Defense Weakness in the Respiratory System
and Its Antihomotoxic Therapy

"We receive a twofold blessing upon drawing every breath:
Taking in the air -- allowing it to discharge its load;
The one a precious burden, the other a refreshing relief:
How wonderful life is in its doubly mixed blessing."

(Goethe)

Indeed, breathing is the very essence of life itself: rhythmical accepting and giving in bipolar interplay. In ancient languages, we find the same word used for breath as for soul. In Latin, spir- are means to breathe, and spiritus means the spirit. In Greek, psyche stands for both breath and soul. In Sanskrit, atman is also the term for breath as well as for soul. The Sanskrit for a person at a very high level of spiritual development is mahatma, a great soul -- or, a great breath. Atman, breath, was the bearer of life -- of prana -- not only in ancient India; in Christian mythology as well it was the divine Odem which made a living spiritual being out of the clump of the Earth.
Breath is therefore something which comes into us from without. Breath does not essentially belong to us; rather, we exist in breath. Through breath, our form is connected with the formless -- with something which extends far beyond our delimited being.

Breath also prevents the limits of our innermost being from complete seclusion from the surrounding world. Through breath, the ego is forced to maintain its connection with the non-ego outside us -- is forced to take part in the rhythm of the universe.

And who can deny the unescapable reality that all of us must intimately share the same life-giving air -- even with our worst enemies? The very same air that animal and plant breathe.

This contact is, indeed, intensively close and profound, as it takes place in rhythmical manner between our personality and the biosphere of our environment. The surface area of our lungs which provides this contact is, after all, approximately eighty square meters in size. The surface area of our skin, in contrast, is tiny: only one and a half to two square meters -- and skin contact with the outer world can, to an extensive degree, be granted and denied as we voluntarily wish. Quite different, the contact of air with our lungs: an involuntary meeting through the alveolar mucous membrane which touches our very souls. This is why someone can "take your breath away" or "take the wind out of your sails." And
why another can provide us a "breath of fresh air," even in the most polluted atmosphere.

Such figures of speech sometimes illustrate in wonderfully simple fashion the intimate relationship between the psyche and breathing. I "breathe a sigh of relief" when I escape from an emotionally constricting situation. And the experiencing of a new, unknown dimension which one willingly accepts, although perhaps overexciting and alarming at the same time, can "leave you breathless."

The first breath we draw marks the beginning of our individual earthly lives -- the great separation from the symbiotic unity with the mother. This first great loss, the first pain, followed by a powerful exclamation into the atmosphere. And when we "breathe our last," we "give up the ghost."

Indeed: breath is life itself.

This applies on all levels of being. But let us now turn from the more spiritual aspects of breath to the more material ones, the vital processes involved with the biological vessel which we steer through the cosmic field of breath, and to which we are bound again and again by the rhythm of breathing.
Man, as a little world among cosmic life, is -- thermodynamically considered -- an open system. For maintenance of this microcosm, man is therefore critically dependent on an uninterrupted supply of nutrients -- and, above all, on a continuous provision of oxygen.

The transport system for this supply of oxygen is, of course, the respiratory system. Through functions of the diffusion mechanisms of the lung-blood barrier, it correlates intimately with the cardiovascular system. Critical functions of human life take place here in their most apparent form.

After all, each of us inhales about 9,000 liters of oxygen every day via the alveolar surface area of 80 to 120 square meters. During a life span of 70 years, this amounts to between 200 and 300 million liters. And: the respiratory passages represent the organic substrate through which the processes of biological oxidation and all metabolic oxidation processes are made possible. It is therefore only logical that this system of vital organs -- consisting of mouth, nasal passages, pharyngeal cavity, trachea, bronchial system, and alveolar space -- is therefore equipped with special protection and immune mechanisms.

