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Prostate Cancer, the Long Search for Etiologic and Therapeutic Factors: Dietary Supplementation Avoiding Invasive Treatment

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1. Introduction

The lack of a comprehensive aetiology for prostate cancer (CaP), and the need for an effective and inexpensive biological treatment modality, devoid of side-effects has resulted in a multitude of therapeutic trials. None of these has been very satisfying, and they have vaned from focal to invasive therapies for CaP. The progress has been delayed and hampered by the lack of any thorough effort to elucidate the cause of the disease. Such efforts would have speeded up the introduction of more rational therapy modalities. The different incidence of CaP in populations aroused our interest to proceed in a more physiologic way to empirically test different functional f factors, since they are non-toxic, although such an approach is consuming time. It was ethical to test the effect of these natural alimentary components, and follow the patients´ reaction and laboratory responses. Huggins and Hodges[1] had already, in 1941, decisively proven that CaP was a hormone dependent disease, although castration alone was not curative. In 1945 Huggins and Scott performed bilateral adrenalectomies[2] after the glands were found to produce DHEA, which could be transformed into testosterone, regarded to have caused recurrent disease. All patients died in a short time postoperatively. However, Huggins did not recognize that this showed the central position the adrenals had in regulating prostate cancer. In this paper we shall review some cases of prostate cancer treated patients. For these we especially follow the shift in FSH, LH, PRL, DHEA, DHEAS, SHBG, plus PSA. The involvement of adrenal glands became evident with an orchiectomized prostate cancer patient with excessively high FSH 120 IU/L and immeasurable PSA. In MRI (magnetic resolution imaging) there was no pituitary adenoma which could explain this high level, but both his adrenal glands were evenly hypertophic. Upon laparatomy the enlargement was due to bilateral zona reticularis (ZR) cell proliferation, while the adrenals other cell structures were normal. These bluish cells with strong green fluorescence had via the hypothalamus stimulated the pituitary to greatly increase FSH and LH, see Case A below with normal laboratory ranges displayed (the row at the top in all cases).

The Importance of adrenal ZR cells is unveiled by: A) their marked proliferation after orchiectomy; B) lack of ZR cells in castrated male pigs [eunuchs]; C) markedly decreased number of ZR cells in patients succumb-bing to CaP; D) the hormonal effect in extracts made from ZR cells of castrated boars; E) rapid lethal out-come if CaP patients after

orchiectomy are also *adrenalectomized*. This operation was conclusively remo-ving the adrenal lifesaving functions of unknown fictive factors; Cycloprostatins No. I (increasing FSH-) & No. II (increasing PRL-levels).

FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	SHBG	PSA
IU/L	IU/L	mU/L	nmol/L	µmol/L	nmol/L	pg/ml	pg/ml	µg/L	nmol/L	µg/L
1-9	2.5-12	50-300	3.0-17.0	0.5-8.0	9-38	~60	300-500	16-253	15-55	<4.0
120	53.8	228	3.2	1.9	0.8	<7.8	330	65	48	<1

Case A.

Cancer can be regarded as a complex metabolic deficiency disease. Nutritional therapy has few negative side effects, as it is a non-invasive treatment. In traditional Chinese medicine feeding patients with exotic herbs could help in curing them. At that time it was impossible to analyse the precise functional factors ingested, but we seem now to have reached an academic form of traditional Chinese medicine since we can include specific pure alimentary components to construct a supportive curative diet. Spontaneous regression of cancer is rare[3], and has been called "The metabolic triumph of the host"[4]. It implies that these patients by chance have ingested a complicated combination of bio-modulating natural components to regain the internal balance in their diseased body. The curative effect does not seem to involve apoptosis[5]. These observations signify that the complex metabolic deficiency triggering cancer, and also genetic weaknesses, can be compensated by feeding patients specific functional alimentary components. Strivings to delineate such metabolic factors has finally, after over 35 years, resulted in the present possibility to improve cancer therapies in a physiologic way - by dietary supplementation. This finding naturally also backs screening tests, since overtreatment can now be avoided. The aim of this long empiric study was actually to decrease the need for expensive invasive treatments, which are all marred by grave side-effects. We will in this article give a short resume´ of how these multiple biological factors were found, over the last decades.

Our cancer therapies usually only try to remove the symptoms by surgery or irradiation, but not to correct the actual aetiology, although the most important aim for cancer treatments should be to strive to avoid recurrent disease. Therefore there is a natural motivation to apply active bio-immunotherapy in many cancer forms. Present treatments are like treating a scurvy patient by extracting his loose teeth but not giving him vitamin-C!

Prostate cancer (CaP) is the most common form of malignancy in men. We find 5,000 cases per year in Finland, with 800 men succumbing to CaP in great pain due to multiple bone metastases. In China and India one million men are diagnosed with CaP every year about 15% dying of it. Last year approximately 300,000 cases were found in the European Union while 200,000 patients were diagnosed in the USA, and 27,000 died of CaP. The yearly casualty rate in Germany is 12,000 men, while in England 10,000, and they pass on in a very painful way from bone metastases. They should all have a chance to be clinically tested whether they have a satisfactory positive response from this physiologic treatment modality before they are remitted to different invasive therapies, which are all expensive, not physiologic, and cause grave side-effects.

Over the years, to facilitate the search, we developed working hypotheses to outline the meta-bolic regulating codes for our three main forms of cancer[5] , schematically presented in Fig 1. The diseased body can take up these multiple components and restore the multi-

factorial balance securing normal health. Basically the elementary compounds are composed by natural organic and inorganic and neurogenic lipid molecules forming the correction of multifactorail deficiency disease/cancer. These components are; amino acids, essential trace-element ions, vitamins and lipids.

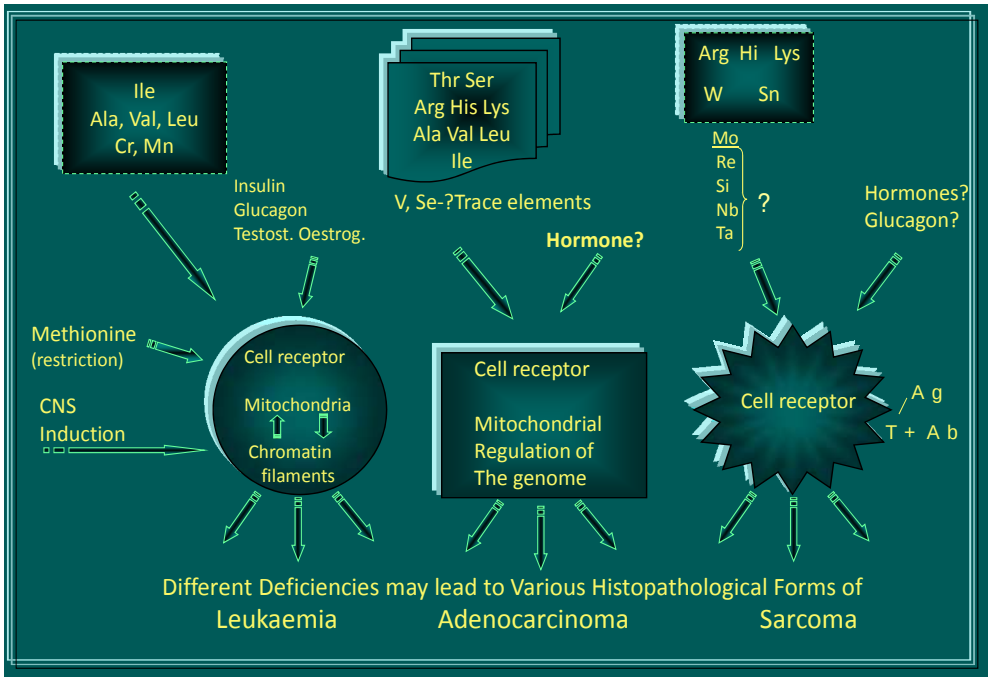

Fig. 1. The hypothetic cell regulatory code for our three main forms of cancer; leukaemia, adenocarcinoma, sarcoma. The combined effect of amino acids, trace element ions and lipids involved in the inductional control mitochondrial Regulation, epigenesist, immune reactions secure normal health.

