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Density-dependent regulatory mechanisms of the generative potency in the males of a bank vole *Myodes glareolus*

Vera MAMINA and Oleg ZHIGALSKY

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Morphological analysis revealed destructive changes in the testicular tissue of bank voles *Myodes glareolus* Schreber, 1780 during different phases of a population fluctuation. The most pronounced changes were observed at the peak phase, when karyolysis of the Leydig cells and degeneration of spermatogenous cells were recorded in 90% of males. During the increase phase, depression of androgenous testicular function was observed in only 50% of males and atrophy of seminiferous tubules in 30% of males. During the low phase, the proportion of males with destructive changes in generative and endocrine portions of the testicles did not exceed 30%. Morphometrical analysis of spermatozoa demonstrated that the size of the head and nucleus were related to the phase of the population fluctuation. Lengths of the middle and main parts of the spermatozoon tail, as well as the size of the acrosome, were not related to phase of the population fluctuation.

Institute of Plant and Animal Ecology, Ural Division of Russian Academy of Sciences, 8 Marta 202, 620144, Ekaterinburg, Russia, e-mail: mamina@ipae.uran.ru

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Introduction

Many species of arvicoline (microtine) rodents undergo high-amplitude fluctuations in abundance. Some population fluctuations are short-term, completing a fluctuation within a few months (Krebs and Myer 1974, Taitt and Krebs 1985), whereas others may take 2–3 years to run their course (Oksanen and Henttonen 1996). The interval between population fluctuations, whether short-term or encompassing 2 or more years, may be annual or erratic; fluctuations that occur at 2–5 year intervals may be described as population cycles (Krebs and Myers 1974, Taitt and Krebs 1985, Krebs 1996, Bjørnstad *et al.* 1998).

Population density is determined by birth rate, immigration, mortality, and emigration (Zhigalsky 1992, Klemola *et al.* 2002, Norrdahl and Korpimäki 2002, Huitu *et al.* 2004, Cerqueira *et al.* 2006, Getz *et al.* 2006, 2007). Hypotheses proposing mechanisms involved in these processes can be divided into two groups: (1) extrinsic factors such as meteorological events, food supply, and predator abundance that regulate birth rate,

immigration, mortality, and emigration; (2) intrinsic factors, including social behaviour, such as territoriality, and other social interactions resulting in variation in the quality of animals that affect reproduction and mortality, as determined by population density. Although population dynamics and the mechanisms driving cyclic fluctuations have been extensively studied, the causes and mechanisms driving this phenomenon remain unknown. Long-term cyclic fluctuations in population density and the mechanisms involved are among the major enigmas of population ecology.

Data on bank vole *Myodes glareolus* Schreber, 1780 population dynamics were used to determine whether the observed fluctuations were stochastic or underlain by biological mechanisms. We conducted a long term study of dynamics of a bank vole population in the subtaiga zone in the Middle Urals. This population underwest periodic fluctuations, with intervals of one and three years. The one-year fluctuations were responses to seasonal changes in environmental factors. Fluctuations with a three-year interval were determined by intrinsic density-dependent mechanisms (Bernshtein *et al.* 1989, Zhigalsky and Kshnyasev 1999, Zhugalsky and Kshnyasev 2000).

In exceptionally dense populations of small rodents, especially in enclosures and cages, aggressive interaction between animals result in increased adrenocortical activity (Christian 1971, 1974). The increased circulating corticosteroids inhibit the reproduction process and reduce survival. Similar phenomena have been observed in free-living populations of *Microtinea* (Lee and McDonald 1985, Aguilera and Rabadan-Diehl 2000). Seasonal stress has an inhibitory effect on male reproductive functions, which are mediated in part by direct actions of glucocorticoids on Leydig cells in the testes (Hardy *et al.* 2005). Leydig cells are the primary source of testosterone in males. Levels of circulating testosterone are determined by the steroidogenic capacities of individual Leydig cells and the total number of Leydig cells in the testes. Stress-induced increases in serum glucocorticoid concentrations inhibit testosterone-biosynthetic enzyme activity, leading to decreased rates of testosterone secretion. High levels of intratesticular testosterone secreted by the Leydig cells are necessary for spermatogenesis. Insufficient gonadotropic hormones provoke a delay or disturbance in the spermatogenetic process that is accompanied by destructive changes, affecting both generative and endocrine aspects of the testes (De Kretser 1984, Mamina and Zhigalsky 2004).

