ORIGINAL RESEARCH

Effects of diets high in whey, soy, red meat and milk protein on body weight maintenance in diet-induced obesity in mice

Xu-Feng HUANG, Yingxu LIU, Gita L. RAHARDJO, Peter L. MCLENNAN, Linda C. TAPSELL and William A. BUTTEMER

1Smart Food Centre, School of Health Sciences, and 2School of Biological Sciences, University of Wollongong, New South Wales, Australia

Abstract

This study examined the effects of different food sources of protein on energy intake, body weight maintenance, and on the responses of plasma leptin, insulin and adiponectin in chronic high-fat diet-induced obese mice. Obesity was induced in 47 mice with a high-fat diet for 20 weeks. They were divided into five diet groups to test the effects of a higher protein proportion (30% energy), achieved at the expense of carbohydrate. For the next eight weeks, four of the groups were fed diets of chow formulated with whey, soy, red meat or milk while the control group continued on their high-fat diet. The results showed that: (i) increasing the protein : carbohydrate ratio (both at 30% energy) in a high-fat diet did not reduce the level of obesity; (ii) the type of protein added, however, did have a significant effect on the level of obesity attained; (iii) whey protein stabilised weight gain the most, had the strongest satiety effects and also stimulated the highest production of adiponectin; and (iv) whey protein also was associated with the lowest insulin values among all proteins tested. Plasma leptin levels were not affected by any of the diets. Dietary fat remains a potent factor in weight management, but the type and amount of protein may also be important through its effects on food intake. In particular, the apparent decreased appetite associated with increased adiponectin in the whey-based high-protein diet may contribute to stabilised body mass in chronic high-fat diet-induced obesity.

Key words: adiponectin, energy balance, high-protein diet, obesity.

INTRODUCTION

Animal model studies provide important theoretical explanations of how foods and food components may affect weight gain and obesity. Obesity is a major factor predisposing individuals to a variety of life-threatening diseases. These include Type II diabetes, hypertension and coronary heart disease, all of which result in enormous financial and social costs. The incidence of obesity has increased substantially in the past 50 years, in part due to the prevalence of high-fat diets among Western societies. There is considerable interest, therefore, in identifying dietary interventions that reduce the development of obesity, but food guidance systems need to be set in context. This means enabling consumers to differentiate between foods that may affect satiety and energy balance, especially in the context of food (and dietary fat) abundance. Most of the knowledge informing this activity concerns nutrients, but research on food is warranted as in the end people eat foods, not nutrients.

Protein, compared with carbohydrate and fat in isocaloric amounts, has been shown to significantly increase satiety. Previous studies have shown that the source of dietary protein can influence subjective satiety and food intake in humans; however, these studies are primarily acute meal-based studies in healthy subjects. For example, compared with casein, whey has been shown to increase subjective satiety and decrease food intake in healthy volunteers. Greater subjective satiety was also found over three hours when young men were fed a 50 g meal of lean fish compared with a meal with equivalent amounts of beef or chicken protein; while a gelatin lunch was found to have a longer...
satiety effect compared with the casein lunch in healthy men. Conversely, other studies have found that dietary protein sources were not a factor influencing later food intake. When various protein sources (egg albumen, casein, gelatin, soy, pea and wheat gluten) were fed in a lunch meal containing 5000 KJ energy, no difference in energy intake was found eight hours later at dinner. However, these negative results may be due to the eight-hour delay until dinner and the low amount of protein in the meals (22% of energy as protein).8

A theoretical understanding of the role of protein in mechanisms affecting food intake would help explain these observed effects. Animal-based experiments have shown that dietary protein source can affect the levels of blood insulin and leptin, which regulate glucose and fat metabolism. For example, soy and cod protein diets lower serum insulin level, whereas high-casein diets produce high levels of insulin, but no difference in fasting or postprandial plasma levels was found between soy and cod protein diets.9–11 It has also been reported that insulin sensitivity significantly increased in rats fed whey protein diets compared with red meat protein. Leptin is an adipocyte-derived hormone that promotes negative energy balance. Rats fed high-casein and high-fat diets had lower serum leptin concentrations than rats fed a high-soy high-fat diet.11

