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Inflammation:

An all-season phenomenon in human pathology

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1 The interest of the scientific community on inflammation has been growing steadily, recently
2 favoring the comprehensive perception of inflammation as a sort of nature's generalized way to cope
3 with the environment.¹ With a group of scientists, we have begun a translational analysis of the
4 evolutionarily conserved process of inflammation. As a result of this work we have elaborated a "Turin
5 concept" of inflammation as an evolutionary and pathologic drift. We have become interested in
6 inflammation because of two main aspects. We first made the point that the clinical presentations
7 (originally comprising the so-called cardinal signs of redness, swelling, and pain) by the end of the 2nd
8 World War have left, as the legacy, a creeping form² made of mildly relapsing fever, weakness, and
9 elevated C-reactive protein, somewhat matching the contemporary overgrowth of "autoimmune"
10 affections of a rheumatologic interest.³ Today, Western Societies are being plagued with such disorders,
11 making important socio-economic issues. On this line, the appreciation of acute/chronic inflammation
12 as an evolutionary carver of life has become of age.⁴ Secondly, inflammatory markers have now been
13 found in unsuspected areas including neuropsychiatry.⁵ In this case, patient's loss of the acquaintances
14 with the outer world is paralleled by inflammation marking the malaise at the organic level. Notably,
15 inflammation is accredited with both archaic and current negative effects on the central nervous system
16 (CNS). Our ancestors experienced historical phases of cerebral weight loss contextually to waves of
17 inflammation,⁶ whereas modern psychiatrists do reckon that inflamed psychiatric individuals are likely
18 to be resistant to standard anti-psychiatric drugs.⁷ At the gut level, inflammatory signals cannot be
19 ignored, in view of the crucial role played by the immune system in a barrier organ such as the gut.⁸
20 Examples of these statements might be the puzzling role of the inflammatory post prandial reactions,⁹
21 the double-sword work of the inflammasomes,¹⁰ and the patrolling action of the sub epithelial
22 lymphocytes.¹¹ On these stages, inflammation is surprisingly being revealed to swing between overt
23 reaction (potentially a host tissue damage) and smart tolerance (protecting host).¹² Moreover, one can
24 only be fascinated by the evidence that the gut immune system is functionally correlated with the

1 airway lymphocyte circuits¹³ through the effects of the still largely unknown microbiome. Wrapping up
2
3 the notes collected above, one can see inflammation as a universally “magmatic” drift: from time to
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5 time permissive conditions may favor its materialization to defined clinical conditions and peculiar
6
7 consequences. Parodontitis,¹⁴ and, intriguingly enough, *Helicobacter pylori* infection,¹⁵ might originate
8
9 CNS degeneration with Parkinson’s like features; the inflammatory bowel diseases (IBDs)¹⁶ may stem
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11 from imbalance of the inflammation physiologically patrolling the borders, and may be at the basis of
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13 significant inflammation beyond the same borders;¹⁷ as an accompaniment to psychiatric disorders it
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15 may underscore individual’s defective adaptation to the surrounding world; all the way to the indolent
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17 yet painful malaise of today’s mankind.³
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23 Finally, we are just at the beginning of the road towards the implication of certain inflammatory
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25 markers as spies of our life expectancy. The intellectual icon of inflammation is now steadily
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27 approaching that of a gear mediating the two-way directed dialogue between the individual and the
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29 surrounding.
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