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Letter to Editor

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Morgellons Disease and Iron Deficiency: Causal Ambiguities, Epidemiological Paradoxes, and Confounding Factors

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To the Editor

We read with interest the retrospective chart review by Gipple et al. [1] documenting a high prevalence of iron deficiency (38%) and insufficiency (17%) in a Morgellons disease (MD) cohort. While the authors identify a valuable, objectively measurable physiological abnormality within a population frequently restricted to psychocutaneous models, their interpretive framework leaves critical systemic and mechanistic questions unaddressed.

First, the study documents iron depletion without investigating its underlying etiology. No clinical assessments were reported for inflammatory markers, hepcidin dysregulation, occult blood loss, gastrointestinal malabsorption, or chronic tissue-specific infections. Consequently, it is impossible to determine whether this metabolic shift is causal, contributory, or secondary to the disease process. This interpretive limitation is compounded by the absence of a matched control group as well as clear selection biases: the data relies on a single specialty clinic, lacks complete testing, and reports ferritin measurements for only 52 of 73 patients.

Furthermore, a critical evaluation of the demographic data reveals a stark epidemiological paradox. The study reports that iron depletion is strictly concentrated among the younger subset of patients (median age 46; p=0.003). However, established psychocutaneous literature consistently demonstrates that MD predominantly afflicts an older, postmenopausal population. If iron-deficiency-driven striatal dopamine dysregulation is the primary phenotypic driver of the disorder as suggested by the authors [2], this model fails to explain why the disease peaks in a demographic that statistically retains normal iron status.

Iron deficiency also fails to explain the physiological impact of the cohort's exceptionally high rates of prescribed amphetamine use (43%) and ADHD diagnoses (38%). Chronic stimulant exposure is a well-documented driver of appetite suppression and systemic nutritional neglect. By treating amphetamine status merely as an isolated statistical confounder rather than a direct cause of dietary iron depletion, the authors overlook a highly plausible alternative hypothesis: the observed iron deficiency may simply be a secondary marker of substance-induced malnutrition rather than an intrinsic, idiopathic driver of cutaneous symptoms.

Finally, the study fails to address chronic inflammatory or infectious states—such as Lyme disease and tick-borne coinfections—which are noted in preceding MD literature [3,4]. Ultimately, because iron deficiency is associated with symptoms including peripheral sensory disturbances, altered dopaminergic signaling, and formication-like sensations, these findings support objective biological models of symptom generation in MD patients just as readily as psychiatric ones. While Gipple et al. should be credited with confirming the presence of iron deficiency in these patients, further rigorous evaluation of metabolic, immunological, and infectious mechanisms is warranted to improve our understanding of this complex illness [5].

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