From a wide pathogenetic point of view, leprosy involves three ecological factors: The leprosy patient, the environment and the Hansen's bacillus which, by reasons that will be explained, must be included in this paper. The epidemiologists call it the "epidemiological triad" or the "host-parasite" interaction.

The infectious pathology considers an ecological triangle formed by the patient, the environment and the causal germ. This triangle does not exist in leprosy since the Hansen's bacillus is not the etiologic element that causes the disease as, for example, the tuberculosis bacillus or the syphilis spirochete. The ecology of this disease is altered since, by a crass conceptual error, the bacillus is constituted as the fundamental centre of the anti-leprosy therapeutic and the treatment led only to the bacillus. This is what obliged to integrate this supossed ecological triangle with the Hansen's bacillus.

I) The leprosy patient

The leprosy is a metabolic disease with a strong genetic component to which, during the patient's life, pathogenic factors of nutritive and metabolic origin are added, becoming from the environment and producing a metabolic disorder consisting in the loss of control of the autooxidatives reactions, named "auto-oxidative disease" (Bergel). This is, as its name indicates, from a metabolic origin and without any infectious componene. During leprosy evolution, complications of different etiology may occur. Specially, the infectious one, which, when appears is apparently the most important sign of leprosy and therefore, puts the Hansen's bacillus, agent of this complication, into the wrong causal place of this disease. The leprosy, without taking into account this bacterial infectious complication, seems to be very simmilar to a collagenopathy, an autoimmune disease or a rheumatic like process. Consequently, it shows many common points with lupus erythematous, panniculitis, arthritis, etc. All the wide symptomatology presented by the leprosy patient with added infection of Hansen's bacillus is of complication type. At this stage, this infection covers and dissimulate the primitive disease.

It is important to bring out these facts:

1) The study of the Weber-Christian panniculitis revealed that this disease, with unknown etiology, presents common points with leprosy such as erythemas, nodes, fever, arthralgias, visceral and humoral alterations, etc.

2) Ibrahim et al. showed in mice that, through a metabolic disorder produced by a diet with a determined quantity of fats, a syndrome very similar to systemic lupus erythematosus can be produced.

3) The "leprous reaction" presenting protein alterations in plasma, arthralgias, painful
nodes, necrotic vasculitis, etc. out of the bacillar component, seems very similar to many collagenopathies.

4) It could be roughly said in order to summarize this concept that leprosy is a "Systemic lupus erythematosus infected with an acid-fast bacillus".

II. The environment

Metabolic disorders caused by the environment must be added to the genetic component to develop leprosy. This metabolic disorder is specially produced when intake of unsaturated fatty acids in auto-oxidative state is increased together with precarious intake of vitamin E. That is to say, when the patient's diet produces a pro-oxidative effect as happens when a diet contains an excess of rancid fats, peroxidated fats, with a low concentration of vitamin E.

The intake of this diet produces a peroxidation state in organic tissues with a flow of lipidic peroxides, which toxicity is well known. These lipidic peroxides play an important role in the appearance of the third conventional component of this ecological triangle: the Hansen's bacillus. This reactualizes the old Hutchinson's ichtiophagic theory; and also the relations between diet and etiopathogenesis of leprosy, established in great regions of the world, such as India, Central Africa, Brazil, Mexico, etc.

III. Hansen's bacillus

When quoting the Hansen's bacillus as the third component of this triangle, we must reiterate that this is not a true ecological triangle of infectious pathology. The Hansen's bacillus appears later to the establishment of leprosy, what does not happen with the bacillus of tuberculosis, which plays an etiological and causal role in the genesis of the disease.

Once established leprosy, when the patient is suffering a flood of peroxides flowing permanently and are spread out through its organs, the appearance of the Hansen's bacillus takes place.

We are going to describe the place of the Hansen's bacillus in relation with leprosy and the cause of its location in these patients.

The Hansen's bacillus is ecologically associated with the leprosy patient in a symbiotic relation known as "commensalism".

Both, the sick organ and the bacillus are associated with mutual benefits, although this damages the host. The situation is as follows: the organism produces lipidic peroxides which causes a permanent flow of these fatty metabolites that the Hansen's bacillus consumes in an obligatory and permanent way.

From this commensalism relation arises two highly important conclusions:

1) The Hansen's bacillus concurs the leprosy patient because it needs the metabolites—fatty peroxides—provided by the organism to live.

2) The organism shelters and does not destroy the bacillus since this is really useful to consume the peroxides that are produced in the organism which, otherwise, would be free causing toxic and inflammatory symptoms.

