

# Abstract

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## Drugs that promote renal excretion of riboflavin.

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**BACKGROUND:** Enhanced urinary excretion of vitamins induced by drugs is a major factor in development of vitamin deficiencies. In addition to increasing urinary excretion, drugs can induce vitamin deficiencies by altering their intestinal absorption, transport, storage, and/or metabolic conversions. Aside from drugs, other factors known to influence urinary excretion of vitamins include the level of the vitamin in the diet, the degree of tissue saturation of the vitamin, and the extent of protein binding of the vitamin.

**DISCUSSION AND FINDINGS:** Alterations in various aspects of flavin metabolism have been observed following administration of certain drugs, namely, antimalarial, antimicrobial, anticancer, and some tricyclic antidepressant and antipsychotic agents. Of these drugs, boric acid and its derivatives as well as the antipsychotic agent, chlorpromazine, have been shown to promote riboflavinuria in both animals and man. Boric acid complexes with the polyhydroxyl ribitol side chain of riboflavin and greatly increases its water solubility. Individuals who have accidentally consumed boric acid or one of its derivatives excrete high levels of riboflavin within the first 24 to 48 hours following ingestion. The phenothiazine ring of chlorpromazine and the isoalloxazine ring of riboflavin have a number of structural features in common and have been shown to form a molecular complex in vitro. In animals treated for a 3- and 7-week period with chlorpromazine, urinary levels of riboflavin are twice that of pair-fed, saline-treated animals.

**CONCLUSIONS:** Recent studies have extended these findings to humans. The administration of certain agents, either therapeutic or toxic, which enhance urinary riboflavin excretion may be of particular concern for high-risk patients who are already nutritionally compromised because of illness or disease.

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