

RIBOFLAVIN STATUS OF ADOLESCENT SOUTHERN CHINESE:  
II. RIBOFLAVIN SATURATION STUDIES

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Studies in Southern China have indicated that adolescent children are at risk to riboflavin deficiency. Riboflavin saturation studies were done on 36 high-school boys in Canton, 14-19 years old, with clinical evidence of riboflavin deficiency. Riboflavin intake was calculated to be about 0.45 mg per person per day (Lo 1982). Subjects were divided randomly into 4 groups. A 1-h urine sample was collected at 0800 hours. Immediately after collection, the boys were given a 2 mg riboflavin loading orally, and hourly urine samples were collected for the next 4 h. Each boy in Group A was then given orally a daily riboflavin supplement of 0.5 mg, Group B 1.0 mg, Group C 1.5 mg. Group D received no riboflavin supplement, but instead an ointment treatment for scrotal dermatitis. The boys were given ordinary school food and participated in usual school activities. At the end of a 14-d period, a 1-h urine sample was again collected, another 2 mg loading test was performed, and hourly urine samples collected for the following 4 h.

The results for 1-h (0700 - 0800 hours) urine riboflavin excretion agreed well with the dietary survey, with very low values before the supplement (1.4 µg riboflavin per h). After 14 d of supplementation, the riboflavin content rose for Group A to 3.4 (P < 0.05), Group B 8.7 (P < 0.01) and Group C 11.4 µg/h (P < 0.01). No change was observed for Group D.

The 4-h excretion of riboflavin after a 2 mg load averaged 8.6% of the load for Groups A, B, C and D prior to supplementation. After supplementation, for Group A it was 11.5% (P < 0.05), for Group B 15.6% (P < 0.1), for Group C 17.8% (P < 0.01) and for Group D 8.4% (P < 0.05).

Clinically, after 17 d supplementation, scrotal dermatitis disappeared in the 1.0 and 1.5 mg, but not in the 0.5 mg riboflavin-supplemented groups.

Four-hourly riboflavin excretion responses and clinical responses to riboflavin supplementation would indicate that the common average intake of 0.45 mg/d should be increased to not less than 1.0 mg/d to avoid clinical riboflavin deficiency.

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