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# The Orgonomic Theory of Cancer

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**Abstract**

We examine in detail Wilhelm Reich's orgonomic theory of the etiology of cancer and his model of the cytological stages of cancer. The orgonomic model of cancer stands alone in linking the persistence and intensification of social and psychosomatic factors that negatively affect emotional expression with the onset of hypoxic conditions, local and systemic, that select for malignant phenotypes. The rarely broached, main novelty of the model suggests that every manifestation of somatic cancer is preceded by increasing RBC dysfunctionality, and that the leukocytosis characteristic of most chronic leukemias is in fact an auto-immune reaction geared against RBC fragments and their (self-)antigens. Even though unproven, these are provocative suggestions that could turn out to be substantially correct. The same cannot be said for the orgonomic model's contention that the real cancer cells are amoeboid cells that arose *de novo* (heterogenically) from the vesicular byproducts of one or more dying or decaying tissue cells - or for the validity of the staging of cancer proposed by Reich. In this context, the vesicle-based concept of a PA bion is found to be wanting and unable to acquire a functional sense - as it is employed to designate such widely different biological phenomena as to become merely a catch-all. In contrast, Reich's identification and isolation of his 'T-bacilli', despite some discrepancies, seems to largely coincide with the identification of mycoplasma. His proposed role for these T-bacilli in chronic inflammation and his experimental induction of leukemoid disease in mice injected with T-bacilli isolates are coadunate with current views of the possible role of mycoplasma in the co-induction of leukemia or leukemoid states.

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**COMMUNICATION**

> "*Resignation* without open or concealed protest against the denial of joy in life must be regarded as one of the essential causes of the shrinking biopathy. *Biopathic shrinking, therefore, constitutes a continuation of chronic characterological resignation in the realm of cellular functioning.*"
>
> W. Reich, "Cancer Biopathy", 1948, p. 210.

**1. Sympatheticonia, energy stasis and hypoxia**

Elsewhere [1] we have presented Reich's biosocial and historical theory of sexual repression, as well as its limitations - in light of both the 'microfunctionalist' criticism addressed to it by Deleuze and Guattari's monist theory of libidinal economy, and of our own aetherometric theory of biophysical systems and processes. We examined how Reich became convinced that aside from infectious diseases and genetic disorders, there was an entire realm of psychosomatic sickness or bioemotive illnesses which - based upon his own clinical research, psychiatric and analytical practices, and original therapeutic methods - he argued were all manifestations of a fundamental sickness or pathology of desire, a socially-induced and generalized disturbance of the autonomic nervous system in each individual body and the emotions of the collectivity at large. In his view, this disturbance had historically become an integral part of social life ever since the State was instituted as forming a collective *organization of power* to repress desire. Irrespective of how Reich articulated the historical process that brought this about, the historical development of this *social repression of desire* across State-societies, barbarism and civilization, eventually led to automatic (unconscious), characterological forms of *self-repression* (variants of a socially-created oedipal complex), or to *sexual repression proper*. With sexual repression, the disturbance had become systemic and unconscious, had become "elemental" to the "act" of "being human", a kind of second nature imposed by the repressive socialization of desire. It was a mold that defined the form of the human, and formed the biological organism of every human being.

The immediate biophysical result of this socially-determined "sexual repression" - also called *secondary psychosomatic repression* [2] - is the induction of a biological energy stasis, a latency of immobilized energy, or what Reich called the "core-reaction basis" of the neurotic character. The *immobilized* (sequestered) and *undischarged* energy was expressed *dynamically* by restricted plasmatic (cellular, organ and body) pulsation and insufficient respiration (retentive exhalation); and *somatically* by the muscular armor (excessive tonus of skeletal and smooth muscle). Deficient blood oxygenation (hypoxia) was the physiological result of this armoring process, while anxiety and depression were the emotional manifestations.

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The mechanism responsible for 'biological energy latency' (ie 'sexual repression') was the disturbance of the autonomic function of the sympathetic nervous system: an exertion of sympathetic innervation (in response to real or imaginary fears) created the chronic condition of sympatheticotonia (chronic contraction), the universal form of the fundamental sickness that was somatically expressed through a variety of neuritic ('nevrotic') organ disturbances. Subsequently, the sickness would develop through increasingly more serious illnesses, forming what one could call an accelerating "biopathic vector". In essence, sympatheticotonia consisted of a progressively chronic systemic contraction of the body and organs; in Reich's terminology - a "shrinking of the organism". The "biopathic shrinking" began with a functional shrinking of the autonomic nervous system, whose direct effect was the spastic restriction of plasmatic pulsation; in other words, the direct consequence of inhibiting the autonomic (parasympathetic) excitations or intensities was the suppression of the vegetative streamings (suppression of pulsation) by the reactive mechanisms of the muscular apparatus.

Between mere over-exertion of the sympathetic innervation and a full fledged condition of sympatheticotonia, there were a series of symptomatic aggravations: hypoxia, transient at first, would become chronic (anoxia); bouts of acute anxiety would eventually lead to chronic anxiety and the desire to die; deficient exhalation would give way to a chronic inhalatory attitude; the central energy stasis, which at first provoked cardiovascular hypertension (in an effort to supply more oxygen and counteract the hypoxia), eventually evolved into deficient circulation (cardiovascular insufficiency), sluggish digestion and chronic constipation.

The 'biopathic illness vector' charted by Reich in 1942 [3] included both somatic and psychic illnesses: on the somatic side - cardiovascular hypertension, muscular rheumatism, pulmonary emphysema, bronchial asthma of nervous origin, peptic ulcer, chronic sphincter spasms (constipation, hemorrhoids, vaginismus, etc), chlorosis and certain forms of anemia; on the psycho-emotional side - all forms and symptoms of neurosis (impotence, frigidity, "symptom neurosis" or actual neurosis, and "character neurosis"), perversions, psychosis and senility (cachexia).

**2. Orgonomic model of cancer: anoxia and erythroid dysfunction**

Reich claimed that the "shrinking biopathy" (sympatheticotonia) was the real dynamic etiology of *nonfamilial or functional* malignancy: aggravation of the biopathic vector affected both the blood system and all the other organs, and the biological response was cancer, above all cancer of the blood. In 1942, Reich enunciates his hypothesis for the emergence of tumors and their organic localization: "organs with chronically impaired respiration and insufficient bioelectric charge are more susceptible to cancer-inducing stimuli than are organs with good respiration. The connection between the exhalatory inhibition of sympatheticotonic "character-neurotics" and the respiratory disturbance

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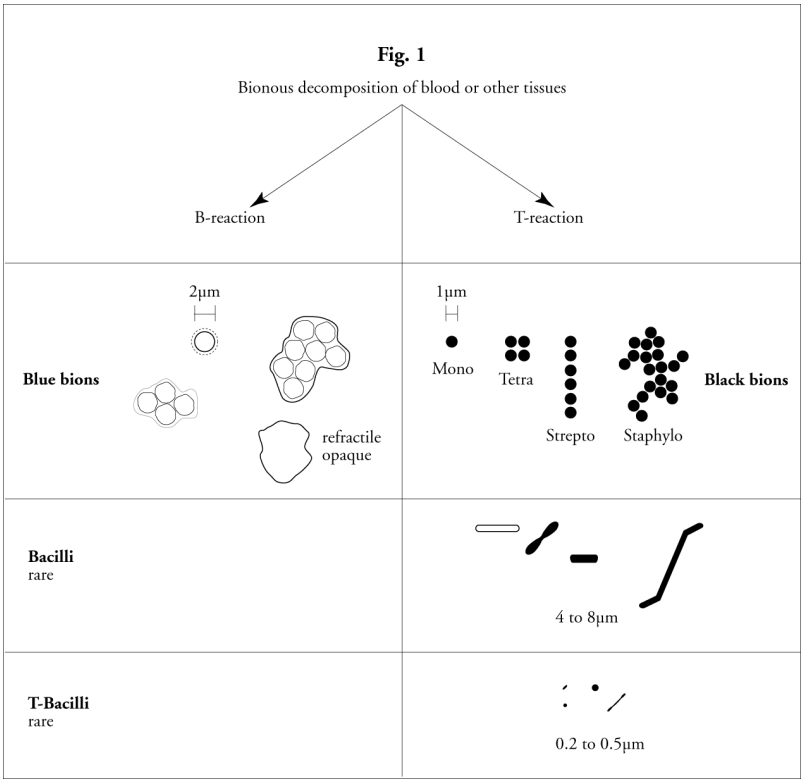


**Fig. 1** - Schematic diagram of the B versus T reactions according to Reich.

of cancerous organs discovered by [Otto] Warburg became the departing point of the sex-economic investigation of cancer" [4]. In 1948, he published his contentious book "*The Cancer Biopathy*", where this hypothesis became the guiding insight in the study of the organic localization of somatic tumors and their metastases. Tissues affected with chronic hypoxia (anoxia) were the target of somatic malignancy.

In the medical orgonomic model of cancer (malignancy), the first organ to be biopathically affected by the "systemic organismic contraction" was the blood system. The process was mediated by hypoxia, the end-product of the sympathetic dysfunction -

sympathetic dysfunction -> restriction of pulsation -> impaired respiration -> hypoxia

Note that in Reich's theory, the impact of the sympathetic dysfunction upon the red blood cell system (the erythroid compartment) was a dual one, since the red blood cells (RBCs, or erythrocytes) were impaired in their ability to hold and convey not just oxygen (chemical energy) but also "Orgone

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energy". Thus the hypoxia was, in his model, functionally identical with a 'hypo-orgonia' of the RBC's [5]. Inability to hold Orgone was, according to Reich, the biophysical basis for the morphological shrinking and the disintegration of RBCs - what he called the "sympatheticotonia of erythrocytes" [6]. This, he claimed, was "the earliest and most general phase in the cancer disease" [5]. Yet, it was also the universal symptom of *all sympatheticotonic disturbances*, cancer being simply the extreme manifestation or consequence of sympatheticotonia, with tumor-formation and metastases localizing in those organs or regions of the body most affected by the muscular armor, ie where the libidinal excitations were blocked and oxygenation the poorest.

**3. The B vs T reactions and their relation to PA bions and T-bacilli**

Reich's experimental and medical documentation of his 'bion theory of the etiology of cancer' is based upon an intended systematic investigation of the Pasteur principles of sterility in microbiology, and in particular upon his own original research into the "bionous" disintegration of tissue and the 'origins of life' (meaning: biopoiesis, comprising both biomolecular and cellular ontogenesis) [7]. From his studies of the thermal destruction of animal and plant tissue and his claim of heterogenic transformation of dead or dying (necrotic) tissue into vesicles and rods ("bions", see [7]), Reich inferred that similar processes of cellular disintegration and conversion occurred in normal tissue subject to oxygen, nutrient and energy starvation. This, he claimed to prove, by long-term *in vivo* examination of processes of inflammation and putrefaction in living, decaying tissue.

We have seen[7] how Reich claimed that all living tissue decomposed into bacterial rods (bacilli) and cocci, forming multiplicities that he collectively referred to as 'PA bions' (see **Fig. 1**). In his mind, these were not pre-existing infectious particles or micro-organisms, but biogenic ('hetero-biogenic') productions. Amongst the PA cocci, he distinguished between the large blue ones (2 to 5 μm in diameter) observed when healthy tissue was stressed chemically, thermally or mechanically; and the smaller blackish ones (~1 μm in diameter or less, see **Fig. 1**), observed when biopathic (diseased) tissue was equally stressed or broke down on its own in the course of an evolving process of disease.

Independently of all the pertinent limitations, doubts and unresolved problems - including methodological ones - regarding Reich's theory of the bions and his experimentation with bion preparations (see [7] for an in depth discussion of these problems), the picture that emerges regarding the medical implications of his theory of the PA bions is that cocci would be heterogenically generated from decomposing tissue cells, some being beneficial to the host, or symbiotic, and others being highly detrimental, but both being the result nonetheless of *auto-infection*. This orgonomic view ran dead counter to Koch's postulates of disease, Schwann's central dogma of cell theory and Pasteur's principles of sterility. Tissue putrefaction in a variety of distinct clinical conditions was not primarily due,

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in Reich's view, to infection with foreign bacteria, nor mediated by the proliferation of bacteria or necessarily promoted by nonsterile environments. Reich was not denying that there are plenty of micro-organisms that only propagated by proliferation and infection, and which obeyed therefore both Koch's postulates and Pasteur's principles. But he was arguing that conditions of tissue putrefaction induced by anoxia-mediated sympatheticotonia resulted in the "bionous disintegration" of tissue and blood cells, giving rise to auto-infection with 'heterogenically generated' 'PA-type cocci and bacilli' that, after all must belong to the kingdom of the prokaryotes.

Reich made a still bolder set of claims than those pertaining to his PA-bion preparations [7]. In coal incandescence and charred blood or blood-soot preparations, and at very high magnifications with apochromatic darkfield microscopy, Reich discovered the presence of very small particles at the limit of microscopic resolution (0.180 to 0.275 μm long), which he termed "T-bacilli" or "T-rods" ("T" for *Todt*, death in German) - the "bacilli of death" - and which were similar to large viral particles. If an incubated culture of PA bions was nutritionally stressed, it would degenerate into "cocci-like pus staphylococci of only 1.5-2 μm, of blackish instead of bluish colour", sometimes even smaller (0.4 to 0.5 μm) and an abundance of these "T-bacilli" [**Reich, 1939**]. In very old 'PA bion' agar cultures, T-bacilli could be extracted from the dark-greenish periphery of the colonies. The T-bacilli were also a type of bions, a member of the bion multiplicity, and like all bions - though more fastidiously - they could be cultured with special media, presenting small rod or coccoid forms depending on the media employed. Note that this fact proved thereby that these virus-like particles which Reich had found were not viruses at all but bacterial cells capable of autonomous self-replication.

