THE EXPERIMENTAL STUDY ABOUT THE CHANGES ON SERUM TRIGLYCERIDES LEVELS UNDER THE INFLUENCE OF SOUND STRESS

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Summary

According to a research study conducted in early 2012 in Europe, noise pollution in enclosed spaces, as a risk factor, acts directly on the auditory organ, exerting negative local and general organic effects. Under sound pressure, the variation of triglycerides occur through a variable synthesis, depending on the intensity and duration of noise, with long term effects on somatic and mental health, with important medical and social impact.

Keywords: stress, sound pollution, triglycerides, noise, auditory organ

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Introduction

Cholesterol (organic alcohol, sterol) is synthesized in the body and found in cell membranes and body tissues, and is carried by the blood. Usually, it is not absorbed from the diet. It concentrates in the liver, spinal cord, brain, and at the level of the plaque, leading to atherosclerosis.

Hyperproduction of cholesterol (into low-density lipoprotein ) influence the production of vascular disease: brain, heart, eye-threatening, with vital risk to the body.

It is almost insoluble in water, therefore it's transportation is carried out by means of lipoprotein (carriers of cholesterol) which are water soluble and carry cholesterol and fats into the body. The proteins is formed in the surface of the lipoprotein particles of which determine the cholesterol to be removed and to be transported.

The first way of transport is the chylomicrons, some large lipoproteins loaded with triglycerides and cholesterol, in the intestinal mucosa, that goes to the liver by vascular blood. (Dietary Guidelines for Americans, 2010)

The triglycerides are synthesized in the liver from glycerol and fatty acids, from the processing of sugar, and certain carbohydrates.

These fatty substances are synthesized by the body from fats in food. They are stored in the body as adipose tissue, so are the main storage form of lipids into the body.

The fat is stored as glycerol, fatty acids and monoglycerides, which are converted in the liver into triglycerides. (Rifai \textit{et al}, 1999)

Materials and methods

Experimental animals used in our model were albino rats of Wistar line, males aged 14 weeks and weighing 200-220 g.

The animals were bred and maintained in Biobase "Ovidius" University of Constanta, in compliance with the rules of hygiene, food and accommodation required by Community legislation. (Ciudin and Marinescu, 1996)

The experimental model consists of five groups, whose characteristics are:

- Control group (M) - animals in this group were not exposed to noise, serving as a reference for the experimental groups.
- Experimental group (E1) - animals in this group were exposed only once to noise (45 ± 2 dB) for one hour, three minutes exposure, 3 minute break.
- Experimental group (E2) - animals in this group were exposed only once to noise (45 ± 2 dB) for 2 hours, 3 minutes exposure, 3 minute break.
- Experimental group (E1-7) - animals in this group were exposed to a cycle of exposure to noise (45 ± 2 db) which lasted seven days, for an hour, 3 minutes exposure, 3 minute break.
- Experimental group (E2-7) - animals in this group were exposed to a cycle of exposure to noise (45 ± 2 db) which lasted 7 days, 2 hours, 3 minutes exposure, 3 minute break.

After exposure to sound stress, animals were sacrified in compliance with the rules of force protection of laboratory animals and blood samples were collected.

For the determination of triglycerides, blood serum was used, obtained by centrifugation for 30 minutes at 5000 rev / min.

Determination of serum triglyceride levels was done using the Biochemistry kit marketed by DiaSys Diagnostic Systems GmbH, Germany.

Spectrophotometric method for the determination and the principle of the method is based on a series of biochemical reactions catalyzed by specific enzymes, that finally gives an quinoneimine, whose absorbance can be measured at 524 nm.

Triglyceride level is expressed in mg/dl.

Data were processed in the program OriginPro 75. The level significance was set at p≤ 0.05.

## Results

<table>
<thead>
<tr>
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<th>Triglycerides</th>
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<tbody>
<tr>
<td>M</td>
<td>X±ES</td>
</tr>
<tr>
<td>n</td>
<td>8</td>
</tr>
<tr>
<td>E1-7</td>
<td>234,16±2,96</td>
</tr>
<tr>
<td>t</td>
<td>-</td>
</tr>
<tr>
<td>p≤</td>
<td>NS</td>
</tr>
<tr>
<td>±M%</td>
<td>-4,03</td>
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<tr>
<td>E2-7</td>
<td>229.58±2.56</td>
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<tr>
<td>t</td>
<td>-</td>
</tr>
<tr>
<td>p≤</td>
<td>NS</td>
</tr>
<tr>
<td>±M%</td>
<td>-5.91</td>
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</tbody>
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**Table no. 1** Statistical variation of serum triglycerides values, after 1 hour of noise exposure per day for 7 days compared with values obtained after 2 hours of exposure to noise per day for 7 days