Seasonal regulation of testicular function in mammals is a well-known phenomenon (Blottner *et al.* 1999, Blottner *et al.* 2000, Gockel and Ruf 2001, Schon *et al.* 2004). Klonisch *et al.* (2006) described the mechanisms of seasonal changes in the testicular tissue of the roe deer. The morpho-functional condition of testes at the different stages of a population fluctuation has been studied insufficiently for voles, in which reproduction begins in the spring and which experience high mortality rates in autumn. Degenerative processes in the seminiferous tubules cause the formation of pathological and atypical polymorphic spermatozoa with reduced fertilizing ability (Roosen-Runge 1977). There are, however, only fragmentary data on the morpho-functional condition of the testes and sperm morphology in murid rodents at different phases of a population fluctuation. Studies of the morpho-functional condition of both the testes and spermatozoa provide an opportunity to evaluate interactions of generative potency and fertility of males. We therefore conducted a cytomorphological analysis of testicles and spermatozoa to evaluate the generative potency of the male bank voles at different population densities.

Material and methods

Study area

Sexually matured male the bank voles were caught on the Middle Urals (57°21'N, 59°48'E) in June and July in years 1990–1995. Based on a complexity of demographic variables (population density, sex ratio and age structure, rate of sexual maturity, and breeding intensity) 1990 and 1993 were regarded as low phases of the population cycle (2.45 voles/100 trap days), 1991 and 1994 as increase phases (4.34 voles/100 trap days), and 1992 and 1995 as peak phases (9.31 voles/100 trap days).

Procedures

Removal of animals from the population in the Sverdlovsk Province was in accordance with the recommendations of the Directorate of the Federal Service for Control in the Field of Natural Resource Use. The numbers of trapped animals were as follows: in July 1990 n = 5 and in July 1993 n = 7, in June 1991 n = 14 and in June 1994 n = 22, in June 1992 n = 12 and in June 1995 n = 23. A total of 83 bank voles were lived trapped. The animals were killed by cervical dislocation and the testes removed and fixed in 10% formalin, embedded in paraffin, sectioned at 5 micron (μm), and stained with hematoxylin-eosin. Both right and left testes were subjected to morphological analysis (n = 166). Cross-sections of the testes were studied with the following results: morphological changes of the Leydig cells, which produce testosterone; morphological condition of the wall of the seminiferous tubules, which includes one of the components of the blood-testes barrier (Sertoli cells); destruction of the wall of the seminiferous tubule that may lead to the breach of penetrability of the blood-testis barrier and to death of Sertoli cells, which bring about the phagocytic and hormonal-regulatory functions of the testes; degeneration of spermatogenic cells; spermatogenous epithelium (disorganization of spermatogenous cells brings about the breach of spermatogenesis); seminiferous tubules (an absence of spermatogenous cells leads to the atrophy of seminiferous tubules).

Spermatozoa extracted from an epididymis of each animal were placed on the object-plate and mixed with a drop of physiological solution. The smears of spermatozoa were stained with azur-eosine and the preparation examined with a use of light microscope (at magnification 1350), with oil immersion. We examined 100 spermatozoa within a smear from an each animal. A SIAMS Photolab (Pattern Approval Certificate of Measuring Instruments Ru.C.31.005. AN 18352, Ekaterinburg, Russia) was used to measure the following parameters of spermatozoa: area of the nucleus (μm^2), area of the acrosome (μm^2), length of the middle part of the tail (μm), length of the main part of the tail (μm). Pathological forms of spermatozoa were registered: eg, those with morphological defects of the head, neck, and tail.

Statistical processing of the data was made with the use of monofactorial, nonparametrics disperse analysis (Kruskal-Wallis ANOVA).

Results

Body mass

During the low phase, the mean body mass of adult males was 19.3 g, during the increase phase 24.5 g, and at the peak 27.4 g. Body mass of males differed significantly between the increase – peak and increase – low (one-dimensional dispersion analysis, $p < 0.001$).

