Axona® (Accera, Inc): A New Medical Food Therapy for Persons with Alzheimer’s Disease

Marian W. Roman, PhD, RN, PMHCNS-BC
The University of Tennessee, College of Nursing, Knoxville, Tennessee, USA

Edited by Marian W. Roman, PhD, RN, PMHCNS-BC
The University of Tennessee, College of Nursing, Knoxville, Tennessee, USA

For many, the title of this month’s column prompts two questions: What is Axona® and what is a medical food? The medical food designation is a U.S. Food and Drug Administration (FDA) category of substances intended for the clinical dietary management of a particular condition or disease. Specific criteria that must be met to receive this FDA designation include that the product must be:

- A specially formulated food for oral or enteral ingestion;
- For the clinical dietary management of a specific medical disorder, disease, or abnormal condition for which there are distinctive nutritional requirements;
- Intended to be used under physician supervision;
- Made with ingredients that have “Generally Recognized as Safe” (GRAS) status; and
- In compliance with FDA regulations that pertain to labeling, product claims, and manufacturing (http://www.cfsan.fda.gov/~dms/medfguid.html).

Nutritional supplements for the general public, nutrient rich healthy foods, and supplements are excluded from consideration for this designation. This would seem to beg the question: In what way does Alzheimer’s Disease (AD) have distinctive nutritional requirements?

The pathology of Alzheimer’s disease (AD) is characterized by cerebral atrophy in frontal, temporal, and parietal regions. The histological changes are now well known: senile plaques, dystrophic neurites, and neurofibrillar tangles within defined areas of the brain. The advent of positron emission scanning (PET) provided evidence that the brains of persons with AD, even in its rather early stages, appeared to have a characteristic deficit of glucose uptake in specific areas (deLeon et al., 1983). The coming of age of imaging studies in the early diagnosis of AD type dementia has confirmed this finding, and added more evidence (Mosconi, 2005). A recent longitudinal study confirmed that progressive reductions in the cerebral metabolic rate for glucose appeared in multiple brain areas. Moreover these reductions were shown to have occurred “years in advance of clinical dementia of the Alzheimer’s type symptoms in patients with pathologically verified disease” (Mosconi et al., 2009, p. 811). Characteristic patterns of regional hypometabolism also have been seen in other degenerative dementias such as frontotemporal dementia and dementia with Lewy bodies (Herholz, Carter, & Jones, 2007).

THE MECHANISM OF ACTION FOR AXONA®

The brain requires glucose for energy; however ketone bodies can be utilized as energy sources for cerebral neurons as well (Morris, 2005). The evidence for ketone body utilization by the brain was the impetus for the work that resulted in this medical food (Axona Product Bulletin, 2009).

According to the website and the written materials distributed by the company that holds the patent, Axona® does not increase metabolism. Instead, it provides an alternative energy source for the brains of patients whose brains cannot metabolize glucose effectively. Axona®, an oral agent, is digested and converted to ketone bodies by the liver. It results in rapidly elevated serum ketone bodies. The resultant elevated level of ketone bodies was found to correlate with improved cognition in AD patients as evidenced by improved scores on the cognitive subscale of the Alzheimer’s Disease Assessment Scale (ADAS-cog) as compared to the scores in the placebo group. The results of two clinical trials have been published in peer-reviewed journals (Reger, Henderson, & Hale, 2004; Henderson et al., 2009). Approval as a medical food was granted by the FDA in 2009 (www.about-axona.com).

ADMINISTRATION, SAFETY, AND SIDE EFFECTS

As a medical food, Axona® carries the GRAS attribution, but must be prescribed, and the patient must be under
professional supervision. A once a day dose of 30 mg of a powder is dissolved in 4–8 ounces of water or other liquid, and consumed in one sitting, after breakfast. The peak effects in alertness and cognition have been measured at two or more hours after consumption. Side effects can include GI distress (e.g., diarrhea, flatulence, and dyspepsia), particularly if not taken on a full stomach. Occasional elevation of blood urea nitrogen (BUN) levels has occurred. This medical food is contraindicated in those who are subject to ketoacidosis, such as certain diabetics, and in persons with renal dysfunction. The food contains milk and soy products. It has not been shown to increase cholesterol levels, although long-term studies are not yet available. It may be taken in conjunction with prescribed AD treatments, such as anticholinesterase inhibitors or the NMDA antagonist, memantine. (Information from insert and educational materials from Accera, 2009).

Readers are encouraged to send feedback on any experiences you have had with this new, novel product.

Declaration of interest: The author report no conflicts of interest. The author alone is responsible for the content and writing of this paper.

**REFERENCES**


