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Intestinal gene expression levels in an epigenetically programmed mouse model of the metabolic syndrome

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Background – The prevalence of metabolic disorders has reached epidemic levels across the globe. Despite the multitude of studies addressing the development of metabolic abnormalities, the picture of aetiology remains complicated.
Aim – To link small intestine gene expression changes in an epigenetically programmed murine model of the metabolic syndrome to the development of the metabolic syndrome (MetS).
Experiment – A murine model of the metabolic syndrome was epigenetically programmed to develop the MetS via maternal over-nutrition[(1)]. Messenger RNA expression levels in the small intestines of programmed mice were then compared to control mice by microarray analysis and GeneGo Metacore™ software analysis.
Results – The small intestines of programmed mice showed a 5–25 fold upregulation in defensin expression, inflammation and an altered redox status, relative to control mice. Genes responsible for carbohydrate digestion, absorption, sensing and metabolism were also altered 3–40 fold in programmed mice. Gut peptide and related receptor gene expression levels were unchanged.
Interpretation – The upregulated defensins may be modulating the small intestinal microflora composition, resulting in bacterially-driven 'metabolic endotoxaemia', increased inflammatory tone and oxidative stress. Post-prandial glucose levels may also be elevated, and nutrient sensing attenuated in this model of the MetS. Further research to confirm the proposed model is required.

1. Samuelsson AM, Matthews PA Argenton M *et al.* (2008) *Hypertension* **51**(2), 383–92.