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Cancer as a Therapeutic Agent?*

Mario Gosalvez

Laboratorío y Servicío de Bioquímica Experimental (1970-2010), Clínica and Hospital Universitario Puerta de Hierro Madrid and Majadahonda, Comunidad de Madrid, Spain.
Email: dmg.secre@gmail.com

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ABSTRACT

The author, concisely and generically, proposes experimental testing on suitable laboratory animals, in state-of-the-art international centers endowed with cancer reversal experimental techniques. Using mild carcinogens to induce more or less benign growths can be reversed back to a normal state by single or dual strategy: in spinal paralysis by medullar contusion in young rabbits; for blindness caused by the severing of the optic nerve in sheep; for neural deafness caused by severing the acoustic nerve in rabbits. If these techniques could be shown to be feasible and successful for higher order primates and if having then already cured for life a sufficient number of malignant cancers by cancer reversal by dual strategy in human beings, of course these methods could be then considered for patients.

Keywords: Mild Carcinogenesis and Cancer Reversal; Animal Recoveries from Spinal Paralysis; Blindness and Deafness; Mitochondrial Filamentation

1. Introduction

I should like to draw your attention to some initial possibilities on the use of cancer as a therapeutic agent. Some years ago, there is going a process which might lead to the understanding of a defect in mitochondrial filamentation in the tumoral cell as a basis for a better comprehension of its nature, cause, diagnosis and treatment [1-10]. My first contribution to these discoveries has simply been assisted with some pilot experiments and first clinical studies in animal and human malignant tumors [11-21]. It is only now that the field of mitochondrial filamentation might begin to go forward, hand in hand, at least, with the most proficient experimental and clinical cancer research [4-6,9].

This field of mitochondrial filamentation augurs well for a good harvest in several applications: from the extension of metabolic life in cases of transitory physiological death [6,22] to an increase of sporting prowess [3], including degenerative illnesses and aging [6,7,10] schizophrenia or obesity [6,7], for example. I have recently published my proposal to explain the intimate mechanism of the aerobic glycolisis of tumoral cells [8], based in our studies on this last incognita of the nature of cancer [1,3,16].

I only have but a few years left in an advisory capacity on mitochondrial filamentation, and its future applications before being overtaken by the new generation of experts. The ball therefore is now in the court of the interested youth. I should now like to explain to your readers, concisely and directly, the possibility of applying cancer to animal experimental treatments for spinal paralysis, blindness and neural deafness. There are three species of creature on which, in my judgment, trials could be started now by experts: the young rabbit for spinal paralysis; the young sheep for blindness and the adult rabbit for deafness. Matters would be ready, possibly, to begin immediately on animals in some centers of clinical and surgical excellence in the USA, Europe and Japan.

2. Spinal Paralysis

Up until now, this problem—the interruption of the spinal cord—has been attempted on rats using neural grafting and stem cells, more or less first-order ones. Among the issues encountered are the impermanence of the solution and the cancerification of the implant. We firmly believe that the exquisite embryonic cell concatenation in time and space for the differentiation continues in the adult organism on account of the suitable molecular correlations for dedifferentiation and redifferentiation.

In such a way, in our opinion, that besides those useful

*An experimental proposal for low order laboratory mammals.

bone marrow transplants in human leukemia, there will probably be only two ways to replace damaged cells in the spinal cord of superior mammals:

1) Cancerify cells locally to cells of low malignancy and subsequently apply reversal techniques back to normality when the problem has been resolved.

2) Locally inducing the dedifferentiation to stem cells so as to redifferentiate them subsequently to normal cells.

In respect of the second way, I believe that decades of study are needed. The first one, I consider, is feasible now in some very capable centers.

Spinal paralysis should be induced in the recently weaned bunny rabbit by spinal cord contusion. As soon as it has been filmed, and its number of movements per hour determined, following the consolidation of the lesion, the pre-carcinogen cocktail-to be metabolized to the final medullar carcinogen suitable for the rabbit, should be injected in few microliters in the neurons immediately in front of and behind the spinal cord hiatus.

Naturally, at the moment there are several possibilities for those local initiators of benign spinal tumor. Only by trial and error on dozens of rabbits will the best ones manifest themselves. The cocktails that may work best in filamented mitochondria and in primary neural cultures, in addition to adult rabbits, will allow us to experiment on the miniature pig with chances of a good success rate. It is considered important, in my opinion, to repeat these experiments on as many different species as possible.

As soon as a success rate of more than 70 percent is guaranteed for the generation of a spinal cord tumor, especially in pigs, reversal back to the normality of a benign tumor could be started, as soon as the slightest reactive movement has been observed in the tails of the stimulated animals.

