0	−2·5
SR	+1·6	+3·0	+11·6	+6·2
FC	−1·6	+0·5	+7·3	+8·3
DW	+1·6	+4·0	+6·6	−1·4

that the differences in heat loss between lean and obese groups on the 'warm' and 'cool' runs could have been explained by differences in physical activity.

It is evident from Table 4 that there is no statistically significant difference in thermogenic responsiveness between the lean and obese groups of subjects. However it is possible that some individuals within each group showed large or small responses. One of the objectives of the study was to find out whether people who showed a large response to one stimulus would also show a similar response to others: if, in fact, people could be classified as 'good' or 'poor' thermogenic responders. To make comparisons within individuals of responses to several stimuli it is necessary to adjust the observed heat losses for sequence effects. Within the obese group the average rate of heat loss decreased by 3·1% with successive calorimetry runs, and in the lean group the average rate of decrease was 2·7% per run. This difference does not indicate a greater rate of decline in metabolic rate among the obese subjects, since the tests on lean subjects were completed within 2 weeks, while those on obese subjects were spread over 3 weeks, so the rate of decrease in metabolic rate per day on the restricted diet was actually more rapid in the lean group.

On the assumption that each member of the obese group would have had a rate of heat loss 3·1% greater than that observed for each place in the sequence a given run came after the control run, and 3·1% less for each place in the sequence before the control run, it is possible to correct the observed value to that which would have been found had the runs all been done on the same day as the control run. If the 'corrected' value for the response to the four thermogenic stimuli is then compared with the control value individual responses can be calculated for each run. In Table 5 the results of this calculation are shown. For the lean subjects a similar calculation is based on a decrease of 2·7% per run.

The responses shown in Table 5 do not support the view that some individuals show a particularly large, or small, response to all the stimuli tested.

DISCUSSION

The most striking difference between the lean and obese subjects in our experiments is that the lean group had a consistently and significantly lower energy expenditure than the obese group, both by direct and indirect calorimetry. The thermogenic response to the stimuli tested was not sufficient to close this gap; the highest rate of heat loss by the lean group

Table 6. *Estimates by indirect calorimetry of diet-induced thermogenesis in (a) obese and (b) lean subjects*

	Reference	*n*	Baseline (W)	Test meal: Nutrient	Test meal: Energy (MJ)	Duration of measurement (min)	Average increase in metabolic rate (%)	Thermic effect as percentage energy in test meal
(*a*)	Pittet *et al.* (1976)	11	86·7	Glucose	0·84	150	5·2	4·8
	Kaplan & Leveille (1976)	4	127·1	Protein	0·82	300	3·1	8·6
	York *et al.* (1980)	8	78·8	Protein	4·18	240	20·0	5·4
			78·0	Protein	2·09	240	8·2	4·4
	Shetty *et al.* (1981)	5	74·8	Mixed	2·33	120	6·4	1·5
	Nair *et al.* (1982)	6	88·2	Glucose	1·25	150	10·9	6·9
			86·7	Protein	1·25	150	28·3	17·7
			87·1	Fat	1·25	150	13·3	8·3
		Weighted mean	87·9					7·8
(*b*)	Pittet *et al.* (1976)	10	80·6	Glucose	0·84	150	13·0	11·2
	Kaplan & Leveille (1976)	4	86·8	Protein	0·82	300	16·7	31·8
	York *et al.* (1980)	8	72·7	Protein	4·18	240	28·6	7·2
			65·1	Protein	2·09	240	21·6	9·7
	Shetty *et al.* (1981)	5	61·7	Mixed	2·43	120	16·2	3·0
	Nair *et al.* (1982)	5	75·8	Glucose	1·25	150	10·5	5·7
			76·9	Protein	1·25	150	21·8	12·1
			76·0	Fat	1·25	150	8·3	4·5
		Weighted mean	74·4					10·0

was 72·0 W on the 'exercise' day, and this still falls far short of the lowest rate of heat loss by the obese group, which was 94·1 W on the 'cool' day. This confirms the results of other workers who, although they may have found a reduced thermogenic response in obese subjects, have always found a lower total energy expenditure in the lean group (Jequier & Schutz, 1981; Shetty *et al.* 1981).

The magnitude of the thermogenic response to two stimuli was similar in the obese and lean groups; 10·1 and 10·3 W for exercise, and 3·4 and 3·0 W for food respectively. The response to exercise is of interest for two reasons. The similarity of the responses for lean and obese subjects supports the conclusion of Whipp *et al.* (1973) and Danforth *et al.* (1981) that the efficiency of mechanical work is relatively fixed, and cannot differ much with the nutritional status or body build of the subject. Obese and lean people may, of course, differ in their habitual pattern of physical activity, and hence in the amount of energy which this activity costs, but if both groups are set identical tasks on a bicycle ergometer the increase in energy expenditure over the resting state is very similar. The other point of interest concerns the suggestion that the energy cost of exercise cannot be estimated from the oxygen uptake during the exercise, since the resting metabolic rate is significantly raised for many hours after the activity has finished (Allen & Quigley, 1977). If this were true we would expect to see an increase of much more than 10 W on the 'exercise' day, since this is the amount which would have been estimated from indirect calorimetry during the exercise. We must conclude, therefore, that any increase in metabolic rate after the cessation of exercise must be small, or brief, or compensated by a decrease in metabolic rate at some later time during the calorimetry period.

