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Zinc deficiency-induced changes in the expression of rat aorta proteins using proteomics

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Damage to the vascular endothelium may initiate atherogenesis, and maintaining the integrity of endothelial cells is therefore key to preventing atherosclerosis. Deficiencies of nutrients such as folate and selenium influence vascular endothelial cell integrity and are therefore thought to be risk factors for atherosclerosis. However, the role of zinc in protecting vascular tissue has not been seriously considered, largely because zinc status cannot be reliably measured and few epidemiological studies relating status with disease have therefore been made. Our approach has been to utilize proteomic techniques to try and detect which vascular metabolic or signalling pathways are affected by physiologically relevant zinc deficiency. Three week old rats were fed semi-synthetic diets containing adequate (35 mg/kg) or marginally deficient (3 mg/kg) levels of zinc for 43 days, and animals in a third group were pair-fed with deficient rats. Two-dimensional gel electrophoresis of aorta tissue using a pH gradient of 3-10 and an acrylamide gradient of 8-16% revealed 1697 proteins, of which 18 showed levels significantly ($p<0.01$) lower in zinc deficiency as compared to adequacy. Although weight gain and food intake of rats in the pair-fed group were not different from the adequate group, aorta proteins affected by both zinc deficiency and pair-feeding were not included in the present analysis. Tryptic digestion and mass spectrometry followed by peptide mass fingerprinting using the Mascot search engine (http://www.matrixscience.com/) and the NCBInr database, revealed that many of the zinc-regulated proteins were related to carbohydrate metabolism. Further work is in progress to establish whether the proteins affected are in smooth muscle and/or endothelial cells.