These findings provide direction for research in humans but at present, there is little information regarding the effects on satiety from long-term dietary intervention and the corresponding blood hormone levels in obese humans. Although studies have suggested that high-protein diets promote weight loss compared with low-protein diets, the results are still inconsistent and long-term studies substituting carbohydrate with protein for body weight reduction and maintenance are very limited.12 The significant investment in these studies, however, warrants strong theoretical development, including that relating to the choice of types of foods that would best deliver the protein. Using a chronic high-fat diet-induced obese animal model, this study aimed to test whether different food sources of protein produced different effects on energy intake, body weight gain and the related hormones leptin, insulin and adiponectin.

**METHODS**

**Animals, diet and experimental procedures**

The protocol used in this research was approved by Animal Ethics Committee, the University of Wollongong, Australia (AE03/31). One hundred and eleven 12-week-old C57Bl/6 male mice were used in this study. Mice were obtained from the Animal Resource Center (Perth, Western Australia). They were housed individually in environmentally controlled conditions (temperature, 22°C; light cycle from 06.00 to 18.00 hours and dark cycle from 18.00 to 06.00 hours) and had ad libitum access to food and water. All mice were fed standard laboratory chow during the first week of adjustment to this new environment. They were then placed on a high-fat diet of specially formulated chow (16% PTN, 44% CHO, 40% fat; 3.86 kcal/g) (Figure 1a).

After 20 weeks on the high-fat diet, 47 mice with the highest body weight gain were designated as chronic diet-induced obese (cDIO) mice according to the method that we and others have used previously.13–15 These cDIO mice were randomly divided into five groups. Over the next eight weeks, four groups were fed high-protein diets with chow formulations based on protein sources of either whey, soy, red meat or milk (30% PTN, 30% CHO, 40% fat; Figure 1b) and where the protein content substituted for carbohydrate content. The fifth group remained on the high-fat diet for eight weeks and served as controls. Foods were made from semisynthetic materials as 1 cm cubes, following the recommendation of ‘AIN93 Diet for Laboratory Rodents’.16

**Food intake and body weight**

Food intake was measured daily during the eight-week provision of high-protein diets and energy intakes were calculated from the composition values for the chow. A weighed amount of food was given at the beginning of the dark cycle and all remaining food was collected and weighed 24 hours later. Animals were weighed weekly throughout the entire eight-week period of high-protein feeding.

**Morphological and endocrinological measures**

The mice were killed at the end of the eight-week dietary period using an overdose of sodium pentobarbitone.
Table 1  Weekly energy intake (kcal/week) of chronic diet-induced obese mice fed five different diets

<table>
<thead>
<tr>
<th></th>
<th>Whey (n = 10)</th>
<th>Soy (n = 10)</th>
<th>Red meat (n = 10)</th>
<th>Milk (n = 9)</th>
<th>Control (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior</td>
<td>108.4 ± 5.4</td>
<td>110.9 ± 2.7</td>
<td>113.4 ± 4.6</td>
<td>100.7 ± 4.0</td>
<td>111.6 ± 2.8</td>
</tr>
<tr>
<td>Week 1</td>
<td>105.9 ± 3.4(a)</td>
<td>114.2 ± 2.2</td>
<td>96.1 ± 3.1(b)</td>
<td>103.2 ± 3.3</td>
<td>116.6 ± 5.9</td>
</tr>
<tr>
<td>Week 2</td>
<td>82.9 ± 5.3(a)(b)(c)</td>
<td>101.0 ± 5.3</td>
<td>99.1 ± 3.4</td>
<td>96.6 ± 3.6</td>
<td>110.9 ± 5.9</td>
</tr>
<tr>
<td>Week 3</td>
<td>106.9 ± 3.1</td>
<td>113.7 ± 3.1</td>
<td>110.5 ± 3.0</td>
<td>112.7 ± 3.3</td>
<td>107.7 ± 3.0</td>
</tr>
<tr>
<td>Week 4</td>
<td>103.7 ± 3.4</td>
<td>113.1 ± 3.5</td>
<td>107.6 ± 2.9</td>
<td>103.3 ± 3.8(b)</td>
<td>104.0 ± 3.5</td>
</tr>
<tr>
<td>Week 8</td>
<td>97.6 ± 4.5</td>
<td>104.3 ± 2.8</td>
<td>95.5 ± 3.0</td>
<td>103.4 ± 3.3</td>
<td>103.0 ± 5.5</td>
</tr>
<tr>
<td>Total</td>
<td>809.1 ± 22.8(b)</td>
<td>891.3 ± 18.0</td>
<td>823.7 ± 17.3(b)</td>
<td>847.2 ± 20.1</td>
<td>873.4 ± 33.2</td>
</tr>
</tbody>
</table>