Let us now attempt to gain a greater understanding of the great
variegation of these means of defense employed by the human organism, by considering the example of the bronchial system. Defense functions here basically involve the three following regulation mechanisms:

1. The alveolar macrophages perform one of the most important defense functions. These cells are also called dust cells, in allusion to the fact that they are practically omnivorous. Their parent cells are the monocytes.

In cases of infections, the number of these phagocytes can increase to five-fold their normal quantity.

Alveolar macrophages perform non-specific defense functions in the form of general resistance to agents -- either directly, or via the intermediary functions of antibodies or complements -- in these latter two cases, by means of the phenomenon of the respiratory burst. In this connection, the macrophages destroy cellular antigens with the aid of activated oxygen stages. Such antigens include, for example, pathogenic agents as well as malignantly transformed cells.

Macrophages also perform specific immunological functions, however, in that they initiate immune responses for T and B cells.
Macrophages also perform functions associated with the healing of wounds, following an inflammation which has run its course in the alveolar wall.

Once, however, the macrophages go too far in conjunction with these intracellular explosions -- once, so to speak, they have overeaten on undigestible food -- they themselves can become a disturbance factor which promotes chronic inflammation.

Foreign matter, furthermore, which enters the pulmonary alveoli is transported away via lymph passages, is decomposed, and is then eliminated -- or it can remain in the Pleura visceralis as visible gray or black spots. In bronchial areas, moreover, lymphoid tissue can be found in the form of lymph follicles, similar to Peyer's patches in the walls of the small intestines.

As a result, all known immunological regulatory processes are found throughout the entire respiratory tract of the human
being: unspecific as well as specific, humoral in addition to cellular.

2. The second important defense process which I would like to mention is implemented in the surfactant system, and involves the anti-atelectasis factor.

From the standpoint of the material substances concerned here, the surfactant is a glycoproteolipid which coats the entire surface area of the alveoli in the form of a thin film. By virtue of its great surface tension, the surfactant not only prevents collapse in the non-ciliary areas of the alveoli and small bronchi, but also inhibits overextension of the alveoli. Certain transport functions are also performed by the surfactant.

In the ciliary areas of the respiratory passages -- i.e., in the area of the larger bronchi -- the surfactant ensures the effective transport of secretion by virtue of furnishing the optimal degree of secretion adhesion.

The surfactant, furthermore, performs essential immunological
functions: it activates the process of phagocytosis and enhances the intracellular destruction of microorganisms by providing an enclosing action.

The surfactant is formed by type II pneumocytes in the alveoli. The surfactant is not only highly essential for the normal respiratory functions of the adult -- it is also critical for the proper development of the lungs while the newborn infant is taking its very first breaths.

3. The third and last regulation mechanism which I would like to treat in conjunction with respiratory defense functions involves bronchial clearance.

The cleansing of the bronchial systems from foreign particles introduced by the environment takes place through secretion and through the ciliary functions of the mucous-membrane cells.

Cilium-equipped columnar epithelial cells, goblet cells, and brush-border cells can all be found in the respiratory mucosa. Most of the cleansing bronchial secretion, however, is provided by the bronchial glands located in the submucosa.
The number of cells here which are provided with cilia is four times greater than the other epithelial cells. The number of cilia decreases in the direction of the alveoli, as a result of which the extent of mucus transport in the periphery of the bronchial system is correspondingly small.

Now, after we have obtained this brief overview of the highly variegated defense system, especially as we find it in the lower respiratory passages, let us now consider the consequences of defense weaknesses in this area. As an aid toward understanding, let us also consider the developments of the morphology of the bronchial system, in conjunction with various stages of respiratory disease. The objective will be, finally, to answer the question as to the possible effective therapy to be employed at these various stages of damage to the respiratory system.

As stated at the beginning of my presentation, respiration is subject to fundamental two-phase interrelationships inherent in our natural makeup -- as illustrated most simply and consciously by the obvious impossibility of being able to breathe in without wanting to breathe out.

