

Environmental pollution and its effect on the living organisms has attracted lots of attention recently. There is a growing body of evidence that we are exposed to environmental pollutants at low concentrations in everyday life. The cells and organisms have tools to identify, neutralize and excrete the majority of the toxic compounds. The most dangerous are those that can escape this process or act at low trace concentrations. Endocrine disruptors (EDs) belong to the latter group.

Endocrine disruptors can be of natural and anthropogenic origin. EDs target corresponding hormonal receptors and can act at low concentrations. A wide family of nuclear receptors recognize steroid hormones. The majority of EDs can pass through the cytoplasmic membrane, use the hydrophobic nature of the receptor-ligand binding, trigger hormone response and change the expression of the sensitive genes. By interfering with estrogen and androgen signaling, EDs can have effect on the whole organism, but the reproductive system is influenced most. In the present work, our aim was to develop the methods for ED detection and monitoring, analyze the estrogenic potency of EDs, and evaluate the effects of natural estrogens and EDs on male reproductive functions, including sperm and testicular physiology and endocrine functions.

First, we prepared a panel of monoclonal antibodies recognizing environmental pollutants and natural estrogens. This allowed fast and reproducible detection of various EDs in environmental water samples. In part of our work we focused on preparation of monoclonal antibodies that recognize surface proteins of the sperm cells interacting with egg envelopes. This allowed us to study in detail the effect of EDs on sperm capacitation and hyperactivation.

Second, we determined the estrogenicity of environmental pollutants *in vitro* and studied the effect of these endocrine disruptors on male fertility and expression of testicular genes during spermatogenesis in a mouse model *in vivo*. We showed that the studied compounds induce changes in testicular gene expression patterns and have a negative effect on the male reproductive system. Our results provide the molecular basis for the underlying mechanisms of EDs action on male reproductive functions during the most susceptible periods of prenatal and pubertal development.

The submitted work has helped us to understand the impact of environmental pollutants on the male reproductive system and sperm maturation.