Caffeine: its effect on catecholamines and metabolism in lean and obese humans

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Summary
1. The metabolic response of lean and obese women to caffeine was studied to see if caffeine could be used to demonstrate the subnormal thermogenesis in obesity previously shown after standard meals or intravenous infusions of noradrenaline.

2. The rise in resting metabolic rate with caffeine was similar in the lean and obese groups and β-adrenoceptor blockade did not reduce the increment in metabolic rate in either group. These responses did not, therefore, correspond with the other subnormal thermogenic responses of the constitutionally obese.

3. In a post-obese group, i.e. previously obese women who were now of normal weight, there was a reduced response of the resting metabolic rate to caffeine.

4. Monitoring plasma substrate concentrations showed that the change in oxygen uptake corresponded to changes in plasma free fatty acids, so that in adults the metabolic effects of caffeine seem to be mediated by increases in adipocyte lipolysis. This effect seems to be mainly independent of the adrenergic system.

Key words: caffeine, catecholamines, free fatty acids, metabolic rate, obesity, phosphodiesterase.

Introduction
It has been known for over 60 years (Higgins & Means, 1915) that caffeine can elevate oxygen consumption in man, but how it does this is still unclear. Caffeine releases free fatty acids from adipocytes, either by inhibiting the action of cyclic AMP phosphodiesterase (Butcher & Sutherland, 1962) or by releasing catecholamines which then enhance lipolysis (De Schaepdryver, 1959; Robertson, Frölich, Carr, Watson, Hollfield, Shand & Oates, 1978). The rise in metabolic rate after caffeine could reflect the response to an increase in plasma free fatty acid, but any release of catecholamines could also act directly on peripheral thermogenic mechanisms.

We have reported a defect in the thermogenic response to noradrenaline in obese women (Jung, Shetty, James, Barrand & Callingham, 1979) and to a standard meal (Shetty, Jung, James, Barrand & Callingham, 1981) unrelated to the prevailing level of plasma free fatty acid. In the present study we have therefore compared the thermogenic response to caffeine in obese and lean women to see whether a subnormal response in obesity occurred with this particular thermogenic stimulus. Since a reduced rise in free fatty acid with oral caffeine has been reported in obese subjects (Oberman, Harell, Herzberg, Hoerer, Jaskolka & Laurian, 1975), we have measured the thermogenic response of lean, obese and post-obese subjects to oral and to intravenous caffeine and related any differences to the circulating substrate and hormone responses.

Patients and methods
Three groups of subjects were studied: lean, obese and post-obese. All were normotensive, euthyroid women of similar age (Table 1). The lean women had a family history of normal
TABLE 1. Age, weight and caffeine intake in the lean, obese and post-obese subjects

Results are means ± SEM. Numbers of subjects are shown in parentheses.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>% above ideal body weight</th>
<th>Loss of weight on low-energy diet for 3 weeks</th>
<th>Estimated daily caffeine intake (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral caffeine</td>
<td>Lean (6)</td>
<td>43.0 ± 4.3</td>
<td>46.0 ± 2.0</td>
<td>−18.3 ± 2.3</td>
<td>251 ± 44</td>
</tr>
<tr>
<td></td>
<td>Obese (6)</td>
<td>39.2 ± 3.0</td>
<td>87.9 ± 3.2</td>
<td>57.1 ± 6.2</td>
<td>218 ± 53</td>
</tr>
<tr>
<td></td>
<td>Post-obese (4)</td>
<td>47.8 ± 6.0</td>
<td>61.3 ± 2.5</td>
<td>9.5 ± 5.7</td>
<td>324 ± 40</td>
</tr>
<tr>
<td>Intravenous</td>
<td>Lean (4)</td>
<td>39.0 ± 3.2</td>
<td>45.7 ± 3.5</td>
<td>−18.7 ± 4.6</td>
<td>242 ± 47</td>
</tr>
<tr>
<td>Caffeine test</td>
<td>Obese (5)</td>
<td>47.4 ± 3.1</td>
<td>93.9 ± 5.7</td>
<td>70.8 ± 4.9</td>
<td>335 ± 104</td>
</tr>
</tbody>
</table>

