Do Protein and Phosphorus Cause Calcium Loss?

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ABSTRACT The widespread opinion that both protein and phosphorus cause calcium loss is examined. Controlled human studies show that commonly used complex dietary proteins, which have a high phosphorus content, do not cause calcium loss in adult humans. Similarly, a phosphorus intake of up to 2000 mg/d does not have adverse effects on calcium metabolism; however, the type of phosphate contained in carbonated beverages may not behave in the same manner. In contrast, a diet low in protein and phosphorus may have adverse effects on calcium balance in the elderly. Studies with adults suggest that high protein foods do not cause calcium loss. J. Nutr. 118: 657-660, 1988.

INDEXING KEY WORDS: protein • phosphorus • phosphorus depletion • calcium • osteoporosis

The belief that protein and phosphorus cause calcium loss in humans is still widespread in the scientific literature (1-3) and in popular magazines. Regarding the effect of protein on calcium metabolism, the purified proteins and amino acid mixtures used in purified diets increase urinary calcium and lead to calcium loss in animals (4) and humans (5, 6). However, commonly used complex dietary proteins do not have these effects in strictly controlled long-term human studies (7, 8) in which large amounts of dietary protein, as red meat, which has a high phosphorus content, were used. When 1 lb. of meat was given daily for as long as 4 mo the urinary calcium did not change significantly. Additional unpublished metabolic balance studies have shown that a high meat intake given for 36-84 d did not significantly increase urinary calcium in eight of nine male subjects (Table 1). The patient who had an increase, patient 5, had received long-term antituberculosis medications, some of which increase urinary calcium (9). These may have interacted with the high dietary protein.

The difference in results obtained with purified and complex dietary proteins may be due to the high phosphorus content of red meat because phosphorus has been shown to decrease urinary calcium regardless of the calcium intake (10, 11). Any increase in urinary calcium that may have occurred because of the acid-ash nature of red meat (12) may have been counteracted by its high phosphorus content. Others have also shown that phosphorus decreases urinary calcium (13). In an animal study in which beef was used as the protein source, there was no adverse effect on bone (14), whereas a short-term 7-d human balance study reported that a high beef intake increased urinary calcium (15). A temporary increase in calciuria may occur, although rarely, during the initial high protein intake given as meat. This was observed in a single case (7) in our 21 high protein studies (7, 8) and was followed by a progressive decrease to levels below the baseline for urinary calcium despite continuation of the high protein diet for another 180 d (7).

Numerous studies in animals and in humans have been published on the calcium-losing effect of protein (16). However, several aspects of the study conditions have to be considered in the interpretation of the effect of protein on calcium metabolism in humans. Some of these are the type of protein, such as purified proteins (5, 6, 17-19) or complex dietary proteins; the duration of the high protein study, i.e., short-term (15, 17, 20) or long-term (7, 8, 18, 21); whether the phosphorus intake remained the same (6), was increased (7, 8) or decreased (18); whether the studies were carried out in out-patient volunteers (20, 21) or under strictly controlled dietary conditions; whether the protein intake was changed from a low to a high protein intake as was done in most human studies (5, 6, 19, 20, 22, 23), or was changed from a normal to a high protein intake (7, 8); and whether excessively high protein intakes were used (15, 19, 22, 23). All these factors affect urinary calcium during a high protein intake, although many other factors play a role (9), such as physical activity and the constancy of the sodium intake. To our knowledge, no convincing data have been published showing

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TABLE 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Body weight (kg)</th>
<th>Length of study (d)</th>
<th>Protein intake (g/kg)</th>
<th>Urinary calcium (mg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Control</td>
<td>High protein (kg)</td>
<td>Control</td>
<td>High protein</td>
</tr>
<tr>
<td>1</td>
<td>69</td>
<td>68.85</td>
<td>67.29</td>
<td>60</td>
<td>1.0</td>
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<td>2</td>
<td>52</td>
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<td>3</td>
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<td>77.28</td>
<td>74.40</td>
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</tr>
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<td>4</td>
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<td>78.91</td>
<td>78.57</td>
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<tr>
<td>5</td>
<td>53</td>
<td>73.22</td>
<td>72.55</td>
<td>24</td>
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</tr>
<tr>
<td>6</td>
<td>66</td>
<td>67.82</td>
<td>65.61</td>
<td>54</td>
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<td>7</td>
<td>56</td>
<td>72.33</td>
<td>72.53</td>
<td>48</td>
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</tr>
<tr>
<td>8</td>
<td>58</td>
<td>86.06</td>
<td>81.48</td>
<td>96</td>
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<tr>
<td>9</td>
<td>47</td>
<td>67.28</td>
<td>70.44</td>
<td>48</td>
<td>1.0</td>
</tr>
<tr>
<td>Average</td>
<td>58</td>
<td>73.28 ± 2.13</td>
<td>72.25 ± 1.77</td>
<td>46</td>
<td>1.0</td>
</tr>
</tbody>
</table>

*High protein intake given as red meat.

that a high protein diet, using complex proteins for prolonged periods of time under strictly controlled dietary conditions, causes calcium loss. Dietary proteins other than red meat, such as milk and cheese, which are also high phosphorus foods, do not cause calcium loss in controlled studies in humans (24). The generalization that "protein" causes calcium loss or may be a risk factor for osteoporosis, without specifying the type and source of protein, is therefore incorrect.