(a) versus control.
(b) versus soy.
(c) versus red meat.
Mean ± SEM; P < 0.05.

anaesthesia (120 mg/kg, intraperitoneally). Euthanasia took place between 07.00 and 09.00 hours to minimise circadian-based variation in blood hormone concentrations. Blood samples were obtained by puncturing the right ventricle of the heart. Plasma leptin, insulin and adiponectin were measured using the LINCOpex Mouse Endocrine Immunoassay Panel (Linco Research, St. Charles). Various fat pads including epididymal, peripheral, omental and inguinal fat masses were removed bilaterally and weighed. Other visceral organs including heart, liver, kidney, intestine and stomach (with and without food contents) as well as the soleus muscle were weighed. The lengths of tibia and intestine were also measured.

**Data analysis**

For energy intake, body weight, fat pads and body weight gain, an analysis of variance was performed followed by post-hoc Tukey–Kramer honestly significant difference (HSD) tests for multiple comparisons among the groups. Unless stated otherwise, values are presented as mean ± SEM.

**RESULTS**

**Energy intake**

Energy intake rates differed significantly between the cDIO mice fed whey, soy, red meat (RM) and milk diets as a percentage of feeding rates in control mice over the first 17 days on the diet. (b) Average total energy intake cDIO mice fed high-protein diets as a percentage of total energy consumed by control mice over the eight-week diet period.

![Figure 2](image_url) (a) Energy intake rates of chronic diet-induced obese (cDIO) mice fed high whey, soy, red meat (RM) and milk diets as a percentage of feeding rates in control mice over the first 17 days on the diet. (b) Average total energy intake cDIO mice fed high-protein diets as a percentage of total energy consumed by control mice over the eight-week diet period.

For energy intake, body weight, fat pads and body weight gain, an analysis of variance was performed followed by post-hoc Tukey–Kramer honestly significant difference (HSD) tests for multiple comparisons among the groups. Unless stated otherwise, values are presented as mean ± SEM.

Energy intake rates differed significantly between the cDIO mice fed whey, soy, red meat or milk-based diets over the eight-week diet period (Wilks’ Lambda = 0.25, F_{13,1} = 14.95, P < 0.005, multivariate partial eta squared = 0.79). Post-hoc tests showed that the whey and red meat protein diet groups had significantly lower rates of energy intake compared with the soy protein diet group, with the most pronounced differences occurring in the first two weeks (Table 1, Figure 2a).

The total energy intake over eight weeks of dietary intervention was significantly lower in the whey (~9%) and red meat (~8%) groups than in the soy group. Compared with the baseline, the average energy intake throughout the entire eight weeks of high protein feeding was significantly differ-

**Body weight gain and fat deposits**

Although there appeared to be differences in body weight after eight weeks of dietary intervention (Table 2), these differences did not reach statistical significance among all
Table 2 Body weight and fat deposit in chronic diet-induced obese mice treated with high-protein diets for eight weeks