According to Reich, the heterogenic production of PA-bions, rod bacteria and T-bacilli from living tissue, whether stressed or diseased, was the subject of a disjunction of polar reactions, what he called the B vs T reactions (see **Fig. 1**). The large blue bions and the T-bacilli constituted *differential* processes of tissue decomposition, and were the hallmark signs of the kind of tissue that had been decomposed, since each reaction presented a certain multiplicity of micro-organisms. When stressed mechanically or thermally, healthy tissue broke down mostly into large PA bions - the so-called B reaction, whereas biopathic tissue spontaneously decomposed into T-bacilli, putrefacting bacteria and small cocci or PA bions - the so-called T-reaction. The difference was embodied by the antagonistic *in vitro* interaction of the large PA bions and the T-bacilli, when they were mixed together: according to Reich, the former would attract and paralyze the latter.

### 4. Orgonomic model of leukemia: uncontrolled response to hemolysis

Since, according to Reich, the first target of sympatheticotonia was the red blood cell system (the erythroid compartment) which functioned as the body's main energy system, an erythroid dis-

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order, of necessity, underlaid all biopathic illnesses, including every form of cancer or malignant tumor formation. Moreover, according to this model, pre-leukemic conditions - such as anemia, and in particular, hemolytic and aplastic anemias - would necessarily antedate and accompany manifestations of somatic cancer. Erythrocyte starvation of oxygen and energy entailed osmotic fragility, and a faster and greater disintegration of circulating erythrocytes or red blood cells (RBCs), progressively loading the spleen and the immune system. The unchecked proliferation of one or both of the main types of white blood cells (WBCs) - myelocytes and lymphocytes - characteristic of nearly all leukemic conditions was the inevitable consequence of the disintegration of RBCs, the blood system mounting an auto-immune response in the hope of getting rid of dysfunctional and dying RBCs. In other words, in its broadest trait, Reich's theory of cancer is that somatic cancer always presents a certain leukemogenic potential, cancer being inherently and ultimately "leukemic".

Reich's theory of leukemia is perhaps the most interesting and provocative of his thoughts. The biopathic disintegration (that he erroneously believed to have proven [8]) of erythrocytes into T-bacilli , presented the equivalent of the introduction of foreign bodies, a form of auto-infection of the host by the heterogenesis of T-bacilli from dead and dying host tissue. Furthermore, and irrespective of T-bacilli being, or not, *de facto* produced from dying erythrocytes (which we have at length shown is *not* the case, see below and [8]), the disintegration of RBCs enforced local and generalized anoxic conditions - and thus favored the putrefaction of tissues while promoting the growth of non-symbiotic, saprophytic, anaerobic bacteria. Thus, the T-reaction involved both (1) auto-infection with T-bacilli heterogenically formed from disintegrating RBCs - and eventually, later on in the same degenerative process, from all tissue cells - and (2) the local and systemic anoxia-induced promotion of saprophytes (whether opportunistic or also heterogenically produced). Confronted with this dual attack, as well as with an increasing mass of cytoplasmic fragments from dead and dying RBCs, the body mounted a vigorous immune reaction against T-bacilli and other putrefacting bacilli, as well as against the self-antigens of erythroid cell fragments [5]. Being an auto-immune response and liable therefore to wreak havoc with any and all hematopoietic regulatory circuits, the result would inevitably lead to disordered, discoordinated, "wild" hyperplastic responses of myelocytes and/or lymphocytes. Thus, the leukocytosis characteristic of most leukemias was simply a symptom of a much more profound "malignancy" or disease, a "malignancy" of the red blood cells - their failure to provide oxygen and energy to the body.

In this model, the fundamental pre-cancerous disposition is the so-called 'T-reaction of erythrocytes', specifically their dysfunction and disintegration. According to Reich, this condition antedates and underlies all forms of neoplastic disease, but is not fully expressed or made patent until an auto-immune leukemic condition finally erupts, with or without prior somatic tumor formations.

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This view, of course, dovetailed with the medical knowledge of his time, as it was by then apparent that so-called "leukemoid reactions" involving a neutrophilic expansion could be elicited by hemolytic anemia [9]. Of course, back then, no lymphocytic leukemoid reaction against hemolytic anemia was definitely ascertainable; and it was unknown that, for example, in chronic lymphocytic leukemia (CLL), there is a CLL-clone-*independent* disturbance of T-suppressor lymphocyte activity that takes over the autoimmune humoral responses of B-cells and causes hemolytic anemia even before the CLL phenotype is manifested [10]. But, since at least the late 1940's, it was apparent that there was an acquired auto-immune hemolytic anemia with an unknown cause, and which presented dysplastic RBCs - most frequently anisocytosis, macrocytes and poikilocytes (pear and spindle RBCs), but also echinocytes (acanthocytes and burr cells - also called spur cells), spherocytes and ovalocytes (see **Fig.s 2-4**). Fig. 3-22 of [11] exemplifies some of these forms found in the peripheral blood of CLL patients.

However, one may *not* designate the underlying erythroid dysfunction that Reich has in mind as an erythroleukemia (or as a real malignancy in the technical sense) - since it is not characterized by the uncontrolled proliferation of RBC progenitor or precursor cells, as is observed in "genuine"

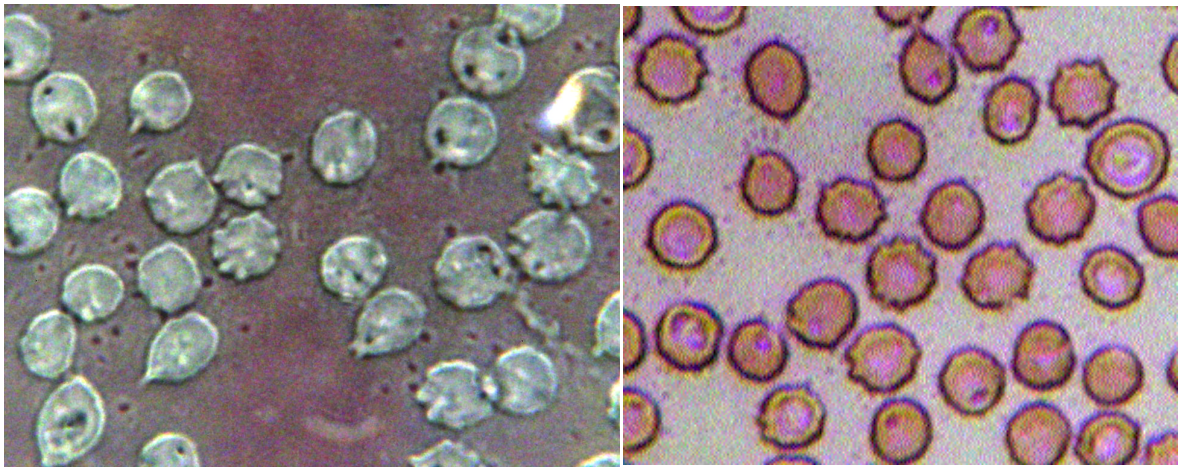

**Fig. 2** (**left**) - Echinocytes and poikilocytes in a human peripheral blood smear. Note the out-puckering of RBC membranes. These are what Reich called T-spikes. Nomarski interference contrast. Sony TVR68 CCD camera. Print magnification: 1,200x.

**Fig. 3** (**right**) - Anisocytosis, echinocytes and poikilocytes in a human peripheral blood smear, Wright's stain, brightfield (40x achromat). Note the radiating-like projections emanating from most RBCs. Excess of RBC membrane covering the cytoskeleton leads to the out-puckering of membrane, outward displacement of lipids, extrusion of lipids and membrane, resulting in detachment of small fragments or 'cytoplasts'. These fragments present coma-like shapes and measure in length from 0.3 to 1µm, most frequently 0.7µm. They can be easily mistaken for Reich's 'T-bacilli' or for mycoplasma. Flooding of the bloodstream with such hemolytic fragments can induce an auto-immune response against RBC antigens attached to them. The RBC fragments lack DNA and RNA, as shown histochemically.

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erythroleukemia (AML-M6) or in erythrocytotic dysfunctions or disorders (secondary polycythemia, polycythemia *rubra vera*, familial polycythemia). Rather, the underlying erythroid disease that Reich had in mind was manifested by the osmotic fragility *and* the weak or dysfunctional hemoglobin content of RBCs that together, and eventually by the shedding of RBC fragments, elicit auto-immune hemolysis, in essence, forming a mixed condition of aplastic and hemolytic anemias (rather than microcytic anemias per se). The underlying 'true' "malignancy" of all cancers and leukemias would, in the orgonomic model, be attributed simply to the erythrocyte's chronic deficiency of (orgone) energy and oxygen. This is the main paradigm of the orgonomic theory of cancer and leukemia.

Intensification of the dysfunctional erythroid condition would then lead to the biopathic breakdown of RBCs into T-bacilli, the second paradigm of the orgonomic model of neoplasia: "leukemia is nothing but a reaction of the organism to the shrinking and T-disintegration of the erythrocytes" [12]. Ultimately, the foreign body responsible for leukemia was the disintegrating erythrocyte and its byproducts, in particular the T-bacilli. Once the myeloid:erythroid ratio tipped over, and the leukemia could finally be diagnosed, the condition had evolved already from the first stage (leukemogenic) of systemic erythroid disintegration, to the second stage, of myeloid and/or lymphoid hyperplasia in the course of an auto-immune reaction, when not to a stage of neoplastic transformation of myeloid and lymphoid cells. By then, the diseased system would be too weak - or completely unable - to supply fully developed, mature and functional RBCs, and pre-mortem necrosis would set in.

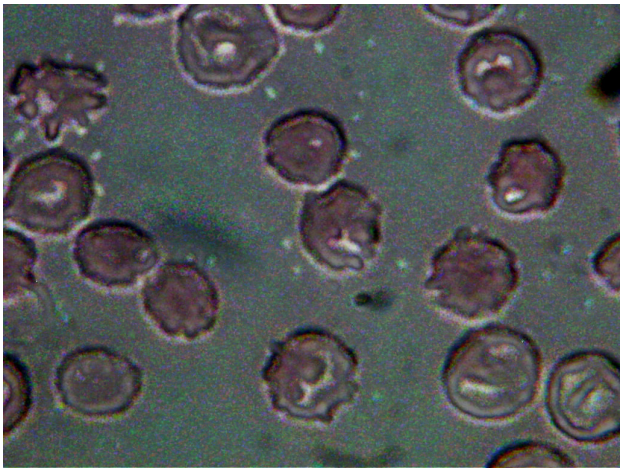

**Fig. 4** - Using compacted differential between two phase-contrast frames with a depth of ca 0.5µm, renders the extruded, lipid-rich RBC fragments clearly visible (oil immersion). Print magnification: 1,350x.

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**5. The B vs T reactions and Reich's model of cancer stages**

According to Reich - and as already examined above - there were two distinct types of tissue response to mechanical or energy stresses, the B vs T reactions, each associated with the emergence or selection of different multiplicities of prokaryotes from dying and disintegrating tissue. Normal blood and tissue cells subject to stress tests (chemio-osmotic, autoclavation) broke down into large, blue PA (Packet-Amoeboid) bions. This was the functional basis of the B reaction. However, when blood and tissue cells broke down on their own (spontaneously, without being subject to an external stress) into such large blue PA bions, the B reaction indicated the presence of a biopathic process. It was, in his view, the very first indication of the presence of such a process, and it represented an attempt of the "normal organism" to react against an internal stress. In this sense, Reich claimed that "large PA bions" attacked T-bacilli, this being the reason why "blue PA bions" "surrounded by dead T-bacilli adhering to them, are often seen in the blood of healthy persons" [13]. But, as we shall see ahead, this stated claim can only be valid if one accepts to make the same illegitimate, dysfunctional amalgamation of blood platelets (which are natural fragments of megakaryocytic cytoplasm, ie physiological cytoplasts) to PA bions as was overtly claimed by Reich - an absurd identification that would be forced to assume that platelets would be identical to bacterial cocci and also carried DNA...

So, it is on totally tenuous grounds that Reich claims a host-symbiotic relation for PA bions as helpers in the organism's fight against disease. Be this as it may, in Reich's model of the cancer stages, a "spontaneous" B-reaction marks the first stage in the onset of a functional (nonfamilial or acquired) disposition towards cancer - what he called the first precancerous stage (Ca Ia). This corresponded - in Reich's theory - to the onset of any acute inflammatory stage, whether mechanically caused or the result of hypoxia (be its source external, viz exposure to high-altitude or very low pressure, or internal, viz caused by autonomic sympatheticotonia in its early stage).

For reference throughout the following discussion, the reader should consult the orgonomic model of the stages of cancer (**Fig. 5**) and the complex map of the various pathways that, according to Reich, would lead to the production of malignantly transformed cells (see **Fig. 6**).

