



16 February 2009
BioPharmica (ASX: BPH) ASX Announcement

HLS5 presented at the 21st Lorne Cancer Conference 2009

Dr Louise Winteringham presented a poster at the Lorne Cancer Conference during 12th to 14th February 2009 in Victoria entitled 'HLS5/TRIM35 is down-regulated in breast and ovarian tumours and can regulate transactivation by steroid hormone receptors'.

Louise Winteringham, Robin Scaife, Jean-Philippe Lalonde, Jennifer Beaumont, Rachel Ramsdale (Molecular Discovery Systems) and Peter Klinken from The Western Australian Institute for Medical Research (WAIMR) and The University of Western Australia (UWA) have researched together in collaboration with BioPharmica Limited under a Joint Venture.

Abstract from the poster:

Transformation of normal cells into malignant tumour cells depends on progressive acquisition of genetic alterations resulting in either activation of proto-oncogenes or inactivation of tumour suppressor genes. Haemopoietic lineage switch 5 (Hls5) is a novel tumour suppressor gene located on chromosome 8p21, a region associated with a number of cancers including the steroid dependant breast and prostate cancers. The requirement for the steroid hormones estrogen and androgen, respectively, in the development of these cancers is well characterised. Our studies have shown Hls5 mRNA is decreased in the majority of breast cancer cell lines and in a number of breast and ovarian tumours compared to normal tissue. We have shown that Hls5 is able to inhibit transactivation by both the estrogen and androgen receptors and can inhibit the activity of estrogen receptor co-activators *in vitro*. These data suggest that Hls5 has a role in down-regulating cellular steroid hormone receptor levels and reduced Hls5 expression may contribute to the increase in steroid hormone receptor levels required for the development and progression of disease. Screening of a chemical library to identify compounds that activate the Hls5 promoter has yielded compounds that increase Hls5 mRNA levels. These compounds are undergoing secondary screening to determine their effects on estrogen- and androgen-mediated activation of transcription and cell proliferation (*see attached full poster for further information*).

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Yours sincerely,

David Breeze
Chairman

Hls5/TRIM35 is down-regulated in breast and ovarian tumours and can regulate transactivation by steroid hormone receptors.

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Abstract

Transformation of normal cells into malignant tumour cells depends on progressive acquisition of genetic alterations resulting in either activation of proto-oncogenes or inactivation of tumour suppressor genes. Haemopoietic lineage switch 5 (Hls5) is a novel tumour suppressor gene located on chromosome 8p21, a region associated with a number of cancers including the steroid dependent breast and prostate cancers. The requirement for the steroid hormones estrogen and androgen, respectively, in the development of these cancers is well characterised. Our studies have shown Hls5 mRNA is decreased in the majority of breast cancer cell lines and in a number of breast and ovarian tumours compared to normal tissue. We have shown that Hls5 is able to inhibit transactivation by both the estrogen and androgen receptors and can inhibit the activity of estrogen receptor co-activators *in vitro*. These data suggest that Hls5 has a role in down-regulating cellular steroid hormone receptor levels and reduced Hls5 expression may contribute to the increase in steroid hormone receptor levels required for the development and progression of disease. Screening of a chemical library to identify compounds that activate the Hls5 promoter has yielded compounds that increase Hls5 mRNA levels. These compounds are undergoing secondary screening to determine their effects on estrogen- and androgen-mediated activation of transcription and cell proliferation.