<table>
<thead>
<tr>
<th></th>
<th>Whey (n = 10)</th>
<th>Soy (n = 10)</th>
<th>Red meat (n = 10)</th>
<th>Milk (n = 9)</th>
<th>Control (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior to</td>
<td>40.9 ± 0.9</td>
<td>41.8 ± 1.1</td>
<td>41.4 ± 0.7</td>
<td>41.8 ± 0.8</td>
<td>40.8 ± 1.1</td>
</tr>
<tr>
<td>After 8 weeks</td>
<td>46.9 ± 1.3</td>
<td>50.9 ± 0.7</td>
<td>50.3 ± 1.1</td>
<td>48.5 ± 1.2</td>
<td>45.5 ± 1.2</td>
</tr>
<tr>
<td>Body weight gain</td>
<td>6.1 ± 0.7**(b)(c)</td>
<td>9.0 ± 0.8**(a)</td>
<td>8.9 ± 0.7**(a)</td>
<td>6.7 ± 1.1**(b)</td>
<td>4.7 ± 0.6</td>
</tr>
<tr>
<td>Fat deposits (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visceral</td>
<td>5.68 ± 0.25**(b)(c)</td>
<td>6.31 ± 0.17**(a)</td>
<td>6.45 ± 0.19**(a)</td>
<td>5.98 ± 0.26**(a)</td>
<td>5.31 ± 0.21</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td>2.04 ± 0.18</td>
<td>2.28 ± 0.12(4)</td>
<td>2.36 ± 0.12**(a)</td>
<td>2.22 ± 0.10</td>
<td>1.86 ± 0.13</td>
</tr>
<tr>
<td>WAT</td>
<td>7.72 ± 0.38**(b)(a)</td>
<td>8.50 ± 0.22**(a)</td>
<td>8.81 ± 0.28**(a)</td>
<td>8.20 ± 0.29**(a)</td>
<td>7.17 ± 0.32</td>
</tr>
<tr>
<td>BAT</td>
<td>0.34 ± 0.02**(b)</td>
<td>0.44 ± 0.03</td>
<td>0.37 ± 0.03</td>
<td>0.33 ± 0.02**(b)</td>
<td>0.40 ± 0.02</td>
</tr>
</tbody>
</table>

**(a)** versus control.  
**(b)** versus soy.  
**(c)** versus red meat.  

Mean ± SEM; P < 0.05; visceral fat = epididymal + perirenal + omental fats; subcutaneous fat = inguinal fat; white adipose tissue (WAT) = visceral fat + subcutaneous fat; brown adipose tissue (BAT) = interscapular fat.

**Visceral organs, muscle and bone**

The liver weight was significantly heavier in the soy group than in the control and whey groups, averaging 42% and 27% higher, respectively, and the weight of filled stomachs was 29% heavier in the soy than control groups (Table 3). None of the other organ masses or structural lengths measured differed significantly among treatment groups (Table 3).

**The levels of plasma insulin, leptin and adiponectin**

Plasma insulin concentrations varied significantly among the protein diets (F<sub>4,36</sub> = 3.668, P = 0.013; Table 4). The soy, red meat and milk diets had plasma insulin concentrations that were 79%, 71% and 94% higher, respectively, than the levels we measured in the control group. In contrast, there was no difference in the plasma insulin levels between the whey and control groups.

The levels of plasma leptin were not statistically distinguishable among all groups (F<sub>3,36</sub> = 0.444, P = 0.776; Table 4).

Plasma adiponectin concentrations varied significantly among the treatment groups (F<sub>4,36</sub> = 5.18, P < 0.001; Table 4). The whey group tended to have higher plasma adiponectin levels than all other groups, but these did not differ significantly from those for controls or the milk protein diet (Table 4). Conversely, the soy group had significantly lower levels of plasma adiponectin than all other diet groups (P < 0.05; Table 4).

**DISCUSSION**

This study has examined satiety, body weight change and key blood-borne hormones in chronic high-fat diet-induced obese mice fed with whey, soy, red meat and milk protein diets for eight weeks. The results showed that:
Table 3 Organ morphometrics of chronic diet-induced obese mice fed various high-protein diets for eight weeks