Morphological analysis of testis

Destructive changes in the testes of mature males are listed in Table 1. The percentages of animals with destructive change during different phases of a population fluctuation are presented in Fig. 1. Destruction of the wall in seminiferous tubules (Fig. 2) resulted in a partial death of the Sertoli cells that promote an intensification of destructive processes in spermatogenous epithelium. The gradual spreading of the degenerative changes within testicular tissue at the peak resulted in the diffuse spermatogenesis in solitary seminiferous tubules and karyopycnosis of the spermatogenous cells (Fig. 3). Disturbance of spermatogenesis and death of spermatogenous cells also causes atrophy of some seminiferous tubules. At the population peak,

Table 1. The types of destructive changes in the testical tissue.

Morphofunctional indices of a testis	Destructive changes	The type of changes
Interstitial tissue (the Leydig cells)-androgenesis	Cytoplasm oxyphility	I
	Karyolysis	II
The seminiferous tubule wall, the Sertoli cells – structural components of the blood-testicular barrier	The destruction of the seminiferous tubule wall and Sertoli cells	III
Spermatogenous epittelium- the process of the sexual cells development	Karyopycnosis	IV
	Karyolysis	V
Seminiferous tubule – the spermatogenesis activity index	Diffuse aspermatogenesis	VI
	Atrophy tubule	VII

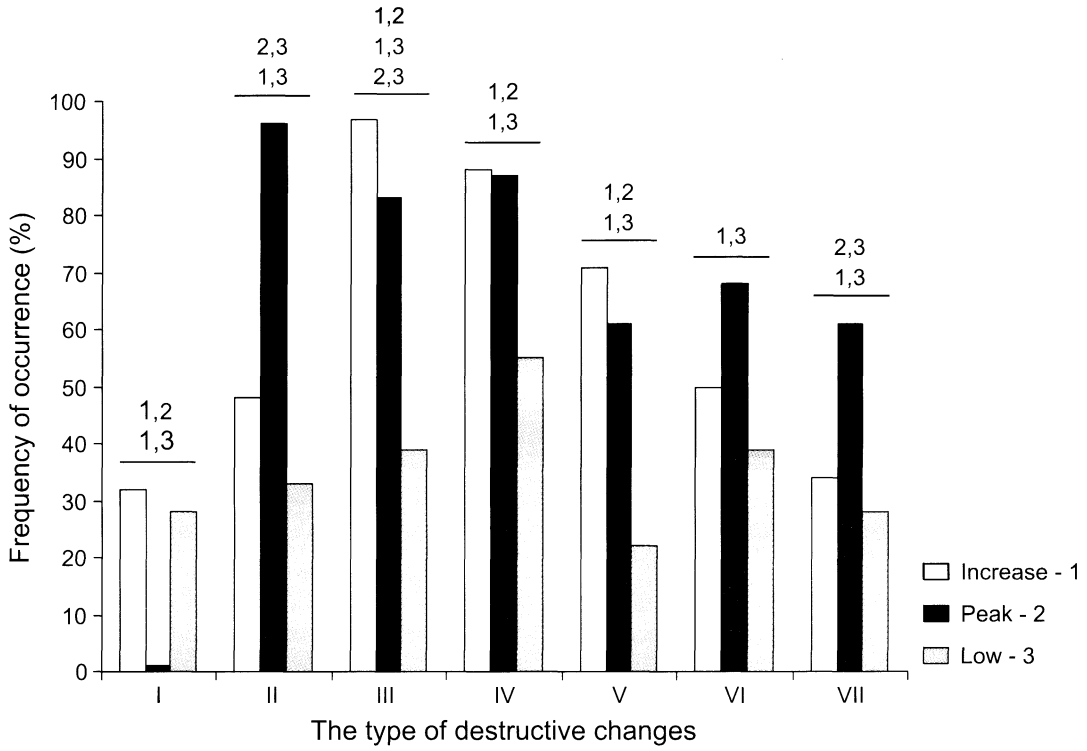

Fig. 1. Percentage frequency of occurrence of destructive changes of different types in the testicles of bank voles in relation to the phase of population cycle. Numbers above bars: a phases of a cycle according to a legend. Dispersion analyses were used to significant differences between phases (1–2, 1–3, 2–3). Statistical significance was determined at $p < 0.005$. See Table 1 for descriptions of changes I–VII.