According to Reich's model of the progression of the cancer vector, the next step is taken when a chronic T-reaction sets in. If blood and tissue cells break down instead into T-bacilli, then the disposition towards cancer has already evolved to a second "precancerous" stage (Ca Ib), so-called 'T-lysis', characteristic of a chronic inflammatory stage [14]. T-bacilli were only directly responsible for the completion of the precancerous process (Ca I). Note that Reich does not attribute causation of cancer to T-bacilli, as Reichian followers and writers often state that he does. Rather, the T-bacilli were at once symptoms, markers, warning signals of a self-induced (autonomic) disease process, and medi-

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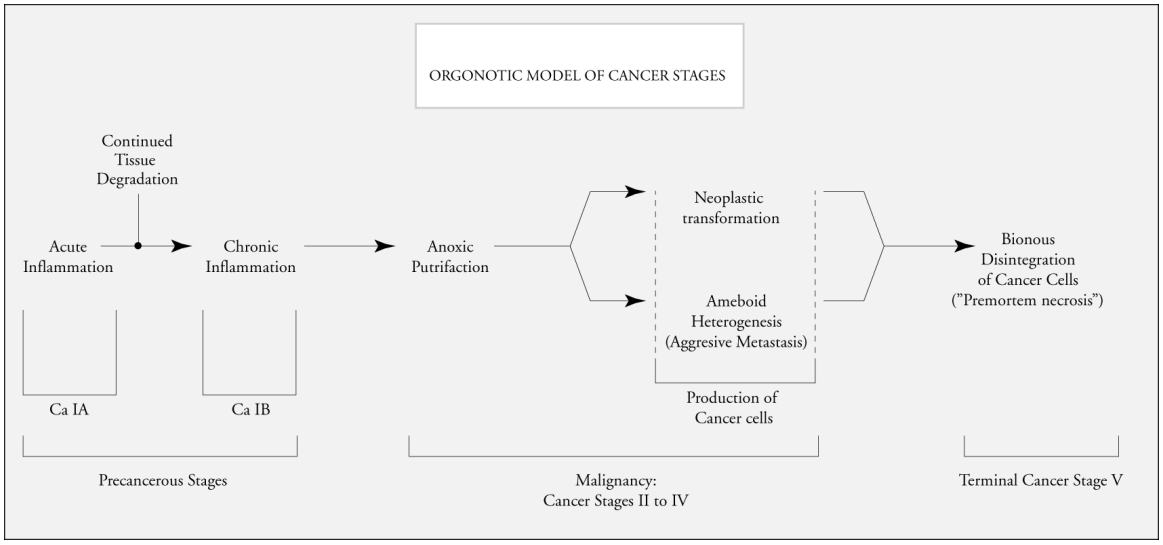


**Fig. 5** - The orgonomic model of the stages of cancer.

ators of the process of oncogenesis: it was the response of blood and tissue cells to self-generated, heterogenic T-bacilli and other rot bacteria, and RBC fragments, *that eventually led some of those cells to mutate by engaging into a process of neoplastic transformation*. This was the essence of his reasoning.

Thus, in Reich's view, cancer proper emerged as a biological response to advanced bionous disintegration of tissues. There were three different types of responses that characterized the Ca II stage, and its subsequent evolution into the more aggressive and terminal stages III to V (see **Fig.s 5 & 6**), as inferred by Reich from his autopsy of the tissues of healthy and cancerous mice in his experimental studies [15].

1) The first oncogenic (or cancer-forming) response consisted of the *neoplastic transformation of a single cell*. Unable to sustain aerobic metabolism under conditions of chronic inflammation, autoinfection and tissue decomposition, individual cells escaped the constraints of normal tissue differentiation and function, and circularized their shape (oblong, tear-drop or club-like), to become, in essence, *independent amoeba-like cells*, as if they had reverted from the metazoic to a protozoic stage (see **Fig.** 7). This formed a Ca II stage. Eventually, the transformed cells would become flagellated and highly motile (caudate and spindle cells, see **Fig.s 8 & 9**), and engage in the uncontrolled proliferation ("crazy mitosis") responsible for tumor-formation and primary metastases (Ca III and Ca IV). In this first scenario, *there is no stage of bionous decomposition linking the original tissue cell to the cancer cell that it transformed into*. We should note that even though Reich described this process of neoplastic transformation of a single cell, he virtually ignored it in the main body of his research into the

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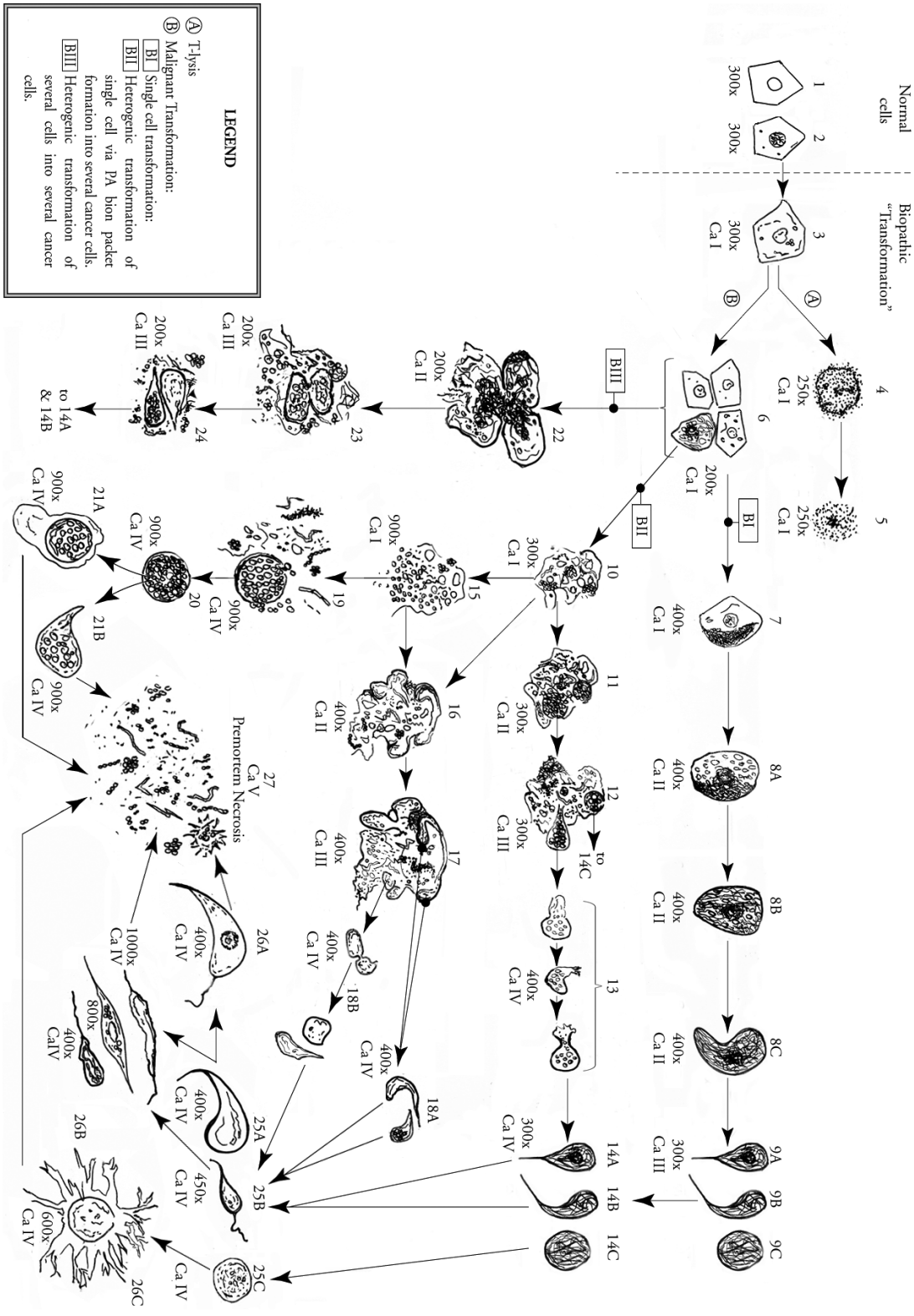


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**Fig. 6 (page on the left)** - Pathways that, according to the orgonomic model of cancer and its stages (see **Fig. 5**), lead to malignancy. The first stage of cancer (CaI) is characterized by bionic vesiculation of a sphericized cell (#4) and its decomposition into mostly T-bacilli (#5). This is the pathway shown as A, at the top of the figure. In pathway BI, a single cell engages in malignant transformation. It vesiculates and loses its rhomboid geometry to circularize or acquire a spindle or caudate shape, as shown by #s 7 to 9. This is the only pathway of single cell transformation considered by Reich. It spans the cancer stages CaI to CaIII. The transition from #9 to #14 below it, is made by the acquisition of motility. In pathways BII and BIII, Reich invokes heterogenesis as the origin of the transformed cell or cells. In BII, a single cell undergoes bionic decomposition, and from the reorganization of the bions thus created into a packet or packets, either a single, large, aggressive cancer cell is produced (#s 13 to 21A and 21B), with the process jumping from CaI to CaIV, or instead, sheltered within the membrane of the decomposing cell, two or more cancer cells are produced. From #s 10 to 14, two motile cancer cells are heterogenically produced from a single cell. And from #s 10 to 16, and to 18A and 18B, four smaller cancer cells are heterogenically produced also from a single cell. In both instances, progressions from CaI to CaIV takes place, with these pathways leading to large and small, amoeba-like caudate, spindle or spherical cells, frequently flagellated (CaIV stage, #'s 25A, B and C), that may develop pseudopodia (#26C). All of the CaIV transformed cells engage in wild mitosis and, during the CaV stage of *premortem necrosis* (#27), they undergo, in turn, bionic decomposition that heterogenically gives rise to "black bions" (ordinary staphylococci, streptococci, micrococci, etc), rot bacilli and T-bacilli. In the BIII pathway (#s 22 to 24), two or more cells decompose bionically to generate two bion packets that turn into two or more spindle cells, as cancer evolves from CaII to CaIII. Once motile, these rejoin the CaIV stage, at #'s 14A and 14B. Only in pathway BI does Reich envisage the malignant metamorphosis of a single normal cell into a cancer cell. All other transformed cells arise by heterogenic processes. Notice that his entire 'oncological cytology' is fundamentally morphological. From CaII to CaIII, morphological changes take place without acquisition of motility, the latter being characteristic only of CaIV cells. Presence of fully amoeboid characteristics (curtate swimming, flagella, pseudopodia,etc) are also a marker of CaIV. Whereas pathways A and BI can be seen as having some reality in the form of apoptosis for pathway A and the malignant transformation of a single cell in BI, the other pathways (BII and BIII) that invoke heterogenesis have not been observed, and their existence is doubtful at best.

"cancer biopathy", going as far as plainly contradicting it by stating that "the cancer cell is in reality a product of the many blue PA bions that originated from blood cells or tissue as a defense against the local autoinfection with T-bacilli" [13]. This, as explained in [16], was based on the wrong premise that protozoa and protozoon-like cells arise *de novo* and heterogenically from the decomposition of normal tissue cells - a premise that (wrongly) appeared to be confirmed by Reich's observations of the behaviour of sheathed or encapsulated prokaryotes that form colonial packets [7], what he called 'packet amoebae'.

A note of caution must be introduced at this point regarding the extraction of illegitimate ilations from morphological and motional observations. Caudate and spindle cells are not necessarily an indication of malignantly transformed cells. Anymore than amoeba-like cells are necessarily cancer cells. The entire lineage of monocytic-macrophage cells is replete with perfectly functional and healthy amoeboid cells that move through the tissues by means of pseudopod formation. The Langerhans cells (discovered by Paul Langerhans in 1868) have been shown to daily travel tremen-

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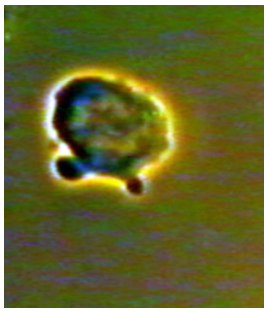


**Fig. 7 (left)** - Live Friend virus (FV-P) transformed cell (745 cell line) extruding pseudopods upon contact and adhesion to glass. Pseudopod formation and retraction takes from seconds to minutes. Though spherical in liquid culture, Friend cells become amoeboid in shape and motion upon contact with a substrate such as glass. Phase contrast (63x Ph3 objective), Hitachi VK-C2000 CMOS camera. 1,875x.

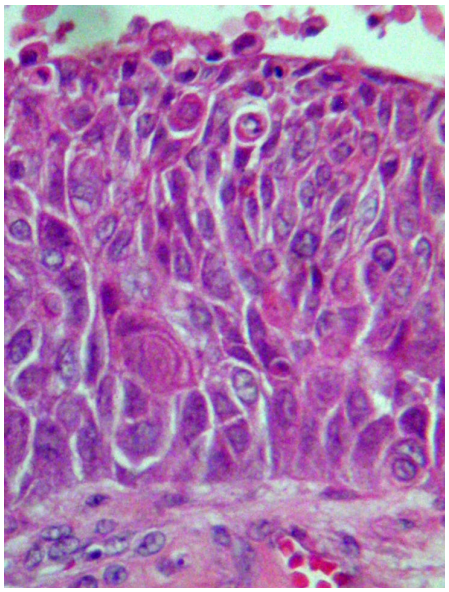


**Fig. 8A (right)** - Malignantly transformed spindle cells in a human sarcomatous growth that formed a nodular infiltrate. Caudate cells are also apparent. Mallory-azan stain. Nikon Coolpix P5000 (10 megapixel). 250x. **Fig. 8B (below left)** - Malignantly transformed spindle cells in a human sarcomatous growth that replaced the germinal culture of a lymph nodule. Some extramedullary hemopoiesis is taking place at the center of the tumor where transformed blast cells can be observed. Mallory-azan stain. Nikon Coolpix P5000. 350x.