The incidence of CaP has increased world-wide, and already 5000 new cases were diagnosed in Finland last year, and 800 died in intensive pain due to incurable bone metastases. In China and India 1 million cases are detected yearly, while 300,000 in the European Union, and 200,000 in USA, of which 40,000 and 27,000 respectively succumb to this painful disease. This high incidence has naturally led to a lot of therapeutic trials. None of these treatments for CaP has been completely satisfactory.

An economical, biological treatment modality which does not cause side-effects seems therefore to be urgently needed. This suggested physiologic dietary therapy is inexpensive devoid of side-effects and has the potential to contribute significantly to a comprehensive response to cancer. Curative clinical results obtained by feeding patients with exotic herbs in traditional Chinese medicine have given a positive clinical results, and must be regarded as a clear signal that mammals have a physiologic capacity to reverse malignant cells back into healthy transcription without apoptosis. In modern biotherapy this biological effect can be simulated by a balanced oral intake of the

numerous missing alimentary components, in pure form. They act as a specific functional food. The regulating code is certainly more complex than the iodine deficiency causing endemic goitre. Our studies have indicated that co-operation by several organs is involved with the adrenal glands in a central position[6,7]. Figure 2. These unknown human adrenal biological factors are harboured in zona reticularis cells and they can be activated by feeding the alimentary components,[8] listed in Table 1.

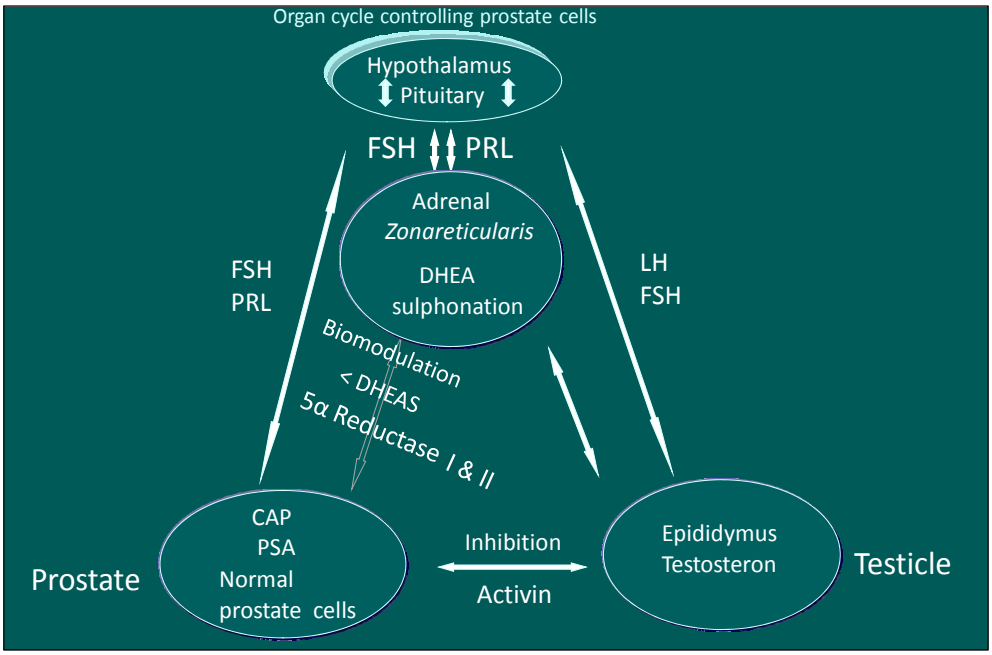

Fig. 2. A schematic "organ cycle" controlling cancer of prostate gland. Adrenal zonareticularis (ZR) cells are in a central position responsible for keeping prostate cells in normal function.

1.1 Present prostate cancer treatments

Prostatectomy, External Beam Radiation therapy (EBRT), Brachytherapy (intensity modulated), Three – Dimensional conformal, Low-Dose Brachytherpy, Cryotherapy, Local therapy, Lumpectomy, Systemic Therapy LHRH analogue implants, ADT androgen depletion therapy, Watchful Waiting,High-intensity focused ultrasound (HIFU) therapy, Vascular - Targeted (VTP), Photodynamic Therapy (PTD) using light-sensitive agents with local laser activation, leading to cell-destruction, Nonspecific dietary therapy (except a la, Tallberg), BMI risk, 5-alpha I & II reductase inhibition (Dutasterid, Proscar, Finasterid), Robotic-Assisted Radical Prostatectomy, Hyperthermia, Immune therapy, Life-style Trial, etc.

1.2 Adverse effects of treatments

Anaemia, Cognitive decline, Depression, Erectile dysfunction /libido loss, Fatigue / General weakness, Gastrointestinal symptoms, Gynecomastia, Bone fractures, hormonally related, Hot flashes, Lipid abnormalities, Osteoporosis, Weight gain/redistribution, Muscle wasting,

Increased fat deposition, General decrease in quality of life, and Marital Problems due to the hormone therapy, Stress, etc.

1.3 Clinical results

A regression of cancer reveal that cancer this complex chronic metabolic deficiency disease, may be cured by non-toxic dietary restoration of the healthy internal bodily milieu of the patient 5[4,5]. Specific mitochondria restore tumour cells to normal healthy transcription without apoptosis or cell destruction[6]. The incentive to try to delineate certain of the physiologic factors entailed in malignant transformation of patients suffering from CaP was bearing successful clinical results obtained with bio-immunotherapy for renal cancer[9], cutaneous and choroideal melanoma[8] in bio-immunotherapy. A positive clinical effect implicated a combined effect caused by specific metabolic dietary substitution, supported by specific active immunological stimulation of the patients' immune defense. The vaccine made from the patient´s own tumour tissue will be directed against autologous antigenic markers, representing the finger print of pertinent tumour markers[9]. For prostate cancer active immunotherapy *is not prescribed* since PSA is a serine protease metabolite, and not a true tumour marker. PSA could decline during immunization but the PSA antibody titer had not sufficient capacity and therefore PSA regained pathologic levels after 8 months[10].

2. Dietary amino acids in attempts to correct the metabolic deficiency causing cancer

The causes of CaP seem rather to be environmental than genetic, and dietary habits have a pronounced effect on prostate cancer incidence. The low incidence of CaP in Japan and Italy may be linked to the local ingestion of the amino acid serine (Ser) contained in soy and parmesan in Italy, further supported by the vitamin-like substance lycopene contained in tomatoes. The original idea that cancer represented a deficiency disease affecting amino acids was presented by Dr Howard Beard, already in 1941- 42[11,12]. He could cause impressive regression of established sarcoma in rats from daily injections of the three basic amino acids (Arg, His, Lys). Our efforts were, therefore, based on studies on the effect of natural non-toxic alimentary components. The effect, based on our rat leukaemia model, showed that the regulatory code was completely different from that of sarcoma. In both experimental models there was, however, a trend effect caused by feeding rats with Threonine (Thr) and Serine (Ser). Our efforts were therefore based on studies on the effect of natural non-toxic alimentary components.

