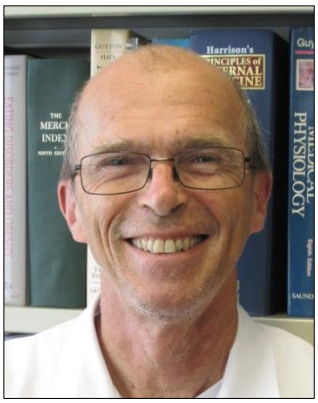

Luc Tappy, MD

Luc Tappy was born in Lausanne in 1957. He graduated from medical school and obtained his MD degree at Lausanne University in 1981. He was then trained in the Department of internal medicine, Centre Hospitalier Universitaire Vaudois, and in the Diabetes section, Temple University Hospital, Philadelphia, PA.

Since 1988, he has been a senior researcher at the Institute of Physiology, Lausanne University School of Medicine. His studies focused on nutrition, physical exercise and metabolism in healthy individuals and in various clinical conditions, such as diabetes, obesity, organ transplant patients and critically ill patients. In 2002, he was appointed full professor of physiology at the Department of Physiology of the University of Lausanne, and associate physician at the Division of Endocrinology and Metabolism of the CHUV. He has also been invited professor at the Centre Hospitalier Sart Tilman in Liège, Belgium (1998-2001), and in the Department of Nutrition,at the University of California at Berkeley (1995).

His present research is essentially focused on the environmental factors involved in the present epidemics of obesity and type2 diabetes. Several studies are thus conducted to evaluate the role of dietary sugars (more specifically fructose in carbonated beverages) in the development of obesity and insulin resistance. Several other studies are aimed at assessing and evaluating the role of sport and physical activity in the prevention of metabolic disorders.

Fructose in diabetes: friend or foe?

Fructose is a natural sugar with high sweetening power, which can be metabolized in liver cells without requiring insulin, and which does not increase glycemia to any great extent. It was initially proposed as a sweetener of choice in type 2 diabetes mellitus in the 1980', but this recommendation was rapidly withdrawn due to potential insulin resistance and dyslipidemia.

Insulin resistance plays an important role in the development of cardiovascular and metabolic diseases associated with obesity. More than total body fat, ectopic fat depots in the omentum, liver, muscle, and skeletal/muscle/epicardium are closely associated with insulin resistance and/or cardiac dysfunction. Omental fat volume and intrahepatic fat concentrations are closely interrelated, and constitute the most powerful predictors of insulin resistance.

Whether specific nutrients, such as saturated fat, trans-fat, and sugars are involved in the development of ectopic fat depots independently of excess energy intake remains an important, unsolved question. In healthy humans, intrahepatic fat concentrations increase to a similar extent with excess fat, fructose or glucose, while increasing dietary protein intake partially prevents this effect. Intrahepatic fat concentrations observed in healthy subjects after overfeeding remain largely lower that those observed in obese, insulin resistant subjects with non-alcoholic fatty liver disease. In obese subjects, deposition of ectopic fat in the liver is mainly related to plasma non-esterfied fatty acid concentration. Recent observations further indicate that, in subjects consuming large amounts of sugar-sweetened beverages, intrahepatic fat concentration is related to body fat and omental fat masses, but not to sugar intake. These observations suggest that dysregulation of adipose lipolysis, more than fructose intake, may be a major pathogenic factor in the development of ectopic fat depots and insulin resistance.

Learning objectives:

1. Identify the key steps for metabolism of fructose in humans
2. Understand that fructosae is initially metabolized without insulin and whithout increasing glycemia
3. get acquainted by the characteristics of type-2 diabetes: insulin resistance, ectopic fat deport
4. get acquainted with tracer methodology for measurement of whole body metabolism
5. critically assess the effects of fructose on ectopic lipid deposition and on diabetes control