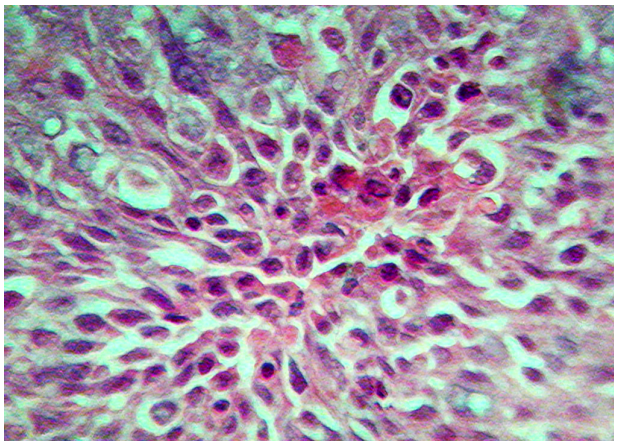
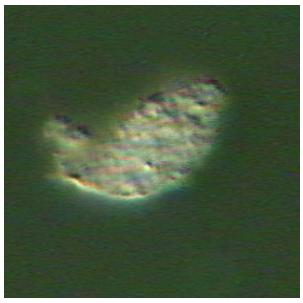
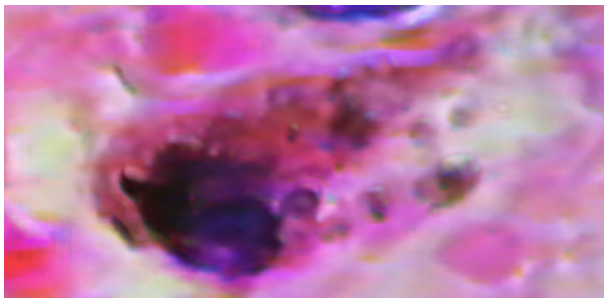


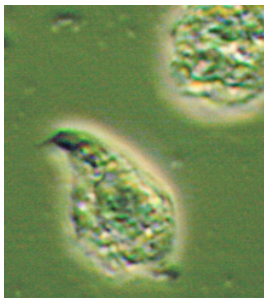
**Fig. 9 (bottom left)** - Live caudate-spindle cell from a murine plasmacytoma (P3) B-cell line, swimming free in liquid culture. Darkfield, oil immersion. Hitachi VK-C2000 CMOS camera. 2,200x.

**Fig. 10 (bottom middle)** - Normal human alveolar macrophage with phagocytosed hemosiderin deposits. A profusion of such cells occurs in chronic pulmonary congestion, the brown colour being due to the iron deposits. Nuclear fast red stain. Hitachi VK-C2000 CMOS camera. 1,500x.

**Fig. 11 (bottom right)** - Live normal caudate cell shed from human ureteral epithelium and containing large cytoplasmic granules. Phase contrast. Luminera Infinity-1 CCD camera. 1,150x.







 ISSN 1920-3799

dous distances between lymph nodes and skin, and over the skin, by employing amoeboid motion. Whether in pathological conditions or as part of the daily lavage of the lungs, macrophages scavenge particles and cell fragments by infiltrating through the bronchial tissue (see **Fig. 10**). In parallel, and with respect to morphology, plenty of cells in the body are spindle-like - fibroblasts and smooth muscle cells are examples - or caudate-like - as is the case with the ureteral epithelium (see **Fig. 11**).

Next, according to Reich, both the *second* and *third* oncogenic pathways - those that he claimed were the source of 'real cancer cells' - involved the 'bionous disintegration' of affected host cells (see **Fig. 6**), and subsequent "spontaneous" reorganization of the bions and their packets into aggressive, "fully mature" amoeboid cancer cells responsible for tumorigenic metastases. Thus, the second and third oncogenic pathways directly invoke the orgonomic model of protozoal heterogenesis [16].

2) In the second oncogenic pathway, packets of bions formed directly inside the disintegrating cell, either in the cytoplasm or in the cortical (ectoplasmic) zone, underneath the cellular plasma membrane. Intra-cellular formation of such packets of cocci was staged as Ca II. According to Reich (who claimed direct observation of this), the packets became enveloped by a 'membrane' they secreted, to heterogenically form amoeboid cells (caudate and spindle shaped) that were released from within the disintegrated host cell. This process of amoeboid cell "generation" defined a Ca III stage. The Ca IV stage was then crossed when these amoeboid cells became highly motile, either by developing flagella or pseudopodia. According to Reich, "mechanistic cancer pathology regarded [these forms] as 'parasites'!" [17] - viz Korotneff's *Rhopalocephalus carcinomatosus*, Pfeiffer's *Cancriamoeba macroglossia*, or Schaudinn's *Leydenia gemmipara* [18].

3) Finally, the third oncogenic pathway was in essence analogous to the second one, but instead of one or several amoeboid cells heterogenically arising from a single decomposed cell, now one or several amoeboid cells arose from the inter-cellular communication of two or more synchronously decomposing host cells. Again it encompassed stages Ca II to IV.

What then were the characteristics of the last stage, Ca V? It defined the terminal stage of what Reich called "premortem necrosis" [19]. The cancer cells and the tumors they formed now disintegrate, in turn, into rot bacteria and T-bacilli, swamping the body with a generalized putrefaction. Thus, premortem necrosis was the result of a systemic bacteremia and toxemia, as even more aggressive rot bacteria and T-bacilli heterogenically arose from decomposing tumor cells.

### 6. The relationship of Reich's T-bacilli to mycoplasmas (class *Mollicutes*)

The microbiological characteristics of Reich's T-bacilli - their changing morphology, fastidious cultivability, and indirect implication in the development of cancer in experimental mice - led us

 ISSN 1920-3799

to suggest, back in 1984, that T-bacilli were likely related to the same sized mycoplasmas and other members of the class of the *Mollicutes* (nearly submicroscopic, "soft-skinned" or pliable prokaryotes that lack a bacterial wall) implicated in primary atypical pneumonia and nongonococcal urethritis, and found in association with a variety of leukemias and auto-immune disorders. Coulson and Boadella, who were not scientific researchers but investigators of Reich's work, made a similar suggestion in 1973 [20-21], citing five characteristics of mycoplasma that tallied with T-bacilli: the Gram-negative reaction; the same size range; production of acid upon incubation in culture; adsorption of mycoplasma to RBCs causing aggregation similar to that observed by Reich with T-bacilli; and the claim that mycoplasma produced leukemia in mice. The last claim was an hyperbole of Coulson's and Boadella's interpretation, since even those researchers who entertained such a possibility at that time did not manage to prove causation or "production" of leukemia by mycoplasma infection. Moreover, even though by the mid 1960's mycoplasma-induced oncogenesis was an hypothesis under active consideration in light of the then recent isolation of mycoplasma from child and adult leukemias, to this day the only cells transformed by infection with some strains of *M. fermentans* appear exclusively to be B lymphocytes with prior exposure to EBV (Epstein-Barr virus) [22].

There are, of course, much deeper parallels between T-bacilli and mycoplasmas not mentioned by Boadella or Coulson. Reich could well have isolated mycoplasmas from the decomposition of normal and diseased plant or animal tissue, as well as from soot, since mycoplasmas from these sources have been isolated and grown in broth media, in serum-containing broth and broth-agar, and are known to be highly pleomorphic according to the chosen culture conditions [23], varying between rod, coccoid, L-shape and spiraloid forms. Moreover, high-power darkfield techniques identical to those used by Reich to observe T-bacilli were employed since 1935 to detect and observe the morphology of *Mycoplasma mycoides* (then known as "pleuropneumonia organisms", PPLO) [24].

Reich reports profuse generation of T-bacilli in soot incandescence experiments, and this is consistent with the observation that certain types of mycoplasma - which belong to the highly pleomorphic genus *Thermoplasma*, are as small as 0.3 μm and grow well at pH 1-3 (very acidic) - are true saprophytes that have been isolated from burning coal refuse and acid hot springs [25].

Mycoplasmas have been found in association with a variety of diseases in plants, arthropods, animals and humans, including a large panel of leukemias. And they are suspected of being the main unwitting cause of the spontaneous immortalization of primary cells in tissue culture and of the eventual, long-latency, neoplastic transformation of cell lines. Most importantly, the Barile hypothesis regarding mycoplasma-induction of auto-immune hemolysis tallies well with Reich's central notion that the proximal cause of leukemia is an auto-immune attack against unviable RBCs and their hemolyzed fragments [26].

 ISSN 1920-3799

Mycoplasmas are also a common contaminant of serum and serum-containing products, as well as of egg and egg-containing media, and this has raised a variety of questions as to the validity of observations performed with cell lines, or the source of mycoplasma isolates in mycoplasma cultures. Reich often employed horse serum and egg preparations in his culture media, and accordingly his results are not immune to such doubts. Indeed, he was confronted with an uncontrolled variety of sources for these mycoplasmas in his different T-bacilli preparations. Even beef broth can be a source of mycoplasma.

However, no matter how insightful might be the correlation between T-bacilli and mycoplasmas, there are considerations that cast doubt on the identification of T-bacilli and mycoplasmas. For instance, Reich reports the detection of T-bacilli in a variety of "biopathic disorders", including as the primary extract from oral cold sores, which today are known to be caused by endogenous (nerve cell-released) infection of epidermal tissue with the double-stranded DNA virus, *Herpes simplex 1* (HSV-1), whose plasma membrane-encapsulated virion measures 0.2 to 0.3 μm - also in the same size range of mycoplasmas and T-bacilli. Thus, it is likely that, in this instance, Reich optically mistook viruses for his T-bacilli. Viruses, of course, cannot be grown in culture - certainly not without co-cultivation of host cells that are targets of infection - since they are unable to autonomously replicate. If Reich's cultures of "pure T-bacilli preparations" were effective microbiological growths, their colonies would still have to be colonies of mycoplasmas or mycoplasma-like prokaryotes, as viruses cannot replicate to form colonies.

Another objection to the assimilation of T-bacilli to mycoplasma, is the complete lack of evidence there is to suppose that mycoplasma, which are not spore formers, can either survive extreme moist-heat sterilization or heterogenically re-assemble *de novo* from heat-killed tissue. With high-power light microscopy one cannot distinguish - certainly not by vision or observation alone - between large DNA viruses, retroviruses, mycoplasma, other *Mollicutes*, and bits or fragments of the cytoplasm and plasma membrane of dead or dying cells that might have the same size range and are capable of autonomous movements [27]. Reich indulged in repeatedly taking visual observations at face value, as if seemingness and analogy were sufficient to identify species of micro-organisms and the actual events of biological processes. He had increasingly become uncritical of the observations he'd made, as if the senses or optical sense-perception never lied, or was not subject to illusion or to "apparently objective movements". Thus it is more than likely that in some instances what he called T-bacilli were actual virus (eg in cold sores), that in others they were cell fragments ("microplasts" or "cytoplasts" [27-28]) devoid of DNA (eg the "T-spikes" of crenated erythrocytes or burr cells), and that in still other instances what he called 'T-bacilli' were mycoplasmas, other *Mollicutes*, or other submicroscopic virus-like bacterial particles (such as *Chlamydia* or *Rickettsia*).

 ISSN 1920-3799

A major objection to the correlation of T-bacilli with mycoplasmas is Reich's erroneous notion that crenation of human RBCs produces spikes that are heterogenic formations of T-bacilli. Indeed, adsorption of certain mycoplasmas to RBCs (eg in rodents) cannot be confused with either a viral-like replication of T-bacilli inside RBCs (which was *not* Reich's claim), or with the heterogenic formation of T-bacilli from dying RBCs (which *was* Reich's claim). Moreover, crenation or acanthocytosis of RBCs (see **Fig.s 3 &** 4) does not require infection or adsorption of any mycoplasmas (or viruses, for that matter). Not only are the crenation or burr spikes *not* cultivable - since they lack all nucleic acids and, once detached, are mere "cytoplasts" - but mycoplasmas do *not* multiply inside of *human* RBCs. That's simply because human RBCs (unlike rodent RBCs) are enucleated cells that have lost the entirety of their metabolic and DNA-replicative machinery and, therefore, are not suitable hosts for mycoplasma replication. If T-bacilli are mycoplasma, they may adsorb to RBCs, but *not* bud from them, which was the essence of Reich's claim (*viz* that T-bacilli emerged heterogenically from RBCs, by budding off them).

To our minds, however, the most important objection to the correlation of T-bacilli with mycoplasmas is Reich's contention that T-bacilli were only encountered in biopathies, either as markers of the T-lysis that characterized his theory of the first cancer stage, or as a result of the decomposition of the cancer cells themselves. This totally departs from what is known today about mycoplasmas. In fact, the latter are, for the most part, *symbiotic parasites* that, though present in all body cavities, do not cause disease, nor are associated with disease. Only saprophytic strains have been shown to cause disease or be associated with it.

