Abstract
Enhancing the ACE Pathway

Background: For a role in viral infection in the pathogenesis of Alzheimer’s disease there has largely been dismissed due to the lack of an accompanying inflammatory reaction. Relative few components in viruses are actually targeted by the cellular immune system. Deletion or mutation of these components can yield derivative viruses; which can cause cellular damage, yet are not effectively recognized by the host’s immune system. This generic immune evasion mechanism is termed “stealth adaptation.” Stealth adaptation can potentially occur with all human and animal viruses. It is most commonly observed in herpes simplex virus (HSV) infected fibroblasts supporting the presence of stealth adapted viruses. While some investigators have suggested a possible infectious origin of Alzheimer’s disease, it is usually argued that the actions of the virus must be indirect and quite possibly delayed. For example, common infections occurring in pregnancy can lead to delayed cytokine levels that may affect fetal brain development, with potential later life consequences. Certain infectious agents might trigger the assembly of an ongoing or an ongoing virus. In addition, certain components, thereby interfering with normal brain function. Such self-reacting antibodies may form as part of an ongoing autoimmune process. Some of these scenarios envision viruses as the direct cause of ongoing cell damage.

Results: Positive cultures were consistently observed in patients with neurodegenerative illnesses, including patients clinically diagnosed with Alzheimer’s disease. The results of this study suggest that conventional and stealth adapted viruses can be suppressed by a non-immunological defense mechanism involving the alternative cellular energy (ACE) pathway. For example, local activation of the ACE pathway using red dye plus intravenous gamma globulin and statins.

Conclusions: Studies are urgently needed to pinpoint the potential benefit of enhancing the ACE pathway in Alzheimer’s disease patients. If successful, these studies can be followed by protocols at least delaying the onset of Alzheimer’s disease and other neurodegenerative illnesses.

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