The effect, based on our rat leukaemia model, showed that the regulatory code was completely different from that of sarcoma. If patients ingest the natural amino acid Ser, PSA which actually is a serine protease - the PSA activity declines. The Ser absorbed into the patients´ blood decreases PSA, through substrate inhibition. This is more physiologic and effective than the tyrosinase inhi-bition caused by Glivec. This stimulated our interest to advance in a more physiologic and rational way, selectively feeding patients natural alimentary factors and analysing the effect on CaP, although it would take a long time to delineate pertinent physiologic metabolic factors, linked to the hormonal balance. Huggins and Hodges had decisively proved that CaP was a hormone depen-dent disease, although castration as a single treatment was not curative[1].

Spontaneous cures of cancer are so rare because the etiological dietary deficiency leading to cancer is more complex than the simple lack of iodine causing endemic goitre. The statistical

chance of getting multiple components in the right proportion, and long enough, to compensate a longstanding metabolic deficiency is extremely small without external active help. These positive findings based on dietary effects should lead us to a new paradigm for cancer therapies founded on restoring the physiological internal balance of the body. It has really improved our under-standing and biological means for treatment and prophylaxis of malignant cell proliferation. We are optimistically reforming prevailing toxic clinical treatment modalities, based on mistaken paradigm to kill all cancer cells with cytostatics instead of trying to regain the curative healthy internal balance.

The lack of an effective biological non-invasive treatment alternative, has led to the risk of over-treating patients. Our standard therapies are un-physiologic, and few methods are curative if app-lied in an advanced stage of CaP. The lack of aetiological understanding has led to a multitude of not well founded therapeutic trials. The progress has been hampered by the lack of thorough strivings to elucidate the cause of the disease. Such efforts would have made it possible more rapidly to find rational therapy modalities. The aim of our study, for over 35 years, has therefore been to clarify aetiology and prognostic traits in patients' suffering from different stages of pros-tate cancer. A better understanding of the causes for CaP could improve biological treatments and dietary schedules[13]. Presently, there is a new incentive for screening, since there is now an applicable biological treatment for CaP. In an early stage, while it still is possible to get a positive clinical response[14], patients should be tested for their response to this physiologic treatment modality.

2.1 The importance of administering trace-element ions

In Dr.H. Beards original studies[11,12] causing complete regression of experimental rat sarcoma from daily injections of 18 mg of all three basic amino acids (Arg, His, Lys). Regression of sarcoma stemmed from a complex formed between the basic amino acids and wolfram (W) ions. A related signal system was found with experimental leukaemia. In mammals the required trace-element ions were different, Cr and Mn formed the regulating signal with Ala, Ile, Leu, & Val preventing induction of leukaemia. In both these studies there was a positive trend effect with Serine and Threonine, hinting that the adeno-carcinoma regulatory code could comprise supplementation with Ser to male patients, while Threonine may be involved in female adeno-carcinoma. The PSA level decreased most likely due to substrate inhibition caused by the thus increased level of the natural amino acid Serine in the patients´ serum neutralizing his protease enzyme (PSA). We got very valuable support from Prof. Klaus Swartz the head of the only trace-element free laboratory in the world in Los Angeles. He kindly extrapolated from his rat experiments how much a minimal daily amount of essential trace-element ions a human 70 kg body would need. The mg amounts and ionic form listed in Table 1 are based on his suggestions.

An interesting observation was that Strontium may have been involved in regression of bone metastases. It was revealed when the ash a patient ingested, whose bone metastases had regressed, were analyzed for metals by proton-induced X-ray emission. It revealed that he had got 7mg of strontium (+ rubidium and 40 mg Zn daily), in addition to the trace-element ions we originally used (Table 1). Radioactive strontium was earlier used to scan bone-metastases. If repeatedly used it did not work because only ten percent of the i.v. bolus (of 85 mg) was actually radioactive. The non-active strontium molecules could block the tumour cell receptors and render the scanning unreliable. This is why we presently use technetium. It hinted that Sr played a role in the healing system of bone metastases in CaP

patients[7,15] . Vanadine and Arginine (V & Arg) had previously been shown to arrest bone metastases in renal cancer cases[16].

Furthermore, Gly and Glu were fed to act as substrate inhibitors and prevent inflammatory reactions in the prostate gland (chronic Pin) caused by splitting the tri-peptide glutathione activating the inflammatory leukotriene cascade (Figure 3). Boron (B) is also an inhibitor of Gammaglutamyl transpeptidase which splits glutathione and may thus prevent sterile inflammatory reactions caused by the Leukotriene - B_4, C_4, D_4 & E_4 cascade - ending in the slow reacting substance of anaphylaxis (SRS-A)[17].

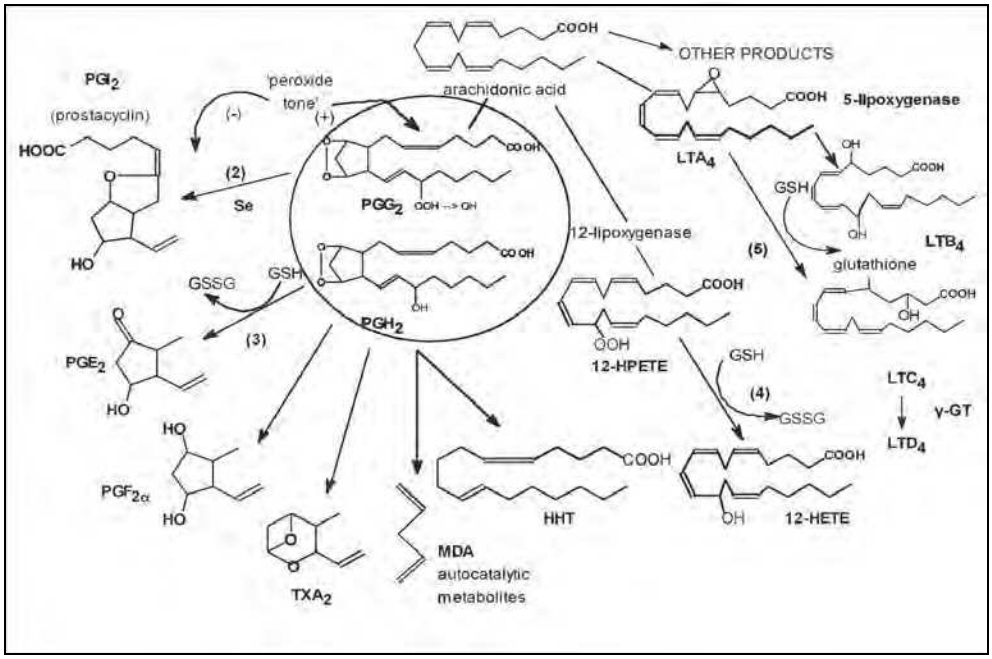

Fig. 3. Possible roles of the tri-peptide GSH in arachidonic acid metabolism (1 to 5). GSH reduses PGI_2 and increases PGE_2 formation. PGE_2-forming isomerases (3) require GSH as essential cofactor (cosubstrate). PGs are involved in the hyperalgesia of inflammation[31]

Molybdate (Mo) had an effect on the female menstrual cycle[13] to make it completely regular, and could perhaps activate a minute oestrogen production even in male patients. Due to side-effects oestrogen therapy for CaP has been stopped but a small physiologic stimulation may be beneficial? Vanadate together with Arg seemed to prevent and cure bone metastases in renal cancer patients and were therefore included as a possible natural co-factor complex related to the renal tissue.