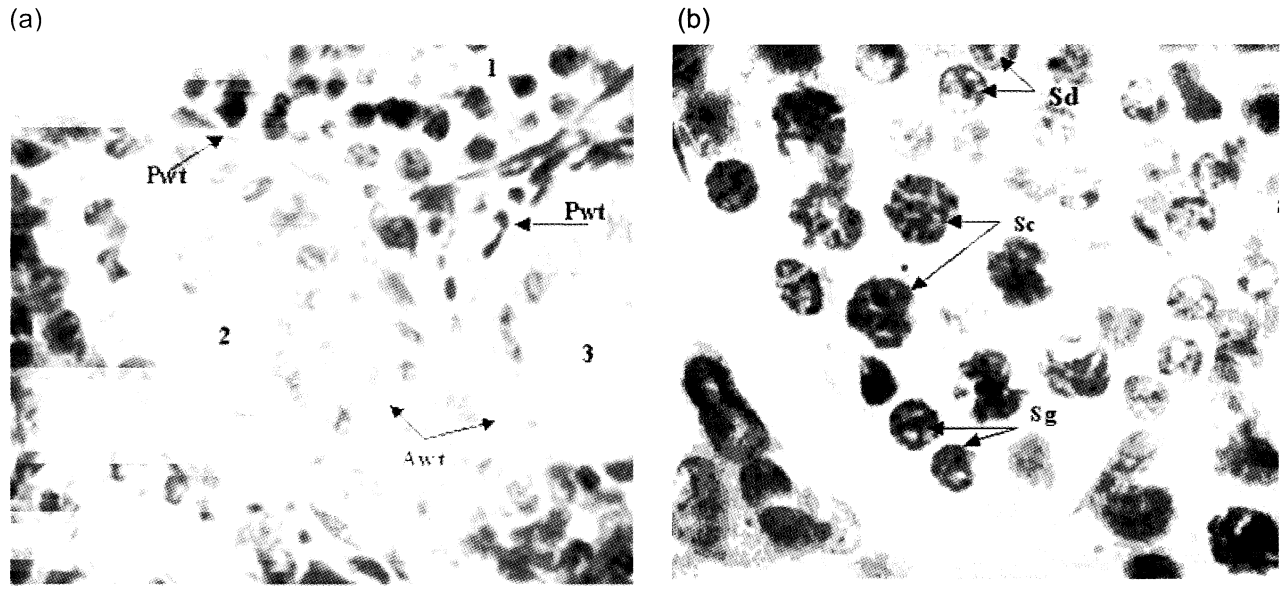

Fig. 2. Testis of a bank vole. (a) – a cross-section of a seminiferous tubules: 1 – a seminiferous tubule with normal spermatogenesis; 2, 3 – Atrophy of seminiferous tubules; Pwt – a wall of seminiferous tubules without changes; Awt – the destruction of seminiferous tubules wall; (b) – a fragment of seminiferous tubules with normal spermatogenesis, there are all types spermatogenic cells (Sg – spermatogonii, Sc – spermatocyte, Sd – spermatid spermatogenesis). Strictly concentric layers locate cells.

