MAEDICA – a Journal of Clinical Medicine
2022; 17(4): 755-756
https://doi.org/10.26574/maedica.2022.17.4.755

MÆDICA - a Journal of Clinical Medicine
EDITORIAL POINT OF VIEW

Give Me an Islet... and I'm Yours

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What a beautiful song had Joe Dollan once: "Take me and break me and make me an island, I'm yours"!

What a genial discovery was communicated by Paul Langerhans (1), in 1869, about "his" one million or more islets in each pancreas! This discovery mainly explained the metabolism of glucose.

However, these islets are often "broken" (or pushed to latency), which produces a terrible illness: diabetes mellitus (DM) – be it of type I or type II.

Of course, those islets are broken in a different way in type 1 or type 2, but the consequences are similarly hard. However, the specific non-substitution therapy is different for each type.

Type 1 DM (T1D) is an autoimmune disease with a genetic component (2), which accounts for about 5-10% of all diabetes cases. The genetic component is proven by the fact that children whose both parents have T1D are more than 10 times likely to develop the illness than the general population.

The trigger for the immune aggression against the self beta cells are not well understood. Some viruses have been implied, without a clear conclusion (1).

Today, the non-substitution therapy in T1D has many very interesting directions which develop, none with final priority till now.

Of course, substitution with insulin is a valid therapy. In these days, insulin pumps and the sensors serving them are so sophisticated that they were compared with an artificial pancreas. Of course – for the beta-cells production substitution (2).

The main alternative way is the success in allo-transplant of beta cells in clinics (3). The first step was to prepare beta-cells from donors, inject and inseminate them; afterwards, adding long term immunosuppression to maintain the injected cells functional. Good results were achieved sometimes, with independence from insulin replacement for a long period (3). The main cost was to support long term immunosuppression.

Another alternative was when the patient received in the same time kidney transplant, with the consecutive immunosuppression for the

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Article received on the 29th of September 2022 and accepted for publication on the 30th of September 2022

kidney. No specific immunosuppression for beta-cells became necessary. However, the results were not as good as those obtained in the case of beta-cells transplant only.

A third attempt is to encapsulate the injected beta-cells in order to protect them from an immune aggression. Studies are ongoing (3).

It was some success to transform self alpha-cells or even delta-cells in the own pancreas to produce insulin *de novo*. Studies are ongoing as well (3).

Finally, attempts to obtain functional beta-cells starting from stem cells began (3).

In type II diabetes mellitus (T2D), the pathogenesis is different and the attempts for non-conventional therapies as well. In T2D, beta-cells in the islets are aggressed by the conditions producing the illness (4). The first step is the increase in insulin resistance, which oblige beta-cells to over work. The aggressive factors, often inflammatory, which are present in T2D-associated conditions, like obesity, act directly in a negative way on beta-cells. It seems that a genetic predisposition may have a role too (4). In all these conditions, some beta-cells may survive only in latency.

If the outside ambient improves – such as after bariatric surgery – beta-cells may recover functionally, later on even in quantity – and T2D may have long term remission (4).

In this regard, treating T2D today is directed into the resuscitation of beta-cells, even acting on cell molecular traits, which could become a way to reduce this terrible disease of civilization. ❑

Aknowledgements: I kindly thank dr. Ariel Florentiu for his precious advice.

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