Abdominal obesity and diabetes

It has been known for about 50 years that different obesity phenotypes do exist. Nonetheless, how abdominal, namely visceral, obesity is burdened by metabolic and cardiovascular diseases has been established only in recent years. The association between abdominal obesity and diabetes, which is well documented, has been mainly explained by the lower insulin sensitivity of subjects with excess visceral fat. However, more recent studies support the hypothesis that several molecules released in greater or lower amount by visceral adipocytes can exert also a detrimental role on beta-cell function. Among these molecules free fatty acids and adipokines should be mentioned. The latter include inflammatory cytokines, such as tumor necrosis factor-alpha and interleukin-6 and hormones synthesized by adipocytes, such as adiponectin, leptin and resistin. Visceral obesity is also associated with an excessive depot of triglycerides and other lipid products (e.g., ceramide) within the key organs of glucose metabolism (liver, skeletal muscle, pancreatic islets). This phenomenon seems to contribute to both insulin resistance and beta-cell dysfunction, favoring abnormalities of glucose homeostasis.