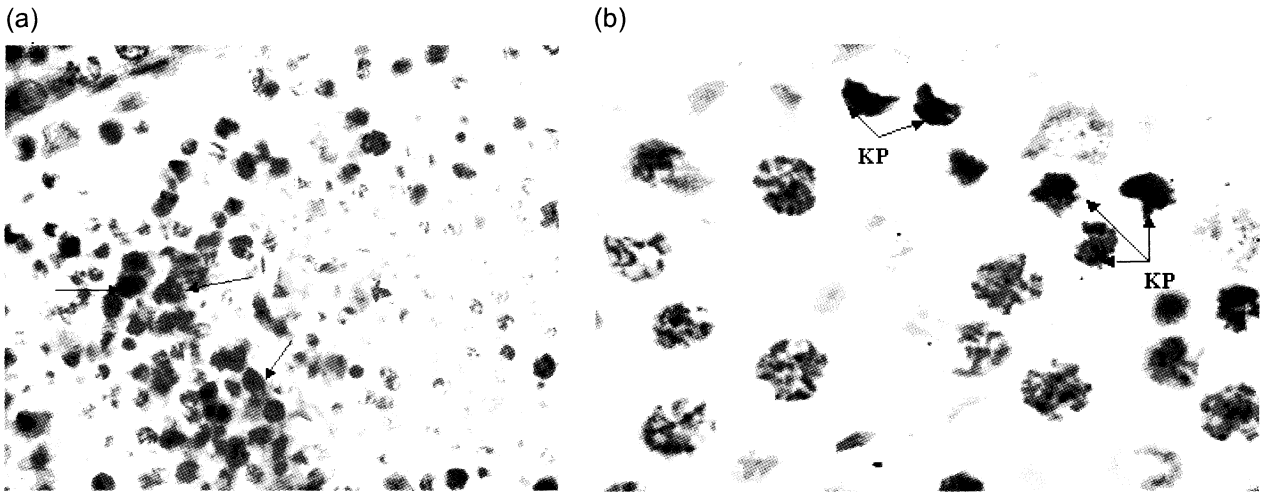

Fig. 3. Testis of a bank vole. (a) – a seminiferous tubule with diffuse a spermatogenesis (desorganization of layers, a chaotic arrangement spermatogenic cells), arrows specify degenerate cells; (b) – a seminiferous tubule contains spermatogenic cells with karyopycnosis – KP (the destroyed structure chromatin).

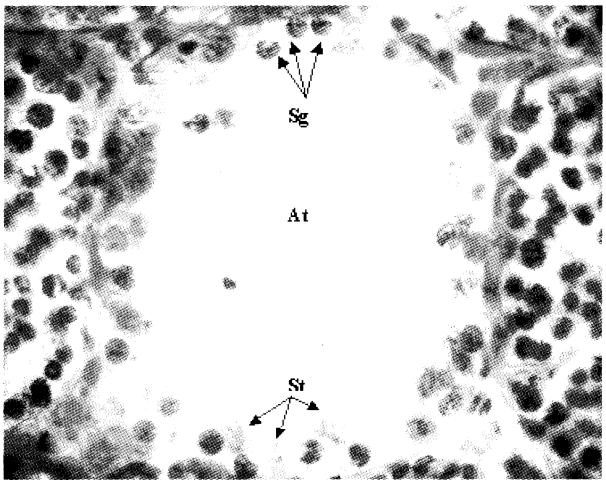

Fig. 4. Atrophy of seminiferous tubules (At) in testis of a bank vole, there are only Sertoli cells (St) and spermatogonii (Sg).

60–70% of the males displayed seminiferous tubules with diffuse aspermatogenesis and complete atrophy (Fig. 4).

Spermiogram analysis

Spermatozoa consist of a head, which includes a nucleus, acrosome, and the middle and main parts of the tail. Study of smear preparations revealed variation of the following parameters of the spermatozoa: (1) size of nucleus, from 5 to 25 μm^2; (2) size of an acrosome, from 4 to 17 μm^2 (Fig. 5); (3) length of the middle part of the tail, from 17 to 31 μm; and (4) length of the main part of the tail, from 41.0 to 70.5 μm. Acrosome size, as well as length of the middle and main parts of the tail appears to be independent from the phase of population fluctuation (Table 2). Variation in the nucleus of spermatozoon were related to phase, with values of 18.0 μm^2 during

