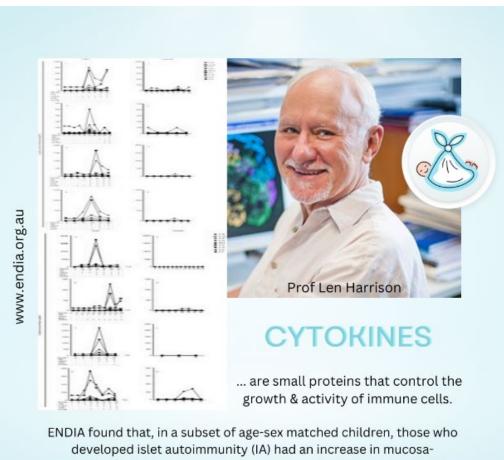
## Harrison et al (2023) A surge in serum mucosal cytokines associated with seroconversion in children at risk for type 1 diabetes. Journal of Diabetes Investigation: <u>https://doi.org/10.1111/jdi.14031</u>

Young children at risk of developing type 1 diabetes can be identified by antibodies in their bloodstream. These antibodies indicate an attack on the insulin-producing beta cells of the pancreas. Although genes predispose to type 1 diabetes, environmental factors are thought to initiate or trigger T1D. Prime environmental candidates are viruses, particularly enteroviruses, that infect the intestinal tract.

Professor Len Harrison and ENDIA colleagues hypothesised that if enteroviruses triggered autoimmunity to the beta cells, this may be reflected by inflammatory proteins, called cytokines, in the blood coming from the intestinal tract. The Environmental Determinants of Islet Autoimmunity (ENDIA) Study, in which children at genetic risk for type 1 diabetes are followed from pregnancy through early life, provided the opportunity to investigate this idea.

By testing blood samples of a small group of ENDIA children over time, our researchers found a sharp rise in cytokines related to intestinal inflammation. This was found around the time children developed antibodies to beta cells. This was <u>not</u> observed in another eight children, matched for sex and age, who did not develop islet autoantibodies.

This new finding supports the idea that intestinal infection, e.g., by an enterovirus, triggers the onset of immunity to the beta cells. This paper was published in the Journal of Diabetes Investigation and can be read online here: <u>https://doi.org/10.1111/jdi.14031</u>



developed islet autoimmunity (IA) had an increase in mucosaassociated cytokines compared to children who did not have IA. This supports the theory that a mucosal infection (e.g. an enterovirus) is a potential trigger of type 1 diabetes.