2.2 The importance of administering lipids (CNS) to cancer patients

The enormous abundance of lipid molecules in the central nervous system (CNS) suggests that their role is not limited to be structural and energetic components of cells. Over the last decades, some lipids in the CNS have been identified as intracellular signalers, while others are known to act as neuromodulators of neurotransmission through binding to specific

receptors. Neurotransmitters of lipidic nature, currently known as neurolipids, are synthesized during the metabolism of phospholipid precursors present in cell membranes. That central nervous lipid molecules were involved in keeping malignant cells in healthy transcription was observed already in the seventies[11]. CNS-lipid molecules were detected by thin layer chromatography to be present in the serum of cancer patients, following Herpes virus infections. The viral infection had caused lesions in the blood-brain barrier, and vital lipids had leaked out into the patients´ blood. The minimal idea was to try to compensate this depletion by feeding patients with the lipids they had lost to corrected the depressed enzyme activity, on the patients´ buffycoat cells in three days[17] and restored his natural immunity [lymphopoiesis] thanks to the activity of an un-identified titanium containing CNS-lipid molecule. The importance and vital function of millions of CNS-lipids the "lipidome" has constantly been overlooked. They have a variety of crucial functions, from embryogenesis to securing healthy mental and motor balance as presented earlier [13]. One of the most important factors is this Ti containing CNS-lipid, which stimulates the patients´ immune reactions and alleviates neurologic pain. These CNS-lipids can be absorbed by nerves from the serum and normalize neuronal function. Millions of vital lipids present in the CNS can preserve the inductional control over all cells in a cancer patient. Pain is actually a warning signal that the nerve tissue cannot produce certain CNS-lipids, but when these become available through ingestion they circulate in the blood and are absorbed by the nerves, while decreasing the patients´ distress. Following alimentary correction of the metabolic deficiency activated cell specific mitochondria can then be seen, by electron microscopy, to force cancer cells to regain a healthy form, without apoptosis[18, 19]. This Bio-Immunotherapy method has successfully been applied in randomized prospective studies in hundreds of patients suffering from melanoma (103 skin + 54 eye)[12], or metastasized renal cancer (127 cases)[15,16] a study was performed at Helsinki University[20].

3. Mitochondrial involvement linked to the healing reaction, without causing tumour cell lysis

The effect of specific dietary supplementation for prostate cancer is usually fairly rapid and seen in 6-8 weeks; the laboratory profile is shown here. These transformed mitochondria can be revealed since they become electron dense (black) because the enzymes in their crista gather significant amounts of metal ions [Cr, Fe, Zn, Ti, & Rb] when they become activated[6,18].

FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	SHBG	PSA
IU/L	IU/L	mU/L	nmol/L	µmol/L	nmol/L	pg/ml	pg/ml	µg/L	nmol/L	µg/L
1-9	2.5-12	50-300	3.0-17.0	0.5-8.0	9-38	~60	300-500	16-253	15-55	<4.0
4.2	4.5	269	2.1	1.5	5.7			130	61	53.7
7.8	6.9	151	< 2.0	1.3	11.1			149	55	3.7

Case B. Typical laboratory profiles of a patient on specific functional dietary supplementation. FSH and Testosterone increase at the same time as PSA, DHEA, & DHEAS declines, indicating a good prognosis.

A biopsy taken from same lobe as previously showed a Gleason score of 7, revealed in EM that the tumour cell nucleus was surrounded by transformed mitochondria, Figure 4. Two

of them empty their electron dense activated enzymes into the nucleus when PSA had decreased to 3.7 µg/L, and he was clinically in good condition [Magnification x 10,000.]. They healed without apoptosis. If the initial PSA level is over 15 ng/mL the therapy has usually been started with combined hormone (e.g. LHRH Zoladex 3.6mg for months duration) supplemented by dietary measures. When PSA decreases rapidly as in Case B, following supplementation with both prostate powders (No1 & No2) in synergy with intermittent total anti-androgen blockade (Zoladex, 3.6mg + Androcur, 50mg x 2/day, for only 10 days) the PSA-level fell also rapidly from 34.3 ng/mL to 2.3 ng/mL in six weeks. In EM the tumour cell nucleus is often seen to be surrounded by transformed mitochondria which are electron dense, due to the increased content of Cr,Fe, Zn, Ti, & Rb [18] when the tumour progress is arrested Similar mitochondrial transformation with activation in the curing phase of mammalian malignant cells, triggered by this physiologic non-toxic biologic therapy has been seen in EM with experimental animal models (rat & horse[13]), in addition to episodes with human patients suffering from melanoma, and malignant histiocytoma[18,22]

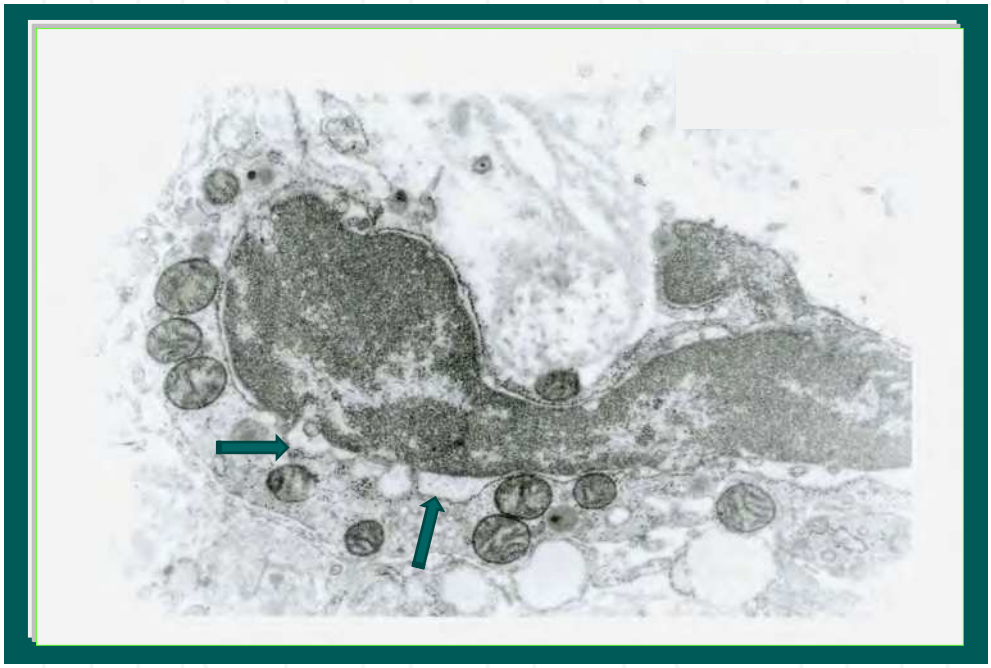

Fig. 4. Electron microscopy showing (magnification 10.000) with electron dense organ specific mitochondria Surrounding the tum cell nucleus when PSA normalized. Two mitochondria seem to empty their electron dense material into the nucleus when the patient was cured, without apoptosis.

4. Adverse effects caused by certain biological components

Alanine administration may increase the PSA-level and food items high in Ala should be avoided. Administration of DHEA 25mg per day is definitely contraindicated since it led to intensive pain and the patient died in three weeks from multiple bone metastases. Hormone

refractory prostate cancer (HRPC) is not due to the emergence and selection of a hormone refractory cell clone. It seems to be the result of a too intensive LHRH treatment (continuous injections with 3 months duration) when the hormone therapy has been used to reach PSA nadir. Instead FSH and LH have decreased to <0.1 IU signaling adrenal feed-back exhaustion involving adrenal ZR cell function. The pituitary is not malfunctioning as PRL can increase to > 1000 mU/L. A brooding HRPC is seen when PSA, is forced to levels under 0.9 μg/L, due to excessive anti-androgen therapy, while PRL exceeds 600 mU/L indicating that the pituitary is not exhausted.