<table>
<thead>
<tr>
<th>Visceral organs</th>
<th>Whey (n = 10)</th>
<th>Soy (n = 10)</th>
<th>Red meat (n = 10)</th>
<th>Milk (n = 9)</th>
<th>Control (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver (g)</td>
<td>2.16 ± 0.16(b)</td>
<td>2.75 ± 0.23a</td>
<td>2.27 ± 0.17</td>
<td>2.26 ± 0.17</td>
<td>1.93 ± 0.18</td>
</tr>
<tr>
<td>Heart (g)</td>
<td>0.16 ± 0.00</td>
<td>0.16 ± 0.01</td>
<td>0.18 ± 0.01</td>
<td>0.16 ± 0.01</td>
<td>0.18 ± 0.01</td>
</tr>
<tr>
<td>Stomach</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With filling (g)</td>
<td>0.35 ± 0.03</td>
<td>0.44 ± 0.03a</td>
<td>0.40 ± 0.05</td>
<td>0.40 ± 0.05</td>
<td>0.34 ± 0.03</td>
</tr>
<tr>
<td>Empty (g)</td>
<td>0.15 ± 0.01</td>
<td>0.15 ± 0.01</td>
<td>0.15 ± 0.01</td>
<td>0.15 ± 0.01</td>
<td>0.15 ± 0.02</td>
</tr>
<tr>
<td>Intestine (g)</td>
<td>1.70 ± 0.13</td>
<td>1.85 ± 0.10</td>
<td>1.74 ± 0.12</td>
<td>1.80 ± 0.14</td>
<td>1.73 ± 0.10</td>
</tr>
<tr>
<td>Intestine (mm)</td>
<td>441.4 ± 6.8</td>
<td>456.2 ± 8.0</td>
<td>462.7 ± 10.7</td>
<td>466.1 ± 9.0</td>
<td>447.0 ± 7.8</td>
</tr>
<tr>
<td>Kidney (g)</td>
<td>0.50 ± 0.03</td>
<td>0.52 ± 0.03</td>
<td>0.52 ± 0.02</td>
<td>0.52 ± 0.03</td>
<td>0.48 ± 0.04</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soleus (mg)</td>
<td>10.4 ± 0.6</td>
<td>9.1 ± 1.0</td>
<td>9.5 ± 0.8</td>
<td>9.4 ± 0.9</td>
<td>9.7 ± 0.8</td>
</tr>
<tr>
<td>Tibia (mm)</td>
<td>23.4 ± 0.4</td>
<td>23.0 ± 0.3</td>
<td>23.3 ± 0.3</td>
<td>23.5 ± 0.2</td>
<td>22.9 ± 0.3</td>
</tr>
</tbody>
</table>

(a) versus control.
(b) versus soy.
Mean ± SEM; P < 0.05.

Table 4 The levels of plasma insulin, leptin and adiponectin in mice on five different diets

<table>
<thead>
<tr>
<th></th>
<th>Whey (n = 8)</th>
<th>Soy (n = 9)</th>
<th>Red meat (n = 8)</th>
<th>Milk (n = 8)</th>
<th>Control (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin (ng/mL)</td>
<td>1.26 ± 0.20(b)(d)</td>
<td>2.56 ± 0.43a</td>
<td>2.45 ± 0.43a</td>
<td>2.77 ± 0.43a</td>
<td>1.43 ± 0.19</td>
</tr>
<tr>
<td>Leptin (ng/mL)</td>
<td>11.85 ± 1.51</td>
<td>13.77 ± 1.37</td>
<td>14.52 ± 1.17</td>
<td>12.72 ± 0.77</td>
<td>12.79 ± 0.74</td>
</tr>
<tr>
<td>Adiponectin (µg/mL)</td>
<td>16.47 ± 1.78(b)(c)</td>
<td>9.95 ± 0.31(a)</td>
<td>12.89 ± 0.72(b)</td>
<td>13.86 ± 0.78(b)</td>
<td>14.63 ± 1.25</td>
</tr>
</tbody>
</table>

(a) versus control.
(b) versus soy.
(c) versus red meat.
(d) versus milk.
P < 0.05.

(i) substituting carbohydrate with protein to a level of 30% energy in a high-fat diet did not reduce the level of obesity; (ii) the type of protein used, however, did have a significant effect on further weight gain; (iii) whey protein appeared to stabilise weight gain the most; (iv) whey protein stimulated the highest production of adiponectin; and (v) whey protein was associated with the lowest insulin values among all protein diets tested.

