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BLADDER CANCER: NEW CONFIRMATION FOR AMYGDALIN

It was not so long ago that American scientists declared that amygdalin (which for practical purpose can be called “laetrile”) had zero activity against cancer and indeed could have no such activity (as Daniel S. Martin, MD claimed in 1975).

Yet in August 2014, Jasmina Makarevic and eight other German scientists (including senior scientist Roman A. Blaheta) showed that amygdalin stopped the growth of three different bladder cancer cell lines. Their article appeared in PLOS ONE, arguably the most famous open access scientific journal in the world. The nine authors in question came from the Department of Urology, Goethe University Hospital, Frankfurt am Main, Germany and the Institute of Human Genetics, University Medical Center Göttingen, Germany. These are two outstanding institutions: there have been 44 Nobel laureates associated with the city of Göttingen (Makarevic 2014).

In the present study, Makarevic and colleagues incubated three separate lines of bladder cancer cells with pure amygdalin. They looked at tumor growth and proliferation, clonal growth and cell cycle progression. Their key finding was as follows:

“Amygdalin dose-dependently reduced growth and proliferation in all three bladder cancer cell lines...”

To be on the safe side, all experiments were performed three to six times. The consistent reduction in tumor cell growth was caused by an interruption in the normal cell cycle and particularly in an arrest of the first step in cell division. Further studies then suggested that amygdalin blocked tumor growth in two ways: by down-modulating cyclin A and cyclin-dependent kinase 2 (cdk2). Cyclin A is a member of a group of proteins that regulate a cell’s progression through the growth cycle.

This was the first study to show the effectiveness of amygdalin against bladder cancer cells. But earlier test tube studies have shown that amygdalin induces programmed cell death (apoptosis) in human prostate cancer cells (Chang 2006) and HeLa cervical cancer cells (Chen 2013).

One of the reasons that I wrote *Doctored Results: The Suppression of Laetrile at Sloan-Kettering Institute* (2014), and participated in Eric Merola’s recent film, *Second Opinion*, was to reopen the debate over the effectiveness of amygdalin in the laboratory and the clinic. I firmly believe that objective scientific research will ultimately prove this class of compounds to be a lost opportunity for the world of oncology. I am therefore highly encouraged when these German authors wrote:

“In vivo [animal, ed.] investigation must follow to assess amygdalin's practical value as an anti-tumor drug.”

It is very exciting that well qualified researchers have taken up the challenge of testing this controversial substance as an anti-cancer agent.

--Ralph W. Moss, PhD

Chang H-K, Shin M-S, Yang H-Y, et al. Amygdalin induces apoptosis through regulation of Bax and Bcl-2 expressions in human DU145 and LNCaP prostate cancer cells. *Biol Pharm Bull.* 2006;29(8):1597-1602.

Chen Y, Ma J, Wang F, et al. Amygdalin induces apoptosis in human cervical cancer cell line HeLa cells. *Immunopharmacol Immunotoxicol.* 2013;35(1):43-51.

Makarević J, Rutz J, Juengel E, et al. Amygdalin blocks bladder cancer cell growth in vitro by diminishing cyclin a and cdk2.