The Compromised, Pre-Diseased or Post-Diseased Terrain, Malaria and Germ Terrain Dualism

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Introduction

The Germ-Terrain duality theory of disease states that the aetiology of certain diseases/diseased states is better explained as a complex interplay between germs and the inherent anatomical/physiological integrity of the body cells [1]. It argues that the aetiology of certain diseases is not fully explained merely by the presence of germs (Germ Theory) or by a mere loss of cellular integrity (Terrain Theory) [2-6]. As a result, the prevention and treatment of such diseases should focus not just on fighting germs but on maintaining/restoring the anatomical/physiological cellular integrity [7-11]. The Germ-Terrain duality theory is a harmonization of the current Germ Theory (popularized by Louis Pasteur) and the hitherto discarded Terrain Theory (popularized by Pierre Bechamp) [11-14].

If an unhealthy/pre-diseased person is infected with malaria, what happens? What is the effect of malaria in an individual whose anatomical/physiological terrain (integrity wise) has been compromised prior to (or after) infection? (Table 1).

Table 1: The above show terrain has a role to play in the aetiology of malaria.

<table>
<thead>
<tr>
<th>Conditions that provide resistance to malaria</th>
<th>Conditions that provide no resistance to malaria, encourage malaria to thrive and/or are themselves adversely affected by malaria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Group O [3-6]</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>AIDS</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Type 2 Diabetes (Disputed) [7,8]</td>
<td>Vitamin A deficiency</td>
</tr>
<tr>
<td>Pyruvate kinase deficiency</td>
<td>Excess Iron</td>
</tr>
<tr>
<td>Duffy antigen receptor negativity</td>
<td>Vitamin B1 (Thiamine) deficiency [11]</td>
</tr>
<tr>
<td>Gerbich antigen receptor negativity</td>
<td>Zinc deficiency [11]</td>
</tr>
<tr>
<td>Human leucocyte antigen polymorphisms</td>
<td>Folate deficiency [11]</td>
</tr>
<tr>
<td>Cancer [10]</td>
<td></td>
</tr>
<tr>
<td>Glycophorin A and B protein mutations</td>
<td></td>
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</tbody>
</table>

References

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