3rd Immunonutrition Workshop, 21-24 October 2009, Girona, Spain

## No effect of vitamin D supplementation on serum fibrinogen concentrations in adults aged $\geq 64$ years

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High serum concentration of the acute phase protein fibrinogen is associated with tissue inflammation and is an independent risk factor for cardiovascular disease (CVD). Low vitamin D status is associated with an increased risk of CVD and the active form of the vitamin, 1,25-dihydroxyvitamin D<sub>3</sub>, is a potent immunomodulator. Furthermore, vitamin D supplementation has been shown to reduce serum concentrations of inflammatory markers such as C-reactive protein in vitamin D deficient individuals. The aim of this study was to assess the effect of vitamin D supplementation on serum fibrinogen concentrations in a group of apparently healthy adults aged  $\geq 64$  years recruited in Cork and Coleraine.

A total of 202 individuals (males, n = 81; females, n = 121) were randomly assigned to receive either 5, 10 or 15 µg vitamin D<sub>3</sub>/d or placebo for 22 weeks. Serum vitamin D status (25-hydroyvitamin D (25(OH)D)) and fibrinogen concentrations were measured at baseline and post intervention using commercially available ELISA kits.

Vitamin D status did not significantly correlate with serum fibrinogen concentrations at baseline or post intervention. One-way analysis of covariance (adjusted for age, sex, centre, body mass index and baseline concentrations) revealed that while vitamin D supplementation significantly increased vitamin D status, it did not alter fibrinogen concentrations.

|            |         | Treatment group ( $\mu g$ vitamin $D_3/d^*$ ) |       |                             |       |                              |       |                               |          |
|------------|---------|-----------------------------------------------|-------|-----------------------------|-------|------------------------------|-------|-------------------------------|----------|
|            | Plac    | Placebo $(n = 54)$                            |       | $5 \mu g/d (n=48)$          |       | $10 \mu g/d (n = 52)$        |       | 15 $\mu$ g/d ( <i>n</i> = 48) |          |
| 25(OH)D (  | nmol/l) |                                               |       |                             |       |                              |       |                               |          |
| Pre        | 59.07   | (43.37, 78.64)                                | 51.84 | (40.28, 71.34)              | 55.53 | (43.00, 72.26)               | 55.09 | (39.39, 70.82)                |          |
| Post       | 42.59   | (27.82, 55.86) <sup>a</sup>                   | 53.19 | (45.57, 68.73) <sup>b</sup> | 70.32 | (57.98, 81.81) <sup>cd</sup> | 73.86 | (61.87, 90.20) <sup>cd</sup>  | < 0.0001 |
| Fibrinogen | (g/l)   |                                               |       |                             |       |                              |       |                               |          |
| Pre        | 1.86    | (1.25, 2.91)                                  | 2.12  | (1.44, 3.54)                | 1.78  | (1.25, 2.85)                 | 2.22  | (1.31, 3.08)                  |          |
| Post       | 2.19    | (1.60, 3.07)                                  | 2.10  | (1.47, 3.79)                | 2.02  | (1.40, 2.81)                 | 2.18  | (1.74, 3.20)                  | 0.889    |

25(OH)D, 25-hydroxyvitamin D

\* Values are median (IQR).

† Effect of treatment assessed on log transformed data by ANCOVA. Different superscript letters denote significant differences between treatment groups (ANOVA).

In conclusion, vitamin D supplementation had a significant dose-response effect on vitamin D status, but did not affect serum concentrations of the inflammatory marker fibrinogen in healthy older adults. These findings concur with previous research in vitamin D deficient adults. However, it has been suggested that 25(OH)D concentrations >100 nmol/l may be required for modulation of immune responses; concentrations higher than those observed in the current study, even after vitamin D supplementation.

This work was supported by the UK Food Standards Agency.