weight and claimed to eat ad libitum without gaining weight. The obese subjects were defined as those weighing 20% or more above ideal body weight. Ideal body weight was taken as the midpoint of the medium frame size weight range as listed in the Metropolitan Life Insurance Company (1960) Tables. The obese women had a strong family history of obesity and were investigated as in-patients initially after 7 days on a weight-maintenance high-energy diet and again after 3 weeks on a low-energy diet. The high-energy diet was calculated to provide 167 kJ (40 kcal) of energy day⁻¹ kg⁻¹ ideal body weight; the low-energy diet 39 kJ (9.2 kcal) day⁻¹ kg⁻¹ ideal body weight. This intake on average amounted to 9.27 MJ (2390 kcal) daily on the high-energy diet and 3.62 MJ (865 kcal) on the low-energy diet. Both diets contained similar amounts of vitamins, minerals, protein and fat, the reduction in energy intake being achieved by a reduction in carbohydrates alone; carbohydrates made up over 85% of the energy intake on the high-energy diet. Sodium intake was kept constant at 52 mmol/24 h. The post-obese subjects were those who had previously been obese, i.e. 81 ± 5.2 kg and 45 ± 10% above ideal body weight (mean ± SEM), but had returned to normal weight on a slimming regimen and had re-equilibrated for at least 3 months on a new weight-maintaining diet.

Questionnaire

To assess the possibility that habitual caffeine intake might differ in obese and lean women, 50 obese and 52 normal-weight women filled in a questionnaire concerning their tea, coffee and cocoa intake. Twelve women filled in the questionnaire when obese and after they had returned to normal weight (post-obese). The questionnaire asked about the quantity of each beverage consumed per day, the quantity of coffee (heaped or flat teaspoon) added per cup and whether they drank ‘instant’, decaffeinated or ground coffee. The strength of tea was assessed as weak, medium or strong. Caffeine (mg) was allocated as follows: ground coffee 85 mg and instant 60 mg per heaped teaspoon, with half these quantities being taken for a flat teaspoon; caffeine in black tea was assumed to be 30 mg in weak tea, 40 mg in medium, 50 mg in strong and 30 mg in instant tea (Stephenson, 1977).

All the subjects tested with oral and intravenous caffeine were coffee and tea drinkers, but all three groups had similar caffeine intakes as calculated from their recorded consumption of tea and coffee (Table 1). The obese subjects, while in-patients, were allowed beverages equivalent to 250 mg of caffeine/day on both the high- and low-energy diets. The subjects were investigated after an overnight fast and a total abstinence for 16 h from any caffeine-containing beverage, this time being sufficient to clear the blood of caffeine, given its biological half-life of 3.5 h (Stephenson, 1977). The subjects, wearing similar clothing, were initially equilibrated for 45 min while lying supine in a room set at thermoneutrality (27–28°C). During this time a 19 G Abbott cannula was inserted into a brachial vein for blood sampling and kept patent with a few millilitres of sterile 3.8% sodium citrate solution. After equilibration and 30 min after cannula insertion, the resting metabolic rate was measured by the ventilated hood technique (Ashworth & Wolff, 1969); 1 min readings over 30 min were taken.

Caffeine (British Drug Houses; British Pharmacopoea, Codex) was given either as a drink dissolved in 150 ml of warm water (37°C) or as an intravenous injection dissolved in sterile water. The dose of caffeine given by either route was 4 mg/kg ideal body weight and similar amounts were given to each group (see Table 2). The resting metabolic rate was then measured for 1.5 h after the intravenous caffeine injection and for 2 h after the caffeine drink. Blood samples were taken before and after the administration of
Metabolic effects of caffeine

Caffeine and analysed for caffeine (Axelrod & Reichenthal, 1953), catecholamines (Callingham & Barrand, 1979), free fatty acids (Trout, Estes & Friedberg, 1960; Salaman & Robinson, 1961), glycerol (Boehringer Mannheim Enzymatic System), insulin (Amersham Kit no. IM78) and glucose (Beckman Glucose Autoanalyzer). For catecholamine estimation, blood was taken into ice-cold heparinized tubes containing reduced glutathione (20 μg/ml of blood) and for free fatty acid and glycerol into ice-cold tubes containing EDTA. Total body water was calculated from the equation of Hume & Weyers (1971): total body water = 0·344 x height (cm) + 0·184 x weight (kg) - 35·27. Total body caffeine, therefore, was calculated as follows: total body caffeine = total body water x plasma caffeine.

