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Type 2 diabetes affects ~10% of the UK population. This syndrome can be best desribed as a failure of insulin-secreting beta cells to functionally compensate for insulin resistance. Therefore, strategies to preserve beta cell function remain central to the treatment of T2D. Most mechanistic studies and drug development programmes work on the assumption that all beta cells are the same. However, recent single cell screening and imaging studies have shown that beta cells are in fact organised into discrete subpopulations. The current presentation will discuss how beta cell subpopulations may influence insulin release. The relevance of this for treatment of T2D, as well as engineering of islets from stem cells, will then be broached