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Role of influenza virus infection in the etiopathogenesis of diabetes

Principal investigator: Ilaria Capua

This project was ignited by applying knowledge generated from influenza pathogenesis in animals transposing it to possible outcomes in human medicine. Basically, if under natural conditions of exposure influenza viruses can colonize the pancreas (from severely to moderately) in a vast range of animals, we believe that this may also occur in humans and that it may have some consequences which result in some cases in the onset of a diabetic condition.

The aim of our study is to demonstrate that under certain circumstances influenza virus infection can trigger the onset of diabetes. The originality of our approach resides in proposing the hypothesis that influenza virus can do this by directly infecting pancreatic cells, including endocrine cells.

Infact, the association between the onset of diabetes and an influenza like illness is a common clinical finding that had been always explained by the increased insulin resistance due to the presence of infection related inflammatory mediators like TNFa, IL6 or IL1b. Here we propose that, similarly to what occurs in animals, in which the tropism of influenza viruses also includes pancreatic tissue, that influenza virus could damage beta cells and trigger diabetes (i) directly by inducing beta cell death and/or cell secretory disfunction or (ii) by indirectly damaging beta cells by starting a deleterious inflammatory program in pancreatic microenvironment.

These mechanisms could be relevant both at the time of autoimmunity appearance or at the time of clinical onset of T1D, but also in the pathogenesis of T2D. On this direction we have inserted in our workplan multiple in vivo approaches including the virus infection associated both with immunological and metabolic stressor