

# New strategy in tumor treatment

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A new strategy proposed by researchers at Dartmouth Medical School and Amtek, Hanover, NH may treat tumors that do not respond to conventional treatment. The study, which was published on May 29th in the open access, peer reviewed journal *PLoS ONE*, uses a combination of two agents to selectively kill tumors while protecting healthy cells.

In previous studies, researchers discovered that a specific enzyme - known as methylthioadenosine phosphorylase (MTAP) - is missing in 35 to 70 percent of lung, pancreatic and biliary tract cancer, melanoma, glioblastoma, osteosarcoma, soft-tissue sarcoma, mesothelioma, and T-cell [acute lymphoblastic leukemia](#). Although information on the incidence of MTAP-deficiency in [breast cancer](#) is still limited, researchers believe it is probably significant.<sup>1</sup> Since the discovery of MTAP-negative tumors, there have been several proposals, over the years, to take advantage of the frequent absence of MTAP in so many lethal cancers. None of these proposals, however, has led to successful clinical use.

Dr. Martin Lubin, Professor of Microbiology, Emeritus, and co-author Adam Lubin of Amtek have offered a new approach. They say, "Our strategy consists of two agents. One drug is given that is toxic both to cancer cells and to normal host tissues. A second, but non-toxic, drug is also given, which protects normal tissues from the toxic action of the first agent. This two-drug combination therapy kills [tumor](#)+cells/" rel="tag" class="textTag">tumor cells while normal tissues are well protected." They go on to state, "Among the drugs used to kill the tumor cells, two -- thioguanine and fluorouracil (or its prodrug Xeloda) - are

already in clinical use. In general, the dose of these drugs is limited by toxic side effects. However, with our strategy, greatly increased doses might be used and tumors not susceptible to low doses could be attacked successfully at higher doses, without harm to host tissues."

To assess the selective killing of tumor cells when they were present in excess of normal cells, the researchers designed co-culture experiments in vitro and animal studies are now in progress.

"We hope that successful animal studies will lead to clinical application as soon as possible," Dr. Lubin said.

More information:

Lubin M, Lubin A (2009) Selective Killing of Tumors Deficient in Methylthioadenosine Phosphorylase: A Novel Strategy. *PLoS ONE* 4(5): e5735. [doi: 10.1371/journal.pone.0005735](https://doi.org/10.1371/journal.pone.0005735)

Della Ragione F, Russo G, Oliva A, Mastropietro S, Mancini A, et al. (1995) 5'-Deoxy-5'-methylthioadenosine phosphorylase and p16INK4 deficiency in multiple tumor cell lines. *Oncogene* 10: 827-833.

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