**Table 1** lists the biological similarities between Reich's T-bacilli and mycoplasma. They are certainly closer than are T-bacilli and viruses [29] - whose biological similarities are shown in **Table 3** for comparison purposes. The closeness between Reich's T-bacilli and mycoplasma strongly suggests that what Reich was doing, in this respect, was conducting pioneering research on the relationship of mycoplasma to cancer, and to experimental cancer in mice. As discussed above, there are, however, very significant differences between Reich's theory of the properties of T-bacilli and the known biological abilities of mycoplasma. These contrasts are shown in **Table 2**; aside from those already examined, we should note that Reich never observed the typical fried egg aspect of mycoplasma cultures in broth agar. This casts some doubt on whether Reich's cultures of T-bacilli were genuine mycoplasma cell cultures. Reich's claim that T-bacilli presented zigzag motion in broth may actually not be incompatible with the identification of T-bacilli with mycoplasma. Although the latter are conventionally considered to be mostly immotile and only display gliding motion, few mycoplasma studies have observed mycoplasma *in vivo* with optical microscopic techniques (given their submicron size and the need for special microscopic optics and techniques). Moreover, if zigzag motion were

 ISSN 1920-3799

**Table 1**
Biological similarities of Reich's T-bacilli with mycoplasma.

| | Reich's theory of T-bacilli | Known properties of mycoplasma |
|---|---|---|
| 1 | • T-bacilli are cultivable in broth agar | • Mycoplasma are cultivable in broth agar |
| 2 | • T-bacilli form *in situ* from disintegrating cells, budding from them | • Mycoplasma *can bud* from cells they have infected |
| 3 | • T-bacilli do not cause cancer, but mediate its expression | • Mycoplasma can promote cancer, with a long latency |
| 4 | • T-bacilli are saprophytic, the saprophytes being anaerobic | • Mycoplasma are saprophytic, the most aggressive being anaerobic |
| 5 | • T-bacilli size: 0.2 to 0.3μm (0.6μm max) | • Mycoplasma size: 0.2 to 0.3μm (0.7μm max) |
| 6 | • T-bacilli stain Gram-negative | • Mycoplasma stain Gram-negative |
| 7 | • T-bacilli are associated with subclinical anemia | • *M. pneumoniae* is associated with subclinical anemia |
| 8 | • T-bacilli induce hemoagglutination and hemolysis of RBCs | • Mycoplasma induce hemoagglutination and hemolysis of RBCs |
| 9 | • T-bacilli can adsorb to cells | • Mycoplasma can adsorb to cells or infect them |
| 10 | • T-bacilli can be isolated from cancer and leukemic patients and animals | • Mycoplasma have been isolated from cancer and leukemic patients and animals |
| 11 | • SC injection of T-bacilli cause lymphomatous, leukemoid disease in mice | • IP injection of $10^8$ CFU/mL of mycoplasma causes fatal leukemoid disease in mice |
| 12 | • Injection of T-bacilli subcutaneously has direct cytopathic effects within 8 days | • IP injection of $10^9$CFU/mL of mycoplasma is cytopathic in 24-48 hrs |
| 13 | • Injection of T-bacilli causes carcinoma | • Mycoplasma have been isolated from bladder papilloma and renal cell carcinoma |

 ISSN 1920-3799

**Table 2**
Basic biological differences between Reich's T-bacilli and mycoplasma.

| | Reich's theory of T-bacilli based on his observations | Known properties of mycoplasma |
|---|---|---|
| 1 | • T-bacilli are produced in the T-spikes of acanthocytes or burr-cells in human RBCs | • Mycoplasma do not bud from human RBCs and are not found in the spikes of acanthocytes or burr cells. |
| 2 | • T-bacilli are found in epitheloid cells of cold sores | • HSV-IV, not mycoplasma, are isolated from infected cells in cold sores |
| 3 | • T-bacilli are heterogenic *de novo* productions from dying or decomposing cells | • Mycoplasma do not heterogenically arise from host cells; the filamentous mode of replication appears microscopically as a *de novo* emergence |
| 4 | • Reich never reported fried-egg colony appearance for T-bacilli cultures | • Most mycoplasma colonies have distinct "fried egg" appearance |
| 5 | • Zig zag motion | • For the most part, immotile, but some have gliding motion |
| 6 | • Injection of T-bacilli causes carcinomatous, epithelioid growths | • Injection of mycoplasmas does not induce carcinomas or sarcomas; tumor formation is rare in nude mice |
| 7 | • Presence of T-bacilli is always pathological | • Most mycoplasmas are symbiotic and not agents of disease |

observed, it would have been most likely attributed (and, in our view, erroneously so) to Brownian motion. Our own observations of live mycoplasma in liquid cultures [30] have shown that their motion presents zigzag movements, as well as flea-like jumps. Finally, in **Table 4**, we present the significant differences between Reich's T-bacilli and viruses in general that preclude any identification of the former to the latter.

### 7. Reich's theory of cancer in light of molecular and clinical oncology

It is fascinating to cross-relate - confront and differentiate - the orgonomic model of cancer, and leukemia in particular, with accepted models of oncogenesis, including those that invoke causation or promotion by viruses and mycoplasmas. Similarly, a cross relation with our own aetherometric and experimental approach to neoplasia [31-32] will prove to be just as provocative.

 ISSN 1920-3799

**Table 3**
Superficial similarities of T-bacilli and viruses.

| | Reich's theory of T-bacilli | Properties of DNA and RNA tumor viruses |
|---|---|---|
| 1 | • T-bacilli form *in situ* from disintegrating cells budding from them | • Replicating retroviruses and DNA viruses arise *in situ* from *infected* cells by budding from them. |
| 2 | • T-bacilli do not cause cancer, but *mediate* its expression | • Viruses (that are integrated genomically) promote *induction* of cancer |
| 3 | • T-bacilli size: 0.2 to 0.3 μm | • Size of retroviruses and herpes viruses: 0.2 to 0.35 μm |
| 4 | • Hypoxia causes production of T-bacilli ("T reaction") | • Hypoxia promotes viral infection of susceptible cells, and viral replication |
| 5 | • T-bacilli are a heterogenic production from cells, each time arising *de novo* | • In their *evolutionary origin*, viruses are *endogenous* productions from cells |
| 6 | • T-bacilli induce agglutination and hemolysis of RBCs | • Viruses that express agglutinins agglutinate and may hemolyze RBCs |
| 7 | • T-bacilli can adsorb to cells | • Viruses adsorb and penetrate susceptible host cells in the course of *infecting* them |
| 8 | • T-bacilli can be isolated from cancer and leukemic patients or animals | • In a minority of neoplasias, viruses can be isolated from tumors and blood cells of patients or animals with cancer or leukemia |
| 9 | • Reich claimed T-bacilli were isolated from sarcoma | • RSV is isolated from sarcomas |

**Table 4**
Fundamental differences between T-bacilli and viruses

| | Reich's theory of T-bacilli | DNA and RNA tumor viruses |
|---|---|---|
| 1 | • T-bacilli are cultivable & self replicate | • Viruses cannot replicate by themselves and are not cultivable without host cells suitable for infection |
| 2 | • T-bacilli are Gram negative | • Viruses do not have a Gram-stain reaction |
| 3 | • T-bacilli are saprophytes | • Viruses are not saprophytes |
| 4 | • T-bacilli have independent metabolism | • Viruses have no metabolism |

 ISSN 1920-3799

The first cross-relation is that afforded by the identification of T-bacilli with mycoplasma, and concerns the evidence for mycoplasma-mediated oncogenesis - from experimental animal models and from in vitro studies of cell transformation following mycoplasma infection. We have succinctly reviewed these lines of research [33], and concluded that, while mycoplasma can transform cells in vitro, these cells are not normally tumorigenic, and likely only become such if another event or set of events has already occurred or survenes (eg infection with EBV, exposure to mutagens, etc). Thus, mycoplasma infection does not cause cancer, but serves as a promoting factor in a cellular environment starved for oxygen and energy.

This, of course, actually fits well with Reich's contention that T-bacilli did not cause cancer, but mediated the onset of a cancerous process. The fact that injection of mice with either T-bacilli and mycoplasma causes leukemoid disease is provocative, but may reflect solely the body's immune response to a massive infection. Moreover, as we have seen, Reich was mistaken in thinking that the "T-spikes" of echinocytes (acanthocytes, burr cells, crenated cells) were T-bacilli. This mistake further led him to design a test for the energy-health or vitality of blood, which is entirely artifactual in method (how T-spikes are formed in RBCs) and conclusions (that they indicate the shedding of T-bacilli from RBCs) [8].

In light of the above, we must conclude that Reich did not succeed in actually isolating mycoplasmas in all the in vivo or in vitro instances where he claimed that T-bacilli were present. Similarly, we are obliged to conclude that he did not isolate T-bacilli as such, since in most instances these T-bacilli were most likely mycoplasmas, but in others they were most likely viral particles. Since Reich conducted this work in the early 1940's and since, as late as the 1950's, research in oncology was still confusing viral particles with mycoplasma, this mistake has no adverse reflection on the quality of Reich's work, even if it led him to a variety of errors.

Lastly, on this subject of the relationship between T-bacilli and mycoplasma - and in light of what has been found during the 1970's and 1980's regarding mycoplasma contamination of serum-containing media, organic media (such as Reich's egg or potato media, etc), and cell lines (see [33]) - we should note that Reich's media preparation methods could easily introduce mycoplasma into his cultures, irrespective of any actual isolation of mycoplasma from diseased animals or human beings.

It is also interesting that Reich considered T-bacilli not to be infectious but their presence to be always a marker of a pathological (biopathic) process of cellular decomposition. In contrast, mycoplasmology has discovered that, while most mycoplasma are benign parasites that don't behave as infectious particles, those mycoplasmas which are saprophytic, cause acute disease (eg *M. mycoides* in pleuropneumonia ), or are involved in cancer promotion (eg *M. fermentans*), are highly infectious.

The second set of cross-relations concerns Reich's key notion of an active biological antago-

 ISSN 1920-3799

nism of the B and T reactions of peripheral blood, upon which he build the entirety of his cancerous and pre-cancerous diagnostics. A "spontaneous B reaction" that "heterogenically produced" blue PA bions, was also and still a healthy reaction against T-lysis of RBCs, with the PA bions (supposedly) attacking and killing the T-bacilli. Such a claim had to be based upon the valid and correct double identification of these "large PA bions" and the T-bacilli. Such identifications were precluded since Reich did not determine either the immunophenotype of the T-bacilli that induced hemoagglutination, or of the PA bions (likely the platelets) that "attacked" the T-bacilli (ie aggregated with the agglutinated cluster).

In the preceding paper [7] we saw how - with reluctance and qualifications - Reich *eventually* concluded that PA bions were functionally identical to the staphylococci and micrococci of microbiology. The B reaction produced large blue PA bions or large beneficial cocci, in the range of 2 to 4 µm, whereas the T reaction produced both T-bacilli and small PA bions, 1µm or less in diameter and blackish in color. Irrespective of the "vital" color problem and the associated obvious objection - that depending on focus, depth, type of slide, light source and illumination method most cocci can be shown under light microscopy to look either milky, bluish, transparent or blackish! - there is a still more profound difficulty with this conceptualization of the B vs T reactions. It stems from the sense that Reich's microscopic observations were not as accurate as he claimed, and that he unforgivably mixed biological entities or systems which are or were provenly different, only very grossly similar, and certainly not susceptible to confusion when high-power microscopy is properly employed, along with standard microbiological techniques of investigation (isolation, immunotyping, cultivation, cloning, etc).

This criticism applies to Reich in light even of the state of biological and microbiological research contemporary with his work. A glaring example of such failures is Reich's contention that "blood platelets are nothing other than blue PA bion bodies surrounded by dead T-bacilli adhering to them" [13]. Even back in 1944-1948, this was clearly a most erroneous assertion, since platelets, aside from their size (2 to 4 µm) and their agglutination into clumps, *can be easily distinguished* from cocci using either live microscopic techniques or simple staining methods for microscopy. Moreover, platelets have phagocytic properties that cocci lack. Whereas cocci may appear blue, milky or even hollow in darkfield, platelets have a central blue granular zone, the granulomere, that never appears hollow (they also have a lighter periphery, the hylomere [34]). Worse still, the production of platelets from the fragmentation of the cytoplasm of megakaryocytes in bone marrow was well known in Reich's time, since Wright, the inventor of the routine hematological polychrome stain that bears his name, discovered it in 1910 [35]! In fact, technically speaking, a platelet is as much a bit of cytoplasm (of a megakaryocyte) as the human RBC is a bit of cytoplasm of an erythroblast. Analogous to

 ISSN 1920-3799

bionous decomposition as the process of production of platelets from megakaryocytes may seem to be ('a release of bions' from regionalized bits of cytoplasm), it is *certainly not* a biopathic process of cellular decomposition, but an essential, normal hemopoietic process! This example underlines the kind of mistake that Reich began making more and more frequently in his experimental work - the premature identification of elements or even the claimed functional identification of processes on the basis of gross formal or optical analogies. Platelets are not blue cocci or PA bions, anymore than megakaryopoiesis is an heterogenic process of bionous disintegration. When the cytoplasm of megakaryocytes breaks down, it does not produce cocci, but cytoplasts with no DNA. Thus while cocci (and PA bions) are cultivable, platelets cannot be cultivated, since they neither grow nor replicate (nor do RBCs, for that matter).

