Type 2 diabetes is a chronic disease in which the body does not produce enough insulin, or does not respond to insulin, or both. Type 2 diabetes is extremely common. The Centers for Disease Control and Prevention (CDC) estimates that over 29 million children and adults in the United States have some form of diabetes. That is about 9 percent of the population. The vast majority of these people have type 2 diabetes.

Medically speaking, Type 2 diabetes is characterized by insulin resistance and Beta-cell failure, the latter resulting from reductions in Beta-cell function and/or Beta-cell mass. Together, these contribute to impaired insulin release and the inability to maintain euglycemia without glucose-lowering therapy. A pathological hallmark of the pancreatic islet in type 2 diabetes is islet amyloid deposition. These deposits occur in the majority of patients with diabetes, but have also been reported in a small proportion of subjects who are apparently non-diabetic, and especially in those who are older.

Islet Amyloid Polypeptide (IAPP)

IAPP is a normal product of the Beta-cell that is co-secreted with insulin in response to glucose and nonglucose stimuli. Islet amyloid deposition involves aggregation of the normally soluble IAPP and contributes to the deterioration of Beta-cell function and reduced Beta-cell mass observed in the disease.

Neprilysin

Neprilysin is a zinc metallopeptidase enzyme. Neprilysin is also secreted by mesenchymal stem cells from adipose tissue but only in small amounts from other stem cells such as bone marrow. In the Med Cell Europe laboratory, Neprilysin bound exosomes and free Neprilysin can be achieved in very high amount through the production process. This high quantity coupled with Med Cell Europe’s GMP grade quality ensures the base to successful prevention of diabetes type 2 and Alzheimer disease. Current observational studies in the treatment of these diseases are encouraging.

Degradation of IAPP by Neprilysin

The physiological role of Neprilysin depends on its tissue localization.

Cell Loss and Cell Death by IAPP

Islet amyloid deposition involves aggregation of the normally soluble IAPP and contributes to the deterioration of Beta-cell function and reduced Beta-cell mass observed in the disease. Given that IAPP is co-secreted with insulin, factors such as insulin resistance that increase insulin secretion also result in elevated IAPP secretion. The frequency of Beta-cell death in diabetic subjects was 4.5-fold higher than that for non-diabetics. Increased amyloid severity was significantly associated with Beta-cell death.

Prevention of Diabetes

"Our data supports a potential therapeutic role for neprilysin in preventing type 2 diabetes mellitus."1

"Our data therefore support a role for the accumulation of amyloid in the loss of Beta cells in type 2 diabetes and suggest that interventions aimed at limiting or preventing islet amyloid deposition may have beneficial effects in preserving Beta-cell mass in type 2 diabetes. 4"

Ref.1: Degradation of Islet Amyloid Polypeptide by Neprilysin
H. Guan et al., Diabetologia. 2012 November ; 55(11): 2989–2998

Ref.2: Prevalence and Clinicopathological Characteristics of Islet Amyloid in Chinese Patients With Type 2 Diabetes
Hai-Lu Zhao et al. DIABETES, VOL. 52, NOVEMBER 2003

Ref.3: Neprilysin Impedes Islet Amyloid Formation by Inhibition of Fibril Formation Rather Than Peptide Degradation
Sakeneh Zraika et al.
JOURNAL OF BIOLOGICAL CHEMISTRY, VOLUME 285•NUMBER 24•JUNE 11, 2010

Ref.4: Beta-Cell Loss and Beta-Cell Apoptosis in Human Type 2 Diabetes Are Related to Islet Amyloid Deposition