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### Comparison of dairy and non-dairy sources of calcium on thermogenesis and substrate oxidation in humans

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**Background** – Calcium homeostasis is important to a large number of physiological processes in the body. Recently, calcium has been linked to the regulation of adiposity. However, it remains to be confirmed whether this occurs through modulation of energy expenditure or energy intake.

**Objective** – To compare the acute effects of different sources of calcium on energy expenditure and substrate utilisation.

**Design** – 8 subjects (mean  $\pm$  SEM, age  $53.8 \pm 2.3$  yr, weight  $92.4 \pm 7.0$  kg, and BMI  $32.5 \pm 1.0$  kg/m<sup>2</sup>) participated in a randomised, single blind, 3-way crossover design. Subjects were provided a low calcium (dairy)- low vitamin D meal (LD), a high calcium (dairy)- high vitamin D meal (HD), and a high calcium (non-dairy, calcium citrate)- low vitamin D meal (HC). The energy, macronutrient content and volume of meals were matched. The LD, HD and HC diets contained 175 mg, 531 mg and 575 mg of calcium, and 40 IU, 364 IU and 45 IU of vitamin D, respectively. Diet induced thermogenesis (DIT), fat oxidation (FOX) and carbohydrate oxidation (COX) were measured using the Deltatrac II (Datex, Finland), that is based on indirect calorimetry. Results were analysed as change from fasting (resting) values. Statistical analysis employed a repeated measures ANOVA with a LSD post-hoc procedure, when appropriate.

**Outcomes** – Change in glucose concentrations were not different between meals, when examined over 2h and over the entire postprandial period. Change in respiratory quotient ( $\Delta$ RQ) was significantly different between meals ( $P < 0.05$ ) with a lower rise following the high calcium meals (LC  $0.3 \pm 0.1$ , HD  $-0.013 \pm 0.1$ , HC  $-0.025 \pm 0.11$ ). Consequently,  $\Delta$ FOX was significantly higher following the high calcium meals (LC  $-6.5 \pm 2.2$ , HD  $3.3 \pm 2.5$ , HC  $2.93 \pm 2.34$  g.6h,  $P < 0.01$ ), and  $\Delta$ COX was significantly lower (LD  $34.1 \pm 7.7$ , HD  $15.2 \pm 7.1$ , HC  $13.6 \pm 7.5$  g.6h,  $P < 0.05$ ). There were no statistical differences in DIT between meals, though a trend for a 10 % higher DIT was seen on the HD and HC meals (LD  $6.5 \pm 1.1$  %, HD  $7.0 \pm 0.8$  %, HC  $7.2 \pm 1.4$  %).

**Conclusions** – Calcium acutely stimulated postprandial fat oxidation and suppressed carbohydrate oxidation. Both dairy and non-dairy calcium meals were equipotent in their effects when examined over the 6 h postprandial period.

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### Folic acid deficiency is genotoxic and increases sensitivity to chromosome damage by gamma-radiation

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**Background** - Folic acid deficiency can alter DNA-methylation, lead to excessive uracil incorporation and an increased level of DNA strand breaks. Therefore, it was hypothesized that folic acid is an important micronutrient in the prevention of both spontaneous and radiation induced chromosome damage.

**Objective** - To determine the impact of folic acid deficiency on spontaneous and radiation induced chromosome damage and chromosome 21 aneuploidy.

**Design** - Chromosome damage and aneuploidy were determined using the cytokinesis-block micronucleus assay in long term WIL2-NS cultures in cell culture medium with four different folic acid concentrations (0.2 nM, 2 nM, 20 nM and 200 nM). WIL2-NS cells were exposed to 0 Gy or 1.5 Gy of gamma-radiation.

**Outcomes** - Micronucleus frequency increased significantly (55.5%) with decreasing folic acid concentration ( $P < 0.0001$ ). Micronucleus frequency and nucleoplasmic bridge frequency showed a significant difference of 46.5% and 50.1%, respectively, between the control (0 Gy) and irradiated (1.5 Gy) group ( $P < 0.05$ ). Folic acid deficiency caused an increase of 51.7% in micronucleus frequency ( $P < 0.0001$ ) and of 7.1% nucleoplasmic bridge frequency ( $P = 0.0280$ ) in irradiated cultures when compared to irradiated cultures that were folic acid replete. Folic acid deficiency and gamma-radiation interact significantly with respect to micronucleus frequency (Two-way ANOVA,  $P < 0.0001$ ). Apoptosis and necrosis were increased by folic acid deficiency but were not significantly altered by exposure to ionising radiation. Chromosome 21 aneuploidy was significantly increased ( $P < 0.05$ ) by folic acid deficiency but not by ionising radiation and there was no significant interaction between those two factors.

**Conclusions** - Folic acid deficiency induces chromosome 21 aneuploidy by non-disjunction as well as chromosome breaks and chromosome rearrangements and it interacts significantly with ionising radiation in inducing chromosome damage that leads to the formation of micronuclei (eg chromosome breaks and/or chromosome loss).