SMW Young Investigator's Award 2003: Clustering of cardiovascular risk factors mimicking the human metabolic syndrome X in eNOS null mice

The publisher and editorial staff of the Swiss Medical Weekly have pleasure in announcing the winner of the Swiss Medical Weekly Young Investigator's Award 2003.

Dr. Stéphane Cook of the Department of Internal Medicine and the Botnar Centre for Clinical Research, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland is the Award winner for the following publication:

Cook S, Hugli O, Egli M, Vollenweider P, Burcelin R, Nicod P, Thorens B, Scherrer U. Clustering of cardiovascular risk factors mimicking the human metabolic syndrome X in eNOS null mice. Swiss Med Wkly 2003;123:360–3.

The authors present the new finding that eNOS$^{-/-}$ mice have elevated plasma leptin concentrations which were not associated with increased body weight. They interpret this as a possible counter-regulatory mechanism opposing the insulin resistance in these animals. It appears possible that the elevated leptin levels merely reflect hyperinsulinaemia, since insulin is known to stimulate leptin production. Consistent with this interpretation, it is postulated that hyperleptinaemia associated with the human metabolic syndrome may be related to insulin.

In addition to blood pressure and insulin sensitivity, the authors measured the plasma concentration of leptin, insulin, cholesterol, triglycerides, free fatty acids, fibrinogen and uric acid in 10–12-week-old eNOS$^{-/-}$ and wild type mice. They also assessed glucose tolerance under basal conditions and following metabolic stress with a high fat diet. They found, as expected, eNOS$^{-/-}$ mice to be hypertensive and insulin resistant. Moreover, cholesterol, triglyceride and free fatty acid plasma concentrations were elevated in eNOS$^{-/-}$ mice.

The findings show that a single gene defect, eNOS deficiency, causes an important clustering of cardiovascular risk factors in young mice. The authors speculate that a defect in nitric oxide synthesis may trigger many of the abnormalities constituting the metabolic syndrome in humans, which thereby may represent a new target for pharmacological agents which deliver and/or modulate the bioavailability of endogenously produced nitric oxide.

We warmly congratulate Dr. Cook and her colleagues on a publication of high scientific quality and relevance. The work is interesting and well written, and was a pleasure to read. We also take the opportunity of thanking the authors for a submission of outstanding quality.

For the Editorial Board:
Peter Gehr, Professor of Anatomy

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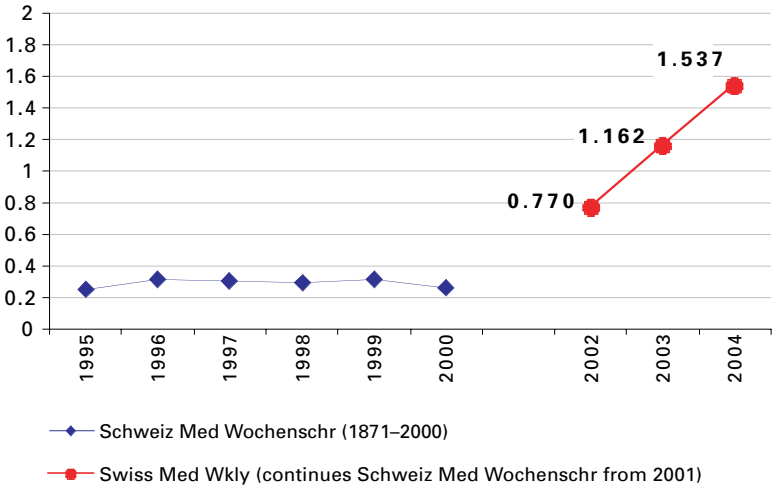

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