Three of the obese subjects while on the high-energy diet were also studied as controls for the effect on the resting metabolic rate of drinking and of circadian rhythms; they received a drink of 150 ml of warm water only. The effect of β-adrenoceptor blockade on the metabolic response to oral caffeine was also tested in a further three obese women while on a high-energy diet and after 5 days of propranolol (80 mg orally every 6 h). The three women were aged 45·7 ± 3·4 years and weighed 95 ± 2·6 kg, an average 79% above ideal body weight. The dose of caffeine given was 212 ± 6 mg; this test was done during the winter season (January and February).

The lean and obese groups were studied between May and September whereas the post-obese was tested in November. Control tests for circadian rhythms were conducted in the winter. These different times were noted, since our previous studies suggest seasonal shifts in basal venous noradrenaline concentrations in obese and post-obese women.

Statistics and ethics

Statistical analyses were made with the paired and single Student’s t-test and the tests were done with fully informed consent. Ethical approval was given by the Dunn Nutrition Unit’s Ethical Committee.

Results

Plasma caffeine (Table 2)

After oral caffeine, the peak concentration of plasma caffeine was seen within 15 min in the post-obese subjects, but at 30 min in the other two groups. Plasma caffeine remained elevated throughout the test period, the slow rate of decline being similar in all groups. Whereas the obese patients on both high- and low-energy diets and the post-obese groups had similar levels of plasma caffeine at all times, the lean group had higher plasma caffeine levels than the obese and post-obese groups during the test; these differences were statistically significantly higher in the lean as compared with the obese on the high diet at 60 min and on the low diet at 90 min.

Table 2. Plasma caffeine concentrations and calculated total body caffeine in lean subjects, obese patients on a high- and low-energy diet and in post-obese patients

Results are means ± SEM. Significance of differences: *P < 0·05; **P < 0·02; ***P < 0·01; ****P < 0·001 compared with lean subjects at the same time interval after caffeine.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Caffeine dose (mg)</th>
<th>Absolute rise in plasma caffeine (pg/ml) at different times (min) after caffeine</th>
<th>Calculated total body water (l)</th>
<th>Calculated total body caffeine (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>15</td>
<td>30</td>
<td>45</td>
</tr>
<tr>
<td>Oral</td>
<td>Lean</td>
<td>227·0</td>
<td>5·07</td>
<td>8·66</td>
</tr>
<tr>
<td>test</td>
<td>Obese on high-energy diet</td>
<td>± 5·9</td>
<td>± 0·67</td>
<td>± 0·94</td>
</tr>
<tr>
<td></td>
<td>Obese on low-energy diet</td>
<td>± 7·6</td>
<td>± 1·07</td>
<td>± 0·68</td>
</tr>
<tr>
<td></td>
<td>Post-obese</td>
<td>224·5</td>
<td>5·46</td>
<td>5·63</td>
</tr>
<tr>
<td></td>
<td>± 9·7</td>
<td>± 0·74</td>
<td>± 1·94</td>
<td>± 1·50</td>
</tr>
<tr>
<td>Intravenous caffeine test</td>
<td>Lean</td>
<td>220·0</td>
<td>7·56</td>
<td>7·29</td>
</tr>
<tr>
<td></td>
<td>Obese on high-energy diet</td>
<td>± 6·7</td>
<td>± 0·87</td>
<td>± 0·89</td>
</tr>
<tr>
<td></td>
<td>Obese on low-energy diet</td>
<td>± 7·9</td>
<td>± 0·39</td>
<td>± 1·01</td>
</tr>
<tr>
<td></td>
<td>Post-obese</td>
<td>219·8</td>
<td>5·98</td>
<td>6·02</td>
</tr>
<tr>
<td></td>
<td>± 7·4</td>
<td>± 0·74</td>
<td>± 0·97</td>
<td>± 0·50</td>
</tr>
</tbody>
</table>
After intravenous caffeine, the lean subjects again had significantly higher plasma caffeine levels at 45 and 60 min compared with the obese subjects on both high- and low-energy diets, but the rate of fall in plasma caffeine was similar in the two groups. Since caffeine is totally and rapidly absorbed from the gut and takes about 60 min to equilibrate with the total body water (Graham, 1978), a smaller volume for distribution may have accounted for the higher plasma caffeine levels in the lean subjects. The calculated amount of caffeine in the body, based on an assumed value for total body water, was not statistically different between the lean and obese groups after oral caffeine at 60 and 90 min or after intravenous caffeine at 45 and 60 min, but the tendency for the calculated values in the obese groups to be lower than in the lean might suggest that the estimated total body water in the obese and post-obese groups was too low.