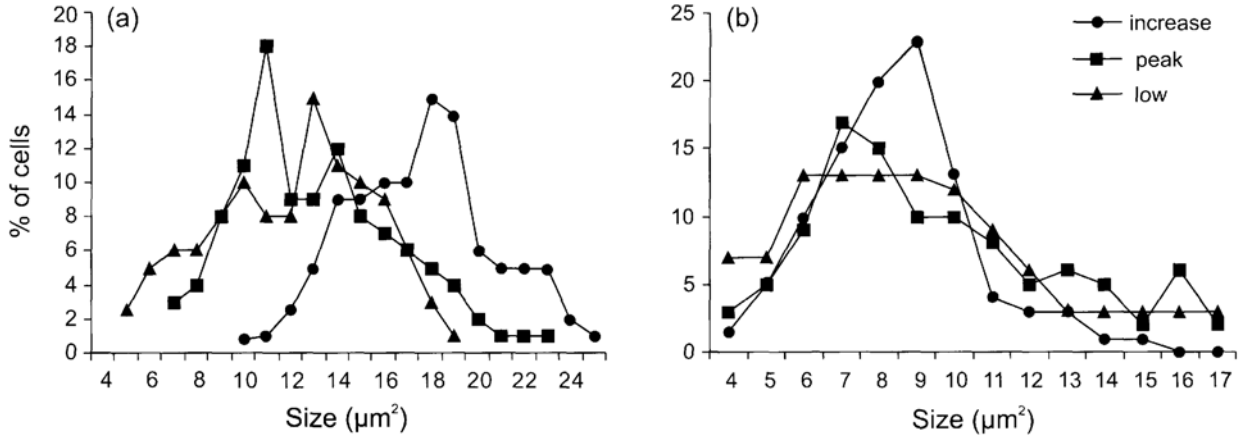

Fig. 5. Frequency of spermatozoa sizes: the nucleus (a) and the acrosome (b) in a bank vole in different phase of the population size.

Table 2. Morphometrical indexes of bank vole spermatozoa in different phases of population cycle. The analysis of a variance has shown essential distinctions of a size of the nucleus spermatozoa between phases: Increase-Peak and Increase-Low ($p < 0.001$). MS_b – mean square between groups, $MS_{w.}$ – mean square within groups intrasample variance.

	MS_b	df	$MS_{w.}$	df	F	p
The size of the nucleus	112.1	2	4.4	39	25.3	< 0.001
The size of the acrosome	0.53	2	2.9	39	0.18	0.83

the increase, 13.6 μm^2 at the peak, and 11.7 μm^2 during the low phase (Table 2). The high variability of the size of the head appears to be related to differences in DNA content, which determines the level of spermatozoa maturity (Bouters *et al.* 1976). During the increase phase of the cycle, males have spermatozoa nuclei with both loose and dense chromatin condensation that suggest different levels of maturity. Spermiogram analysis showed a presence of pathological forms of spermatozoa (the tail in the shape of a loop), but in only 10–15% of the males.

At the peak phase of the cycle, most spermatozoa had nuclei with dense chromatin packing that testified to a high level of maturity. Fifty percent of the males displayed up to 35% pathological epididymal preparations. During the low phase, the oligopyrene spermatozoa (with the size of nuclei – from 5 to 9 μm^2) did not exceed 10–15%.

Discussion

An important biological feature of cyclic population fluctuations of voles is phase-related changes in body mass, with adults in high-density phases 20–30% greater than those in low-density phases of a cycle. This observation, called the "Chitty effect", is considered to be a ubiquitous feature of cyclic populations. There is, however, no agreement among biologists regarding the causes of the "Chitty effect". Oli (1999) presented a hypothesis to explain the "Chitty effect", based on phase-related, dynamic allocation of energy between reproductive and somatic effort.

The effects of social stress on the immune and reproductive functions of small mammals have been well-documented. Increased glucocorticoid levels disrupt and suppress endocrine signaling in the male reproductive axis (Hales and Payne 1989). Glucocorticoids have been designated

stress hormones because circulation levels rise sharply in response to stress, resulting in testicular involution and a significant drop in testosterone secretion (Orr *et al.* 1994). These changes are accompanied by diminished libido and fertility. The changes in hormonal functions caused by population density is the consequence of the previous phase of a population fluctuation. At the peak of population cycle disruption of the neuroendocrine complex is observed. The neuroendocrine mechanisms slowing or stopping populations growth, are initiated during increase and are connected with early differentiation of "hypothalamus-hypophysis-adrenal gland" systems (Christian 1963, Lee and MacDonald 1985). Restoration of the mechanisms promoting population growth during the low phase are connected with early differentiation of "hypothalamus-hypophysis-thyroid gland" systems, leads to decreased thyroid hormone production and activation of reproductive functions (Vladimirova *et al.* 2006).

Thus, functioning of the gonad-adrenal systems may define reproductive success of a population. In our study, during the population increase, reduction in androgenous functions of the testes was observed in about 50% of the males and at the peak up to 90% of the males (karyolysis of the Leydig cells, wall destruction in the seminiferous tubules). The reduction of the androgenous function apparently was due to inhibition of gonadotropic hormones through an enhancement of adrenocortical activity. Suppression of androgenesis may lead to either inhibition or to complete cessation of spermatogenesis in the seminiferous tubules. Reduction of generative activity of the testes is most likely conditioned by both suppression of spermatogenesis and degeneration of the spermatogenous cells. During the increase phase, changes in spermatogenesis (diffuse aspermatogenesis) and the atrophy of seminiferous tubules were observed in 50 and 30% of the males respectively, and at the peak in 70 and 60% of the males. During the low phase, diffuse spermatogenesis and atrophy of seminiferous tubules was observed in 50 and 30% of the males respectively.