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>M</td>
<td>X±ES</td>
</tr>
<tr>
<td>n</td>
<td>8</td>
</tr>
<tr>
<td>E1</td>
<td>722.92±5.27</td>
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<tr>
<td>t</td>
<td>13.15</td>
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<tr>
<td>p≤</td>
<td>0.001</td>
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<tr>
<td>±M%</td>
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<tr>
<td>E2</td>
<td>535.58±8.75</td>
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<tr>
<td>t</td>
<td>13.07</td>
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<tr>
<td>p≤</td>
<td>0.001</td>
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<tr>
<td>±M%</td>
<td>+119.49</td>
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**Table no. 2** Statistical variation of serum triglycerides values after a single exposure to noise for an hour, compared with values obtained after a single exposure to noise for 2 hours

**Symbols and abbreviations:** (Senedecor, Cochran, 1980)

"X ± SE" - standard error;
"N" - number of individual samples that were finally constituted the arithmetic mean,
"t" - the value of "t" test of Student,
"p" - materiality based on the value of "t" (the change was considered significant in terms of statistical value of 'p' ≤ 0.05),
"± M%" - the percentage difference between the batch in question, and controls,
"NS" - change not statistically significant,
"p" ≤ 0.001 - > ***
Discussion

Graphic no. 1 Statistical variation of serum triglycerides values, after 1 hour of noise exposure per day for 7 days compared with values obtained after 2 hours of exposure to noise per day for 7 days

Graphic no. 2 Statistical variation of serum triglycerides values after a single exposure to noise for an hour, compared with values obtained after a single exposure to noise for 2 hours

Exposure, on short and long term, of a biological system to a noise source with constant intensity can produce physiological changes in the body, changes that can lead to acute and chronic disease.

In the experimental model presented, the laboratory animals have been exposed to a noise source with a constant intensity of 45 ± 2 dB, but with different exposure periods of time.

The effect is due to stimulation of the hypothalamic-pituitary-adrenal (HPA), which is a dynamic system adapted to answer to the constantly changing of body interrelation with the environment.

HPA stimulation of a single noise exposure produces significant changes (p ≤ 0.001) of triglycerides (chart 2), with a significant percentage difference between the batch in question and controls.

This can be explained by the fact that HPA axis stimulation produces a discharge increased amounts of adrenaline, as an effect of stimulation of the autonomic nervous system. (Cole and McNamara, 1997)

Due to increased secretion of adrenaline, the pituitary gland generates HGH (human growth hormone) which favors the transformation of fats (lipids) in carbohydrates causing hyperglycemia even if the subject has not consumed food. (Guder, et al., 2001)

Chart no. 1 shows statistically insignificant, with a slight percentage difference among the group concerned and controls.

This can be explained by the fact that a long exposure, it causes the release of cortisol, which is the enzyme indicator of stress.

Cortisol stimulates the activity of glycogen synthase, stimulates aminoacids uptake and synthesis of regulatory enzymes of gluconeogenesis, and also potentiates the action of glucagon and epinephrine in the liver.

The adipose tissue lipolysis increase cortisol, both directly and indirectly by potentiating other lipotropic hormones like adrenaline and somatotropic hormone and decrease glucose uptake at this level. (Cole, and McNamara, 1997)

We summarize that, after exposure to the experimental conditions of laboratory animals to sound stress, accounted for 45 ± 2 db, after activation of the spindle HPA, in substance humoral, hormonal control, increased serum triglyceride levels after a single exposure to sound stress for one hour and for 2 hours compared with exposure over a period of 7 days, for an hour, and 2 hours, where serum triglyceride levels remained within physiological limits.
Conclusions
1. Pathophysiological effects of exposure to sound stress is based on a complex mechanism that includes, among other things, a change in triglycerides levels.
2. When, some subjects have high, idiopathic, serum triglycerides without a favorable response to specific medication would be useful elimination of external factors that can cause a noise.
3. Serum triglyceride levels of animals exposed only once, for an hour or to 2 hours, increase the downloading massive adrenaline, the hormone that helps mobilize triglyceride from stores.
4. After exposure for a period of 7 days for an hour and two hours, serum triglyceride levels returned to normal, due to the action of cortisol, which induces lipolysis emphasis, and normalization of serum triglycerides levels.

References