**Metabolic rate**

After oral caffeine (Fig. 1) all groups showed a rapid rise in metabolic rate within 15 s and the resting metabolic rate remained elevated for the 2 h test period. The response of the obese group over the total period was less than that of the lean, but not significantly less. The post-obese group, however, showed a significantly lower response, amounting to a third of that seen in the lean women (Table 3, Fig. 1). From the tests with more directly administered, intravenous, caffeine it was apparent that in the obese patients there was no defect in thermogenesis; the response was greater than in the lean subjects despite the lower
Fig. 2. Metabolic response to intravenous caffeine. Symbols are as given in Fig. 1.

**Table 3. Effect of caffeine on plasma free fatty acid and glycerol and the resting metabolic rate**

Results are means ± SEM. Significance of differences: *P = <0·05; **P = <0·02; ***P = <0·01; ****P = <0·001 when compared with the post-obese group given oral caffeine. †P = <0·05; ††P = <0·01; †††P = <0·001 when compared with the lean group given intravenous caffeine. †††P = <0·001 when compared with the obese group on the high-energy diet.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Basal resting metabolic rate (kJ/min)</th>
<th>Total plasma free fatty acid at different times (min) after caffeine (µmol/l)</th>
<th>Incremental rise in plasma free fatty acid at different times (min) after caffeine (µmol/l)</th>
<th>Basal plasma glycerol (µmol/l)</th>
<th>Incremental rise in plasma glycerol at different times (min) after caffeine (µmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral caffeine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>3·267 ± 0·093</td>
<td>53·9*** 612</td>
<td>209 366</td>
<td>356* 497** 351 50·5</td>
<td>13·3 ± 0·7 13·6* 24·9 38·3 11·1</td>
</tr>
<tr>
<td>Obese on high-energy diet</td>
<td>4·520*** 0·201</td>
<td>9·8 ± 65 111 87 ± 5·1</td>
<td>0·8 ± 62 122 ± 87</td>
<td>313 325 372 85·8</td>
<td>8·1* 18·2 30·4 29·2 22·7</td>
</tr>
<tr>
<td>Obese on low-energy diet</td>
<td>4·123* 0·150</td>
<td>6·2 ± 102 90 ± 20</td>
<td>0·4 ± 58 54 ± 66</td>
<td>345* 315 274 ± 13·1</td>
<td>25·4 63·4 57·2 46·5***</td>
</tr>
<tr>
<td>Post-obese</td>
<td>3·433 ± 0·203</td>
<td>17·3 544 55 208</td>
<td>— 106 132 174</td>
<td>65·5 25·3 40·5 33·0 17·2</td>
<td>± 11·2 16·9 ± 11·1 14·0 13·3 11·3</td>
</tr>
<tr>
<td>Intravenous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>3·190 ± 0·154</td>
<td>6·5 154 2·0 ± 8</td>
<td>— 2·9 ± 64 105 ± 108</td>
<td>396 441 50·7 36·0 19·5 23·5</td>
<td>34·2 38·7 —</td>
</tr>
<tr>
<td>Obese on high-energy diet</td>
<td>4·569*** 0·312</td>
<td>8·0 ± 105 54 ± 99 108 ± 2·1</td>
<td>3·4 ± 8·2 8·1 ± 10·3 9·7 ± 8·8</td>
<td>25·4 63·4 57·2 46·5***</td>
<td></td>
</tr>
<tr>
<td>Obese on low-energy diet</td>
<td>4·253*** 0·212</td>
<td>6·6 133*** 767 722 545 459 359 396 441 50·7 36·0 19·5 23·5</td>
<td>34·2 38·7 —</td>
<td>12·4 22·2 71·1 47·9 46·1 —</td>
<td></td>
</tr>
</tbody>
</table>

Intravenous Lean caffeine test

Intravenous Lean caffeine test
plasma caffeine concentration achieved. The initial response in the obese seemed to be enhanced by a 3 week period on a low-energy diet (Fig. 2), but the increase calculated over a 2 h period did not achieve statistical significance.