In a like manner, human defense processes are also subject to a two-phase law, as we physicians have learned from Hoff's concept of the mobilization of the autonomic nervous system. The studies pro-
vided by Ricker and Speranski on the biorhythm of inflammation at the substrate of the connective tissue also provides morphological confirmation of the double-pole concept of defense functions.

In addition, the normal biorhythmic phenomena involved here represent at the same time the very expression of intact immune response, and are controlled through the restraints provided by two synergistically acting antagonists: the sympathetic and parasympathetic nervous systems.

Now let us take a closer look at this dual play of power, by considering Selye's alarm reaction curve, as modified by Perger.

We see here the so-called shock phase, in the form of response to vaccination by agents, as it is measured by the decrease of eosinophils and calcium in serum. The shock phase occurs under the direction of the sympathetic nervous system, and the following countershock phase takes place under the guidance of the parasympathetic nervous system.

Through examining the basic regulation processes of patients with chronic illnesses, researchers have determined characteristic deviations in the course of these biorhythmic alarm reactions. Such
chronic diseases as covered in this study are initially characterized by loss of biorhythmic quality: i.e., chronological demarcation is lacking. In addition, the double-phase characteristic of the alarm reaction is not observed.

As test parameters for patients which such chronic illnesses clearly confirm, the organism can pathologically react in either of the following three ways:

1. The organism remains arrested in the shock phase, with the result that an acute inflammation cannot develop back to the phase of mesenchyme synthesis.

2. Or, as a second possibility of pathological reaction from the standpoint of the ground substance, the organism can remain arrested in the countershock phase. This mode of abnormal reaction represents the cause of all chronic proliferative inflammatory processes.

3. A third mode of pathological deviation in the basic regulation curve is demonstrated in the form of a complete lack of reaction. We encounter this type of reaction among the autoimmunity disorders: for example, with primary chronic polyarthritis (PCP) and with all destructive neoplastic processes.
All of these pathological alterations in the mode of reaction of the organism are accompanied at the same time by a reduction in the stimulus threshold for total reactions.

For a healthy person, an agent count of 500,000 of a vaccine is necessary before a total reaction can at all take place, as Perger -- a colleague of Pischinger's -- has most impressively documented. If the reaction form demonstrates arrest in the shock phase, however, the threshold is significantly reduced: to a level of 20,000 to 30,000. With arrest in the countershock phase, reduction is even lower -- down to around 10,000. With complete lack of reaction -- as we almost always encounter for PCP, for example -- the stimulus threshold is below 5,000 agents. For a chronically ill patient, in other words, the organism cannot properly regulate itself locally in response to low-level stimuli, as is naturally expected among the healthy. The chronically ill therefore require correspondingly earlier mobilization of the defense powers of the entire organism -- a process, however, which is uneconomical and which leads further to progressive weakening of the defense capabilities, to the point at which they are fully immobile.

Now, if we consider the morphological alterations to the bronchial
mucosa of the human being, we can follow the consecutive characteristic stages which may begin with acute bronchitis and lead through exudative, chronic proliferative, and metaplastic phases. In a manner analogous to the progressively weakening defense system, we can observe the progressive damage to the organism, up to the destruction phase, and we can thereby use the particular stage of the disease observed to inversely infer as to the degree of the reduction in defense capabilities which has led to the apparent damage.

I would like to emphasize here that it is precisely such conclusions which provide the critical criteria for the proper choice of therapy. And this is, of course, the purpose for conducting such experiments and making such considerations -- efforts which otherwise would be of merely academic interest and would represent only an interesting way for physicians and researchers to spend their time.

Now, let us attempt to employ clinical symptom pictures and the associated morphological alterations, for the disorders being considered here, and by way of their example -- namely, respiratory illnesses -- to illustrate the individual stages of the corresponding defense weakness. The purpose here will be to derive the logical justification for optimal antihomotoxic therapy of the various phases of disease.
To begin, we see here the undamaged lung of a young man and, further, the intact, cilia-equipped bronchial mucosa characteristic of a healthy person.

