

## ICCN Poster Presentations

### Molecular nutrition for the clinician

#### **Niacin metabolism and Parkinson's disease**

T Fukushima\*, M Ohta, K Tanaka, S-Y Kaneko, T Maeda and A Sasaki

*Department of Hygiene & Preventive Medicine, Fukushima Medical University School of Medicine,  
1 Hikarigaoka, Fukushima 960-1295, Japan*

Previous epidemiological results show the importance of niacin in relation to the cause of Parkinson's disease, that is the nutritional condition which causes pellagra, niacin deficiency, might protect people from Parkinson's disease. Because maize (*Zea mays*) contains niacytin which the human being cannot use as niacin, and because maize contains low tryptophan, and abundant leucine which inhibits quinolinate phosphoribosyl transferase, the key enzyme of converting from tryptophan to NAD, niacin deficiency is observed in the people who obtain most of their energy from maize. Correlation coefficients among the prevalence rate of Parkinson's disease, maize yield, niacin intake and selenium intake by each province in China were analyzed. Positive correlation was seen between selenium intake and niacin intake. Niacin deficiency also could be seen in the Keshan disease prevalent area. Negative association was seen between maize production and niacin intake and between maize production and prevalence rate of Parkinson's disease. Retrospective study of preventive effect of maize on mortality from Parkinson's disease in Japan was also demonstrated. Absorbed niacin is used for synthesizing of NAD in the body, and in the metabolic process, NAD releases nicotinamide by poly (ADP-ribose)ylation which activation has been reported to mediate MPTP-induced Parkinson's disease. Nicotinamide N-methyltransferase (EC2.1.1.1) activity was assayed with cytosolic fraction of rat brain, and nicotinamide could be methylated to 1-methylnicotinamide (MNA) via this enzyme in the brain. The deficiency of mitochondrial NADH: ubiquinone oxidoreductase (complex I) activity is believed to be a critical factor in the development of Parkinson's disease. MNA destroyed several subunits of cerebral complex I, and it was suggested that MNA was concerned in the pathogenesis of Parkinson's disease. From these results, niacin is expected to be the causal substance of Parkinson's disease through following process, NAD produced from niacin releases nicotinamide via poly (ADP-ribose)ylation which is activated by hydroxyl radical. Released excess nicotinamide is methylated to MNA in cytoplasm, and superoxides formed by MNA via complex I destroys complex I subunits directly or indirectly via mitochondrial DNA damage, and stimulates poly (ADP-ribose)ylation. Any hereditary or environmental factors may cause acceleration of this rotation and consequently cause neuronal death.