5. Attempts to actively increase FSH and PRL in patients, or the use of autologous vaccines failed

The positive prognostic effect of increased FSH- and prolactine (PRL) levels led to attempting active stimulation. Ingestion of extracts made from ZR cells of castrated male pigs can cause a marginal positive effect on FSH and PRL for a short time but no human forensic material was available for testing. Forensic human adrenal ZR-cell extracts should be tested as they probably contain these biologic stimulatory factors. If they could be characterized it could lead to a natural substitution therapy for CaP, like insulin is for diabetics.

Daily Injections with human FSH (75 -150 U/L) could increase the FSH-level for a short time, but would be impracticable in the long run, and too expensive. FSH-releasing factor was even less effective. Then again injection of human prolactin PRL or HCG had only a marginal effect on PSA. Attempts to externally increase FSH or PRL-levels did not seem to be functional. Physiologic stimulation directed at the adrenal glands appears to be the only practical therapeutic way.

Immunisation using autologous tumour polymer particles can decrease PSA-levels for some months but is not curative because PSA is only a metabolite, and not a regular tumour marker[10].

5.1 Description of some prognostic features in cases of specific dietary supplementation for CaP

The most important laboratory test to be taken immediately when CaP is suspected, or actually diagnosed, is to analyze the serum levels of FSH, LH, PRL, DHEA, DHEAS, SHBG, and f/t PSA. PSA as a single marker is not alone sufficient, since it can be held down by an increased FSH- or PRL-level. A high FSH-level and low DHEAS with decreasing PSA is a good prognostic sign, and this effect caused by alimentary supplementation is usually fairly rapid. In one patient of the earliest cases found by screening tests his PSA rose from 4.3 ng/mL to 6.4 in two months with a Gleason of 7. He was then routinely scheduled for prostatectomy. With informed consent he preferred to test this biological alternative, safeguarded by regular PSA controls, and immediate change back to the invasive treatment option if his PSA rose. On a double dietary daily dose and CNS-lipids, his PSA declined to 2.3 ng/mL in half a year, followed by diminished urinary distress, and without any side-effects. He opted then to take the prostate supplements only once a day. In half a year his PSA had once more started to rise and the double dose was reinitiated. The dose response was again positive, as his PSA-level declined to normal levels. His CaP became then stable for 6 years in continuous dietary therapy, but a slight increase in his PSA-level was seen (from 6 – 7 ng/mL over four months), when Arg substitution had been stopped for some

time. This was to test if this component really was necessary to keep his PSA-level stable. The urologists persuaded him then immediately to accept prostatectomy, although we would have preferred to reinstate Arg to see if it would reduce PSA anew. His prostatectomy specimen showed that his Gleason score had declined to 4, and no growth of the prostate tumour tissue had occurred. This unnecessary operation stopped anyhow further biological treatment for CaP, as all tumour cells had been removed. We did not pay sufficient attention to the fact that this patient had, before his prostate cancer was diagnosed, also suffered from Crohn´s disease, however, most clinical symptoms had almost disappeared during his biological treatment for CaP[23]. Four years later his symptoms of Crohn´s disease of Crohn's had recurred, and he was consequently operated for colon cancer. This awoke our interest for sterile inflammatory diseases like, ulcerative colitis, rheuma, fibromyalgy, psoriasis polyarthritis[24] etc. The probable common denominator was the effect caused by supplementation with CNS-lipids which we had included to quell chronic prostatitis as a precancerous state for CaP. Administration of this lymphopoietic, Ti containing CNS-lipid molecule was aimed to normalize inflammatory reactions with the aid of this vitamin-like CNS factor, which now also seemed to affect other sterile inflammatory diatheses, as mentioned above.

In some patients the effect of a sole dietary supplementation is slow and protracted as shown in Case C. In this case ingestion of prostate powders (our cocktails) 1/day, alternatively 2/day, and CNS-lipids increased slowly the patients FSH- and LH-levels & constantly low DHEAS and rising testoteron while PSA is low, in the normal range. Dietary supplementation alone has kept him in stable disease, now for over five years. His starting Gleason score was 8.

	FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	HBG	PSA
	IU/ L	IU/ L	mU/ L	nmol/ L	µmol/ L	nmol/ L	pg/ ml	pg/ ml	µg/ L	nmol/ L	µg/ L
	1-9	2.5-12	50-300	3.0-17.0	0.0-8.0	9-38	~60pg /ml	~500pg /ml	16-253	15-50	<4.0
2005	5.4	<0.01	48	5.5	1.5	4.9			455	77	<0.2
2006	8.4	2.5	83	3.2	1.2	5.6			376	82	<0.2
2007	13.8	6.2	114	3.5	1.0	10.2			313	61	0.22
2010	20.5	12.3	103	3.5	1.1				347	60	0.49

Case C.

A positive reparative reaction seems to be flagged by an increase in the FSH- LH- and prolactin (PRL) levels. Revelation of these unknown stimulatory factors could form a decisive prevention for the emergence of a "hormone refractory state", leading to improvement.

Case D. This patient was succumbing to CaP, 11.1996, [T4NxM1] with multiple bone met - 11/94] had orchiectomy -94, three years earlier. Initially PSA 92.6 decreased after orchiectomy 1994 to 5.7- 5.3 (-95) in combined bio-modulation but without Sr. He stopped the intake of all dietary components and became androgen independent (-96). PSA rose to

119 ng/mL, and he died showing extreme laboratory values, as measured from a blood sample taken the same day before his exitus.

FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	SHBG	PSA
1-9	2.5-12	50-300	3.0-17.0	0.5-8.0	9-38	~60	300-500	16-253	15-55	<4
<0.1	<0.1	1060	7.2	6.8	0.6				2145	364

Case D.

The marked increase in PRL affirm that the pituitary function is not depressed and it is feverishly trying to save the patient, but without a concomitant FSH increase the case is lost.

His laboratory values have oscillated during all these years but conspicuously FSH has stayed high all the time indicating a continuous adrenal feed-back reaction which could involve in preventing recurrent disease.

FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	SHBG	PSA
IU/L	IU/L	mU/L	nmol/L	µmol/L	nmol/L	pg/ml	pg/ml	µg/L	nmol/L	µg/L
1-9	2.5-12	50-300	3.0-17.0	0.5-8.0	9-38	~60	300-500	16-253	15-55	<4.0
67-30	37-16	159-95	< 2.0	< 0.8	1.0	< 7.8	330	109-99	58-61	< 0.1

Case E. CaP detected with PSA 30 µg/L with multiple bone metastases. Orchiectomy was performed 1992. Dietary bio-modulation + Strontium (7mg/day) started 1993. His periostal pain subsided in four months. All bone metastases disappeared 1996. He is now in excellent clinical condition 2011.

In patients with hurting bone metastases to get these to regress they may need to be orchiectomized if during the dietary substitution they have recurrent bone metastases. After castration the bone metastases could totally regress in half a year. This improved clinical result after recurrent CaP may signal that some factor in the testicular tissue is produced which during a prolonged biological therapy may arrest the positive effect of the full dietary substitution although it supplies many of the required factors; Sr, V, Ser, Arg etc. If this is required in selected cases for s cure it may be worth the castration, a fairly simple operation compared to prostatectomy, since the survival time of CaP with multiple hurting bone metastases is otherwise only 8 months [?].

Of the more than ten patients who were orchiectomized , all who not have got any adjuvant supportive dietary treatment have died. Some of them lived up to 8 years after castration, which is better than Huggins and Hodges got in the early forties. On the contrary, castrated patients who continuously have got our supportive metabolic functional alimentary factors are alive in good clinical condition, as well as those cases that have not been epidydectomized, but have been on dietary supplementation in synergy with short time intermittent hormone treatment. In certain cases the continuous hormone treatment with LHRH has been possible to stop completely, without leading to recurrent disease, with follow-up presently already over 5 years, as in Case B.