In the case of acute, catarrhal bronchitis, we find all the signs of exudative inflammation, with increased mucus production, greater bronchial clearance as a result of coughing, augmented surfactant activity, and intensified macrophage action.

After elimination of the initiating noxae, we observe complete re-synthesis of the bronchial system.

The Selye would then demonstrate a normal progress. In Reckeweg's six-phase Table of Homotoxicosis, we can categorize the illness under the second heading: the reaction phases.
If the noxa is too strong, or if it attacks the organism on a repeated basis, or if it inflict damage on the organism while it is in a state of reduced defense capability, then acute, catarrhal bronchitis will develop into the chronic form -- a situation which continually relapses without achieving a decisive outcome. In other words, the exsudative inflammation processes never develop to the point of being able to eliminate the responsible noxae.

Two typical characteristics for chronic exsudative or catarrhal bronchitis are excessive secretion and dyscrinism. These are exhibited on the surface epithelium and at the peribronchial glands.

From the histomorphological standpoint, lymphocytic and plasmacellular infiltrates appear only slowly, in addition to the exsudative signs of inflammation. The stimulus response has deviated from its normal biorhythmic progress, and the organism remains in a weakened posture for attack -- i.e., it remains arrested in the shock phase, and resynthesis of the inflamed mucosa cannot take place.

This situation comes about because the initiating noxae were not
able to be completely eliminated by exsudation, and because the inflammation stimulus remains in effect.

This form of chronic exsudative inflammation is characterized by intensive macrophage activity, which extends even to the point of self-destruction, and to the point of increased activity of fibroblasts. Cellular defense processes are therefore involved here.

To use the terminology of Hans Heinrich Reckeweg, the illness is now in the deposition phase. Selye would say that it is arrested in the shock phase.

Critical defense functions can be further blocked by any of the following developments:

1. The defense system cannot sufficiently recover.
2. The homotoxic agent cannot be removed from the system.
3. Additional damages are inflicted -- for example, by inhalation of toxic material. One of the most widespread examples here is the action of tobacco smoke, which places a great burden on macrophage activity.
In addition, the surfactant system is additionally damaged. As a result of these influences, chronic exudative bronchitis can progressively develop into the chronic proliferative form.

Chronic proliferative bronchitis -- i.e., bronchitis marked by fibrous degeneration -- demonstrates a process of reconstruction in the subepithelial zone of connective tissue, in addition to various degrees of excessive secretion and dyscrinism. These processes are accompanied by progressive fibrosis. The proliferative processes of the local connective-tissue cells -- as expression of the protracted clinical progress of the disease -- characterize the histomorphologic picture. Although the fibrous degeneration involved here also affects to a particularly extensive degree the capillary bed of the bronchial mucosa, it also significantly inflicts the nerve structures, the glands, and the fibers of the smooth muscles.

As with all processes of fibrous degeneration, this proliferation of the connective-tissue fibers leads to shrinkage of and to progressive reduction in the patency of the bronchi, and to loss of elasticity.
This protracted process aggravates a chronic obstruction disease and therefore also contributes to the degree of resistance increase.

In the sense of Selye's alarm curve, this clinical form of development and its morphological substrate correspond to a disorder in the defense system which has become arrested in the countershock phase. Resynthesis of the tissue does not take place. The organism's functions of cleansing and repair go out of control.

Using the nomenclature of Reckeweg, the illness is in Phase 4, which involves the deposition and impregnation of homotoxins in connective tissue. Upon progress into this phase, however, we have crossed the Rubicon represented by Reckeweg's Biological Section. This means that the disease has reached a point at which the organism is no longer capable of spontaneous self-healing.

For this reason, such cases of chronic bronchitis with fibrous
degeneration will unavoidably and as a matter of natural course progress toward bronchitis with mucosa alteration if decisive therapeutic action is not taken.

