



ASX & Media Release

Publication Highlights Role of Patrys' DNA-Damaging Antibodies in Cancer

- A subset of lupus autoantibodies, including assets licensed to Patrys, can penetrate cells, translocate to nuclei, and inhibit DNA repair
- These DNA-damaging lupus autoantibodies are selectively toxic to cancer cells
- Lupus autoantibodies may have a direct role in mediating DNA damage, thereby affecting the cancer risk profile of people with these circulating autoantibodies

Melbourne, Australia; 31 March, 2016: Patrys Limited (**ASX: PAB**) is pleased to announce the publication of a scientific article regarding its newly licensed technologies Deoxymab and 5C6 in the leading scientific journal *Nature Reviews Rheumatology*. The article is currently available online and will be included in a future print edition of the journal.

Deoxymab (3E10) and 5C6 antibodies were originally isolated from lupus-prone mice. The Deoxymab antibody was first considered for use as a vaccine for patients with systemic lupus erythematosus (SLE), and it was safely tested in humans in 1999. In the evaluation process Deoxymab was found to have the unusual capacity to penetrate into live cell nuclei and bind DNA. The mechanisms by which Deoxymab and similar antibodies cross the cell membrane are diverse, but it was recently discovered that once inside the nucleus they can inhibit DNA repair or directly damage DNA, making them promising potential cancer therapeutics because these effects are toxic to cancer cells with defects in DNA repair and also increase the sensitivity of cancer cells to radiation and chemotherapy.

This review article examines the role of DNA-damaging antibodies, such as Deoxymab and 5C6, in lupus and suggests that these antibodies may suppress the growth of and reduce the risk of development of certain cancers and are a promising new approach to cancer therapy.

Patrys' Chief Executive Officer Dr. James Campbell commented: "We are excited about the newly licensed technologies from Yale University and we look forward to reporting on progress with the optimized variant of Deoxymab". Deoxymab and 5C6 are currently the focus of preclinical development programmes sponsored by Patrys.

A link to the article is available at

<http://www.nature.com/nrrheum/journal/vaop/ncurrent/full/nrrheum.2016.23.html>

-Ends-

For further information, please contact:

Patrys Limited:
James Campbell
Chief Executive Officer
P: +61 3 9670 3273
info@patrys.com

Patrys IR:
Kyahn Williamson
Buchan Consulting
P: +61 3 9866 4722
kwilliamson@buchanwe.com.au

Patrys Media:
Kellie Stanborough
Buchan Consulting
P: +61 3 9866 4722
kstanborough@buchanwe.com.au



About Patrys Limited:

Based in Melbourne, Australia, Patrys (ASX: PAB) is focused on the development of antibodies as therapies for a range of different cancers. Patrys has a pipeline of anti-cancer antibodies that enable both internal development and partnering opportunities. More information can be found at www.patrys.com.

About 3E10 and 5C6 anti-DNA antibodies:

A nuclear-penetrating lupus anti-DNA autoantibody 3E10 (Deoxymab) is novel cancer therapy that has the capacity to penetrate cancer cell nuclei, inhibit DNA repair, and kill DNA repair-deficient cancer cells. The antibody preferentially localises to tumours and has the ability to sensitise cancer cells to radiation and chemotherapy and interfere with their ability to sustain themselves through DNA repair. Furthermore, 3E10 when used alone can selectively kill cancer cells with DNA repair deficiencies such as those with mutations in the BRCA2 and PTEN gene. These characteristics of 3E10 open up new possibilities for treating BRCA2 and PTEN-related cancers. Yale University has also found that 5C6, an anti-DNA autoantibody that can penetrate cells, has a toxic effect on BRCA2-deficient cells in colon cancer. In March 2016 Patrys licensed worldwide rights to both Deoxymab and 5C6 from Yale University.

About BRCA2 and PTEN Gene Mutations:

Certain types of cancers occur when DNA repair goes awry because of inherited gene mutations. BRCA2 is a tumour suppressor that, when damaged or deficient, can lead to malignancies such as breast, ovarian, pancreatic, and prostate cancers while PTEN mutations are related to breast, brain gliomas and astrocytomas, head and neck carcinoma, endometrial and thyroid tumour development.

Further publications related to this work can be found at the following websites:

<http://cancerres.aacrjournals.org/content/75/11/2285.long>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4496662/>

<http://www.nature.com/articles/srep05958>