INFLUENCE OF MAGNESIUM ON THE DEPOSITION OF CADMIUM IN RATS

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Abstract

Magnesium chloride was given via drinking water (500 mg Mg/L) to rats exposed intragastrically to cadmium (cadmium 109) at a dose corresponding to 10 mg/kg diet for 7, 14, 21, and 28 d. The results provided the evidence that magnesium decreased cadmium retention in the duodenum, kidneys, and liver after 7 d. Significant reduction in cadmium retention was also found on days 14 d and 21 in kidneys. No significant decreases in cadmium retention in the organs examined were noted after 28 d. The results suggest that beneficial action of magnesium on body cadmium accumulation ceased with time after the two metal administration.

Key words: rat, cadmium, magnesium, interaction.

Cadmium is a widespread environmental contaminant with unknown metabolic functions. This metal reveals cumulative properties and may retain in the body for a long time. A long biological half-life of cadmium means that even a small decrease in its bioavailability may result in lowering distant toxicological manifestations (9, 11, 14).

The bodily uptake and toxic actions of cadmium seem to be proportional to its environmental and dietary concentrations. Several studies have well documented that the rate of cadmium absorption from dietary sources and cadmium toxic manifestations may be influenced by nutritional status of trace elements including zinc, selenium, iron, manganese, or calcium. The protective role of these trace elements involves alterations in cadmium absorption from the gastrointestinal tract and its toxicokinetics and toxicodynamics in the body (2, 5, 8, 10, 12, 16, 17).

The studies referred to magnesium and cadmium interaction are not very popular although magnesium plays a significant role in people and breeding of animals. It was also reported that feeding magnesium over the standard level is well tolerated by animals (6, 9).

In earlier studies, Van Barnevels and Van den Hamer (19) found that feeding magnesium deprived or magnesium supplemented diet did not affect cadmium metabolism in mice. More recent reports have provided the evidence that magnesium pretreatment protects the kidneys against toxic doses of cadmium (4). Moreover, Bulat et al. (3) found that magnesium supplementation of rabbits exposed to subacute doses of cadmium reduced cadmium retention in several organs of the animals.

The aim of the study was to examine the effect of magnesium supplementation on cadmium distribution in the body of rats treated with cadmium in doses resembling those reported in the areas contaminated with this metal (11).

Material and Methods

Seventy five male Wistar rats initially weighing from 230 g to 255 g were used. The animals were randomly assigned into three dietary groups (the controls – group I. Cd treated – group II, and Cd plus Mg treated – group III) each of 25 rats after an acclimatisation period of one week. Rats were given a tap water (about 19 mg Mg/L) supplemented with dissolved magnesium chloride (POCh, Poland) up to 500 mg Mg/L and a standard rodent chow LSM ad libitum (Fodder Manufacture Motycz, Poland) containing about 19 mg of Mg/kg according to the manufacturer’s instruction. The total daily magnesium intake in these groups was twice that offered for rats fed non-supplemented tap water and the standard LSM diet. The consumption of feed and tap water was recorded. The animals were on diets for the whole experimental period. Rats in groups II and III were given intragastrically cadmium chloride labelled with cadmium 109 (Polatom, Poland) in a 0.5 mL water solution comprising about 20 kBq per rat daily for 7, 14, 21, and 28 d except weekends. The daily dose of cadmium corresponded to 10 mg Cd/kg diet. Body
weight gains and organ to body ratios were recorded weekly during the 28-d feeding period. Animals were killed by immersion in gaseous carbon dioxide 7, 14, 21, and 28 d after dosing.

The blood, duodenum, liver, kidneys, spleen, heart, testicles, brain, and muscles were removed for weighing and radioactivity measurements. Radiocadmium in homogenised organs (1 g samples or whole organs) was measured by a Wizard 1480 automatic scintillation counter (Perkin-Elmer).

Data were analysed statistically with the use of Student’s t-test at P<0.05.