The chronological development here will depend on the individual patient, as well as on the following factors:

1. Genetic bases as represented in the quality of the respiratory system
2. The degree of damage to the organism's defense system
3. The personal lifestyle of the patient, with its greater or lesser degree of self-discipline
4. The quality of the air which we breathe.

In dwelling slightly longer on the last factor, I would like to point out that we are more severely restricted in exerting our personal influence on the menu of toxins which we inhale from our biosphere. No one forces us to consume pork chops or strawberry shortcake, but it is not so simple to refuse the cocktail of daily urban smog. After all -- as I have touched on before -- we cannot simply stop breathing.

In addition to protracted fibrosis, this form of bronchitis is also
associated with a progressively increasing degree of alteration in the bronchial mucosa. This process begins in the central bronchial sections and develops farther on into the periphery.

These alterations progress over extensive areas of the bronchial system, and lead to the interruption of secretion. This results in dangerous impairment of bronchial clearance.

The alteration of mucosae here is the consequence of their faulty repair. This was the process which was originally influenced by inflammation -- at a stage at which resynthesis could not take place while the organism was arrested in the countershock phase. The consequence is that pathological alteration of the epithelium occurs, accompanied by a reduction in the complement of cilia.

So-called compound cilia develop: these are ungainly ciliary complexes which are hardly capable of function. In conjunction here-with, more and more surface areas, of various size, begin to develop without cilia. The cell cytoplasm of the epithelial cells begins to demonstrate degenerative alterations. Irregular formation of secretion, without discharge, takes place in individual epithelial cells.
The intercellular capillary gaps open, and in this manner there follows the progressive formation of squamous-epithelium-type metaplastic alteration of the bronchial epithelium.

At this point the development of a clinically detectable bronchial carcinoma is not longer a mere possibility: it is, rather, only a question of time.

But what about the organism's defense system?

At this point, it finds itself in a situation of arrest, more or less unable to respond.

In the six-phase Table of Homotoxicosis, we have fatefully passed -- via the degeneration phase -- into the phase of destruction.

Now, if we observe this development while the organism's defense system -- with its functional and morphological substrates -- gradually exhausts itself, we should rightfully pose the following, almost desperate question: Is there anything we can do?
At medical school, we learned to do the following:

In case of catarrhal bronchitis, physicians traditionally administer antipyretic and anti-inflammatory agents, in addition to secretolytics and antitussives. If bacterial or bacterio-viral mixed infections are suspected, we were taught to administer antibiotics. Such therapy is, of course, extremely effective in alleviating the patient's troublesome complex of symptoms.

But what effect does such therapy have on the organism's defense system?

Antipyretic and anti-inflammatory agents, as well as antibiotics for the most part, inhibit the first phase of defense reaction -- the shock phase. In effect, they suppress and prevent the process of inflammatory exsudation which serves to eliminate toxins. As a result, the organism cannot proceed toward the antishock phase and the final process of resynthesis. These classical measures therefore represent a form of self-deception, since the causative agents have not been eliminated. And the result: the organism must exert itself additionally to initiate a new inflammation in an attempt to destroy the toxins by pyretic action. Defense capabilities, however, have been weakened: less, I must emphasize, by the action of homotoxins, than by our well-meaning therapy. After all, in case of doubt -- "What, is he still coughing?" -- classical therapy will once again go on the offensive -- this time, on a broader field.
As a result of defense weakness, the next episode of inflammation is definitely attenuated. Chronic exudative bronchitis consequently develops in this manner -- a process which often enough leads to bronchitis marked by fibrous degeneration. The way is now paved toward chronic obstructive bronchitis, or even to the final state of metaplasia.

In conjunction with the description of traditional modern therapy of bronchitis, I used the phrase, "... to go on the offensive." I did this intentionally. But -- dear colleagues -- we are truly lost if we try to fight against natural forces. If we are really interested in healing, and not in mere suppression of symptoms, we must come around to a consideration of another viewpoint.