**Plasma catecholamines (Table 4)**

The catecholamine response to caffeine was very variable. Overall, there was no significant rise in plasma noradrenaline, adrenaline and dopamine in any group after either oral or intravenous caffeine. Individual variations in response were seen, but in only two lean women was there a rise in both noradrenaline (of 355 and 140 pg/ml) and adrenaline (of 405 and 185 pg/ml respectively); these two also had elevated metabolic responses and developed an initial tachycardia on drinking caffeine, followed after 5 min by a slight bradycardia. The rest showed a bradycardia from the outset of the test. Other increases in noradrenaline or adrenaline were not accompanied by unusually marked responses in metabolic rate and did not relate to their having previously consumed an unusual amount of caffeine. Many subjects showed no change or even a slight fall in plasma catecholamine concentrations after caffeine.

**Plasma glucose and insulin**

After oral caffeine a similar but small rise in plasma glucose was seen in the lean, obese (on high-energy diet) and post-obese groups; the peak rise occurred at 90 min in both the lean group (incremental rise of glucose $= 0.33 \pm 0.12$ mmol/l) and the obese group on high-energy diet ($0.35 \pm 0.12$ mmol/l) and at 60 min in the post-obese group ($0.24 \pm 0.08$ mmol/l). When the obese group were slimming on a carbohydrate-reduced, low-energy diet they showed no rise in plasma glucose after oral caffeine.

After intravenous caffeine, the increase in plasma glucose was less than after oral caffeine but the obese subjects on the high-energy diet did have a significant response by 90 min ($0.3 \pm 0.1$ mmol/l); obese patients on the low-energy diet and the lean subjects had no significant rise in plasma glucose after the intravenous stimulant. Plasma insulin showed no significant rise in any test group.

**Plasma free fatty acid and glycerol**

The rise in plasma free fatty acid (Table 3) seemed to parallel the responses of the resting metabolic rate (Fig. 1) to oral and intravenous caffeine. The plasma free fatty acid and glycerol increments after oral and intravenous caffeine were similar in the lean and obese subjects on the high-energy diet (Table 3). The post-obese group, who had a lower rise in resting metabolic rate after oral caffeine, also had a reduced free fatty acid response but a normal increase in glycerol. Reducing the energy intake of the obese subjects
led to a greater response in plasma free fatty acid and glycerol concentrations after both intravenous and oral caffeine, which again simulated the effect on the metabolic rate (Fig. 2, Table 3).

**Controls**

The three obese women who were given only water showed no significant increases in resting metabolic rate, plasma noradrenaline, adrenaline, dopamine, caffeine, glucose, insulin, free fatty acid or glycerol (results not shown).

**Propranolol (Table 5)**

β-Adrenoceptor blockade reduced the basal resting metabolic rate (by 15%) as well as the basal plasma free fatty acid and glycerol concentrations. Oral caffeine given while the obese patients were maintained on propranolol led to a similar rise in plasma caffeine but the responses in metabolic rate, plasma free fatty acid and glycerol were enhanced on the propranolol regimen.

**Questionnaire (Table 6)**

All three groups of subjects consumed about 300 mg of caffeine/day. Although the normal-weight women drank fewer cups of coffee per day, their coffee was stronger. The caffeine intake remained unchanged with weight loss, although two women showed a change of preference, one from coffee to tea, the other vice versa. Intake of drinking cocoa was negligible.

**Discussion**

The literature for man suggests that coffee or caffeine (oral and intravenous) will increase the urinary excretion of adrenaline by about 80% and noradrenaline by 20% (Levi, 1967; Bellet, Kostis, Roman & De Castro, 1969). Work in rats and dogs has confirmed that caffeine stimulates the adrenal glands to secrete mainly adrenaline (De Schaepdryver, 1959; Berkowitz & Spector, 1971). With caffeine, the adrenaline content of a rat's heart increases, owing to adrenaline extrac-
tation from the circulation, and adrenalectomy abolishes this effect (Berkowitz & Spector, 1971). Caffeine also appears to stimulate synthesis of noradrenaline in the brain and the heart of rodents (Berkowitz, Tarver & Spector, 1970; Waldeck, 1971, 1972) and probably activates release of catecholamines by an effect on intracellular calcium concentrations (Rall & West, 1963).

