Spleen Hypertrophy in Small Mammals: 
An Ecological and Histological Analysis

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The spleen is a multifunctional organ taking part in hematopoiesis and immune reactions in mammals. It is known that the spleen is sensitive to different damaging factors [1–3], but since its weight and size are highly variable, it is not recommended as a morphophysiological indicator of natural population state [4]. Spleen hypertrophy is sometimes seen in small mammals; it is a pathological enlargement of an unknown etiology, when the organ index is 100‰ or more, while usually it is 1–10‰ [5, 6]. It was proposed that this phenomenon be used as a marker of certain damaging factors in populations [7], but this requires knowledge of the cause(s) of splenomegaly (SM).

In medicine and veterinary, it is considered that the spleen size increase or SM has a multiple etiology and, in general, is not primary (it accompanies hepatic diseases, hematological neoplastic dysfunctions, inflammations, etc. [8–10]). Determination of SM causes in humans and domestic animals does not involve any difficulties since the key tools of medical diagnosis (life history and clinical studies) are available. These studies are not applicable to small mammals taken from natural populations. Therefore, the analysis of the relationships between SM frequency and animals condition, environment quality, and histomorphological data are essential. The results of ecological and morphological analysis allow the range of the possible causes of the pathology to make as narrow as possible.

The goals of this study were to research SM prevalence in several small mammalian species, to estimate the relationship with sex and reproductive—age status, and to characterize the micromorphology of the enlarged spleen. Data from small mammal field collections (2004–2009) (n = 1900) from forest ecosystems in the Middle Urals were used. Entrapments were conducted in natural (the Visimskii Reserve and the Pripyshminskie Bory National Park) and anthropogenically transformed habitats (the vicinities of the Middle Urals and Kirovgradskii copper smelters). Depending on the degree of industrial pollution, areas were classified as follows: background, buffer, and impact zones. The phytocenosis condition and levels of pollutant accumulation in soil and small mammal reservoir organs suggest pessimization of the quality of the buffer and, especially, impact zones [11–13]. Seven species of four genera of two rodent families, Muridae (Sylvæmus uralensis and Apodemus agrarius) and Cricetidae (Clethrionomys glareolus, Cl. rutilus, Cl. rufocanus, Microtus arvalis, and M. oeconomus) were studied. The animals were divided into reproductive—age groups: immature individuals (II), mature individuals (MI), overwintered individuals (OI). Data on the body weight and indices (the ratios of the organ weight and body weight in per mille) of the liver and spleen were used. The micromorphology of the enlarged spleen (n = 50) was studied in the bank vole (Cl. glareolus) which is the most widespread species in the collections. On the basis of the frequency distribution (Fig. 1), the animals were divided into two groups: those with a normal spleen weight (≤10‰) and those with SM (>10‰) (table). Logit regression was used for statistical analysis.

The animals with SM were found in all areas studied, and the SM frequency did not depend on industrial pollution (χ^2(2) = 1.07, p = 0.57). Therefore, SM cannot be considered a direct indicator of biotope pessimization.

Splenomegaly was almost absent in the family Muridae; In the family Cricetidae, it was not genus-, species-, or sex-specific and was in all reproductive—age groups. Among Cricetidae, SM was more prevalent in Microtus (18/54) than in Clethrionomys (89/1532). Remarkably SM was often found in MI and
OI ($\chi^2(2) = 10.4, p < 0.005$): the chances to detect SM in MI (Clethrionomys) are 3.5 times (95% confidence interval is 1.8–6.9) higher than in II; in OI they are 2.5 times (1.5–4.2) higher than in MI. In reproducing animals, SM depends on the sex: in male bank voles, it is detected 1.7 times (1.2–2.4) more frequently than in females ($\chi^2(1) = 10.4, p < 0.001$). Probably, the differences in the frequency of SM in Cricetidae are connected with distinct reaction to disease factors, as well as with their regional and physiological (reproductive) activity (and correspondingly different durations and frequencies of negative contacts).

The external and internal properties of animals with a normal spleen weight and with SM are similar, and they cannot be considered associated symptoms of pathological spleen enlargement. For example, SM in the bank vole is not connected with the hepatic index (Fig. 2).

From the histological point of view, SM in bank voles is a result of hyperplastic processes (i.e., an increase in the cell number). Therefore, we may use the term hypertrophy (an increase in the cell volume) only in the broadest sense. Among the diversity of histomorphological changes in the spleen, the most common are the ones when plasma cell reaction (mass trasformation of lymphocytes into plasmocytes) was

<table>
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<tr>
<th>Family/genus</th>
<th>Reproductive– age group</th>
<th>The level of pollution</th>
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<tr>
<td></td>
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<tr>
<td>Muridae/sylvaemus, Apodemus</td>
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<td></td>
<td>MI</td>
<td>0/21</td>
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<tr>
<td></td>
<td>MI</td>
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<td></td>
<td>OW</td>
<td>32/164</td>
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<tr>
<td>Cricetidae/Microtus</td>
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detected. In two-thirds of the cases, lymphoid follicles (taking part in the immune humoral response) retained its contours and germinal centers; i.e., they were functional; in other cases, they were at different stages of reduction. There were also cases of extreme disorganization of spleen tissues. Tissue degeneration and follicle reduction in the increased spleen may be interpreted, though with caution, as a sign of an immunodeficient condition of the animals.

Therefore, SM is a result of a pathological increase in cell number and their degeneration, it is widespread among small mammals and is not related to the level of industrial pollution or to hepatic disorders accompanied by hepatomegaly. Further search for SM causes should be conducted in the field of pathology associated with pathologies of immuno- and haemopoiesis organs.

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