At that point, very possibly, the comparison of movements per hour would show a marked increase and the detection by PET-SCAN of the spinal cord tumor would have yielded some indication of its malignancy. Therefore, very shortly afterwards, the beginning of the tumor reversal with techniques solely directed at benign tumor cells, or by dual strategy, (including therefore cytotoxics for tumor reducts) could be shown [13-16,20,21].

Following the complete recovery of movement, without any other added toxicity, an observation period of at least eight weeks will be set up, to ensure that there is no tumor reappearance and then to progress to the ape and monkey.

When sufficient monkeys may have been cured completely the therapy could be considered in volunteers. Especially in those young tetraplegics previously well-prepared in suitable centers if these persons could blink their approval. Having taken the due assessorial information, family advice and insurances, of course, these procedures could be followed, only if a sufficient number of human malignant cancers had then cured for life duration [4-6,9].

3. Neural Blindness

A young sheep would be the animal of choice. All the optic nerves would have to be cut very cleanly at the best height of their length, leaving it without sight in one eye. Neural atrophy would be expected, taking due ophthalmologic measures at regular intervals for both eyes, the sightless and the healthy one.

From that moment, the retinal hyperplasic cocktail suitable for sheep, a specialization directed at the cells with aerobic glycolysis, would begin to be administered. The observation of pre-carcinogenic hyperplasia in the cells of the retina of both eyes would need to be done daily at the back of both eyes. The intestinal crypts would also have to be checked.

In the healthy eye the reversal treatment would be performed locally. For the sightless eye only a local reversal treatment would be done for the benign cancerification. When the photon charge tests were almost identical for both eyes using intracerebral electrophysiological Register, the process would be stopped until complete cure had been instated and registered by complete vision returning to the eye that was sightless, with the healthy eye covered.

Naturally, the adaptation of the hyperplasic cocktails and the local benign cancer which function best on sheep, in order to progress to the ape and monkey and then on to the human being, would need to be proven with primary cultures and filamented mitochondria of retinas of the species in question.

4. Neural Deafness

The cochlea contains neurons that charge metals very well.

The cocktails of benign cancerification of this organ must be manufactured as metal complexes suitable to be digested intracellularly in their target cells. In such a way as to be able to distinguish similarities between the ear whose nerve has been cut and the healthy one by intracerebral sound charge resister. Reversal, whether by single or dual technique, should begin as soon as the deaf ear can hear. The treatment of the healthy ear must be local and start from the beginning. At the final stage of recovery the procedure would be the same as for neural blindness.

5. Important Considerations

The accumulated research on agents acting on cell hyperplasia is very extensive. The collection of agents, hypoplasic, hyperplasic, pre-carcinogens and soft carcino-

gens, is very large. The primary culture techniques, of cells, tissue and organs, is well embedded. Trials on filamented mitochondria [6-10,21] are easy to perform and will represent a very rapid screening of hundreds of compounds per month capable of returning more or less neoplastic cells to their state of normality.

It is recommended that the proposed studies be performed, in a calm and serene manner, with the collaboration of all, especially doctors and chemists of different specializations, to achieve this possible step forwards. If at some future point these studies were successful on apes, the techniques could be considered for patients. Of course, only when the complete cure of cancer diseases had been obtained in humans [4-6,9].

6. Discussion

A new adventure of ideas that surpasses the avenues of habitual thinking may be taken as too daring by those experts who have the right equilibrium between the past and the path to the future. Our proposition, first of all, for animal experimentation, comes after having observed not too much progress with the stem cell approach in the last decade, with science still not counting on localized techniques of adequate stem cell formation to resolve clearly these problems.

We have recently published on-line a concise account of our methodological expertise in the isolation and assay of filamented mitochondria and some proposals for possible future applications of this now emerging field [1,2, 4-10,21,22]. A search for private national and international financing for this and other future possible applications in this area of molecular physiology and medicine has already been started.

7. Conclusions

The due opportunity for this proposal in laboratory animals is timidly requested to the scientific community. It would be almost ready for the task with the security that if it should result in another failure, at least we would have clarified possible new ways. This proposal brings out a humble request for a combination of neurologists and oncologists of various types working closely together for some years on several animal species.

I considered that this, perhaps too daring an idea, could be contemplated with attention by the Journal of Cancer Therapy. Other journals declined previously rapidly and kindly to publish this paper. Most surely based on the lack of expertise on one or another of the two aspects considered in the proposal: Cancer and Neurology.

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REFERENCES

[1] M. Gosalvez, "Oxygen Production by Filament Mitochondria. Importance in Cancer and Neurodegeneration," *Anales de la Real Academia Nacional de Medicina* (*Madrid*) *Spain*, Vol. 115, No. 3, 1998, pp. 747-756.

[2] M. Gosalvez, "Reversion of Cancer and Mitochondrial Filamentation," *Anales de la Real Academia Nacional de Medicina* (*Madrid*) *Spain*, Vol. 117, No. 4, 2000, pp. 825-834.

