Commentary

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Thalassemias Validate Germ Terrain Duality of Malaria

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Introduction

The Germ-Terrain duality theory of disease states that the etiology of certain diseases/diseased states is better explained as a complex interplay between germs and the inherent anatomical/physiological integrity of the body cells [1,2].

It argues that the etiology 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) [1,2].

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.

The Germ-Terrain duality theory is a harmonization of the current Germ Theory (popularized by Loius Pasteur) and the hitherto discarded Terrain Theory (popularized by Pierre Bechamp) [1,2].

Thalassemias [3-5] reduce the size and/or change the shape of red blood cells thus hindering and limiting the activity of plasmodium [6-10] **(Table 1)**.

There is a need for competent scientists to create an index of the relative resistances of abnormal haemoglobins (obviously with HbS as the standard) to malaria so that we may come to a fuller understanding of the germ terrain duality of malaria-and hopefully of other diseases as well [11-15].

Table 1 Anatomical or physiology variation of different samples.

Abnormal haemoglobin	Anatomical/Physiological Variation	Effect vis-à-vis resistance to malaria
S	Sickle shaped cells; base substitution of glutamic acid with valine in beta chain	Provides resistance to malaria
C	Forms crystals; base substitution of glutamic acid with lysine in beta chain	Provides resistance to malaria
D	Mutation on codon 121	Provides resistance to malaria
E	Point mutation at position 26 of glutamic acid to lysine	Provides resistance to malaria
Lepore	Crossover between delta and beta chains	Provides resistance to malaria
F	2 alpha, 2 gamma structure	Provides resistance to malaria
Persistent F	As above	Provides resistance to malaria
J	Alpha globin variation	Provides resistance to malaria
O	Mutation at codon 121	Provides resistance to malaria
G	Alpha chain mutation	Provides resistance to malaria
Hasharon	Alpha and beta chain mutation	Provides resistance to malaria
M	Beta globin gene codon 92	Provides resistance to malaria
Hope	Alpha and beta chain mutation	Provides resistance to malaria
Pisa	Protein related	Provides resistance to malaria
N-Baltimore	Protein related	Provides resistance to malaria
I	Protein related	Provides resistance to malaria
Hopkins - 2	Histidine replaced with aspartic acid	Provides resistance to malaria
Bart	Alpha globin gene dysfunction	Provides resistance to malaria

Embryonic Gower 1	2 zeta chains and 2 epsilon chains	Dearth of good, widely available studies on this subject
Embryonic Gower 2	2 alpha chains, 2 epsilon chains	Dearth of good, widely available studies on this subject
Embryonic Portland 1	2 zeta, 2 gamma chains	Dearth of good, widely available studies on this subject
Embryonic Portland 2	2 zeta, 2 zeta chains	Dearth of good, widely available studies on this subject

Normal haemoglobin (Hb A) provides no resistance to malaria [16]. This proves the germ terrain duality nature of malaria.

I repeat, in the light of the above, it is suggested that since there are several other (hundreds) [17-21] abnormal haemoglobins it will be appreciated if competent professionals study their effects or lack thereof vis-à-vis resistance to malaria so that we can come to a greater understanding of the germ-terrain duality nature of this malady.

Already work has been done on utilizing foetal haemoglobin to better understand and treat sickle cell anaemia.

If more work is done with the other abnormal haemoglobins it is not impossible that very effective therapies against sickle cell diseases could be developed which could make the disease a thing of the past.

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