

# Abstract

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## Marginal Biotin Deficiency is Common in Normal Human Pregnancy and Is Highly Teratogenic in Mice

Mock DM.

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**BACKGROUND:** In studies of marginal biotin deficiency induced experimentally in adults, increased urinary excretion of 3-hydroxyisovaleric acid (3HIA), which likely reflects decreased activity of the biotin-dependent enzyme beta-methylcrotonyl-CoA carboxylase, and decreased activity of the biotin-dependent enzyme propionyl-CoA carboxylase (PCC) in peripheral blood lymphocytes have been validated as indices of biotin status.

**OBJECTIVE:** About half of pregnant women excrete increased amounts of urinary 3HIA. However, interpretation of urinary 3HIA excretion rates is problematic, because renal function is altered by pregnancy per se.

**RESULTS:** In a recent pilot study, activity of PCC in peripheral blood lymphocytes was decreased in 18 of 22 pregnant women. In 4 of 4 pregnant women with decreased PCC activity, biotin supplementation caused increased PCC activity by a mean of 95%. Taken together, such studies provide evidence that a substantial proportion of pregnant women are marginally biotin deficient. In mice, degrees of biotin deficiency that are metabolically similar to those seen in pregnant women are very teratogenic. Moreover, in mice, a marginal degree of biotin deficiency in the dam causes a much more severe degree of deficiency in the fetus.

**CONCLUSION:** These observations further raise concerns that biotin deficiency does occur and does cause human birth defects.

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