Abstract

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Metabolism of mycotoxins, intracellular functions of vitamin B12, and neurological manifestations in patients with chronic toxigenic mold exposures. A review.

Anyanwu EC, Morad M, Campbell AW.

Neurosciences Research, Cahers Inc., 8787 Shenandoah Park Drive, Suite 122, Conroe, Houston, 77385 TX, USA.

BACKGROUND: This paper evaluates the possible reasons for consistent vitamin B12 deficiency in chronic toxigenic mold exposures and the synergistic relationships with the possible mycotoxic effects on one-carbon metabolism that lead to the manifestations of clinical neuropathological symptomology.

SUMMARY: Vitamins are first defined in general and the nutritional sources of vitamin B12 are evaluated in particular. Since patients with chronic exposures to toxigenic molds manifest vitamin B12 deficiencies, the role of mycotoxins in vitamin B12 metabolism is assessed, and since vitamin B12 plays important biochemical roles in one-carbon metabolism, the synergistic effects with mycotoxins on humans are reviewed. An outline of the proposed mechanism by which mycotoxins disrupt or interfere with the normal functions of vitamin B12 on one-carbon metabolism is proposed. The overall functions of vitamin B12 as a source of coenzymes, in intracellular recycling of methionine, in methionine synthase reaction, in the prevention of chromosome breakage, in methylation, and in maintaining a one-carbon metabolic balance are reviewed. Signs, symptoms, and clinical neurological indications of vitamin B12 deficiency are also cited.

CONCLUSION: By implication and derivation, it is likely that the interruption of the structure and function of vitamin B12 would in turn interfere with the one-carbon metabolism leading to the neurological manifestations. This review is an attempt to formulate a basis for an ongoing research investigation on the subject.

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