Robertson et al. (1978) have measured the plasma catecholamine response to caffeine (250 mg) in non-coffee drinkers by a sensitive radioenzymatic technique which is similar to ours. Some of the subjects had abstained from caffeinated beverages for 21 days before the test whereas others normally avoided these beverages because of unpleasant side-effects. In the whole group caffeine produced a mean rise of plasma noradrenaline of 102 pg/ml (75% rise) and a rise of plasma adrenaline of 53 pg/ml (20-7% rise). In our group of habitual consumers of caffeine we were unable to find any consistent and significant increase in plasma catecholamines after oral or intravenous caffeine. A similar rise in plasma adrenaline to that found by Robertson et al. (1978) was observed in only three of our 22 subjects after oral caffeine and in three of our 14 subjects after intravenous caffeine. In the two lean women with an appreciable rise in both plasma noradrenaline and adrenaline there was an associated tachycardia, which might indicate some degree of hypersensitivity to caffeine. As tolerance to caffeine’s effects on diuresis, salivary gland secretion, central nervous system stimulation and negative chronotropic actions have been reported (Graham, 1978), it is conceivable that the regular, normal consumption of coffee induced tolerance in the adrenal medulla (Colton, Gosselin & Smith, 1967); this could explain the unresponsiveness of our subjects. In the present investigation the test was deliberately conducted on those who were regularly drinking caffeine as this is likely to be more nutritionally relevant. Given the failure to suppress the metabolic response to caffeine by propranolol it seems unlikely that caffeine’s effects are mediated by catecholaminergic mechanisms in subjects who habitually consume caffeine.

Caffeine exerts its effects in part by inhibiting phosphodiesterase activity and increasing lipolysis. The free fatty acid response to caffeine was not reduced, but was enhanced by β-adrenoceptor blockade; animal work also shows the independence of free fatty acid release from catecholamine stimulation, since sympathectomized animals have a normal response to caffeine (Hynie, Krishna & Brodie, 1966; Strubelt, 1969). Phosphodiesterase activity may itself be altered by β-adrenoceptor blockade and it is reduced in hypothyroidism (Reckless, Gilbert & Galton, 1976). However, an effect of caffeine on catecholamine sensitivity or thyroid metabolism could not explain the observed effects and it is probable that caffeine increased plasma free fatty acid mainly by inhibiting phosphodiesterase. The reduced free fatty acid release of the post-obese was not due to an impairment of lipolysis as glycerol response was normal. Enhanced free fatty acid re-esterification was probably occurring and could relate to increased insulin sensitivity developing after weight loss.

In addition to the thermogenesis associated with activation of the lipolytic system there can also be a rise in the metabolic rate in man in response to the increase in circulating free fatty acid (Jung, Shetty & James, 1981). Increases in plasma free fatty acid, induced by infusions of heparin and triglyceride, can be used to predict the effect of increases in plasma free fatty acid (and glycerol) without incurring the metabolic cost of stimulating adipocyte lipolysis. When this is done the increase in resting oxygen consumption for the observed rise in plasma free fatty acid of 200–400 μmol (Table 3) amounts to only 0.1 ± 0.15 kJ/min, i.e. 30–50% of the observed metabolic response (Jung et al., 1980). The additional metabolic response to caffeine may therefore relate to its direct metabolic action on adipocytes and other cells.

Previous work has shown a reduced free fatty acid response to oral caffeine in obese subjects (Oberman et al., 1975), but a normal response to intravenous caffeine (Ratzmann, Riemer, Männchen & Paul, 1976). These studies did not include a measure of the metabolic rate in association with these changes and the present work on the post-obese suggests that subnormal rates of free fatty acid release and metabolic rate response may indicate an adaptation to a reduced food intake rather than inherent differences in the rates of free fatty acid release and metabolism in the obese. In other studies (Jung et al., 1981) we have found no suggestion of impaired free fatty acid oxidation in the obese, a finding in keeping with that of Issekutz, Paul, Miller & Bortz (1968). The prompt metabolic response to caffeine in lean and obese groups does suggest that caffeine may have an appreciable effect on energy balance, but the equivalent intake in the lean and obese subjects suggests that caffeine drinking is unlikely to explain differences in the body-fat content of individuals.

We conclude that the metabolic response to caffeine appears to result mainly from an effect
on adipocyte phosphodiesterase and lipolysis and not from mechanisms involving catecholaminergic stimulation of adipocyte or other cellular metabolism.

Acknowledgments

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References


