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Effect of increasing cow's milk consumption on riboflavin intake and plasma riboflavin measures in women of child-bearing age

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Milk and dairy products are excellent sources of riboflavin (vitamin B2), estimated to contribute 25–27% to average daily intakes in adults⁽¹⁾. There are concerns regarding low riboflavin intakes in young women in the UK⁽²⁾. The effect of milk consumption on riboflavin status has not been previously investigated, primarily because riboflavin biomarkers are rarely measured in human studies and the gold standard EGRac (erythrocyte glutathione reductase activation co-efficient) assay demands laborious pre-analysis sample preparation. Plasma riboflavin has been proposed as an alternative and more accessible measure of riboflavin status⁽³⁾. The aim of this study therefore was to investigate the effect of increasing cow's milk consumption on riboflavin intakes and plasma riboflavin concentrations in young women.

A convenience sample of data were analysed from a randomised controlled trial where healthy females were assigned to one of two groups, to consume either 3L semi-skimmed milk weekly (approximately 430mls/d milk; intervention, n = 39) or continue with their habitual milk intake (control, n = 39) for 12 weeks4. Retrospective power calculations indicated sufficient sample size to investigate the current study aim. At weeks 0 and 12, participants completed a 24hr dietary recall and provided a blood sample with plasma analysed for riboflavin and its coenzyme forms, flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) by isotopedilution liquid chromatography-tandem mass spectrometry. One-way analysis of covariance was used to compare between-group effects adjusting for baseline measures. Chi-square test was used to compare riboflavin intakes between groups when categorised as below/above the RNI (</ $\geq 1.6 \text{mg/d}$).

Fifty-four completed the intervention with a mean \pm SD age of 28.1 \pm 7.6. The change in riboflavin intake from week 0 to 12 was significantly greater in the intervention (1.3 to 1.7mg/d) compared to the control (1.6 to 1.1mg/d) (F (1) =5.12, p = 0.02). Following the intervention, the proportion of those above the RNI for riboflavin intake was significantly greater within the intervention compared to the control (57% vs 25%; x2 = 7.6, p = 0.006). The change in plasma riboflavin from week 0 to 12 did not significantly differ between the intervention (median (IQR) 12 (8.5–16.6) to 13.4 (9.15–22.1) nmol/l) and control (12 (9.15–17.05) to 12.3 (6.9–10.9) nmol/l) (F (1) = 0.18, p = 0.67), nor was there any effect of the intervention on plasma FMN or FAD. There was no significant correlation between riboflavin intake and plasma measures in either group.

Increasing cow's milk consumption resulted in a significant increase in riboflavin intake and an increase in the proportion of women meeting the RNI compared to the control. No changes, however, were observed in plasma riboflavin as a result of the intervention. Further studies should investigate the association between dietary riboflavin intake and measures of riboflavin status in population-based cohorts.

References

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