Ft. Lauderdale Notebook Series

Notebook #2: Interdisciplinary Studies

From a series of notebooks
chronicling the

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Prepared by Kendra Dayger, BS, MS
CFIDS Rochester Research Project

Serum Levels of Carnitine in CFS: Clinical Correlates

Audrius Plioplys, MD, CFS Center
Mercy Hospital, Chicago, Illinois

Thanks to Dr. Plioplys for his review and editing.

Notes

CFS Center was opened in Chicago in 1993. Works with wife. See poster on L-carnitine treatment (in Conference Proceedings book): reports significant improvement with L-carnitine *but only 5 of 14 chose to remain on it after the trial*, possibly indicating that the patients did not perceive such a great improvement. Also not a blinded trial.

Mitochondria review: final output is energy production. The input can be in the form of glucose or fatty acids. For the transport of long chain fatty acids carnitine is essential, which goes into a cycle of acylcarnitine back to carnitine transporting in the long chain fatty acids. Simplistically, carnitine can be seen as the worker pushing fatty acids into the mitochondrial energy production oven and taking out some of the toxic substances.

These data have similarities and differences with the Japanese data from Dr. Kuratsune.

N=35 CFS vs. Mayo Clinic norms (established by Dr. Engel, researcher in muscle diseases and carnitine deficiency conditions; based on normal controls, not patients). No one was taking L-carnitine supplements. Frozen and courier-delivered to Mayo Clinic for analysis with a radioactive labeled enzymatic procedure, which is different from the Japanese technique [see Dr. Kuratsune in preceding paper].

Results:

1. Total carnitine showed shift downward although most were in lower limits of normal.
2. Free carnitine showed same shift.
3. Acylcarnitine, which is the difference between the total and the free, does not have normative data from Mayo, and can't simply subtract the norms from above.

Comparison with Dr. Kuratsune: acylcarnitine decreased in both, using Kuratsune's controls. Note that these are two geographic areas and using two different techniques. Conclusion: very supportive.

Comparison with Dr. Kuratsune: free carnitine in Japan was normal, in Chicago was decreased in CFS but low normal. Total carnitine was also decreased but low normal.

Correlation with symptomatology: severity scales and carnitine. Fatigue with both free carnitine and total carnitine = weak but significant. CFS Impairment Index with free carnitine weak but significant. Less symptoms with higher carnitine.

Summary: Able to confirm Dr. Kuratsune's result about acylcarnitine levels being depressed and that basically the results are identical, emphasizing the fact this is found in two different geographical locations and using different laboratory techniques.

However there were some differences: depression of free carnitine level, also total carnitine level.

Saw clinical correlates with free and total carnitine levels, but did not see correlation with acylcarnitine levels.

In Dr. Kuratsune's Albany report [*CID*. January 1994; 18:S62.] he looked at symptomatology over time and relations to carnitine levels; this is not what was done here. This looked at a group of patients at one point in time. Issues of what is going to happen to their symptoms over time will be addressed in the future.

There are other studies underway. One study is a muscle biopsy research program, looking ultrastructurally at abnormalities in the mitochondrial membrane which will be correlated with L-carnitine levels. Another study involves an *unblinded* L-carnitine vs. amantadine treatment program, and preliminary results from 14 patients is available in the poster section of these proceedings: only 5 of the 14 patients decided to continue on L-carnitine after the study and only 1 on amantadine (and one on both).