With regard to the alleged adverse effect of phosphorus on calcium metabolism or bone, these results primarily apply to lower animal species (25, 26) but not to subhuman primates (27) or strictly controlled human metabolic studies (10, 11). When the phosphorus intake was increased by a factor of 2.5 and was as high as 2000 and 2700 mg/d no adverse effects were observed on any parameter of calcium metabolism, the only significant change being a decrease in urinary calcium in the absence of decreased intestinal absorption of calcium (11). The calcium balance did not change significantly during the high phosphorus intake, regardless of the calcium intake (10, 11). In human studies, ortho- and polyphosphates did not affect bone resorption (28), although one study reported increased bone resorption in osteoporotic women during a high phosphorus intake in the absence of increased serum parathyroid hormone [PTH] levels (29). Recently, the intermittent use of 2000 mg phosphorus in conjunction with other therapeutic measures has been reported to increase bone mineral density in osteoporosis (30). This is not surprising because the bone crystal hydroxyapatite contains calcium phosphate and the use of calcium alone, without the addition of phosphate, cannot be expected to improve the bone structure (31). Beneficial effects of phosphorus such as a decrease in urinary calcium (13), promotion of fracture healing (32) and reduction of the hypercalcuiuria induced by purified proteins (22) have been reported in humans. In animal studies, phosphorus depletion increased bone resorption, whereas phosphorus decreased bone resorption (33); it increased bone mineralization (34); and in in vitro studies it increased collagen synthesis (35) and inhibited bone resorption (36).

The effects of phosphorus in humans that we have cited apply to dietary phosphorus and not to phosphate contained in carbonated soft drinks. In young adults the use of soft drinks led to a decrease in ionized serum calcium, an increase in the serum PTH levels [although not significant] and an increase of urinary hydroxyproline and cyclic AMP, changes that have been interpreted as increased parathyroid function (37). In a survey of a large number of women, the frequency of skeletal fractures was increased by a factor of 2.3 in athletic women who consumed carbonated beverages (38). The reason for these changes (37, 38) is not clear. Most carbonated soft drinks contain phosphoric acid as well as citric acid, and these acid components may adversely affect bone because acidosis leads to an increase in urinary calcium (39–41).

Statements that high protein and high phosphorus intakes have adverse effects on calcium metabolism may lead physicians to recommend a low protein and low phosphorus diet to prevent calcium loss. This regimen may then particularly apply to the elderly who are most prone to develop or to have osteoporosis. Low protein diets are usually also low phosphorus diets by nature of their composition, and this type of dietary intake would lead to a deficient intake of both of these nutrients. Elderly persons may already be in a suboptimal, if not poor, nutritional state (42). Restriction of dietary protein and particularly of dietary phosphorus would lead to further debility and to adverse effects on general health and on the skeleton (43). Although food restriction may prolong the life span (44), a further de-
crease in food intake of already nutritionally compromised elderly persons is undesirable, particularly the decreased intake of phosphorus [43] and of protein [45, 46]. The low intake of phosphorus associated with a low supply of protein is a greater risk factor for bone loss and even for complications such as skeletal fractures than an adequate or relatively high phosphorus and a high protein intake. A diet-induced low phosphorus status may have adverse effects similar to those due to drug-induced phosphorus depletion [43, 47]. Chronic undernutrition has adverse effects on the bone structure in young adults [46, 48], and this effect may be exaggerated in the elderly. Certain parameters of calcium metabolism, such as low levels of the vitamin D metabolite, 1,25-dihydroxycholecalciferol [49] and PTH levels [50], are also altered in the elderly, which can lead to inevitable bone loss. The elderly are also prone to use more medications than younger persons and some drugs induce a low phosphorus status. Commonly used aluminum-containing antacids induce phosphorus depletion and secondary calcium loss [43, 47]. A low phosphorus diet used in conjunction with these drugs would exaggerate this effect and would also lead to generalized physical debility [43].

Because long-term controlled human studies do not support the view that increased dietary protein [7, 8] and a high phosphorus intake [10, 11] cause calcium loss, these nutrients should not be considered as risk factors for calcium loss and osteoporosis. A normal protein intake and a normal or relatively high phosphorus intake, preferably in conjunction with a normal or high calcium intake, derived from natural foods, would be more protective than harmful, particularly for the elderly.

**LITERATURE CITED**


