CURRICULUM VITAE

Geppo Sartori, born on May 16, 1966, is a Researcher of Molecular Biology at the Medical School of the University of Padova since february 2001 and currently conducts his research activity at the Department of Biomedical Sciences in the same University. In the year 1992 he obtained the degree in Biological Sciences at the University of Padova by discussing a thesis concerning his contribute to the sequencing of the yeast *Saccharomyces cerevisiae* genome. In the year 1994 he started a PhD course in Biochemistry and Biophysics, which was devoted to the functional analysis of a yeast gene, named *YNL234w*, coding for a protein belonging to the hemoglobins family but characterized by an unusual structure. His work has clarified some aspects of the *YNL234w* transcription regulation and of some biochemical characteristics of the corresponding protein. After the PhD degree he continued his work (partly in the J. Verdière laboratory at C.G.M. of C.N.R.S., Gif sur Yvette, France) to study in detail some aspects of gene transcription regulation by oxygen in yeast.

When he returned to Italy in 1997, he participated in a research project focused on the large-scale functional analysis of unknown yeast genes funded by the European Community. Among the results obtained, the most relevant is the characterization of Bud32, an evolutionarily conserved protein kinase which is an important component of a multiprotein complex (KEOPS) involved in transcription regulation and telomere homeostasis.

In 2009 he started a collaboration with Dr. Leonardo Salviati (Dept. of Woman and Child Health, University of Padova) and participate in the development of yeast models for the biochemical and functional characterization of mutations in human genes related to rare mitochondrial and metabolic genetic diseases, as ASL (Deficiency of Argininosuccinate Lyase), PCH6 (Pontocerebellar Hypoplasia type 6) and gyrate atrofy of choroid and retina.

This collaboration has continued to the present day by extending to other groups involved in the study of genetic disease related to mitochondrial disfunctions, as Friedreich Ataxia