Beyond Laetrile (Vitamin B-17) Controversy-Antitumor Illusion or Revolution

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Abstract

Many researchers have discovered amygdalin or laetrile to be clinically inefficient in curing cancer, as well as potentially toxic or fatal when orally ingested due to cyanide poisoning. Nevertheless, hypotheses highlighted the role of amygdalin as an anticancer treatment causing programmed death in prostate cancer and human cervical cancer cells. Moreover, one study reported that amygdalin blocked bladder cancer cell growth by down-modulating the cell cycle regulating proteins cdk2 and cyclin A.

Keywords: Vitamin B-17; Amygdalin; Laetrile; Kernels; Rhodanese

Introduction

Amygdalin was extracted for the first time in 1830 from bitter almond seeds [1]. Laetrile (patented 1961) [2] is a mostly human-made form of the substance amygdalin, existed naturally in Prunus fruits pips like apricot, beach, plum and bitter almond as well as raw nuts. It also is existed in clover, lima beans, and sorghum [3,4].

Nearly 60 years ago, both amygdalin and a modulated version named laetrile have been consolidated as an alternative treatment for tumors, often utilizing the misnomer vitamin B17 [5]. But some researchers have explored them to be clinically inefficient in controlling abnormal cell growth, as well as implicitly lethal when orally ingested because of cyanide toxicity [6].

Rhodanese (thiosulfate sulfurtransferase) enzyme is found in healthy cells and has the ability to neutralize benzaldehyde and hydrogen cyanide in B-17. It transforms these materials into the utilizable compounds thiocyanate (rhodanide) and benzoic acid.

Laetrile which is delivered to cancer cells along with glucose is not neutralized to rhodanide and benzoic acid due to lack of rhodanese enzyme in neoplastic cells. Instead, neoplastic cells release cyanide and benzaldehyde from delivered glucose by the action of β-glucosidase found in cancer cells. In turn, the released cyanide and benzaldehyde attack neoplastic cells by formation of a killing targeted poison [3].

Natural amygdalin with the R configuration can be racemized to the unnatural form, neoamygdaline, during inexperienced extraction, bad packing or storage. Neoamygdaline is therapeutically as inefficient as amygdalin and a commercial combination of each is called isoamygdaline [7,8].

Both laetrile and amygdaline are metabolized by hydrolysis via the alkaline duodenal and intestinal juice enzymes forming D-glucuronic acid and L-mandelonitrile. L-mandelonitrile is further hydrolyzed to hydrogen cyanide and benzaldehyde that cause cyanogenic toxicity when laetrile is ingested in an abundant amount [9].

Hypotheses regarding the role of amygdalin as an anticancer is controversial. Some theory suggested that neoplastic cells contain plenteous β-glucosidases, which emancipate hydrogen cyanide from vitamin B-17 through hydrolysis. Normal tissues were reportedly uninfluenced as they contain low levels of β-glucosidase enzyme and high concentrations of the transforming enzyme, rhodanese, which transforms hydrogen cyanide to the less toxic thiocyanic acid salts. However, it was demonstrated later that both neoplastic and normal cells contain only traces of β-glucosidases and equal amounts of rhodanese [10].

Another untenable hypothesis suggested that, after taken orally, amygdalin is metabolized to a cyanohydrin compound, the so-called mandelonitrile, which is wholly transported to hepatic tissues to be transformed to a beta-glucurononide compound. This compound is in turn being conveyed to neoplastic cells to be further catalyzed by β-glucuronidases to liberate mandelonitrile and then hydrogen cyanide. A third hypothesis suggested that cancer is due to laetrile or the discovered vitamin B-17 deficiency. It assumed that steady dietary administration of laetrile would prevent the incidence of cancer. Hence, Ernst T. Krebs, an American biochemist, indicated laetrile as a vitamin to be categorized as a nutritional supplement rather than being a drug [5].

In the early 1970s, Memorial Sloan-Kettering Cancer Center persuaded some researchers to carry out a study on experimental animals for the anticancer efficacy of laetrile. Dr. Sugiura, the researcher who performed the study, discovered that vitamin B-17 stopped the secondary tumors in experimental...
animals. In spite of these promising findings, laetrile failed to devastate the primary tumors. He reiterated the experiment many times and had similar results. Nevertheless, some other investigators failed to emphasize Sugiura’s results [11].

Meanwhile, G. Edward Griffin’s research evolved into his groundbreaking book, World without Cancer. He discovered that the Sloan-Kettering Cancer Institute had inhumed some documentation from researcher’s findings, which evidenced laetrile was highly efficient at curing cancer [12]. Conspiracy theory then appeared after conclusion features of Sugiura’s study being leaked to laetrile advocates. They claimed that the US Food and Drug Administration and the medical community, including the American Medical Association and the American Cancer Society as well as the pharmaceutical industry are responsible for this conspiracy to exploit cancer patients [13].

Recently, from the Cochrane Collaboration review carried out by Milazzo et al. concluded that the facts that vitamin B-17 has profitable effects on cancer are no longer propped clinically. In contrast, there are substantial hazards if laetrile ingested orally owing to cyanide poisoning. The risk–benefit equipoise of amygdalin or laetrile in curing cancer was therefore unambiguously negative [14].

In spite of this depressive report against amygdalin, many studies showed that amygdalin had an antitumor properties [15] that caused programmed death in prostate cancer [16] human cervical carcinoma [17], weakened activation of kidney fibroblast and interstitial fibrosis [18] and blocked bladder cancer cell growth by down-modulating the cell cycle regulating proteins cdk2 and cyclin A [19].

References

1. A chronology of significant historical developments in the biological sciences. University of Hamburg, Department of Biology.
8. http://users.navi.net/~rsc/ismoyg.htm