5.1.1 Short time intermittent hormone treatment blocks hormone refractory prostate cancer induction

Short time intermittent LHRH hormone treatment is definitely recommended as it has prevented the development of hormone refractory states.

FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	SHBG	PSA
IU/L	IU/L	mU/L	nmol/L	µmol/L	nmol/L	pg/ml	pg/ml	µg/L	nmol/L	µg/L
1-9	2.5-12	50-300	3.0-17.0	0.0-8.0	9-38	~60pg/ml	~500pg/ml	16-253	15-50	<4.0
(Short time intermittent androgen ablation treatment with Zoladex 3.6mg + cyproteronacetate)										
15.2	16.1	993	3.8	2.4	6.4	75	410	1500	45	13.4
(Three months after Androcur 50mg x 2/day for 10 days to avoid flare-up after Zoladex 3.6mg injections.)										
4.2	7.3	1490	2.5	1.6	5.8	72	430	1100	2.5	
(Three months later when the activated adrenal feed-back has time to increase FSH and sometimes also PSA)										
15.2	17.7	1520	2.2		< 0.8	76	500	993	14.5	

Case F. Lab assay profiles during recommended short time intermittent LHRH treatment, Zoladex 3.6mg,+ Androcur 50mg X2/day with many months intervals, in synergy with specific dietary supplements.

Zoladex 3.6 mg inj can then be repeated, if also PSA has increased as in this case. If PSA stays low Zoladex would not need to be repeated as it stays low due to synergy with supplementation. One should not strive to reach a PSA nadir. Avoid injection of Zoladex 10.8mg every third month becau-se the adrenal feed-back will be exhausted, and FSH declines to < 1.0ng/mL. This is not due to pituitary dysfunction because PRL can start to increase when the body in vain is trying to defend itself (see Case C). This ominous turn is not due to selection of a hormone refractory cell clone, but to ZR exhaustion, as the adrenal glands have not been allowed to function during a necessary intermission in the hormone therapy. If the FSH-level is depressed to under 1 ng/mL it is a signal that the adrenal feed-back has been exhausted, but not the pituitary since PRL can be markedly increased (600 – 1060 mU/L), indicating a brooding HRPC.

Growth factors, activin & inhibin. Dramatic changes were seen after castration, during pregnancy and estrogen substitution therapy. CaP diagnosed by soft tissue biopsies had also specific profiles Inhibin and activin patterns changed dramatically in patients detected from soft tissue metastases, and not from biopsies of their prostate gland. A shift characterized by a very depressed inhibin level was seen in castrated patients, as well as during normal pregnancy, and in healthy females on estrogen substitution.

CaP patients diagnosed from the presence of *soft tissue metastases represent a special from* of prostate cancers (possibly neuroendocrine?). They may have markedly high serum activin-levels ((1890- 2180 pg/ml) paired with low inhibin-levels (25-34 pg/ml). Initially their serum ferritin levels are high (1164-2499 µg/ml), while DHEAS (< 0.8 – 1.8) and DHEA (2.2 - 2.4) are low. FSH-levels are normal or low (1.9 -5.1 IU/L) while LH-levels are undetectable (<0.1 IU/L). Case G. had a PSA-level of 10070 µg/L, while the other similar case showed a PSA of

246 µg/L. In both patients their PSA decreased to 5.3 µg/L, and 2.1 µg/L respectively in half a year, following intermittent short cycle LHRH therapy, in synergy with bio-modulating dietary measures. The high activin levels were not appreciably affected by the therapy, while PSA was decreased markedly, to normal levels.

FSH	LH	PRL	DHEA	DHEAS	Testost	Inhibin	Activin	S-Ferrit	SHBG	PSA
IU/L	IU/L	mU /L	nmol /L	µmol/L	nmol/ L	pg/ml	pg/ml	µg/L	nmol /L	µg/L
1-9	2.5-12	50-300	3.0-17.0	0.0-8.0	9-38	~60pg/ml	~500pg/ml	16-253	15-50	<4.0
5.1	<0.1		2.4	1.8	<0.8	25	2900	1164		10070.0
combined hormone and dietary therapy led to CR of metastases (CR) and to normal PSA										
6.2	<0.1					22	2650	316		5.3

Case G. CaP was detected from neck metastases and he had also bone metastases. A special characteristic was extremely high activin- and PSA-levels 10,070 µg/L which decreased to 5.3 µg/L, in 6 months following combined intermittent short time LHRH plus dietary therapy. All soft tissue metastases regressed and he was in good health.

His extremely high activin 2900 and PSA-level of 10,070 µg/L, decreased in intermittent short time hormone therapy to 5.3 µg/L, in 6 months in combined LHRH + biological therapy. Patients don't die of high PSA, but of depressed FSH (< 0.1 ng/mL, due to adrenal feed-back exhaustion).

Orchiectomized CaP patients generate an increase in their FSH-levels to, 40 – 130 IU/L, in some months, but show also a characteristically manifestly *depressed serum inhibin level (<7.8 pg/ml)* while their activin level (~ 560 pg/ml) is normal. Surprisingly healthy *pregnant females revealed a similar change* in their inhibin / activin levels, with a correlation value of 1:70, until parturiency when the "growing cell-mass" – the healthy child is borne and the related value again becomes normal. Alarmingly also estrogen substitution (already 50 µg plasters) in postmenopausal women can show a similar dramatic change in their inhibin / activin correlation values. Such a provoked change in a females serum growth factor levels – usually actively made to *last more than nine months* by their gynaecologists – could easily fool the body that it should produce growing cells. This anomalous change may explain the observed increased breast-cancer and lymphoma incidence generated by estrogen substitution therapies in otherwise healthy females. The effect of *estrogen substitution therapy*, prescribed to any female patient, *should therefore obligatory be monitored by inhibin / activin assays,* to see *if it causes a reaction simulating pregnancy,* since it may physiologically be misinterpreted, and consequently generate proliferating (malignant?) cells.

The growth factor "activin" is appreciably increased (5-6x) in the serum of patients suffering from a rare special form of CaP, primarily diagnosed from soft tissue metastases, and not from the prostate gland. Strontium (Sr) is an essential component of the periost, and this trace-element is involved in curing bone metastases caused by ingestion of Sr 7mg/day. It

has eradicated multiple metastases, now with a follow-up of over eighteen years - without recurrent disease. Ingestion of DHEA is contra-indicated since it can activate CaP. Autologous vaccines applied for CaP is not cura-tive and has only a short-time effect, since PSA is not a regular tumour marker but a metabolite.

5.2 Unfavorable clinical events

Excessive hormone treatment is unfavorable, e.g. LHRH analogue treatment using continuous injections with three months duration (> 10mg) in an effort to reach a PSA nadir exhausts the adrenal feed-back reaction and causes HRPC. FSH will then also decline to < 1.0 ng/mL. Excessive needle biopsies (12 -24 cores) may spread malignant CaP cells and could be the reason for the high recurrency rate of 35 -40 % following prostatectomies [25]. The use of specific serum markers [26], MIR, PSA velocity etc. should decrease the need for un-necessary cores, and the few biopsies actually needed should be directed more precisely. The side effects with pain, and caused inflammations would diminish. This biological treatment schedule would anyway be the same disregarding the numbers of cancer focuses in the different lobes of the prostate gland.

The dietary effect which increases FSH is sometimes protracted. In Case E. it took 9 months of continuous prostate powder ingestion to cause an increase from 5.4 to 8.4, and after 5 years it had risen to 20.5 IU/L. The response to dietary therapy is **fairly progressive** so that one can judge, in months, if the patient will respond to this specific dietary supportive therapy based only on standard laboratory tests, FSH increases and DHEAS decreases etc. Initially increased FSH or PRL-levels are good prognostic signs and should always be analyzed when CaP is found by screening tests, since invasive treatments can be avoided. The human adrenal biological factors harbored in the zona reticularis (ZR), fictively called cycloprostatin I & II, should be purified from forensic healthy human adrenal material aided by assays for their stimulatory effect on the pituitary. The clinical use of such purified factors could form a biological compensatory medical treatment for CaP.

