

# Abetalipoproteinemia and Related Disorders Foundation (ABL + Foundation)

## Template Letter of Medical Necessity for VITAMIN E Coverage

Patients with abetalipoproteinemia and other familial hypobetalipoproteinemia disorders possess gene mutations required to package lipids into particles for absorption and transportation throughout the body (1, 2). Fat-soluble vitamins, including vitamins E, A and K are dependent on these pathways for absorption. Consequently, patients develop severe deficiencies of these vitamins and the clinical outcomes can be catastrophic, including debilitating neurologic impairment, blindness and life-threatening bleeding.

**Vitamin E deficiency** has been demonstrated in relation to the neurologic manifestations observed in hypobetalipoproteinemia disorders. Reduction and eventual loss of deep tendon reflexes followed by proprioceptive and cerebellar abnormalities can lead to severe disability and immobility. Neurologic defects can be identified on testing as early as 7 months of age; emphasizing the necessity for early and lifelong intervention with appropriate vitamin E supplementation (3, 4). An exceptionally high dose of vitamin E (100-300 IU/kg/day) is required to halt the progression and potentially reverse adverse complications (2).

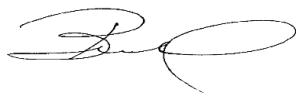
For patients with abetalipoproteinemia and related hypobetalipoproteinemia disorders, vitamin E is not a micronutrient supplement, instead an essential therapeutic treatment.

As the ABLRDF Medical Advisory Panel, our members include established researchers and medical professionals who care for patients with these disorders. We attest that a high dose of vitamin E taken lifelong is medically necessary to prevent devastating complications in patients with abetalipoproteinemia and other hypobetalipoproteinemia disorders.

Sincerely,



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