Response to Comment on: Padmavathi et al. (2010) Chronic Maternal Dietary Chromium Restriction Modulates Visceral Adiposity: Probable Underlying Mechanisms. Diabetes;59:98–104

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We thank Vincent and Rasco (1) for showing interest in our work. In this present study (2), diets were prepared as per AIN 93G formulations, which use chromium (Cr) at a level of 1 mg/kg diet as recommended earlier for rodents (3). Despite this, the finding that the control diet had 1.56 mg Cr/kg diet suggests that this could be due to contributions from other dietary ingredients. This is corroborated by our observation that Cr-restricted diet, in which Cr salt was deleted, had 0.51 mg Cr/kg diet. As indicated by Vincent and Rasco, if we assume that Cr requirements of humans and rats are comparable, it is true that both these diets provide more than adequate Cr to rats.

Plasma Cr levels in mothers (prepregnant) and offspring (at different postnatal ages) in our study ranged between 1 and 3 ng/ml in those fed the control diet and between 0.65 and 1.3 ng/ml in the restricted diet–fed rats. Considering that *1*) literature on plasma Cr levels in rats is scanty, *2*) the control diet in our study had recommended levels of Cr, and *3*) the rats fed Cr-restricted diets had significantly lower plasma Cr levels than controls, we consider the Cr-restricted diet–fed rats to have insufficiency if not deficiency. Thus, our observations on plasma Cr levels in rats, despite abundant dietary Cr content, may suggest that Cr requirements and/or absorption/utilization of dietary Cr may be different for humans and rats.

Unlike Striffler et al. (4,5), the control and restricted diets in our study had identical composition but for their Cr content. Also, diet intake was in general comparable between control and Cr-restricted rats. Thus, in our study Cr-restricted rats were not subject to any additional stress (than Cr restriction) compared with control subjects. Therefore, we feel that the differences observed between the control and Cr-restricted rats could be due to the Cr restriction produced in them. It was interesting that changes in body fat percentage and visceral adiposity in CrR offspring were not mitigated by rehabilitation, which, however, restored their plasma Cr levels. They indicate that body composition changes in offspring may not be due to low Cr levels per se but probably are due to the effects of maternal Cr restriction on the programming of the body composition of the fetus.

In view of the facts and observations mentioned above, we infer that chronic maternal dietary Cr restriction induced irreversible changes in the body fat percentage and visceral adiposity of the rat offspring.

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