Table 1
Total body weights (g) and gains (%)

<table>
<thead>
<tr>
<th>Time of exposure</th>
<th>Non-treated</th>
<th>Cd</th>
<th>Mg plus Cd</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>255 ± 10</td>
<td>253 ± 23</td>
<td>230 ± 30</td>
</tr>
<tr>
<td>1 week</td>
<td>326 ± 33 (29.1%)</td>
<td>272 ± 36 (7.5%)*</td>
<td>300 ± 37 (30%)</td>
</tr>
<tr>
<td>2 weeks</td>
<td>372 ± 35 (46.3%)</td>
<td>298 ± 32 (17.8%)*</td>
<td>337 ± 36 (47%)</td>
</tr>
<tr>
<td>3 weeks</td>
<td>461 ± 46 (80.9%)</td>
<td>321 ± 32 (26.9%)*</td>
<td>376 ± 41 (63%)*</td>
</tr>
<tr>
<td>4 weeks</td>
<td>487 ± 45 (92.2%)</td>
<td>351 ± 42 (38.7%)*</td>
<td>399 ± 40 (73%)*</td>
</tr>
</tbody>
</table>

Cd – rats exposed to cadmium; Mg plus Cd – rats exposed to magnesium and cadmium; * – P<0.05 between the non-treated and Cd, or Mg plus Cd rats

Table 2
Content of cadmium 109 (% dose) in organs

7 d exposure

<table>
<thead>
<tr>
<th>Organ</th>
<th>Duodenum^a</th>
<th>Kidneys^b</th>
<th>Liver^b</th>
<th>Heart^b</th>
<th>Spleen^b</th>
<th>Testicles^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cd</td>
<td>1.48 ± 0.016</td>
<td>0.082 ± 0.02</td>
<td>1.010 ± 0.170</td>
<td>0.002 ± 0.001</td>
<td>0.005 ± 0.002</td>
<td>0.005 ± 0.002</td>
</tr>
<tr>
<td>Mg plus Cd</td>
<td>0.065 ± 0.009*</td>
<td>0.045 ± 0.09*</td>
<td>0.495 ± 0.071*</td>
<td>0.002 ± 0.001</td>
<td>0.002 ± 0.001</td>
<td>0.002 ± 0.001*</td>
</tr>
</tbody>
</table>

^a – 1 mL or 1 g.
^b – whole organ
* – P<0.05 between Cd and Mg plus Cd rats

14 d exposure

<table>
<thead>
<tr>
<th>Organ</th>
<th>Duodenum^a</th>
<th>Kidneys^b</th>
<th>Liver^b</th>
<th>Heart^b</th>
<th>Spleen^b</th>
<th>Testicles^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cd</td>
<td>0.091 ± 0.016</td>
<td>0.053 ± 0.007</td>
<td>0.521 ± 0.122</td>
<td>0.002 ± 0.001</td>
<td>0.003 ± 0.001</td>
<td>0.003 ± 0.001</td>
</tr>
<tr>
<td>Mg plus Cd</td>
<td>0.071 ± 0.017</td>
<td>0.039 ± 0.0004*</td>
<td>0.445 ± 0.158</td>
<td>traces</td>
<td>traces</td>
<td>0.003 ± 0.001</td>
</tr>
</tbody>
</table>

21 d exposure

<table>
<thead>
<tr>
<th>Organ</th>
<th>Duodenum^a</th>
<th>Kidneys^b</th>
<th>Liver^b</th>
<th>Heart^b</th>
<th>Spleen^b</th>
<th>Testicles^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cd</td>
<td>0.082 ± 0.008</td>
<td>0.061 ± 0.007</td>
<td>0.492 ± 0.070</td>
<td>0.002 ± 0.001</td>
<td>0.003 ± 0.001</td>
<td>0.003 ± 0.001</td>
</tr>
<tr>
<td>Mg plus Cd</td>
<td>0.057 ± 0.016</td>
<td>0.036 ± 0.008*</td>
<td>0.369 ± 0.095</td>
<td>traces</td>
<td>traces</td>
<td>0.003 ± 0.001</td>
</tr>
</tbody>
</table>

