

# Alopecia areata plus peripheral neuropathies evoked by emotive stresses may be treated by acetyl D carnitine per os in three weeks

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Sir,

OCTN2 is a widely expressed organic cation transporter. It plays a key role in the oral absorption, tissue distribution, and renal reabsorption of L-carnitine. It is polyspecific, and appears to act as both a Na<sup>+</sup>-dependent and Na<sup>+</sup>-independent uptake transporter of organic cations. It is implicated in systemic carnitine deficiency and in Crohn's disease, as well as in the disposition of cationic respiratory medicines in the lung [1].

OCTN2 can undergo degradation or mutation during senescence or neurological declines, caused by alcoholism, drug addiction or HIV or immunodepression.

OCTN2 has a high degree of sequence homology with OCTN1 [1,2]. Substrates of OCTN2 include TEA, quinidine, verapamil, pyrilamine, choline, short-chain acyl esters of carnitine, zwitterionic beta-lactam antibiotics, L-lysine, and L-methionine [3,4]. OCTN2 mediates the active absorption of L-carnitine in the small intestine and its reabsorption in the proximal tubule. OCTN2 also mediates the uptake of L-carnitine into adipocytes, cardiac myocytes, skeletal muscle cells, neurons, brain, lymphocytes, spermatozoa, and across the blood-retinal barrier. L-Carnitine is an essential component in the mitochondrial oxidation of fatty acids.

OCTN2 is very widely expressed in human tissues as: kidney, ileum, colon, skeletal muscle, lung, mammary gland, ovary, placenta, CNS, cornea, blood-retinal

barrier, macrophages, lymphocytes, spermatozoa, heart, pancreas, prostate, brain.

SLC22A5 that is a membrane transport gene providing instructions for making the protein itself called OCTN2 associated with primary carnitine deficiency. This protein is involved in the active cellular uptake of carnitine. It acts a symporter, moving sodium ions and other organic cations across the membrane along with carnitine. Such polyspecific organic cation transporters in the liver, kidney, intestine, and other organs are critical for the elimination of many endogenous small organic cations as well as a wide array of drugs and environmental toxins. Mutations in the *SLC22A5* gene cause systemic primary carnitine deficiency, which can lead to heart failure.

For these reasons acetylated L-carnitine could be useful to avoid heart failures, but when OCTN2 is deficient (in senescence, alcoholism, immunodepression and toxidependance), the penetration of BEE by acetylated L-carnitine is fully inhibited and so a drug that is produced, advertised and sold in South Italy is not able to maintain mental guidance and might not slow the rate of disease progression, improve memory, or improve some measures of mental function and behavior in some patients with Alzheimer disease and/or mental decline, as the producers of the drug itself promise.

Acetyl L-carnitine can be administered by transfusion or by mouth [1] and can treat many diseases evoked by

**How to cite this article:** Martini L. Alopecia areata plus peripheral neuropathies evoked by emotive stresses may be treated by acetyl D carnitine per os in three weeks. *Our Dermatol Online*. 2020;11(e):e66.1-e66.2.

**Submission:** 26.04.2020; **Acceptance:** 01.06.2020

**DOI:** 10.7241/ourd.2020e.66

emotive stress (in this case report alopecia areata and peripheral neuropathies even if not provoked by drug or alcohol addiction, but simply by emotive stress).

A young man (32 y. old) suffering from alopecia areata since 6 months cause of emotive stress (he was abandoned by his fiancée after 14 years and lost his job) and peripheral neuropathies (skin and legs pains with no evidence of pathological discomforts by x-rays, Cat scan or MRI scan) has been treated for 3 weeks by acetyl Dextrorotatory carnitine (Gensan, Germany; 1000 mg pro day)

Since there is a neat synergism between carnitine receptors and acetylcholine and nicotine receptors, the volunteer has been prayed to inhale pure tobacco many times during the day and smoke handmade cigarettes using the same inhaled tobacco and masturbate almost 1-2 times/day, since canitine encourages utterly spermatozoa migration in testicles (the young man has been suffering from spermatorrhea and nocturnal pollutions during all the treatment).

After three weeks all the disturbs caused by neuropathies disappeared at all and the AA have referred to be enthusiastic for the success of the treatment.

After the very first 6 days of assumption of 1000 mg of acetyl carnitine nerve pains disappear, alopecia begins to ameliorate even if skin remain whitish (perhaps a supplementary cause of vitiligo is present in this skin manifestation), but after three weeks almost all the neuropathies are defeated at all.

It must be stressed that the usage of acetyl carnitine as treatment or cure for mental decline or Alzheimer's disease should be revisited, after 35 years of inappropriate

administration to let the meridional firm to dedicate its employ for other neurological diseases.

Anyway its use as medicament combating peripheral neuropathies is welcome.

## Consent

The examination of the patient was conducted according to the Declaration of Helsinki principles.

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Source of Support:** Nil, **Conflict of Interest:** None declared.