Antineoplaston

Name coined by Stanislaw Burzynski for a group of peptides, peptide derivatives, and mixtures that he uses as an alternative cancer treatment.

Antineoplaston therapy has been offered in the US since 1984 but is not approved for general use. The compounds are not licensed as drugs but are instead sold and administered as part of clinical trials at the Burzynski Clinic and the Burzynski Research Institute.

Burzynski stated that he began investigating the use of antineoplastons after detecting what he considered significant differences in peptides between the blood of cancer patients and a control group.

Burzynski first identified antineoplastons from human blood. Since similar peptides had been isolated from urine, early batches of Burzynski's treatment were isolated from urine.[

Since 1980 he has produced his compounds synthetically.

The first active peptide fraction identified was called antineoplaston A-10 (3-phenylacetylamino-2,6-piperidinedione). From A-10, antineoplaston AS2-1, a 4:1 mixture of phenylacetic acid and phenylacetylglutamine, was derived.

The website of the Burzynski clinic states that the active ingredient of antineoplaston A10-I is phenylacetylglutamine.

Since 2011, the clinic has marketed antineoplastons as "personalised gene-targeted cancer therapy" which stirred further controversy as the treatment bears no relationship to gene-targeted therapy and only superficially incorporates elements of personalized medicine.

Antineoplastons contributed to more than a 5-year survival in recurrent diffuse intrinsic glioblastomas and anaplastic astrocytomas of the brainstem in a small group of patients ¹⁾.

Shows efficacy and acceptable tolerability profile in patients with recurrent pediatric diffuse intrinsic pontine gliomas (RPDIPG)²⁾.

the results of unconventional therapies such as the one proposed by Burzynski et al. for the treatment of DIPG must be read. It is a rule; whenever the rigid progress of knowledge, briefly and in a quite simple way outlined above, is not respected, the level of attention on the credibility of the experiment reported immediately raises. The author and his collaborators present the results of an agent called antineoplastons A10 and AS2-1 (ANP), which are synthetic amino acid derivatives, combining phenylacetylglutaminate (PG) sodium and phenylacetylisoglutaminate sodium in different ratio used to treat DIPG. The overall results reported are as bad as the ones described by other reports. That is not the point however. The point is to question if the evidences and more importantly the steps which should be followed to bring a compound from "bench to bed" have been respected in this case. It raises quite a level of suspicions the fact that a treatment developed and piloted by Greg Burzynski was sponsored by the Burzynski Research Institute, conducted by the Burzynski Clinic (BC) and never reproduced elsewhere. It is not worthy insisting too much of these aspects, which could be argued extensively. The matter of fact that an agentwho brings to a 2-year overall survival of 11.8 % for children with DIPG must be considered clearly ineffective and thus abandoned. The children affected byDIPG deserve to become the focus of serious collaborative researches. For these children, there are many "lacks," which should be promptly corrected, the lack of knowledge, the lack of basic and

clinical scientists' passion to the problem of finding "the solution" for DIPG, the lack of rigid methods to run research in this frustrating field, the lack of research proposals, and the lack of serious, despite not magic, protocols to offer them. All these would help these unfortunate children and their families not to be attracted by dangerous illusions and thus, by the temptation of investing important physical, psychological, and not rarely financial energies to pursue them ³⁾.

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