Indeed: we should try to work along with natural forces, to elicit their help, to support them. After all, the phenomena of illness are natural and fitting regulatory processes which serve for the benefit and the maintenance of life. In this sense, I hope that you will be able to more readily appreciate my following remarks on possible biological therapy -- i.e., measures of treatment conceived in accordance with natural processes. In line with the example covered in my presentation -- bronchitis -- I shall treat the various stages of this disease, up to the state of metaplasia, and shall propose the various forms of biological therapy in accordance with the degree of defense weakening encountered in each of these pathological stages.
Let us begin with common catarrhal bronchitis, which is generally observed in association with polysinusitis. I have had highly effective results with what I call my "flu cocktail." I administer it once my patient's symptom complex here is severe enough -- and it usually is, otherwise he or she would not come to my office.

My flu cocktail consists of the following:

1. Gripp Heel: For support of the organism's defense against colds and flu

2. Traumeel: For control of inflammation

3. Lymphomyosot: For activation of the mesenchyme, and promotion of elimination of toxins

4. Engystol: For enhancement of defense functions, especially for virus infections, as well as for support during the shock phase.

I administer my cocktail intravenously. Or, application is by intramuscular means, as part of progressive auto-sanguis therapy, a technique using iso therapy with the patient's own blood. I administer it three to five times.
And the results? -- the patient gets over his or her bronchitis without conking out. After all, defense resistance is normal at such a stage, and is further strengthened by the therapy outlined here.

For supplementary, symptomatic treatment, I have achieved good results by starting with the medication Aconit Homaccord, followed by Belladonna Homaccord. Then I administer cough medication such as Tartepphedreel, or Drosera, according to the nature of the cough.

I modify my therapy in case of the all too-familiar chronic exsudative bronchitis, especially when children are involved. This modification is necessary because we are confronted here with defense systems which have already been impaired.

You may recall that the organism is arrested in the shock phase in such cases. The therapy administered here must therefore stimulate the inhibited regulation processes of this phase. As a result, my cocktail has a modified and extended composition:

1. Echinacea
   Compositum: For stimulation of defense resistance, and for support during the shock phase
2. Engystol: For revival of latent inflammation by stimulation of the shock phase

3. Lymphomyosot: For elimination of toxins via the mesenchyme

4. Traumeel: For control of inflammation

5. Grip-Heel: For enhancement of resistance with inflammations involving cold, flu, and catarrh.

Since exsudative diathesis is involved here, the physician can make effective use of the heredonosode Tuberkulinum Injeel, as well as the constitutional nosode Calcium Carbonicum Injeel, with one to three applications of each.

In any case, the injections should be administered with a frequency of two to three times weekly, in the form of progressive auto-sanguis therapy, a special method using iso therapy with the patient's own blood. I have found it best to administer Engystol intravenously, in order -- so to speak -- to allow latent focal disorders to flicker up again.

Orally administered adjuvant therapy in the form of complex homeopathic medication ensures that these revived foci do not flame out of control.
In passing, I would like to point out the great importance of proper, whole-food nutrition in cases of chronic exudative bronchitis among children. Abstinence from milk and sugar can be especially critical here. Since the administration of antibiotics and the effects of poor nutrition with such bronchial disorders usually bring about degeneration of the intestinal flora, and disturb the natural processes of detoxification via the intestinal tract, symbiosis assistance is often necessary. These additional measures are especially critical in view of the fact that children in such situations are in particular jeopardy of developing asthma.

In order to logically grasp the sense of these therapeutic efforts in the context of Reckeweg's six-phase Table of Homotoxicosis, one can view these measures as the attempt to assist the organism in leaving the deposition phase and in progressing toward self-healing via activation of the reaction phase.

