

## **Exploring the mechanisms behind the effects of chronic bovine colostrum supplementation on risk of upper respiratory tract infection**

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### ABSTRACT

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Chronic bovine colostrum (COL) supplementation has been shown to reduce the incidence of upper respiratory tract infection (URTI) but the underlying mechanism remains unclear. Fifty seven highly active males (aged 18-62) were randomised to daily supplementation of 20g of COL (N = 28) or an isoenergetic/isomacronutrient placebo (PLA) (N = 29) for 12 weeks under double-blind procedures. Daily illness and weekly activity logs were completed. Resting blood samples collected at baseline and at 12 weeks were analysed for total/differential leukocyte counts and in-vitro stimulated neutrophil oxidative burst. Metabolomic profiling of serum samples at these timepoints was performed using fourier transform infrared spectroscopy and direct-infusion – electrospray-ionisation – mass spectroscopy. Secretory IgA and bacterial load were measured within saliva samples obtained at baseline, 4, 8 and 12 weeks using enzyme-linked immunosorbent assay and real-time, quantitative PCR methods respectively. The mean number of URTI episodes was significantly higher ( $P < 0.05$ ) in the PLA group ( $0.8 \pm 0.7$ ) than in COL group ( $0.5 \pm 0.7$ ). There were a significantly greater proportion of days with URTI symptoms in the PLA group ( $P < 0.001$ ) compared to COL group. No statistical differences were found between leukocyte counts, neutrophil oxidative burst or salivary IgA across time or between groups ( $P > 0.05$ ). However, Discriminant Function Analyses (DFA) of outputs received from the metabolomic profiling showed a significant difference between COL and PLA at 12 weeks ( $P < 0.05$ ). Interrogation of the loading vectors in the DFA models indicated 10 metabolites which contributed most to the difference between COL and PLA which included, but were not limited to, decreased noradrenaline, uric acid, and dehydroascorbic acid with COL ( $P < 0.05$ ). There was a general increase in bacterial load within groups, over the 12 week period, but this increase was significantly lower ( $P < 0.05$ ) with COL. These findings support previously reported evidence regarding the effects of COL on URTI incidence. Although the lack of effect on immune measures does not support some of the previously suggested mechanisms (e.g. improved resting salivary IgA concentration) some further sample analysis is currently outstanding. Also, the effects on the responses to exercise (which were not assessed in this study) may explain the lower URTI incidence and bacterial load with COL in these athletes. The differences observed in metabolomics profiling highlights novel potential mechanisms behind the effect of COL on infection risk, but outstanding analyses mentioned above and further study is required to verify this.