Similarly drastic errors underlie Reich's view of acute and chronic inflammation. Inflammation is fundamentally a response - initiated by vascular (endothelial) and connective tissues, and continued by leukocytes - to mechanical insults, chemical irritants or bacteria. In the absence of pathogenic bacteria, acute inflammation processes will develop successive tissue infiltrates of mononuclear and polymorphonuclear cells without suppuration. The general process of inflammation follows a series of well defined steps: the ground substance of the tissue is depolymerized and lydrolyzed; capillaries and venules become locally dilated to increase blood supply and permit leukocytes (WBCs) to exit from the vessels into the tissue; the permeability of the vascular wall is increased to permit blood plasma to enter the affected area and form an edema, thus creating pressure on the nerve endings. Initially, connective tissue cells like mast cells (histamine producers) control the process by degranulation of their inclusions, but soon the dynamic processes of leukocytes take over. Monocytes are locally activated to become *amoeba-like macrophages* (which are normal and *not* transformed cells) with pseudopodia that phagocytose bacteria and viruses, and they also degranulate to release acid enzymes and control factors (monokines). Polymorphonuclear leukocytes and lymphocytes engage in both cyto-immune (cytotoxic) and antibody-immune (humoral) responses, with neutrophils, eosinophils and basophils degranulating and releasing a variety of microcidal compounds (acid enzymes, lactoferrin, heparin, histamine, serotonin, iodides, chlorides, fatty acids, lysolecithins, and a variety of inflammation control factors, including prostaglandins and interleukin-8 [36]). The role of neutrophils (microphages) is central to the early part of any inflammatory process - both by the release of microcidal compounds and regulatory factors, and by their active phagocytosis of foreign material or micro-organisms. Impaired neutrophils or lack of neutrophils leads to rapid death from bacterial infection. With darkfield microscopy, it is easy to spot bilobate or trilobate mature neutrophils - the nucleus appears dark - with their infernal dance of cytoplasmic membrane-bound organelles, formed by both granules and mitochondria (see **Fig. 12**). The appearance of these

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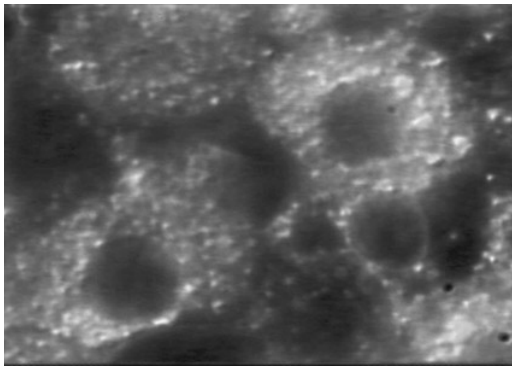

**Fig. 12** - Darkfield view of the vesicular appearance of neutrophils in the human oral cavity (segmented nuclei only apparent by changing focus). The vesicles present an incessant movement, and they are composed of a variety of organelles - some DNA-bearing like mitochondria, others being a variety of microsomes (lysosomes, peroxysomes and phagosomes). Oil immersion, 100x apochromat. Hitachi VK-C2000 CMOS camera. Print magnification: 1,900x.

organelles would easily fit the description of "PA bions and rods" in Reich's language, the neutrophil being "vesiculated" or presenting a vesicular bionic structure; yet, aside from mitochondria, the intracellular vesicles are "microsomal granules", ie either intracellular structures filled with bacteriocidal compounds, or phagosomes containing bacteria (cocci included) at different stages of bacteriolysis. Indeed, neutrophils have essentially two types of granules, the "*specific granules*" which contain lactoferrin (that binds iron to deprive bacteria of it, and feeds back a signal controlling hemopoiesis of neutrophils), microcidal cationic enzymes (alkaline phosphatase, peroxidases), iodides, chlorides and lecithins, and the "*azurophilic granules*" that contain acid hydrolytic enzymes. Granules either fuse with phagosomes, to form active lysosomes, or fuse with the plasma-membrane to release their contents to the intercellular space.

This exploration of the inflammatory processes is of interest to our present assessment of Reich's model of cancer stages and his notion of a precancerous chronic inflammatory stage, because the processes normally involved in evoking and controlling inflammation lend themselves to the kind of confusion with the interpretation suggested by Reich's theory of the B vs T reactions of tissue under stress. Let's make a short list of a circuit of such premature confusions:

• "Cells that are round and become pseudopodial amoebae" - that is the normal process whereby monocytes become activated and "transform" (note the linguistic imprecision) into macrophages, and granulocytes develop anterior pseudopodia [37].

• "Ovoid cells become elongated, oblong, tear-shaped and club-shaped" - that's the process whereby lymphocytes become motile and assume the 'hand-mirror configuration' with a trailing uropod when they move [38].

• "Cells appear vesiculated, and release fluid and the content of their vesicles" - that's the process of degranulation of the microsomal inclusions of granulocytes and macrophages.

• "PA bions are attracted to amoeboid formations and become part of them" or are ingested

 ISSN 1920-3799

by them - that's the process of monocyte and neutrophil phagocytosis of the cocci that opportunistically show up and replicate when the inflammation is caused by viral or bacterial infection, or has become chronic and hypoxic.

• "Cells breakdown into PA bions, releasing individual bions or packs of them" - that's either the process of degranulation of granulocytes, or the still *very different* process of production of pus, when tissue infiltrates made up of dead and dying leukocytes, mostly neutrophils, catastrophically release their content of granules and mitochondria.

• "PA bions attract one another, forming irregular packs that attach to damaged tissue" - that's the platelet aggregation response observed when blood vessels and exposed subendothelial tissue are damaged mechanically or by inflammation, whether its source is infectious or not.

Of course, most of these inflammatory processes were not at all understood or properly articulated back in the 1940's when Reich carried on his bion and cancer research, just before the advent of molecular biology and biochemistry. Yet, most of the histology and histogenesis was already known - that platelets were formed from marrow megakaryocytes (back then known as megalokaryocytes); that neutrophils and other granulocytes prevalent in tissue infiltrates in acute supurative inflammation had phagocytic activity and produced lysozymes in their granules; that these granules were released from these cells; that macrophages prevalent in chronic inflammation were amoeba-like wandering monocytes capable of phagocyting bacteria; and so on.

The next set of cross-relations concerns the biology of malignant transformation (oncology proper) - how does a malignant cell arise, and what does transformation consist of. Molecular and clinical oncology has not validated a single of the biological contentions of orgonomic oncology with respect to the generation of cancer cells (malignantly transformed or neoplastic cells). Those cancer cells which were, back in the 1920's to 1940's, thought by some investigators to be foreign protozoal parasites are today understood to be *metastatic amoeboid-like cancer cells* with highly mutated host genomes, often with pronounced aneuploidy in the most aggressive instances of fast replication and tissue invasiveness. Thus, both histologically and clinically, the orgonomic model of the etiology and staging of cancer - and its reliance on what we discussed above as the second and third oncogenic pathways - has not been verified. In other words, the vector that Reich claimed existed between the decomposition of cells into bions, and then the reorganization anew, heterogenically, of a cancer cell or an amoeba from the spontaneous assembling of these bions, just does not exist. All known instances of transformed cells result from single cell alterations that are transmitted to the daughter cells of a clone. Reich severely mistook the self-aggregation of sarcinoid prolaryotes and their common secretion of an envelope (not a cellular membrane), as discussed in [7], with the "spontaneous generation" of an amoeba or a protozoon. He made an inference from optical observation, which has

 ISSN 1920-3799

no biological reality: the sarcinoid clusters are not protozoa ('packet amoeba'), but colonial bacteria.

The more definitive set of objections to Reich's theory of malignancy concern the identification, by molecular biology and modern clinical medicine, of the molecular and cellular processes that lead to oncogenic transformation. After the initial domination of the field of oncology by the oncogenic theory of viral-induced transformation (see [39] for a review), modern oncology was forced to admit that oncogene activation needed neither viral infection or virally-induced activation. The so-called 'cancer genes' (oncogenes) were normal genes after all, just altered in their expression. The initial in vitro successes in inducing the malignant phenotype by altering expression of oncogenes were soon tempered by the realization that there was no simple one-gene:one protein model of oncogenesis - unlike what Renato Dulbecco once hoped for [40]. From complex varieties of mutagenesis to altered epigenetic control, there were multiple and complex pathways toward neoplasia. But more important still, modern oncology was also forced to admit that malignant transformation was, for the most part, encompassed by a variety of *nonrandom* genetic alterations of metabolism, growth and differentiation (for reviews, see [31-32]). The realization was finally sinking in that cancer was not, except for a minority of instances, a familial or inherited disease - anymore than it was caused by infections with certain viruses. Rather, it formed a complex adaptive system [41-42] that genetically responded to biophysically and biochemically altered cellular and tissue environments. Each transformational event mobilized a variety of adaptive mutations [43-44], most cancers being an acquired disease that selected for these alterations.

We should note that in humans, and to this day, viral *causation* of neoplastic transformation has only been circumstantially accepted (and not proven) for Epstein-Barr virus (EBV) infections (eg infectious mononucleosis) and the B-cell lymphocytic leukemia known as Burkitt's lymphoma [45-47]; and for adult T-cell lymphomatic leukemia (ATL), which is endemic in southern Japan, the Caribbean basin and Central Africa, and associated with HTLV-I (human T-cell leukemia virus I) infection and expression [48]. Claims that human papilloma virus (HPV) causes cervical, uterine, vulvar and penile carcinomas [49-50] still remain controversial.

The majority of acquired human cancers and leukemias is *not* virally caused or virally promoted. And there is no overall accepted model or pathological vector of neoplasia that can stage all known forms of malignancy and "pre-malignancy", not even a conceptual understanding of what "premalignancy" is. What is apparent, however, is that - leaving aside the minority of cancers caused by exposure to ionizing radiation or chemical carcinogens, by viral induction or familial genetics - cancer is a dynamic and acquired ('adaptive') disorder. In this neo-lamarckian sense, then, modern oncology has moved closer to a theory of the dynamic causation of cancer, and thus to Reich's hypothesis regarding the role of sympatheticotonia and its link to hypoxia. But conversely, at a cytological

 ISSN 1920-3799

and molecular level, oncology has moved away from any theory, like Reich's, that invokes 'spontaneous generation' (heterogenesis) of cancer cells. For, it has also become well established that in all specific tumor formations and varieties of leukemic disorders, the neoplastic process always operates by conversion ("transformation") of single cells. There is absolutely no evidence for malignant cells arising heterogenically from the decomposition of normal cells, nor *de novo* from the assemblage of cocci into packet-amoebae, as Reich claimed. Moreover, if there is a biological process to cancer, a vectorial process that involves more or less defined stages, this can no longer be reduced to a random, one-hit transformation of a single cell. Increasingly, the neoplastic vector appears to involve a series of cellular alterations - occurring in sets of cell populations - directed to respond to a neoplasia-promoting environment. Each set of changes is a complex of hits that targets a single cell, from which, if the changes are effectively adaptive, a transformed clone develops. Furthermore, with the progression of the neoplastic vector, the single cell or single cells that are each time targeted by any set of changes is a more and more primitive type of cell, whether in somatic cancer or in cancer of the blood (leukemia). Most tumors present both mutagenized populations of monoclonal origin, and a polyclonal structure. In leukemia, the transformation of more primitive or immature cells gives rise to acute conditions characterized by blastosis.

Thus, a neoplastic vector affects the conversion of a single cell or single cells at any stage of the process, with each stage originating one or more clones of transformed cells, and all the stages being concatenated by a transformation vector that is increasingly malignant and more aggressive. Indeed, there is not a single state of malignancy, or a single definition of such a state. Cancer is a multiplicity. Transformation, aside from the distinction between benign and malignant, is a concept that encompasses rather distinct cell types or types of cellular conversion. In particular, some stages of transformation only exhibit hyperplasia of differentiated cells - seemingly defining solely proliferative disorders (pre-blastic chronic leukemias actually fall under this rubric) - and not neoplastic properties per se - such as metaplasia, tissue invasiveness or tumorigenesis. However, as we have explained in communications presenting the aetherometric models of acquired oncogenesis and leukemogenesis [31-32], when the differentiated phenotypes of these proliferative disorders are analyzed, they always contain some form of dysplasia and dysfunctionality, and often signs of anaplastic reversion to embryonal-type responses. The existence of recurring adaptive genomic mutations (as found in the chronic myeloproliferative disorders, CMPDs, for the *JAK*-2 gene [51-55] encoding a Janus kinase) with their complex crosstalks (eg *JAK-2* with the Insulin-like Growth Factor-I receptor, IGF-IR and the *SOCS* genes in *Polycythemia vera*, PV [55-56]), then attests to the transformed state of these cells. Thus, these diseases may not exhibit tumor formation or any overt blastosis, and yet appear already to be malignant. All happens as if they form *a first stage of malignancy*, when the transformation vector has *not*

 ISSN 1920-3799

*yet* fully arrested differentiation and produced either clones of tumor-forming cells or "blastic crises". The latter only happen when, typically, a more primitive cell undergoes metabolic conversion into a *lactic-fermenter*, suppresses its mitochondria, and acquires amoeboid-like morphology and properties (fibroblast focus assays and growth in liquid media are typical assays of amoeboid properties: loss of topoinhibition and contact inhibition, and acquisition of pseudopodial motion). In other words, when a metaplastic conversion occurs that blocks differentiation entirely and permits even greater rates of hyperplasia ('wild mitosis') than the properly speaking 'hyperplastic stage'.

To simplify, we shall say that the oncogenic vector encompasses at least two distinct stages, one where proliferation becomes hyperplastic and differentiation dysplastic and anaplastic, and another where even greater rates of proliferation coexist with metaplastic conversion to a protozoal state, and the acquisition of high tumorigenic potential. In other words, not all malignantly transformed cells have the same biological characteristics and neoplastic potential. For example, in this sense, the CMPDs are already a neoplastic disease, not a pre-neoplastic disorder.