Spontaneous death of certain classes of germ cells is a constant feature of normal spermatogenesis in a variety of mammalian species, including humans (Kimura *et al.* 2003). This internal death program leads to typical morphological and biochemical changes that have been termed "apoptosis". Germ cell apoptosis is under endocrine, cell social association, and genetic control. Withdrawal of gonadotropins and/or testosterone accelerates germ cell apoptosis (Woolveridge *et al.* 1999). Testosterone, supplied by mature Leydig cells, is a necessary requirement for spermatogenesis. Degeneration of spermatogenous cells increases due to destruction of the wall of seminiferous tubules and the death of the Sertoli cells, which are responsible for nourishing, phagocytosis, and endocrine (estrogen) functions (Sharpe *et al.* 2003). During the low phase, changes in estrogen activity were observed in 40% of the males in our study. The seminiferous tubule walls and Sertoli cells are known to be structural components of the blood-testicular barrier (Russell *et al.* 1989). Breach of permeability of the blood-testicular barrier can initiate the autoimmune processes in the testes, which promote beginning of the destructive changes in spermatogenous epithelium. The low phase of population density, as a rule, follows the peak phase, when the animal's growth and development take place at high population densities and under the high physiological stress. Destructive changes in the testicular tissue, which we observed during the low phase, most likely were a response to endocrine conditions during embryonic development, which in turn was determined by the physiological status of the female during pregnancy (Tkachev 1980).

Degeneration of spermatogenous cells in the testes often gives rise to polymorphic spermatozoon. Degenerative changes in the germinal tissue cause elimination and degeneration of the chromosomes that results in formation of the oligopyrene spermatozoa (Roosen-Runge 1977), ie those with insufficient chromatin (with a small head). During the increase phase reproductive potential of males decreases due to pathological spermatozoa. At the peak, we expected decreased reproductive potential as the number of pathological spermatozoa doubled, and 20% of the spermatozoa have low amounts of chromatin. During the low phase, reproductive