We found that an overall increase in dietary protein from 16% to 30% was insufficient to correct the late stage of diet-induced obesity in mice previously fed a high-fat diet. This is despite of the known effects of protein in stimulating thermogenesis and satiety and its known poorer assimilation efficiency compared with carbohydrates or fats. These results indicate that overall energy intake and perhaps also fat content remain of prime importance. Nevertheless, differences in effects from the food source of protein were observed. For example, when the source of protein was whey, no further weight gain was observed, but this was not the case when the source was red meat or soy. This may have implications in the design of dietary interventions involving humans.

Protein is generally accepted as the satiating macronutrient that can suppress food intake more than fats or carbohydrates and delay the return of hunger. Thus, high-protein diets may function by suppressing hunger to a greater extent and helping to maintain a lower energy intake. This study showed that energy intake was significantly lower in whey, red meat and milk, but not soy, diet groups compared with controls in the first seven days of the study. A lower energy intake continued in the whey and milk groups in the next seven days, but it did not continue in the red meat group. This suggests that the satiety effect of dairy protein persists longer than that of red meat. Between the whey and milk groups, whey appears to have a more profound effect than milk (Figure 2). This is in agreement with previous human studies showing subjects given a preload containing about 50 g of whey protein consumed significantly lower amounts of food than when given a casein preload. A possible explanation is that whey has been found to be digested quickly, resulting in a rapid increase in plasma amino acids that is sustained for more than two hours. Increased brain amino acid concentration may contribute to the suppression of food intake. Also, whey ingestion results in greater release of several gut peptides involved in satiety including cholecystokinin, glucagon-like peptide 1 and glucose-dependent insulinotropic polyptide significantly compared with casein intake. Furthermore, whey prepared
by ultrafiltration as used in this study contains about 15% caseinomacropeptide, a bioactive macropeptide known to stimulate cholecystokinin release, which in turn can inhibit food intake. This brings in the importance of the food matrix when considering food formulations.

The present study showed that the plasma adiponectin levels were significantly higher in the mice fed a whey-based high-protein diet than those based on soy or red meat protein. Adiponectin is a hormone secreted by adipocytes and acts as adipokine in muscle and liver which increases fatty acid oxidation and glucose uptake. Unlike many of the other adipokines such as leptin, TNF-α and resistin, which increase with adiposity, circulating adiponectin concentrations are reduced in obese and in Type 2 diabetes in humans. Although the mechanism of regulation of plasma adiponectin is unknown, it has been shown that circulating adiponectin concentrations are (i) not affected by fasting or leptin administration; (ii) correlated with insulin sensitivity; (iii) increased with the administration of PPARgamma ligand, thiazolidinedione; and (iv) increased when weight is lost. On the other hand, food intake rates of mice were unaffected by intracerebroventricular injections of adiponec
tin. The present study showed that the whey diet group had the lowest body weight gain and visceral fat deposits, and improvement of plasma insulin compared with other diet groups in the late stage of diet-induced obesity in mice. This suggests that these diets differ in their metabolic effects, but quantification of energy expenditure is needed to corroborate this.

In summary, this study showed that there is good reason to consider the food source of protein as well as the level of protein in dietary studies manipulating protein levels. The type of protein used to reach a level of 30% energy did have a significant effect on the level of obesity attained. In the context of a high-fat diet, whey protein was found to prevent further body weight gain, significantly elevate adiponectin production, and result in low levels of plasma insulin. These differentiating factors are informative for human dietary studies. It also may be important to consider the approach to the whole macronutrient proportion in dietary modelling, as decreasing carbohydrate to accommodate the increased protein may be less efficacious than decreasing the fat component. We found that providing more dietary protein without controlling fat intake did not reduce body weight gain in chronic high-fat diet-induced obese mice. Given the metabolic consequences, the type of fat and carbohydrate is also highly likely to warrant consideration. The animal model used in this study mimics human chronic obesity, and is therefore useful in uncovering new knowledge that would support the design of dietary interventions and of strategic development in the functional foods domain.

ACKNOWLEDGEMENT

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CONFLICT OF INTEREST

No conflict of interest has been declared by X.-F. Huang, Y. Liu, G.L. Rahardjo, P.L. McLennan or L.C. Tapsell.

REFERENCES


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