[3] M. Gosalvez and S. Weinhouse, "Control Mechanism of Oxygen and Glucose Utilization in Tumors," *Advances in Experimental Biology and Medicine*, Vol. 75, 1976, pp. 587-596. http://dx.doi.org/10.1007/978-1-4684-3273-2_69

[4] M. Gosalvez, "Mitochondrial Filamentation," 101*th AACR Meeting*, Washington DC, 17-21 April 2010, pp. 3-7.

[5] M. Gosalvez, "Reversal of Cancer by Dual Strategy: A New Molecular Target," 103*rd AACR Meeting*, Chicago, 31 March-4 April 2012, p. 1118.

[6] M. Gosalvez, "Mitochondrial Filamentation: Some Methods of Isolation and Assay," *IOSR Journal of Pharmacy and Biological Sciences*, Vol. 4, No. 4, 2012, pp. 37-39. http://dx.doi.org/10.9790/3008-0443739

[7] M. Gosalvez, "Methods to Be Developed for Some First Applications of Mitochondrial Filamentation," *Open Journal of Biophysics*, Vol. 3, No. 1A, 2013, pp. 51-53. http://dx.doi.org/10.4236/ojbiphy.2013.31A006

[8] M. Gosalvez, "Metabolic Control of Respiration and Glycolysis of Tumoral Cells," *Advances in Biological Chemistry*, Vol. 3, No. 1, 2013, pp. 86-89. http://dx.doi.org/10.4236/abc.2013.31011

[9] M. Gosalvez, "Reversal of Cancer by Dual Strategy?" *Journal of Cancer Therapy*, Vol. 3, No. 4, 2013, pp. 518-520.

[10] M. Gosalvez, "Mitochondrial Filamentation: A Therapeutic Target for Neurodegeneration and Aging?" *Journal of Alzheimer's Disease & and Other Dementias*, Vol. 28, No. 5, 2013, pp. 423-426. http://dx.doi.org/10.1177/1533317513494451

[11] L. Lopez-Alarcon, *et al.*, "Quantitative Determination of the Degree of Differentiation of Mammary Tumors by Pyruvate Kinase Analysis," *Cancer Research*, Vol. 41, No. 5, 1981, pp. 2019-2020.

[12] A. Brugarolas and M. Gosalvez, "Preliminary Clinical Results with Norgamen (thioproline) and Revercan (2-Amino-thiazoline): The First Inducers of Reverse Transformation," *Adjuvant Therapies of Cancer, Recent Results in Cancer Research*, Vol. 80, 1982, pp. 346-350. http://dx.doi.org/10.1007/978-3-642-81685-7_56

[13] H. Cortes-Funes, *et al.*, "Quelamycin: A Summary of Phase I Clinical Trials," *Recent Results in Cancer Research*, Vol. 74, No. , 1980, pp. 2000-2009. http://dx.doi.org/10.1007/978-3-642-81488-4_25

[14] M. Gosalvez and A. Brugarolas, "Treatment of Cancer by an Inducer of Reverse Transformation," *The Lancet*, Vol. 1, No. 8159, 1980, pp. 68-70.

[15] M. Gosalvez y Gosalvez, "Basis for Design of Pharmacological Effectors Acting on Plasma Membrane Receptors," *Biochemical Society Transactions*, Vol. 1, 1981, pp. 151-152.

[16] M. Gosalvez, "The Zipper Mechanism," Comercial Malvar, Madrid, 1982.

[17] M. Gosalvez, "Carcinogenesis with the Insecticide Rotenone," *Life Sciences*, Vol. 32, No. 8, 1983, pp. 809-816. http://dx.doi.org/10.1016/0024-3205(83)90216-3

[18] M. Gosalvez, "Thioproline and the Reversal of Cancer," *The Lancet*, Vol. 1, No. 8333, 1983, p. 1118.

[19] M. Gosalvez, *et al.*, "Spectral and Metabolic Characteristics of Mitochondrial Fractions Isolated from Rotenone Induced Tumours," *British Journal of Cancer*, Vol. 36, No. 2, 1977, pp. 243-252. http://dx.doi.org/10.1038/bjc.1977.184

[20] M. Gosalvez, *et al.*, "Treatment of Intravenous Leukemia 1210 Transplants with Tumor Modifiers. Redoubt Cytotoxics and Minor Tranquilizers," *Aminoacids Chemistry, Biology and Medicine*, Escom Sciences Publishers, Leiden, 1990, pp. 704-711.

[21] M. Gosalvez, "Cancer Reversal by Dual Strategy," Editorial Gober, Madrid, 1992.

[22] M. Gosalvez, "Extension of Metabolic Life. Abstract," *Proceedings of the* 36*th ISOTT Meeting*, Sapporo, 3-7 August 2008, p. 52.