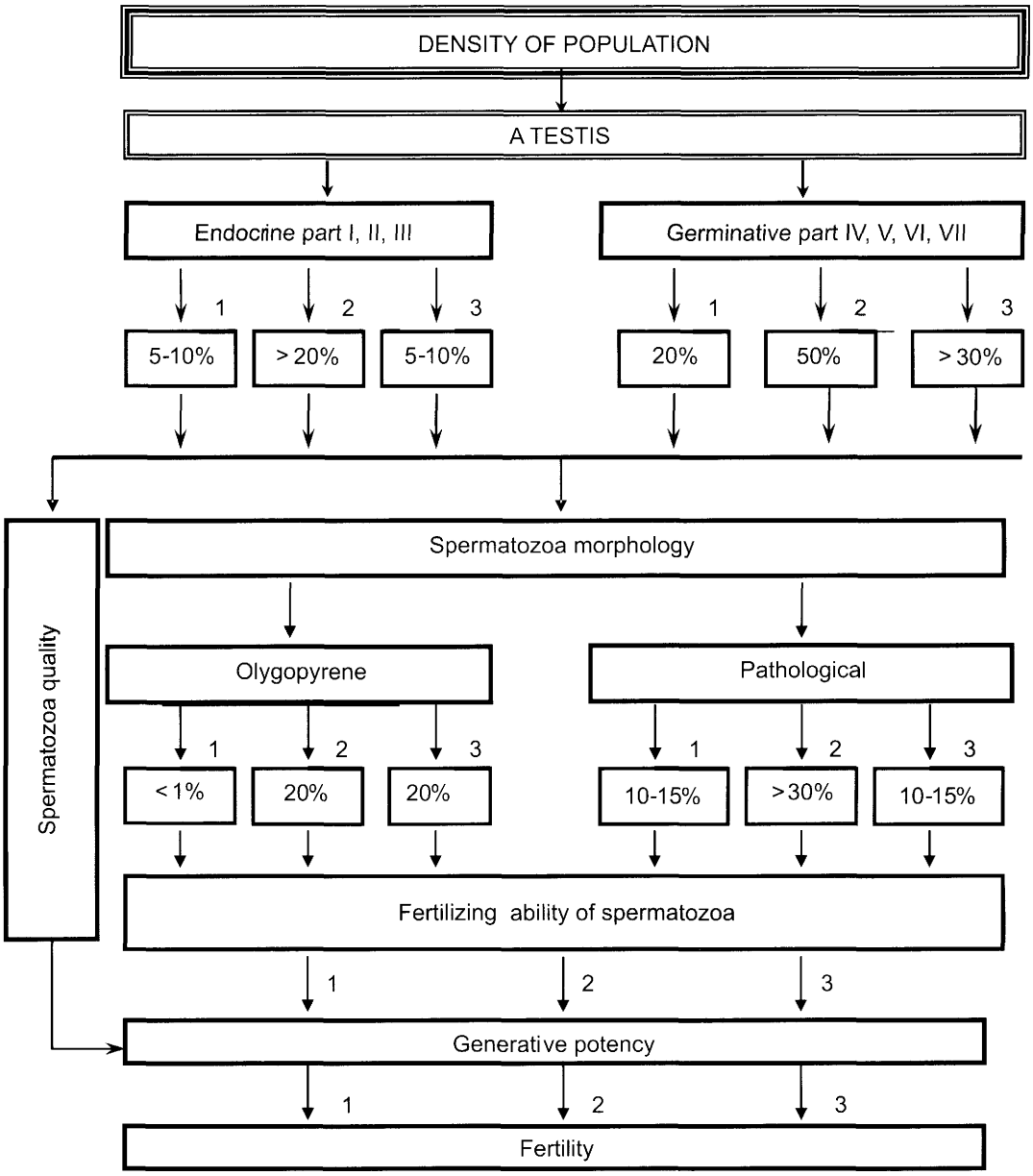

Fig. 6. Schematic representation of the mechanisms during density-dependent changes in the "testicle – spermatozoa – fertility" system in the different phases of population cycle: 1 – increase phase, 2 – peak, 3 – low phase; I, II, III, IV, V, VI, VII – different types of destructive changes (see Table 1 for descriptions).

potential remains low, because of the presence of olygopyrene and pathological spermatozoa.

Pathological forms of the spermatozoa are also the result of degenerative changes in the spermatogenous cells. Pathological spermatozoa with loop-shaped tails lose their mobility, thus reducing the ability to fertilize eggs. As a rule, oligopyrene spermatozoa do not take part in fertilization (Roosen-Runge 1977). Presences of hyper- and oligopyrene spermatozoa reduce fertility of males (Beatty 1970). Degenerative changes in testicular endocrine function result in the reduction of androgen products, which affects fertilization ability of spermatozoa.

Menkveld *et al.* (2003) found a positive correlation between the numbers of pathological, immobile forms of spermatozoa and the number of damaged acrosomes. We observed acrosome vacuolization in a few spermatozoa, which also could favour the reduction of their fertilization ability. We observed the most marked destructive changes in the testes and spermatozoa morphology at the peak phase of a population fluctuation. The cytomorphological analysis of the testes and spermatozoa in the bank vole allowed us to describe the mechanism of changes going in the "testicle – spermatozoa – generative potency of an animal" system during the different phases of a population fluctuation (Fig. 6). Change in population density led to a decrease in total number of spermatozoa and an increase in the frequency of pathological and oligopyrene spermatozoa. The morphofunctional condition of spermatozoa determines their ability to fertilize an egg, which affects reproductive potential of males.

Changes in the level of destruction of the testicular tissue and in the frequency of pathological and oligopyrene spermatozoa, as well as in the fertilizing ability of spermatozoa and the fertility of males as a whole, are one of the regulatory mechanisms in the "density – fertility" system. A similar scheme of interactions between the population density and reproductive ability of males may characterize the population fluctuations of other murid species.

Neurohumoral processes are involved in the development of distrophic processes in the gonads of mammals. Intrinsic factors may result in destructive changes both in the testes and ovaries that are capable of provoking a delay in spermatogenesis and follicular maturation in the young animals, which in turn prevent population growth.

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