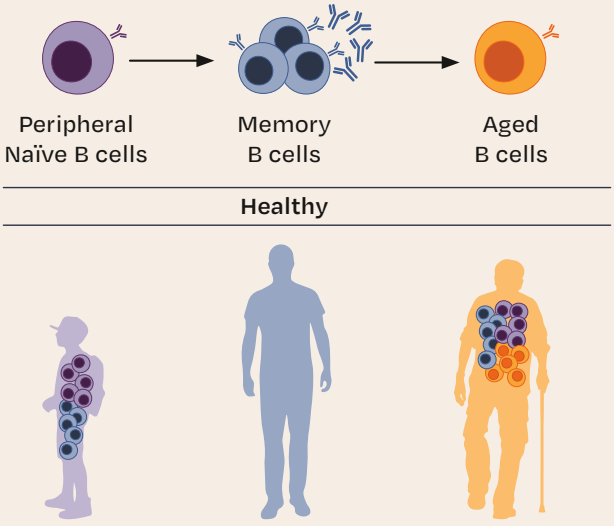
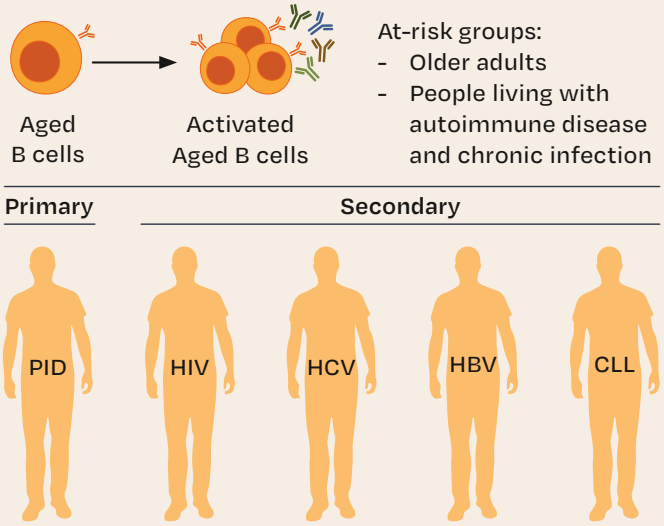
Inflammageing and models of chronic immune system decline



Immunity: Robust vaccine responses, protective immune response



Inflammageing: Poor adaptive immunity, systemic inflammation, malignancies

'Inflammageing', the build-up of low-grade inflammation in the body over time, increases the risk of age-related conditions such as heart disease, arthritis and type 2 diabetes.

Inflammageing results from a decline in naïve B cells and an increase in aged B cells. Aged B cells develop in higher numbers as the body ages, as well as in people living with co-morbidities such as HIV, CLL, and autoimmune diseases.

Burnet Institute has discovered that the release of a certain cytokine transforms B cells into overactive aged B cells. Through this finding, we have developed unique animal models of age-associated B cell disease: the B cells lack a specific cytokine pathway, blocking or reversing progression into aged B cells.

This development paves the way for therapeutics which slow or prevent inflammageing's impact.

Why work with Burnet?

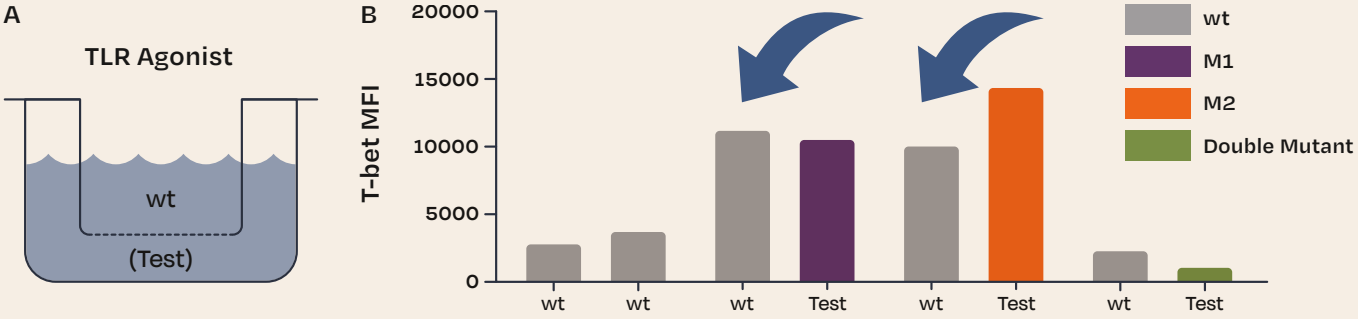
- Long-standing expertise in immunology and cell signalling assays
- Advanced automated equipment for high throughput antibody screening
- AMNIS Image and Multi-Panel Flow Cytometry
- Clinical and implementation research support in multiple regions
- Access to ISO 9001 Quality Management System
- Burnet Institute's record of industry and research partnerships

We are seeking partnership opportunities to investigate new targets and screen new treatments for autoimmune disease and immunodeficiencies.

Our technology

Our transwell culture system, along with our novel mutant mouse models, provides a valuable tool to screen therapeutics that block or reverse the Aged B cell phenotype.

- Transwell culture system to mimic inflammation
- Mutant mouse models to replicate the human condition
- *In vitro* assays to perform drug and antibody screening
- Validation studies in human culture assays
- Validated targets including cytokines, transcription factors and TLRs to stop and/or reverse inflammageing



Disease mutant M1 and M2 B cells are seeded in the test chamber and cause naïve B cells to acquire Aged B cell properties

Double mutant B cells lacking a specific cytokine pathway blocked aged B cell reprogramming of wt naïve B cells

Our expertise and track record

- Cell signalling in T and B cells and mouse models of immunodeficiency
- Aged B cells in inflammageing
- B cell deficiencies and autoimmune disease
- Gugasyan et al (2016) *NFxB1 is essential to prevent the development of multiorgan autoimmunity by limiting IL-6 production in follicular B cells* DOI: 10.1084/jem.20151182

Work with us

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