

Hepatoprotective effect of multivitamins and phospholipids complex

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LCLT ARTICLE

Multivitamins and phospholipids complex protects the hepatic cells from androgenic-anabolic-steroids-induced toxicity

Hepatoprotective effect of multivitamins and phospholipids complex

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Introduction. Androgenic-anabolic-steroids (AAS)-induced hepatotoxicity typically occurs with C-17 alkylated oral agents abused by

exercising individuals at clinically recommended doses. Injectable compounds appear to have the same risk for hepatotoxicity, but are

applied in doses three to six times higher than clinically recommended. AAS users occasionally try to avoid the well-known hepatotoxic

effects associated with the abuse of a multitude of AAS agents, by using the pharmaceutical agent compound N a phospholipid/vitamin

preparation.

Primary Objective. The investigation of the actual hepatoprotective effect of compound N against AAS-induced toxicity.

Methodology. This was an observational cohort study of 320 athletes; 160 were AAS users and the other 160 were not abusing any

substances. Of the 160 users, 44 were using AAS and compound N (group A), and 116 were using solely AAS (group B). The 160 athletes

abstaining from substances abuse acted as controls (group C). All athletes were tested for alterations in serum levels of hepatic enzymes.

Enzyme levels before the study's onset and after the end of the 8-week AAS regimes were compared among the three groups, in order to

delineate the hepatoprotective effect of compound N.

Results. Prior to our research all groups showed normal values in all enzymes except

creatine kinase (CK). After the 8-week period, CK levels were slightly lower in group A, but without variation in Groups B and C; γ ;

Glutamyl Transferase (γ GT) levels remained normal. Groups A and C had no elevations in any of the enzymes, except CK, while in group

B all enzymes' values were elevated above the normal range. The only factor differentiating AAS users in group A from those in group B

was the use of compound N, thus the results being suggestive of the compound's detoxification effect. The severity of AAS abuse was

positively associated with the degree of changes (Δ values) in all measured enzymes except γ GT and CK.

Conclusions. Previous

suggestions that serum hepatic enzyme elevations in exercising AAS abusers are connected to muscle fiber damage rather than the abuse

itself, are contradicted by our results. Since all AAS abusing athletes were prone to exhibit elevations in enzymes' values, the mean values

of group A were to be similar to those observed in group B, exceeding normal values. The group hepatic enzyme values of group B were

significantly higher than the group C (control). Notably, group A did not have any statistically significant difference in the hepatic enzyme

values compared to group C. The effect of exercise on these enzymes'

Compound N was a multivitamin complex which consisted of: gelatine capsule containing 300 mg of natural, essential polyunsaturated phospholipids [polyene phosphatidylcholine] (diglyceride esters of choline-phosphoric acid and unsaturated fatty acids, predominantly linoleic acid in 70% concentration), 6 mg of thiamine mononitrate (vitamin B1), 6 mg of riboflavin (vitamin B2), 6 mg of pyridoxine hydrochloride (vitamin B6), 6 μg of cyanocobalamin (vitamin B12), 30 mg of nicotinamide, 6 mg of DL-alpha-tocopherol acetate (vitaminE).

Compound N is a composite of polyunsaturated phospholipids, mainly Phosphatidylcholine (PC) and vitamins of the B complex, which has been demonstrated to affect detoxification and possess antioxidative effects. PC is a constituent of important biological compounds; it is required for cell membrane integrity, methyl group metabolism, and lipid transport. It has a marked fluidizing effect on cellular membranes, as well as having hepatoprotective effects against toxicity and damage from alcohol, pharmaceuticals, environmental pollutants, xenobiotics, and from infection due to viral, bacterial, and fungal manifestations. In vitro, PC prolongs the survival of adult rat hepatocytes by means of stabilization of the plasma membrane, effectively preventing morphologic degeneration, such as enlargement of cell surface, degranulation of cytoplasm, and multinucleation.