These various plasias or stages of histological disorder do not involve the same cells, and not even necessarily the same tissues. For example - following Spivak's recent revision of Wasserman's long-standing "natural history hypothesis" [57] for *Polycythemia rubra vera* (PV) [58], there appears to be an essential link between PV and the later manifestation of acute myeloid leukemia in the same patients. The marrow erythroid hyperplasia that characterizes PV - and which is driven by an IGF-1R hypersensitivity to IGF-I [59-61] - eventually leads to marrow depletion, though not irreversibly - as Spivak points out - since there are prominent iatrogenic and nutritional-deficiency factors also at work. This depletion is further aggravated by marrow fibrosis, possibly also as a result of the activation of IGF-IR in fibroblasts. The result of marrow depletion and fibrosis is refractory anemia, and ultimately marrow death or sclerosis. It is the depletion and failure of marrow that exacerbate the necessity to produce *not just red blood cells*, *but all other blood cells*, by some alternative process. This pressure is now exerted upon the most primitive stem cells, and their eventual response is to become transformed blast cells. It is at this point that the final neoplastic transformation technically characteristic of leukemia occurs. The marrow does not simply spill over with the resulting blastosis, but the blastosis rather seems designed to seed the body with what are effectively metastatic sites of abnormal hematopoiesis, typically in organs that embryonically were once the sites of hematopoiesis (such as spleen and liver), or in still others (lymph nodes, kidneys), converting one type of tissue (eg spleen) back into another (eg marrow-like) in what is known as myeloid metaplasia. Thus, in the case of PV, the leukemic transformation does not hit the cells of the polycythemic, or hyperplastically transformed, clone, but other blast cells in the patients marrow.

In light of the preceding, the notion of a pre-malignant state or disposition can only refer to

 ISSN 1920-3799

conditions that promote, cause or usher in the first stage of the neoplastic vector. It is in this unexplored respect, that, in our view, Reich's theory of the origins of cancer in the autonomic disturbance of chronic sympatheticotonia has merit and deserves verification and investigation. For Reich's observation of sympatheticotonic disturbances in neurotics provocatively interlocks with both the discovery of the Warburg effect in cancer cells (increased rates of glycolysis shunting pyruvate to lactic acid and acid ion, rather than to the oxidative phosphorylation), and the neo-lamarckian school of thought regarding the post-adaptive nature of malignant transformation.

After all, neoplastic transformation - or, rather, the entire vector of neoplasia - exists only in response to the pressure of an altered biological environment, ie non-physiological constraints that prevent tissue from performing normally. The major neoplasia-promoting stress is hypoxia, and thus the entire vector aims, by directed mutagenesis, at the ultimate or terminal transformation of a cell into a lactic fermenter. This is in line with all those theories of cancer (O. Warburg's, W. Reich's, A. Szenty-Gyorgy's, J. Watson's, etc) which have claimed that the universal trait of all cancer cells is conversion of metabolism to lactic fermentation [62]. But, in fact, just as most tumors are polyclonal and not monoclonal, *not all transformed cells are lactic fermenters*; and not even all tumor-forming cells are lactic fermenters. All transformed cell types of what we denoted, just above, as the first stage of cancer still present mitochondria and intact aerobic respiration. And inside tumors, cells close to the blood supply are typically lactic respirers that also retain mitochondria, but symbiotically import the lactate produced by the more aggressive lactate fermenters in the same tumor [63-64] (for a discussion see [31]). Thus, what one should rather say is that the end-point of the neoplastic vector is the neo-lamarckian development and active selection of protozoa-like lactic fermenters.

Yet, as we have also examined at some length, hypoxia is not the only driver of the neoplastic vector. Once engaged, the neoplastic vector deploys hypoxia-independent cellular control systems that modify the response to hypoxia and to growth factors. We have presented a model that takes hypoxia-dependent and -independent processes into account in both oncogenesis in general and leukemia in particular [31-32].

However, what Reich was fundamentally missing to complete his hypothesis of sympatheticotonia-induced cancer-causing chronic hypoxia, was the link or links between the autonomic disturbance and the pre-neoplastic state. In our own work, we have suggested that this link is not the status of the B-vs-T reaction [8], but the complex of relations between anemia and polycythemia, which likely passes through the induction of a mild, subclinical hypoproliferative anemia, that we have termed the "stress anemia" hypothesis [32]. The central notion is that hypometabolism induced by stress, fatigue and anxiety depresses respiration and thus oxygenation, and that, instead of the normal response being increased erythropoietin (EPO. the hormone normally responsible for RBC dif-

 ISSN 1920-3799

ferentiation) secretion by kidney cells, the EPO secretion is slightly depressed; or that, when EPO secretion is normal, the response of marrow erythroid progenitor cells is slightly more insensitive to EPO (possibly through decreased expression of EPO receptors). Either way, there is a long-term partial failure of the EPO circuit to respond to the hypoxia, with the result that tissue inflammation will become chronic in the most vulnerable organs or tissues (those less susceptible to be properly oxygenated). Subclinical anemia in geriatric frailty seems to be associated with IL-6 induced chronic inflammation [65], and it is possible that pronounced IL-6 levels might be a more general marker of subclinical anemia, expressed whenever the hypometabolic condition is depressed enough that tissue inflammation results. In our model, it is the stress anemia, rather than T-lysis, which marks the *pre-neoplastic* dynamic disposition to neoplasia.

Lastly, we must comment on the fairly useless concepts of bionous organization and bionous decomposition of tissue cells. The eukaryotic cell is not a colonial packet of vesicles, anymore than an amalgam of various structures - vesicles, vacuoles, fibers, and complex membrane systems - in solvated and gelated phases. It is a highly ordered set of processes that uses an assemblage of structures. To say that an eykaryotic cell is an assemblage of vesicles or is bionously organized does not have any real biological sense. And even less, if the vesicles are thought to be a generic concept, as if all vesicles were equal. Even if a cell were merely an assemblage of vesicles, it would matter what vesicles. Any aerobic eukaryotic cell contains a large array of vesicles - some are mitochondrial, others microsomal (which means they may be of a large variety, including peroxisomes, glyoxysomes, lysosomes) or vacuolar, and still others part of interconnected intracellular membrane systems (Golgi apparatus, endoplasmic reticulum). Pigment inclusions are frequently granular and have a vesicular appearance. Yet, it suffices for one to remove from the picture a single of these kinds of vesicles - the mitochondrion - for the cell in question to cease to be aerobic. Thus, none of these vesicles are identical or belong to a 'bion realm'. Some are secretory, others lytic, others are endosymbionts - like the mitochondria, the plastids and proplastids, and the microsome-like hydrogenosomes. An hepatocyte has many vesicles, including mitochondria, but when one stains it with Schiff's periodate and hematoxylin (see **Fig. 13**) the vesicles that light up are not the mitochondria but the glycogen storage granules - which are only one aspect of the cytoplasmic structure of the cell. Other than as a diseased cell, it is impossible to have a cell be an hepatocyte without having glycogen inclusions inside of it. Thus the nature of the prevalent vesicles or their combination is a correlate of the specialized phenotype of the cell in question. Without the correlate or correlates no specific phenotype or distinct phenotypes are possible.

So, not only is a cell not a mere congregation of vesicles, but the nature and composition of any intracellular vesicular multiplicity is a correlate of the cell type and its differentiation, or differentiated state. We also want the reader to understand that vesicular structure and composition, by

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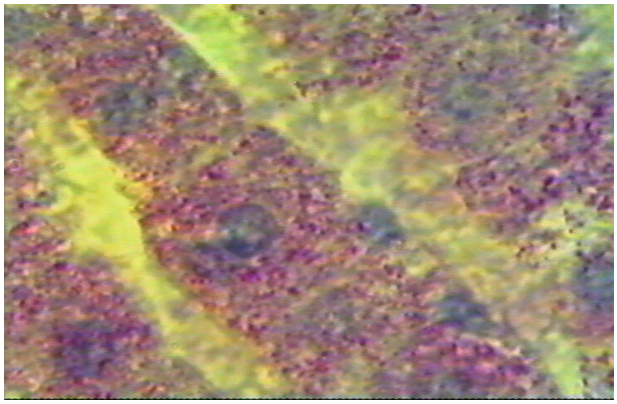

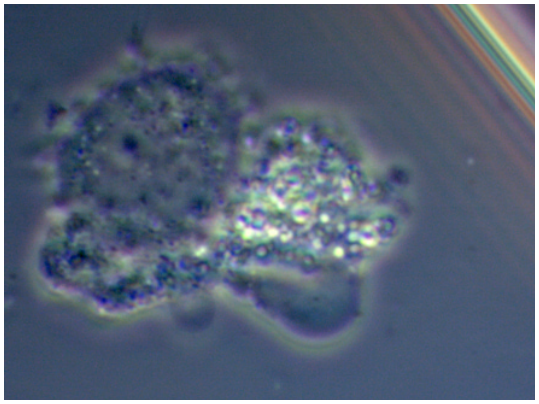

**Fig. 13** (**top left**) - Normal vesiculation of human cells: glycogen storage granules in rows of hepatocytes. Periodic Acid-Schiff and hematoxylin. Oil immersion, 100x apochromat. Hitachi VK-C2000 CMOS camera. 1,100x.

**Fig. 14** (**top right**) - Live oval fat histiocyte (macrophage) in urine, laden with phagocytosed prostatic secretory globules (white) among mitochondria (blue or blue-green) and microsomes (mostly lysosomes, "black"). Oval fat histiocytes may or not be symptomatic of disease, and may or not be malignant. They are observed in association with non-pathological spermatoceles, with benign renal cysts, with renal cell carcinoma, or in inflammatory or infectious prostatitis. Phase contrast, oil-immersion. Luminera Infinity-1 CCD camera. 2,000x.

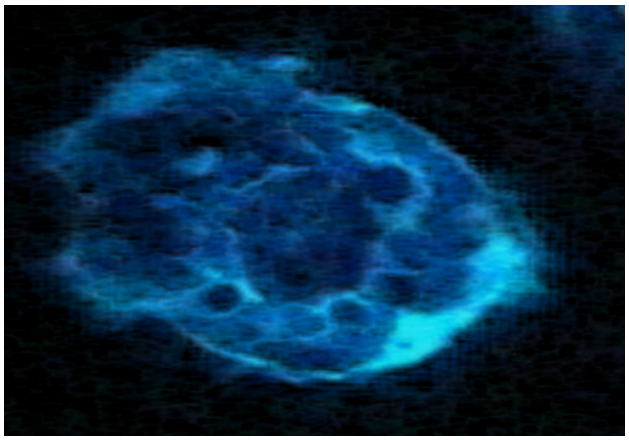

**Fig. 15** - Natural blue fluorescence of a myeloblast in a *Polycythemia vera* cell line cloned from a circulating progenitor cell. The organellar vesicles that fluoresce in the cytoplasm are mitochondria. The outline of the nucleus in dark is apparent. Epicondenser, 40x apochromat with diaphragm. Nikon Coolpix P5000. 3,500x.

itself, is neither an indicator of normalcy or health, or of disease per se. The appearance, for example, in human urine, of macrophages and neutrophils filled with phagocytosed prostatic or spermatic secretion (the so-called oval fat histiocytes, see **Fig. 14**) is diagnostic of a disorder, possibly a disease, but is not specifically a diagnostic of anything. These oval fat histiocytes are doing their normal work of scavenging excess secretory and alkaline material, and will be expelled from the bladder. Yet, they appear in non-pathogenic disorders, like spermatocoeles, or in pathological states (prostatitis, benign renal cysts) that may or may not also include malignancy (renal cell carcinoma, bladder carcinoma) [66]. In other words, there rarely are single markers or symptoms that inequivocably specify def-

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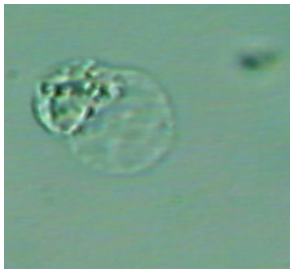


**Fig. 16** - When induced to differentiate, a transformed Friend cell extrudes both the nucleus and all its organelles, leaving the cytoplasma with no or few organelles behind. The differentiation is abortive and the cell dies. Death by self-excision has been documented in a variety of cancer cell lines and termed autoschizis (after Gilloteaux J et al (1998) *Scanning*, 20:564). The vesicles are composed by both mitochondria and microsomes. Before extrusion, the nuclei and the vesicles group together where a pseudopod or 'bleb' will form. Brightfield (40x apochromat). Hitachi VK-C2000 CMOS camera. 1,900x.

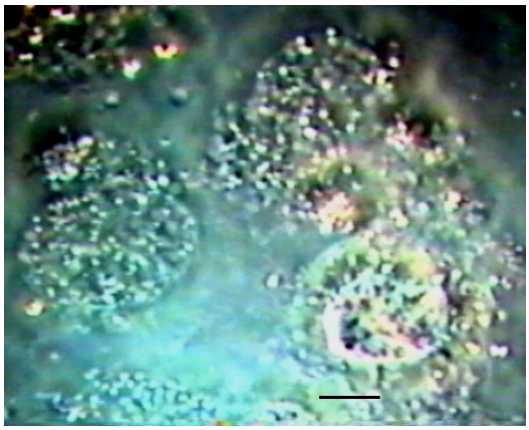


**Fig. 17** - Spontaneous hypertonic lysis of 3 *Strombidia* (*Ciliata*, *Oligotricha*) releases both mitochondria and dinoflagellate endosymbionts. The outlines of the cell plasma membranes are still visible, as well as the partially decomposed nucleus of one of the protozoa. Phase contrast, Sony TVR68 CCD camera. Bar is 15µm.

inite pathological conditions, cancer being no exception. Likewise, as biologists, one should refrain from making rash generalizations. Not only have we just reminded the reader of the fact that cancer cells are not all and always lactate fermenters - and that tumor cells establish a symbiosis between the lactate fermenters and lactate respirers - but one should keep in mind that even very aggressive malignantly transformed cells may still employ mitochondria, and perhaps do so in ways that are not limited by the normal pyruvate-based substrate phosphorylation carried on by mitochondria under physiological conditions. Blast cells from a leukemia cell line derived from a *Polycythemia vera* patient show (see **Fig. 15**) the natural blue fluorescence of cytochromes in the double membrane (the outline) of mitochondrial vesicles closely apposed to the outlined nucleus.

