Endosymbiotic processes were the defining events in the evolution of the eukaryotic organisms. As current chloroplasts and mitochondria evolved in this course, the development of an efficient communication system between the organelle and nucleus was required. Such signaling system is one of the most crucial factors for any "symbiotic consortium" to function properly. Information exchange between chloroplasts, mitochondria, and nucleus takes place by means of anterograde ("forward", nucleus-to-organelle) and retrograde ("backward", organelle-to-nucleus) signaling pathways. This bi-directional communication is necessary for coordination of organelles' development, function, and adjustments to changing environmental conditions. There is evidence that chloroplasts as well as mitochondria can exert an effect on nuclear gene expression. One of the retrograde signaling pathways was proposed to involve reactive oxygen species (ROS), particularly hydrogen peroxide (H_2O_2) and singlet oxygen ($^1O_2$). Although some progress has been made in deciphering the involvement of H_2O_2, our knowledge regarding the mechanisms governing $^1O_2$-induced chloroplast retrograde signaling is scarce and fragmentary. The aim of the project presented here is elucidation of these mechanisms. With the intention to search for the mechanisms responsible for conveying the $^1O_2$ information from the chloroplast to the nucleus, a ***sig**naling **Rep**orter* (*sigRep*) strain of *Chlamydomonas reinhardtii* was produced, which allowed us to monitor the efficiency of the $^1O_2$-signaling in this unicellular organism. Subsequent random mutagenesis performed on *sigRep* resulted in isolation of a series of mutants disrupted in $^1O_2$-signaling. These mutants were named ***g**enomes **un**coupled **S**inglet **O**xygen **S**ignaling* (*gunSOS*). We discovered that one of these mutants, *gunSOS1*, showed significantly altered expression of several genes, followed by altered content of certain metabolites. Among metabolites accumulating in *gunSOS1* relative to *sigRep*, fumarate is a well-recognized oncometabolite in mammalian cells, associated with development of tumors. 2-oxoglutarate was shown to regulate expression of enzyme involved in nitrogen assimilation in *Nicotiana tabacum*, while myo-inositol is a building block for several molecules also involved in the signaling. We decided to test the effect of these metabolites on $^1O_2$-signaling by providing them exogenously to the cell cultures. Fumarate, 2-oxogluatarate, and myo-inositol, always had a concentration-dependent suppressing effect on $^1O_2$-signaling in *sigRep*, with no change in *gunSOS1* and wild type, relative to their respective non-treated controls. Among the metabolites, which showed decreased content in *gunSOS1* relative to *sigRep* were mannose 6-phosphate and glucose 6-phosphate, which are mainly involved in starch and glucose metabolism. Exogenous application of the sugar phosphates had a positive effect on $^1O_2$-signaling in *sigRep*, but no effect could be observed in *gunSOS1* and wild type. However, aconitate was another metabolite deficient in *gunSOS1*, relative to *sigRep*. Addition of aconitate to the cell cultures increased the efficiency of the $^1O_2$-signaling in *sigRep*, but most importantly rescued $^1O_2$-signal transduction in *gunSOS1*. Furthermore, addition of aconitate rescued low expression of the proteins required for the $^1O_2$-signaling. Thus, the main goal of the research presented here is to explain the mechanisms behind observed positive effect of aconitate on $^1O_2$-signaling. There is no information in the literature about direct involvement of aconitate in any of the known signaling pathways, but increasing amount of reports points to the role of aconitase in stress responses. Aconitase is an enzyme interconverting citrate and isocitrate via cis-aconitate in the processes taking place in mitochondria. The expression level of aconitase in *gunSOS1* was significantly decreased relative to *sigRep*, but increased expression was observed after addition of aconitate. I hypothesize that not aconitate, but aconitase is the key player in chloroplast $^1O_2$-signaling in *C. reinhardtii*, while its localization in this organism depends on the redox balance between different compartments of the cell. Aconitase in plants and animals is susceptible to posttranslational modifications. The possibility of such modification and their role in aconitase function and involvement in $^1O_2$-signaling will be examined in *C. reinhardtii*, by means of modifications of the possible active amino acid residues, followed by biochemical analyses. The results of the project will expand our understanding of the $^1O_2$-signaling and its role in regulation of growth, development, and response to various stresses via exerted changes on nuclear gene expression. Additionally, because of the apparent cross-talk and dependence of $^1O_2$-signaling on other processes and signaling pathways, it is also expected that our knowledge regarding important signaling systems will also improve, which will open new routes in study of regulation of gene expression, development, and response to various stresses.