1.	Oral administration of each (2-5g/day) of respective L-amino acids; Arg, Asp, Glu, Gly, Lys, & Ser, all in connection with meals.
2.	Essential trace-element salts prescribed orally as biologically active ions, at dose levels of some milligrams (1-3mg/day); Chromium ($CrCl_2.6H_2O$) 6mg (=1.17 mg Cr), Molybdenum ($Na_2MoO_4 .2 H_2O$) 4mg (≈ 2mg Mo), Rubidium ($RbCl_2 . 2 H_2O$) 1-10mg (≈ 7mg Rb), Tinn ($SnCl_4.5H_2O$) 4mg (=1.35mg Sn), Strontium ($SrCl_2$) 1-7mg (≈ 4mg Sr), Vanadine ($Na_2VO_4.4 H_2O$), 6mg (= 2.5 mg V), Wolfram ($Na_2WO_4. 2 H_2O$), 4mg (=2.3mg W), Zink (= Zn 30mg).
3.	Physiologic dosages of vitamins; A,B,C,D,E,K , folic acid and lycopene.
4.	To improve lymphopoiesis and the immune-defence of patients a diet containing prion-free neurologic lipids (micro-capsulated CNS-lipids).
5.	Dose-levels are adjusted based on the clinical response as measured during the therapy, and correlated to the patients´ body weight.
6.	Transformed organ specific mitochondria participate in the curing phase of CaP.

Table 1. As Cancer is a complex metabolic deficiency disease it is curable by dietary supportive measures.

5.2.1 Un-interrupted hormone therapy may cause HRPC, avoided by short time intermittent treatment

The effect of hormone therapy for CaP, in recommended intermittent short time pulses combined with metabolic bio-modulation activates a feed-back reaction recorded as characteristic changes in the laboratory response profile, with FSH, LH and/or PRL increase trailed by DHEAS and PSA decreases, a reaction in which adrenal ZR cells seem to have a central regulatory function. Orchiectomy will cause FSH to increase, further accentuated by prostatectomy. Prostate cancer patients die in a short time if orchiectomy is followed by adrenalectomy which attests the importance of functional adrenal glands. A dramatic rise in activin levels is recorded in a special form of CaP, diagnosed in biopsies from soft tissue metastases. Inhibin is again remarkably decreased following orchiectomy. A similar depressed inhibin level was surprisingly also seen in normal pregnancy, and during the popular estrogen substitution therapy. If estrogen substitution in postmenopausal females cause a growth factor shift mimicking pregnancy her body may not understand that cells, especially malignant cells, should not be allowed to multiply. The increased breast cancer incidence may possibly be connected with this unnatural shift in the levels of these, inhibin/activin, growth factors [13,15]. Patients on estrogen substitution should be tested for these growth factor levels to see if they inadvertently have been rendered to belong to a risk zone for malignant cell proliferation?

Complete regression of CaP shows that this complex posses chronic metabolic deficiency disease can be treated by non-toxic dietary restoration of the healthy internal milieu in the patient activating organ (cell) specific mitochondria to restore normal healthy transcription of malignant cells without apoptosis or cell destruction as seen in Figure 4. The improvement could be due to activation of cell/organ specific mitochondria which regulate the genome, and can force oncogen transcription back to a normal healthy form[21, 22]. This was first seen in rats in which induction of leukaemia was prevented by activated, electron dense cell-specific mitochondria functioning at body temperature [18]. They were found to lose their inductive regulatory potency if stored at +4 ^{0}C (because we are warm-bloodied?). The enzymes in mitochondrial crista were activated by metal ions; Cr. Fe, Zn, Ti, Rb, and this metal increase made them electron dense, and possible to observe by EM[22].

5.2.2 Mitochondria inducing healing, correcting mutations, activation of stem- cells & transplantation

Most of our standard treatments represent a poor practice alternative often in the form of a toxic therapy for a complex metabolic deficiency disease, and by only removing the symptoms, which make patients prone to suffer recurrent disease in 35 -40% even after prostatectomy. Patients who after the operation later on start to show a biochemical increase in PSA, supposedly due to a non-radical operation, or to the active spread of malignant cells by excessive biopsy cores (12-24). A chronic inflammatory (Pin) reaction may be induced by these bloody interventions, and any inflammatory reaction is potentially carcinogenic [23,24] , like we see e.g. as a sequel of the lack of a lymphopoietic central nervous lipid molecule - linked to titan - resulting in an aberrant immune-response as in; Crohn´s disease, ulcerative colitis, rheuma, psoriasis, & fibromyalgy [24,30] etc. This physiologic deficit can be compensated by the intake of CNS-lipids (and/or butter) [23,24,30]. If these lipid precursors are present in the patients´ blood the depletion caused by daily stress can be compensated during our sleep.

A well directed biopsy core may suffice to evaluate the Gleason score. This functional dietary treatment will principally be the same disregarding the number of actual CaP focuses.

6. Conclusion

The primary effort of this long study was to characterize etiological factors for CaP before starting any big randomized series. Instructive features compiled from approximately 70 patients suffering from different forms and stages of CaP were followed-up for decades. This has resulted in a recommendation to apply these findings in a biological treatment modality. Understanding of the nature of that particular tumour it can help us to optimize therapy or to design therapeutic approaches. Patients after prostatectomy may not respond as well to dietary supplementation with activation of the adrenal ZR feed-back cycle, since this organ cycle may require or involve also normal pros-tate cells to be fully effective. Probably prostatectomy should not be performed before one has had time to evaluate the patients´ responses to this physiologic bio-modulating treatment. The positive clinical effect of continuous dietary treatment is prolonged, and is today already extending over 19 years in a case who initially suffered from multiple hurting bone metastases[13] . This beneficial adrenal fed-back activation has continuously been stimulated by ingestion of the biological factors listed in Table1.

An efficient biological treatment modality devoid of side effects and economical, will give scree-ning for CaP a new rational, since the progress of the disease can now be arrested and even bone metastases be cured in a physiologic way. The dispute over the marginal improvement in survival rates between patients on Watchful Waiting over cases that are prostatectomized could be missed by the fact that neither group has received any biological supportive treatment to compensate the actual aetiological metabolic deficiency. And thus the surgical removal of symptoms of CaP (i.e. prostatectomy) does not censure the aetiological deficiency[7, 15].

The importance of mitochondrial function, linked to the memory of the nucleotide sequence in the chromosomes they have created [21] can explain how identical mutations in both chromosomes can lead to that 10% of the off-springs have lost the mutation, and are healthy. This does not require a change of Mendel´s law since mitochondrial memory could explain this surprising result as they correct the mutation during replication [28] ! Correction of the fault can occur when mitochondria have a memory of what they have created [21,27]. The interesting study by Hohlfelds group on paediatric skin burns may be explained by the possibility that cell specific mitochondria transgressed from the male skin transplant they used into the female receptor-cells, whereby the full genome present in any of her cells could transform into her own skin – a "transplant" which cannot be rejected. This represents a further step of refined "stem-cell" activation, which is not hemmed by restrictions presently affecting stem cell studies [28] . To efficiently learn to use *cell-specific mitocho-ndria and epigenesis* in biology and medicine will be the scientific challenge of this century!

7. Aknowledgements

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8. References

[1] Huggins, C.; Hodges, C.V.: Studies on prostate cancer: I. Effect of castration, estrogen, and androgen injection on serum phosphatases in metastatic carcinoma of the prostate. Cancer Research 1 (1941) 293-97.