Similarly to the concept of 'bionous organization', the concept of "bionic decomposition" of tissue cells is not illuminative of anything in particular. There are interesting details of the behavior of various eukaryotic cells, protozoa included. The most provocative we can think of is the response of transformed murine Friend cells to certain types of induction to differentiate, such as simple exposure to the partial pressure of atmospheric oxygen. Even though murine RBCs do not extrude the nucleus - as human RBCs do - the Friend virus-transformed cells attempt to push the dying or pyknotic nucleus and all vesicles into a pseudopodial bubble (see **Fig. 16**), as if trying to extrude all of its vesicular material at once was the first step in cell lysis, and perhaps a phylogenetic tendency

 ISSN 1920-3799

that human RBCs eventually exploited (model of the arrested or intercepted apoptosis or autoschizis in the production of enucleate RBCs). Clearly, there are different pathways to cell death, and different forms of lysis, some of these pathways having been exploited by normal cells to develop different differentiation phenotypes.

However, it is rather obvious today that from a microscopic viewpoint, the only products of cellular lysis that are apparent are the vesicular ones - all submicroscopic structures, fibers and fibrils, membrane systems, macromolecular aggregates, etc, being invisible. Again, a reliance on the 'apparent truth of optics' is not a friend of the biologist. Once more, we must say that cells do not break down into indistinct and equal vesicles. Arguably, an oval fat histiocyte is bound for destruction and cell lysis. But it will not break down into the same vesicular products as, say, will an hepatocyte or a cell from the so-called higher plants, or a protozoon. Summarily, an oval fat histiocyte will release fat phagosomes, mitochondria and various microsomes; an hepatocyte will release glycogen granules, mitochondria and various microsomes; and a plant cell will also release proplastids and plastids, and the protozoal *Strombidium*, for example (see **Fig. 17**) will release mitochondria and dinoflagellate endosymbionts (very small protozoa, in a definite example of '*emboîtement*').

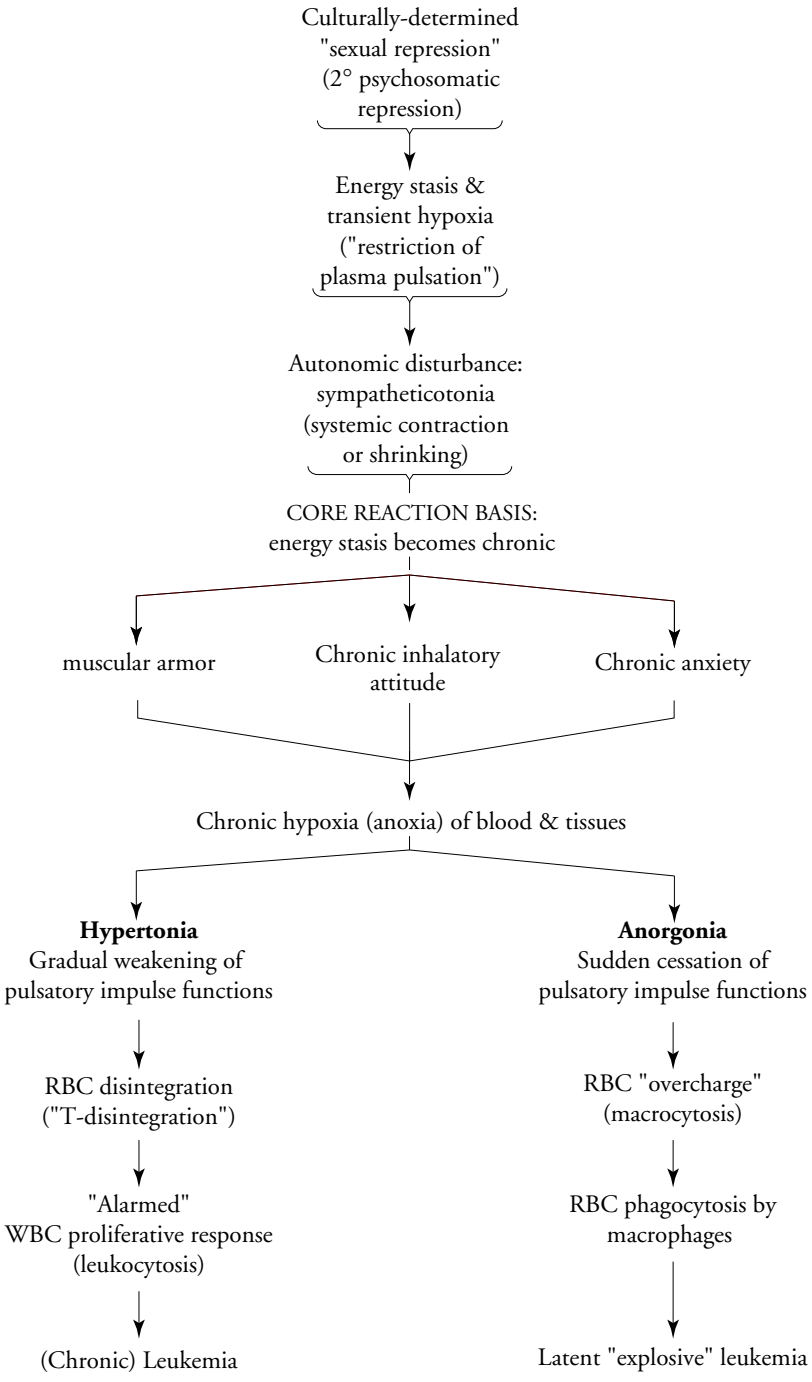
Whether any or some of these vesicles have the properties of hyperthermal resistance that Reich claimed for his PA-type bions, and are or not functional components of normal tissue cells, that is a question that only further experimental investigation may answer.

**8. Conclusion**

Aside from the claim of a primary involvement of the RBC compartment in every form of cancer and leukemia - a claim that may well turn out to be correct, but is in dire need of further investigation and reformulation - the main contribution of Reich's theory of cancer can be ascribed to his set of dynamic linkages between social and sexual repression and the armoring and anxiety mechanisms ultimately responsible for tissue hypoxia. **Fig. 18** presents Reich's complete view of what he called the 'cancer biopathy', with respect to his argument of an inherent erythroid dysfunction that eventually leads to leukemia. Aside from these dynamic linkages, it is noteworthy that Reich also tried to differentiate between an initial leukemic response characterized by an auto-immune reaction against the dysplasia and the dysfunctionality of produced RBCs and their fragments, and a latent 'explosive' leukemia caused by RBC macrocytosis ('overcharge' of the RBCs), with increased RBC phagocytosis and splenic sequestration. This separation largely coincides with the distinction between chronic and acute or fulminating leukemias, with the former generally marked by leukocytosis with differentiated WBC phenotypes, and the latter by leukoblastosis. Reich's notion that a severe RBC

 ISSN 1920-3799

**Fig. 18** - Reich's view of the cancer biopathy as a process that originates in social and sexual repression, involves an autonomic disturbance and a characterological armor, and develops an inherent or subjacent erythroid dysfunction that eventually leads to leukemia, whether chronic or acute.

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dysfunction underlies every form of cancer suggests that an incipient chronic leukemia forms as a response to RBC dysfunction and coexists with any manifestation of somatic cancer. We will return to these original perspectives in the follow-up communications that address various other models of the etiology of cancer.

It is also remarkable that the argument formulated by Reich for cancer induction (not causation) by 'T-bacilli' is mostly coadunate with the current approach to induction of leukemoid disease and malignancy by certain strains and species of mycoplasma (for a review and analysis of this work see [33]). However, the distinction between B and T reactions is not a valid diagnostic technique, nor based on sound foundations, *viz.*: neither T-bacilli nor mycoplasma are shed by RBCs; most mycoplasma, read 'T-bacilli', do not express disease states, but are symbiotic even if parasitic; the identity of blood PA-bions is un-ascertainable, since it may mean a large coccus, a small one, a platelet, a free mitochondrion, a cytoplast; the T-lysis test of RBCs suffers from a constitutive artifact [8]; cancer cells do not assemble *de novo* from PA-bions; and so on. The *Achilles heel* of Reich's theory of cancer lies, therefore, in the cellular biology of oncogenesis or transformation. He thought that he saw a link between his theory of cancer cell production and his own theories of biopoiesis of prokaryotes and eukaryotes [7, 16]. It was this that sunk Reich's theory of the cancer biopathy - irrespective of its contribution of a model of the relation between sympatheticotonia and hypoxia in oncogenesis; of the suggestion that an erythroid dysfunction underlies all forms of acquired or functional cancer; of the notion that cancer is a functional process (which antedates the current view of cancer as an adaptive system); of the proposed dynamic localization of metastases; and of the relation between T-bacilli and mycoplasma. But, as we believe our own work shows, confrontation of current oncology with the medical orgonomic theory of cancer can be productive of new insights. These, increasingly seem to converge toward the concept that cancer is an 'evolutionary laboratory', a neolamarckian process that addresses the biology of systems where the logic of the organism has histologically stifled certain organs or cells, certain parts of the system, submitting them to an adaptive pressure (eg hypoxia). The malignant response seems to be trying to find, at the cytological level, a way out, and in general this seems to be the 'liberation' of an individual cell from the stifling tissue constraints by means of a process of altered epigenetic control and post-adaptive mutations that result in the neoplastic transformation. That it effects what appears to be a reversion - in histological phenotype and metabolism - may well be inevitable, since it is the respiratory system of the cell which, to begin with, became superfluous when hypoxia set in.

 ISSN 1920-3799

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**2.** For Reich, the social repression of desire operated by despotic or 'patriarchical' societies constituted the original form of this psychosomatic repression. See, in this sense, **Reich W (1937) "People in trouble", published in 1953 by the Orgone Institute Press, Orgonon, ME, pp. 44-49 and 86-97**. Reich distinguished between two types of repression - social and sexual. The former was effectuated by specific social institutions, but had - across History - growingly relied upon the sexual repression of desire, upon an unconscious effectuation of repression. Sexual repression was therefore an internalization of social repression, and its original form was secreted together with the institution of a State. This differs from the more consistent analytical treatment given to the matter by Gilles Deleuze and Félix Guattari in the "*Anti-Oedipus*", which actually reinforces Reich's argument while altering its freudo-marxist content and articulation, but yet was *completely lost in the english translation of that book*. The very fact that the vegetative nervous system possesses a sympathetic pole of contraction (in the deleuzo-guattarian language, a "paranoiac machine") is evidence for a *primary* (or 'originary') *form of psychosomatic 'repression'* (drawback or 'refoulement' of the 'partial-objects' or 'partial-organs' of desire). The internalization of social repression could not succeed unless it had found a neurological support (some biological machinery) that it could engraft. Thus, whereas the *primary psychosomatic repression* is simply sympathetic, the *secondary psychosomatic repression* is sympathetico-*tonic*, or tending towards sympatheticotonia. The approach suggested by Deleuze and Guattari - specifically in the "*Anti-Oedipus*" - also distinguishes between the social, cultural and historical stages of development of that secondary psychosomatic repression, until it becomes an achieved castration complex. See, for example, in the original French **"L'Anti-Oedipe" (1975), Éditions de Minuit, pp. 142-143**. In this context, because incest only figures in savage societies as a "displaced represented of desire" that is *unoccupied* by desire (note how this differs from Reich, who claimed that "in primeval times brothers and sisters were *natural mates*", and that "incest was the basis of human society", see **"People in Trouble", p. 89**), *no secondary psychosomatic repression* is formed in these societies, only a "repressing territorial representation" that leaves most of desire uncoded. In contrast, "despotic machines" are established upon a royal prerogative of incest, the "displaced represented" or false image of desire being now *occupied* as the instance of a "repressing despotic representation" that codes and overcodes all desire - the desire of the subjects becoming solely desire of the despot's desire. Ultimately, in the age of civilization, when all codes and overcodes are subject to the axiomatics of

 ISSN 1920-3799

capitalism, the "displaced represented" turns into the "false representative of desire", as the 'analytical content' of the castration complex.

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**6. Idem, p. 234.**

**7.** See our preceding report, **Correa PN & Correa AN (2010) "The PA & SAPA bion experiments and proto-prokaryotic biopoiesis", *J Biophys Hematol Oncol*, 1(2):1.**

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**13. Idem, p. 284.**

**14.** What pathologists today refer to as chronic inflammation is characterized by the appearance of *mononuclear* cell infiltrates (monocytes, particularly macrophages, and lymphocytes) in affected tissues. Since, for Reich, T-lysis marks the threshold when pus-forming or suppurative processes appear, it would seem that his reference was *not* chronic inflammation but *bacterial acute inflammation* characterized pathologically by *polymorphonuclear* cell infiltrates (granulocytes, mostly neutrophils).

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