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Encapsulation of hPSC-Derived Pancreatic Progeny for Cell Therapy

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Description

The *in vivo* maturation of hPSC-derived pancreatic progenitors or pancreatic beta cells indispensably requires a suitable transplantation site, as well as an appropriate encapsulation material or device. The pancreas provides an appropriate microenvironment for the maturation of islets. However, a surgical method for delivery and retreivability has limited its consideration as a candidate for transplantation site. One study transplanted islets in rats and found that normoglycemia is achieved with fewer islets, compared to the extra-pancreatic sites, such as the liver and the kidney. However, it is worth noting that pancreatic progenitors transplanted subcutaneously or under kidney capsules have, nevertheless, resulted in their differentiation into functional beta cells, despite not being exposed to a 'pancreatic' microenvironment. For example, a previous study investigated the effect of the transplantation site on the generation of monohormonal insulin-secreting cells and found that the transplantation of pancreatic progenitors under mammary fat pads or kidney capsules do not affect their maturation into beta cells. It is plausible that the crucial vascular system supplying nutrients and oxygen to these transplanted progenitors carries cues sufficient to facilitate their maturation into beta cells.

An encapsulation device is, therefore, a crucial factor affecting the performance of the transplanted cells and it can control teratoma formation if the transplanted cells are contaminated with undifferentiated cells. Encapsulation of the transplanted pancreatic lineage is crucial in case of T1D to prevent an autoimmune reaction against the transplanted cells. Additionally, if the cell therapy product is from an allogenic source, for example, commercialized off-the-shelf hPSC-derived beta cells, then encapsulation is still a requirement. However, in the case of T2D or monogenic diabetes, there is a possibility of transplanting pancreatic progeny derived from the patient's own cells, which circumvents the need for encapsulation.

Achieving the right design of an encapsulation device requires putting together variables, such as the biocompatibility properties of the membrane, exposure to the blood stream, and availability of nutrition and oxygen for the encapsulated cells amongst others. Studies are being done on modification of the available materials to improve these properties of the biomaterial, and have mainly been developed for islet transplantation, both in macro- and micro-encapsulation systems. The assessment of islet function and viability following their coating with alginate derivatives is being widely investigated for improving islet transplantation outcomes. Purified alginate improved the survival of encapsulated islets and had a moderate effect on necrosis compared to non-purified alginate capsules. Furthermore, certain alginate modifications are particularly interesting to study as they could circumvent immune response following transplantation of allogenic islets. Modification of alginate capsules using triazole-derivatives showed positive results in preventing immune cell activation at capsule surfaces in mice and non-human primates. The incorporation of the chemokine CXCL12 in the alginate capsule protected the islets and improved their function by serving as an immune-isolating material without the need for immune-suppression. Likewise, such alginate-based microencapsulation methods are now being applied for stem cell therapy, such as for hPSC-derived beta cells. CXCL12 coating was recently shown to prolong the viability of hPSC-derived beta cells in immune-competent mice without requiring immunosuppression by preventing fibrotic overgrowth. In addition, the CXCL12 coating enhanced beta cell function by improving their glucose responsiveness, thereby making it an important biomaterial to study further for beta cell encapsulation.