In cases of chronic proliferative bronchitis, however, the way to self-healing is of course correspondingly longer and more difficult -- since we have to summon the strength to re-cross the Rubicon of the Biological Section, in the middle of our table. In this strenuous effort, the organism alone cannot summon the strength to enable resynthesis of the afflicted tissue after the inflammation which has taken place, because the responsible toxins were not completely eliminated. The organism is arrested in the repair -- or
countershock -- phase. As a result, we must further broaden our program of therapy, as follows:

1. Echinacea Compositum
2. Lymphomyosot
3. Traumeel
4. Engystol
5. Mucosa Compositum: For enhancement of defense functions, specifically with respect to the mucosae
6. Bronchus Suis Injeel: For organ-specific enhancement of defense functions
7. Grippe Nosode Injeel: For eliciting specific defense functions, by administration of the originally initiating toxins in highly potentized form.

The most effective heredonosode here would be Medorrhinum Injeel. The injections with this preparation should be administered two to three times per week by means of the auto-sanguis therapy referred to earlier.

Equally critical prerequisites for the success of this therapy are the avoidance of toxin intake to as great a degree as possible, as well as extensive reduction of inhaled pollutants. If the physi
cian is successful in triggering a good, heavy case of catarrhal bronchitis in his patient, then all involved have reason to rejoice: this shows that the hoped-for jump back over the Rubicon of the Biological Section has indeed taken place.

If metaplastic alteration has in fact taken place, therapy is naturally more difficult. In such events, we must additionally apply the entire spectrum of biological cancer treatment -- therapeutic possibilities which I of course will not be able to treat in this presentation.

I can, however, sketch out the antihomotoxic therapy of bronchitis in this degeneration, or destruction, phase. It includes the following preparations:

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1. Echinacea Compositum
2. Lymphomyosot
3. Coenzyme Compositum: For stimulation of blocked enzyme systems
4. Ubichinon Compositum: For stimulation of cellular toxicity defense mechanisms
5. c-AMP Injeel: For stimulation of cell respiration
6. Mucosa Compositum
The program of therapy here can also effectively include the hered-
onosode Luesinum Injeel.

In addition, of course, patients whose afflictions have reached
the neoplastic stage should maintain a diet of whole-food nutrition
as well as a style of life in accordance with their serious dis-
ease.

In conclusion, I would like to call attention to the most critical-
ly urgent therapy of them all -- the one capable of treating de-
fense weakness in the respiratory system, and its consequences, in
the most direct and most sensible manner: the restoration of the
atmosphere of our planet Earth.

The following graffiti was observed in San Francisco in conjunction
with a particularly bad siege of smog:

"I shot an arrow into the air,
    And it stuck up there."

It stuck in a conglomerate of sulfur dioxide, nitric oxide, carbon
monoxide and an excess of carbon dioxide, the entire spectrum of
heavy metals -- especially lead, and other metals in the form of
dust -- as well as aromatic aerosols, radioactive waste, and colon-
ies of airborne particulates. But you know all this better than I.
All of these materials are inhaled toxins. And, as though these were not enough -- tobacco consumption is maintained at a dangerously high level.

This entire spectrum of heterogeneous toxins, once inhaled into the organism, leads in the bronchial system to relatively uniform reactions: inflammation, allergic and autoimmune mechanisms, and chronic inflammation -- with further development toward dysplasia, metaplasia, and finally carcinoma.

A living being -- be it a plant, an animal, or a human being -- can maintain an optimum level of defense capabilities only if it is constantly subjected to the action of those elements which have introduced and accompanied its history of development -- an environment to which its entire organ function has naturally adapted.

Did you know that you can read about smog and radioactive waste in the book of Genesis?

As a result of the toxins in the atmosphere which we breathe, ou.
environment has become a time bomb for us all. And since we alone were the ones who set this time bomb, it is up to us alone to defuse it. Once we succeed, we can finally breathe a sigh of relief once again.

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Our souls will breathe sighs of relief, too, since our breath is our very life.

Thank you very much.