In other words, we are observing a biological association from which there are many examples in human biology, that is the case of lactobacilli and bacteria producers of vitamin K and
B complex, which play an important role in regulating the microbial flora of the intestine and genital tract. Sometimes, acting as "pathogenic facultatives" can produce "circumstantial infections".

Once having cleared up the role of Hansen's bacillus in relation with pathogenesis of leprosy, we will make the therapeutical considerations:

1) In order to act therapeutically in leprosy, it is necessary to operate over the two pathogenic and primary elements of the disease, that is to say, over the leprous organism and the environment.

It is necessary to avoid the production of fatty peroxides in the organism and destroy or neutralize them through the primary or secondary anti-oxidants and with inactivators of metallic pro-oxidants, such as chelaets or sequestrators. All these components in their association, integrate what is denominated an "anti-oxidant system". With reference to the environment of the patient, it is necessary to act through a balanced diet which does not include excess of unsaturated fatty acids in rancid auto-oxidative condition, with adequate contents of natural anti-oxidants as tocopherols or vitamin E. Acting this way, the organism stop producing fatty peroxides progressively without damaging, through its toxic-inflammatory activity, the tissues any more. Therefore, the disease is resolved favorably.

Now, we will analyze the consequence of acting only on the Hansen's bacilli. If only the Hansen's bacilli are destroyed, undesirable situations for the organism could be produced.

The Hansen's bacillus by effect of bactericidal drugs such as rifampicin will loose its viability in the human organism but this organism would go on producing fatty peroxides that was metabolizing, absorbing or neutralizing the alive Hansen's bacillus. Therefore, in absence of the viable bacillus, these fatty peroxides—without being metabolized, absorbed or neutralized—will continue circulating and originating toxic-inflammatory symptoms. It is the probable cause of the "leprous reaction" because it attracts attention that this acute toxic inflammatory episode is only present in leprosy patients with very few bacillar charge and on the other hand, it is almost absent when the patient is covered by bacilli, what produces an ecological equili-brium between the patient and the sheltered bacilli.

This allows us to arrive to the following conclusions:

1) When treating leprosy, it must be used drugs acting on the auto-oxidative disease through mechanisms acting on the formation, metabolization and neutralization of fatty peroxides such as primary and secondary antioxidants, inactivators of metallic pro-oxidants, etc.

2) In the treatment of leprosy, it must not be used bactericidal drugs with direct activity on Hansen's bacillus.

The drug which shouldn't be used is rifampicin because it is highly bactericidal over the Hansen's bacillus and acts over the leprosy patient breaking the ecological balance, perhaps beneficial, established between the sick organism and the Hansen's bacillus. This one, when getting the neutralization of fatty metabolites produced by the sick organism, avoids the appearance of the already mentioned toxic-inflammatory syndrome. The above is stated by the fact that the clinical experience with the use of rifampicin in the treatment of leprosy makes us
conclude as follows:

1) Rifampicin alone must not be prescribed in the treatment of leprosy in spite of its extraordinary bactericidal activity on Hansen’s bacillus.

2) Multiple experiences reported that the association rifampicin-sulphones is not superior at all to sulphones alone in long-term.

3) The use of rifampicin for the last 15–20 years in the treatment of leprosy was the element that has broken the ecological balance which had the patients during the first 25 years of treatment with sulphones alone and induced to present recrudescence of the sulphone-resistance in leprosy. The rifampicin, by breaking the mentioned ecological balance, makes that a commensal germ as the Hansen’s bacillus, is transformed into a pathogenic facultative.

In conclusion, treatment of leprosy must be only aimed to the auto-oxidative metabolic disease through these drugs which act inhibiting the production of fatty peroxides. With reference to the Hansen’s bacillus, can be or not attacked with bactericidal drugs, although we believe it is better not to do that.

It is important to note that many therapeutical errors in leprosy are due to the fact that this disease was considered similar to tuberculosis and therefore, leprosy and tuberculosis bacilli might play protagonic roles in the genesis of the mentioned diseases.

In tuberculosis, Koch’s bacillus attacks the human organism and is the etiologic agent that causes directly tuberculosis. But, in leprosy, the Hansen’s bacillus is a commensal germ that is established in a disease already developed. So, the protagonic roles of both bacilli are completely opposite and this is what must be taken into account to attack both bacilli.

The Koch’s bacillus must be attacked in tuberculosis because it is its causal agent but, in leprosy, the target of attack should be the metabolic disease, without taking into account the bacillus that is not its causal agent but is just added as a commensal agent to the leprosy patient perhaps, in a mutually beneficial relation.