The aim of the study is to unravel mechanism of pathogenesis of ulcerative dermal necrosis (UDN) – a deadly disease affecting skin of salmonid fish, mainly salmon (Salmo salar) and sea trout (*Salmo trutta m. trutta*). Sea trout like salmon (*Salmo salar*) are anadromous fish belonging to the same genus and are considered as one of the most economically, biologically and socially important fish species, ensuring existence of commercial and recreational fisheries along the Atlantic and Baltic coasts. Also Sea trout like other anadromous fish plays an important role in maintenance of the general balance of ecosystems The project focuses on extremely important issue of biodiversity conservation. In the Baltic region the sea trout reproduces naturally in 25 rivers and some of these populations are supported by stocking. In Poland sea trout have appeared in many Pomeranian rivers and the Odra and Vistula basin. The catches of sea trout in total (offshore, river and sea) in the years 1972 - 1994 fluctuated in a small range and reached the size between 50 and 200 tonnes.). Only exceptionally in 1990 it was about 500 tonnes. Starting from 1995 the caches consistently increased reaching in 2002 as much as 863 tonnes. After 2002 the caches systematically dropped. Among reasons of constant decline in sea trout caught observed since 2002 UDN can be seriously considered as after several decades of relative peace in 2007 an UDN outbreak in Pomeranian rivers was noted. Severe, UDN caused losses of sea trout population threaten preservation of genetic resources of this precious species. The most severe course of disease in Poland was observed in fish from the Słupia river where 74.7 % of individuals catched in 2007 showed clinical symptoms of UDN. The clinical symptoms suggest the infectious (fungal/bacterial and/or viral) etiology of the disease. However, what we usually observe, i.e. dramatically affected fish, covered with ulcers and mold is just the final stage of the disease. We do not know what initiates the pathological process. Solving this puzzle is the key to explain the mechanism of disease pathogenesis. No virus has been detected so far as an pathogenic agent associated with UDN. However, it has been demonstrated more than 40 years ago, that filtrates from UDN affected tissues can transmit the disease to healthy fish. Early attempts to search for virus related to UDN, based on transmission and scanning electron microscopy were not successful. Answer to the above mentioned questions and hypotheses and explanation of the pathological nature of the syndrome requires as wide as possible investigation. Comprehensive approach proposed in the project, covers a wide range of methods starting from classical histopathological, electron microscopy and virological research and ending with application of innovative methods in the area of toxicology, proteomics and genomics. To this end we propose study of the infectious nature of the UDN syndrome (search for bacteria, viruses and/or fungi possibly involved in the development of the syndrome). Virological research will consist of two parts: i) in vivo experiments and ii) NGS based virus search. In vivo experiments aim to verify possibility of transmission of the pathogen from UDN affected to healthy fish. NGS based study of the nucleic acid samples from tissues of the UDN affected fish will be performed using a high-throughput sequencing on the illumina platform followed by bioinformatic analysis. Search for bacteria and fungi involved in UDN will be performed using routine, accredited methods (API tests). We also plan histopathological, TEM and SEM study of the UDN lesions. Also eventual toxic component (dioxins and related compounds) as well as the level of trace elements in sea trout tissues will be studied. Finally the proteomic analysis of UDN affected tissues of fish is planned. We believe that such wide range of research, unavailable in the seventies and eighties of the last century when many researchers tried to explain the pathogenesis of the disease, and available now will result in the dissection of the mechanism of UDN.