28 d exposure

<table>
<thead>
<tr>
<th>Organ</th>
<th>Duodenum^a</th>
<th>Kidneys^b</th>
<th>Liver^b</th>
<th>Heart^b</th>
<th>Spleen^b</th>
<th>Testicles^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cd</td>
<td>0.045 ± 0.006</td>
<td>0.046 ± 0.009</td>
<td>0.316 ± 0.085</td>
<td>0.002 ± 0.001</td>
<td>0.002 ± 0.001</td>
<td>0.003 ± 0.001</td>
</tr>
<tr>
<td>Mg plus Cd</td>
<td>0.043 ± 0.012</td>
<td>0.044 ± 0.007</td>
<td>0.252 ± 0.063</td>
<td>traces</td>
<td>traces</td>
<td>traces</td>
</tr>
</tbody>
</table>
Results

All rats demonstrated similar feed and water intake (not shown) although visibly higher water consumption was observed in the animals treated with cadmium or cadmium and magnesium. Table 1 reveals that rats treated with cadmium or magnesium plus cadmium (group III) had significantly lower body gains as compared to those of the non-treated rats at all stages of the experiment except for 1 and 2 weeks in group III. Rats in this group revealed a little higher body gains as those treated only with cadmium but the differences were not statistically significant.

Table 2 presents the organ to body ratio was similar in all rats tested (data not shown).

The highest content of cadmium 109 was found in the liver and kidneys. The content of the element expressed as the percentage of dose decreased steadily with time in all organs examined. Magnesium supplementation reduced renal, hepatic, duodenal, and testicular cadmium concentration after a 7 d exposure. However, after 14 and 21 d exposure, significant decreases in cadmium 109 content were noted only in the kidneys of rats supplemented with magnesium although a visible but not statistically significant decrease in the cadmium content was found in all tested organs. No significant differences between the two groups tested were found after a 28-d exposure to magnesium and cadmium.

Discussion

The mechanism of gastrointestinal cadmium absorption is not entirely understood as no specific uptake for non-essential toxic metals has been reported (13, 16). However, experimental evidence shows that cadmium absorption and its toxic action may be altered if the nutritional status of trace elements including zinc, copper, iron, or calcium is high (2, 5, 8, 12, 17). On the other hand, marginal deficiencies of these trace elements may enhance the absorption and organ accumulation and retention of cadmium (5, 12, 16).

This experiment demonstrated the influence of magnesium supplements on cadmium retention in selected organs of rats. The results indicated that magnesium reduced duodenal, renal, hepatic, and testicular cadmium retention. The decreases were seen especially after a 7 d exposure and then ceased with time and after 28 d of co-administration no significant differences were noted. This finding corresponds with the data indicating that magnesium pretreatment lowered significantly cadmium content in several organs of mice and rabbits exposed to subacute doses of this metal (3, 13). The authors postulated that lowered cadmium content in organs examined could be a result of magnesium influence on cadmium transport from intestinal lumen to portal blood or that magnesium may modify the organ distribution pattern of cadmium. However, the data presented in the report do not support the view that magnesium may affect redistribution of cadmium among organs because the content of cadmium in the organs not only active in the metabolism of cadmium was reduced within the whole experimental period. Thus, it seems more possible that magnesium co-administration with cadmium may influence cadmium absorption. These findings may be confirmed by the observation of Goytaiin and Quamme (7), who concluded that cadmium uptake in human osteoblast cells occurs, at least partly, through calcium and magnesium inhibitable channel of the TRPM family.

Lack of significant reductions in organ cadmium retention following a 28 d exposure found in the study seems a bit surprising. This finding indicates that beneficial effects of magnesium on organ accumulation of cadmium appeared only within few weeks of cadmium and magnesium administration. The observation may be to some extent consistent with the report by Boujelben et al. (1) who demonstrated that protective effects of magnesium on cadmium induced lipid peroxidation in the testicles ceased 5 d and 10 d after cadmium and magnesium treatment.

To conclude, it is worth to suggest that a beneficial action of magnesium on cadmium accumulation is time dependant and ceased with the time of the two metal administrations. However, magnesium supplements may contribute to the therapy of cadmium intoxication and may suggest further research on magnesium and cadmium interaction.

References