[2] Huggins, C.; Scott, W.W. Bilateral adrenalectomy in prostate cancer. Ann Surg. 1945: 22, 1031-41.

[3] Everson T.C. Spontaneous regression of cancer. Ann NY Acad Sci. 1964:114; 721-35.

[4] Cole W.M. Spontaneous regression of cancer. The metabolic triumph of the host. Ann NY Acad Sci 1974:239; 111-15.

[5] Tallberg T. Cancer treatment, based on active nutritional bio-modulation, hormonal therapy and specific autologous immunotherapy. J Aust Coll Nutr & Env Med. 1996: 15 No.1, 5-23.

[6] Tallberg T. Biological cancer therapy, its effect on inductional control, triggered by hormones and mediated by transformed mitochondria. J Austr Coll Nutr & Env Med. 1998: 17; 17-24.

[7] Tallberg Th. Studies on cancer of the Prostate Gland, a search for Aetiological and Prognostic Factors. J Aust Coll Nutr & Env Med. 2003:22 No.2; 11-16

[8] Tallberg T. A Possibility to Prevent Recurrent Melanoma, Renal, Breast and Prostate Cancer Utilizing Inexpensive Powders Containing Specific Dietary Supplementary Factors. J Aust Coll Nutr & Env Med. 2005:24 No.3; 3-9.

[9] Tallberg T. Development of a combined biological and immunological cancer therapy modality. J Aust Coll Nutr & Env Med. 2003: 22 No.1, 1-20.

[10] Tallberg Th., Klippel K.F. Effect on PSA from combined therapy with LHRH-analogue, specific immune- therapy, and active biomodulation. Fourth Eur Urol Winter forum. Abstr. Davos Feb.19-25th 1995.

[11] Beard H.H. The effect of parental injection of synthetic amino-acids upon appearance, growth and dissapperance of emge sarcoma in rats. Arch Biochem. 1942:1; 177-85.

[12] Beard H.H. Effect of subcutaneous injection of individual amino-acids upon the appearance, growth and disappearance of Emge sarcoma in rats. Exp Med Surg. 1943:1; 123-35.

[13] Tallberg Th. Regulation of cancer by therapeutic vaccination and dietary bio-modulation involving organ specific mitochondria. Int J Biotechnology. 2007: 9, ¾; 391-410.

[14] Thomas Tallberg and Mervi Dabek. Prostate Cancer, Aetiological, Therapeutic, Prognostic and Prophylactic Factors. Anticancer Research. 2008 :28 No.5C; Abstr.658, 3507-3508.

[15] Tallberg Th., M. H. Dabek. Dietary substitution therapy of prostate cancer patients: A possible nonivasive treatment. Int J Trends in Med. 2011: 1; 21-27.

[16] Tallberg Th., Tykkä H., Mahlberg K. et al. Active specific immunotherapy with supportive measures in the treatment of palliatively nephrectomised, renal adenocarcinoma. 1985: 11; 233-43.

[17] Tallberg Th., Tykkä H., Halttunen P. et al. Cancer immunity. The effect in cancer immunotherapy of polymerized autologous tumour tissue and supportive measures. 1979: 39; Suppl. 151. 1-35.

[18] Tallberg, T., Stenbäck, H., Hallamaa, R., Dabek, J., Johansson, E., Kallio, E. Studies on mitochondrial regulation of the genome. Deutsche Zschr Onkol. (German J Oncol) 2002: 34; 128 -39.

[19] Tallberg Th., Hallamaa R. Cancer Regulated by Organ-Specific Mitochondria Via Lipidomics, Genomics and Proteomics. Anticancer Res. 2008: 72 No.5C; Abstr.659

[20] Tykkä H. Active specific immunotherapy with supportive measures in the treatment of advanced palliatively nephrectomised renal adenocarcinoma. A controlled clinical study. Scand J Urol Nephrol 1981, pp. 1- 107.

[21] Tallberg Th. Mitochondria seem to regulate the genome in the chromosomes they have phylogenetically created. Trends in Biomedicine in Finland 2000: ISBN 951-98382-1-X, pp. 36-38.

[22] Tallberg Th., Stenbäck H., Dabek J., Palkama A. Complete disappearance of human malignant histioc- cytoma cells following dietary biotherapy, leading to activation of inductional control mediated by mitochomdria. J Austr Coll Nutr & Env Med. 1996: 15 No 2 ; 5-10.

[23] Tallberg Th. Lipidomics, the function of vital lipid molecules forming our brain and spinal cord. J Trends in Biomed. 2008: 3 No1; 6-19.

[24] Tallberg Th. Biological dietary treatment of inflammatory bowel disease. The XL Nordic Meeting of Gastroenterolgy 8-11 June 2009, Stavanger, Norway Abstr P12.

[25] Capitanio U., Ahyal S., Graefen M. et al. Assessment of Biological Recurrence Rate in Patients With Pathological Confined Prostate Cancer. Urology 2008:72 (6); 1208-1213.

[26] Leman L.S., Cannon G.W., Trock B.J. et al. EPCA-2: A Highly Specific Serum Marker for Prostate Cancer. Urology 2007:69 (4), 714-720.

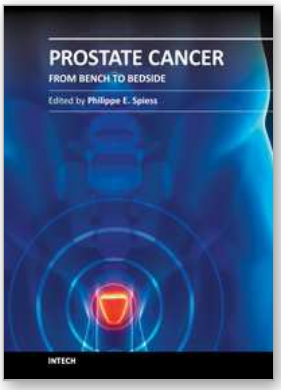
[27] Hohlfeld, J., de Buys Rossingh, A., Hirt-Burry, N., Chaubert, P., Gerber, S., Scaletta, C., and Hohlfeld, P. Tissue engineered fetal skin constructs for paediatric burns´. Lancet 2005 (Research Letters), August 18, DOI: 10.1016/50140-6736(05)67107-3, pp.1-3.

[28] Lolle, S.L., Young, J.M., and Pruit, R.E. Genom-wide non-mendellan inheritance of extra-genomic information in Arabidopsis´, Nature 2005:434;505-509.

[29] Tallberg Th. Constrains linked to stem cell research, as compared with the refined medical regulation of cell-induction caused by organ/cell specific mitochondria. BIT´s 3rd Annual Protein and Peptide Conference (PepCon-2010), Cancer Research, March21-23, 2010.

[30] Tallberg Th., Dabek J., Hallamaa R., and Atroshi F. Lipidomics: The function of Vital Lipids in Embryogenesis Preventing Autism Spectrum Disorders, Treating Sterile Inflammatory Diatheses with a lymphopoietic Central Nervous System Component. J Lipids: Volume 2011, Article ID 137175, 6 pages. doi:10.1155/2011/137175.

[31] Atroshi F., Sankari S., Työppönen J. and Parantainen J. Inflammation related changes in trace elements, GSH-metabolism, prostaglandins and sialic acid. In: Trace Elements in Man and Animals 6 (Hurly LS ; Keen CL; Lonnerdal Bo, & Rucker RB, Editors), Plenum Press, New York & London, 1988, pp.97-99.



Prostate Cancer - From Bench to Bedside

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The present textbook highlights many of the exciting discoveries made in the diagnosis and treatment of prostate cancer over the past decade. International thought leaders have contributed to this effort providing a comprehensive and state-of-the art review of the signaling pathways and genetic alterations essential in prostate cancer. This work provides an essential resource for healthcare professionals and scientists dedicated to this field. This textbook is dedicated to the efforts and advances made by our scientific community, realizing we have much to learn in striving to some day in the not too distant future cure this disease particularly among those with an aggressive tumor biology.

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