

Identification of new therapeutic strategies for aggressive breast cancer



Researchers:

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Project location:

Monash University, Peter MacCallum Cancer Centre and the Victorian Centre for Functional Genomics.

This research team hypothesises that there are different subclasses of TNBC, each exhibiting characteristic changes in chemical signals and kinase activity. If this is correct, then measuring these signals and activities represents a novel way of sub-classifying TNBC. If they can identify the kinases in each subclass that generate key growth-promoting signals, then these kinases represent potential therapeutic targets, which would be a major step towards personalised treatment.

present in TNBCs. In a world first, this will enable them to subclassify the cancers according to the signals that are present, and identify the kinases that emit these subclass-specific signals. This will lead to improved treatments, and ultimately to reductions in morbidity and mortality.

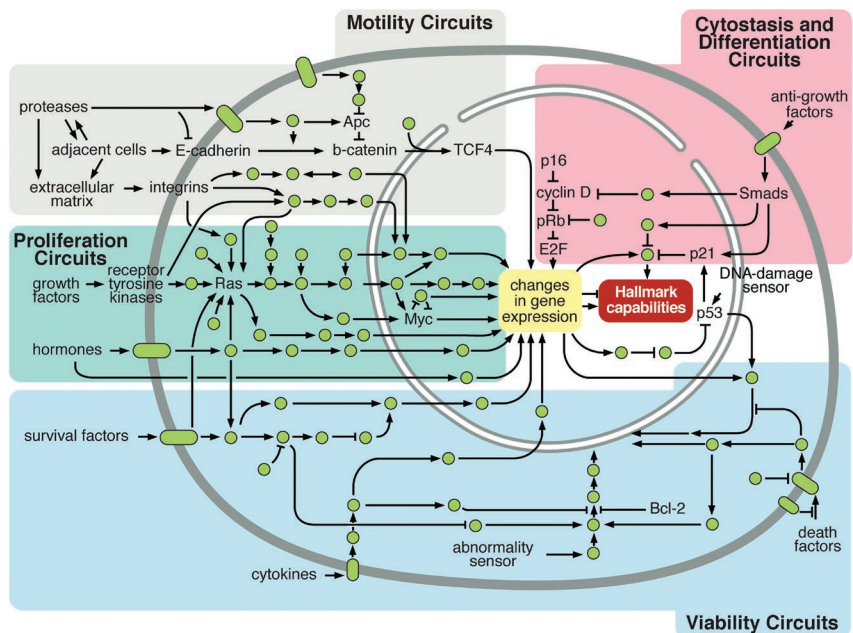
“We want to identify new personalised treatments by advancing molecular sub-classification of triple negative breast cancer – a world first.”

- Professor Daly

To do this they propose to use cutting edge approaches to measure the chemical signals and kinase activities

Triple negative breast cancer (TNBC) is a highly aggressive subtype of the disease that constitutes up to a third of breast cancer cases. It presents a major clinical problem due to its aggressive nature and because targeted treatments suitable for other forms of breast cancer are ineffective. At the moment, the only drug treatment that can be given is chemotherapy, which often has harmful side effects.

While it is becoming increasingly evident that not all TNBCs are the same, we do not understand the molecular basis of these differences. Therefore, we do not know how to treat TNBC patients in a personalised fashion, so that they each receive optimal treatment.



Cancer development requires rewiring of intracellular signalling networks
Image courtesy of Cell Press / Elsevier Inc.