Similar arguments apply to the results of the 'food' stimulus: the observed increase in 24 h heat loss is similar to that which is observed by indirect calorimetry over approximately 4 h after a meal. The results of five such studies, performed by other workers, on obese and lean subjects are set out in Table 6. To simplify comparison with the results of our study the baseline energy expenditure has been expressed in watts; where it was given only as O_2 uptake in the original publication a conversion factor has been used: watts = O_2 uptake (ml/min) × 0·35. This assumes an energy equivalent of 21 kJ/l O_2. The weighted mean baseline values for the thirty-four obese subjects is 87·9 W, and for thirty-two lean subjects is 74·4 W. These values fall within the range observed among our lean and obese subjects. When the thermic effect of the meal is expressed as a percentage of the energy in the meal (Table 6) the weighted mean value for obese subjects is 7·8% and for lean subjects it is 10·0%, and the response with a protein meal is greater than with carbohydrate, fat or mixed nutrients. In our series the thermic effect as a percentage of the meal energy was 7·0% among obese subjects and 5·9% among lean subjects; again these values fall within the range observed by indirect calorimetry.

The response of lean and obese subjects to moderate warm or cool stimuli differed, but not to an extent which reached statistical significance. The only other publication dealing with the effect of mild cold on 24 h energy expenditure is that of Dauncey (1981), who observed an increase of 7·0 ± 1·1% in the metabolism of lean subjects at a temperature of 22°, compared with control measurements at 28°. Our results are very similar: lean subjects at 23·3° showed an increase in metabolism of 7·8% over control values, and calorimetry at the upper end of the thermal comfort zone for these subjects (26·2°) showed no difference from control values.

The response of the obese subjects was surprising. Despite the fact that the chosen comfort zone for obese subjects was almost identical to that of the lean subjects, the effect of cool conditions was to cause a slight decrease in metabolism (−2·1%), while warm conditions caused a slight increase (+3·9%). Since this failure of cold-induced thermogenesis in obese subjects fits so well with the observed defect in genetically obese rodents it is tempting to

believe that the difference between lean and obese subjects is real, but in view of the small sample size, and small response, it would be wise to await confirmation in a larger study.

The authors are grateful to Mr J. Parker for the catecholamine analyses, to Mr R. Hesp for the measurements of total body potassium, and to Dr D. Halliday and Mrs D. Wilkins for estimations of total body water.

REFERENCES

Allen, D. W. & Quigley, B. M. (1977). *Med. J. Aust.* **2**, 434.
Blaza, S. E. & Garrow, J. S. (1980). *Proc. Nutr. Soc.* **39**, 13A.
Crout, J. R. (1961). *Standard Methods of Clinical Chemistry*, vol. 3, p. 6 [D. Seligson, editor]. New York: Academic Press.
Danforth, E. Jr, Daniels, R. J., Katzeff, H. L., Ravussin, E. & Garrow, J. S. (1981). *Clin. Res.* **29**, 663A.
Dauncey, M. J. (1981). *Br. J. Nutr.* **45**, 257.
Davis, T. R. A. & Mayer, J. (1954). *Am. J. Physiol.* **177**, 222.
Fanger, P. O. (1972). *Thermal Comfort: Analysis and Applications in Environmental Engineering*. Copenhagen: Danish Technical Press.
Garrow, J. S. (1978). *Energy Balance and Obesity in Man*. Amsterdam: Elsevier/North-Holland Biomedical Press.
Garrow, J. S. (1981). *Treat Obesity Seriously: A Clinical Manual*. Edinburgh: Churchill-Livingstone.
Garrow, J. S., Durrant, M. L., Mann, S., Stalley, S. F. & Warwick, P. (1978). *Int. J. Obesity* **2**, 441.
Garrow, J. S. & Hawes, S. F. (1972). *Br. J. Nutr.* **27**, 211.
Garrow, J. S., Murgatroyd, P., Toft, R. & Warwick, P. (1977). *J. Physiol., Lond.* **267**, 16P.
Halliday, D. & Miller, A. G. (1977). *Biomed. Mass Spectrom.* **4**, 82.
Jequier, E. & Schutz, Y. (1981). In *The Body Weight Regulatory System: Normal and Disturbed Mechanisms*, p. 89 [L. A. Cioffi, W. P. T. James and T. B. Van Itallie, editors]. New York: Raven Press.
Jung, R. T., Shetty, P. S., James, W. P. T., Barrand, M. A. & Callingham, B. A. (1979). *Nature, Lond.* **279**, 322.
Kaplan, M. L. & Leveille, G. A. (1974). *Am. J. Physiol.* **227**, 912.
Kaplan, M. L. & Leveille, G. A. (1976). *Am. J. clin. Nutr.* **29**, 1108.
Nair, K. S., Garrow, J. S. & Halliday, D. (1982). *Clin. Sci.* **62**, 43.
Pittet, P., Chappuis, P., Acheson, K., De Techtermann, F. & Jequier, E. (1976). *Br. J. Nutr.* **35**, 281.
Shetty, P. S., Jung, R. T., James, W. P. T., Barrand, M. A. & Callingham, B. A. (1981). *Clin. Sci.* **60**, 519.
Smith, T., Hesp, R. & Mackenzie, J. (1979). *Phys. Med. Biol.* **24**, 171.
Trayhurn, P. & James, W. P. T. (1978). *Pflugers Arch. ges Physiol.* **373**, 189.
Trayhurn, P., Thurlby, P. L. & James, W. P. T. (1976). *Proc. Nutr. Soc.* **35**, 135A.
von Euler, U. S. & Lishajko, F. (1961). *Acta physiol. scand.* **51**, 348.
Whipp, B. J., Bray, G. A. & Koyal, S. N. (1973). *Am. J. clin. Nutr.* **26**, 1284.
York, D. A., Morgan, J. B. & Taylor, T. G. (1980). *Proc. Nutr. Soc.* **39**, 57A.

Printed in Great Britain