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Stress and Protists: No life without stress

Vera Slaveykova[a], Bettina Sonntag[b], Juan Carlos Gutiérrez[c,*]

[a] *Environmental Biogeochemistry and Ecotoxicology, Institute F.-A. Forel, Earth and Environmental Sciences, Faculty of Sciences, University of Geneva, 66, Bvd Carl-Vogt, CH-1211 Geneva, Switzerland*

[b] *Ciliate Taxonomy and Ecology Group, Research Institute for Limnology, Mondsee, University of Innsbruck, Mondseestrasse 9, A-5310 Mondsee, Austria*

[c] *Dpto. Microbiología-III, Facultad de Biologia, C/ José Antonio Novais, 12, Universidad Complutense (UCM), 28040 Madrid, Spain*

Abstract

We report a summary of the symposium "Stress and Protists: No life without stress", which was held in September 2015 on the VII European Congress of Protistology in partnership with the International Society of Protistologists (Seville, Spain). We present an overview on general comments and concepts on cellular stress which can be also applied to any protist. Generally, various environmental stressors may induce similar cell responses in very different protists. Two main topics are reported in this manuscript: (i) metallic nanoparticles as environmental pollutants and stressors for aquatic protists, and (ii) ultraviolet radiation – induced stress and photoprotective strategies in ciliates. Model protists such as *Chlamydomonas reinhardtii* and *Tetrahymena thermophila* were used to assess stress caused by nanoparticles while stress caused by ultraviolet radiation was tested with free living planktonic ciliates as well as with the symbiont-bearing model ciliate *Paramecium bursaria*. For future studies, we suggest more intensive analyses on protist stress responses to specific environmental abiotic and/or biotic stressors at molecular and genetic levels up to ecological consequences and food web dynamics.

Keywords: Environmental stress; Epigenetic; Nanoparticles; Oxidative stress; Protists; (Solar) radiation

Introduction

All living beings may have been under some kind of stress throughout their life. In fact, the existence of different environmental stress forms has been a key piece of the evolutionary machinery. Stress and life are closely connected. In general, the concept of "stress" can be considered as any harmful environmental factor that induces cellular physiological changes, disturbing the homeostasis of an organism. Therefore, it has a negative connotation because it induces damage or homeostatic disturbance on the living system. However, the initial negative connotation of stress can become a positive one after cellular acclimatization (recovering the cell homeostasis) and later selection of acclimatized cells, or in other words; "what does not kill it can make it stronger". If acclimatization fails and homeostasis cannot be recovered, cells die (unregulated cell death) or undergo suicide (regulated cell death or apoptosis) (Galluzzi et al. 2016).

From a biological point of view, we can distinguish two types of stresses; abiotic and biotic ones (Fig. 1). Abiotic stress includes all exogenous physicochemical

Abbreviations: ENM, engineered nanomaterial; ENPs, engineered nanoparticles; miRNA, microRNA; MOPS, 3-(N-morpholino) propanesulfonic acid; NPs, nanoparticles; OS, oxidative stress; PAR, photosynthetically active radiation; PER, photoenzymatic repair; QDs, quantum dots; ROS, reactive oxygen species; UVR, ultraviolet radiation.

*Corresponding author. Tel.: +34 913844968; fax: +34 913944964.
E-mail address: juancar@bio.ucm.es (J.C. Gutiérrez).

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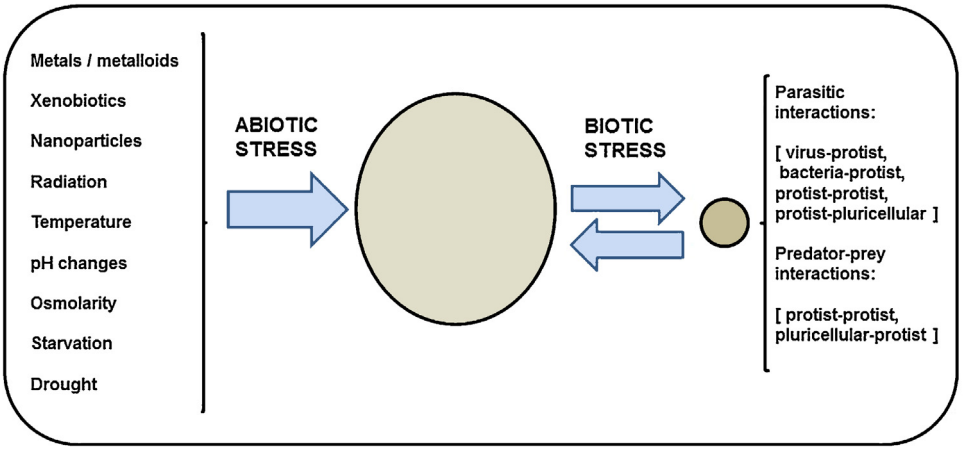

Fig. 1. Schematic representation of the main environmental abiotic and biotic stressors on an organism (big circle). Small circle represents any parasite (living stressor). For details see text.

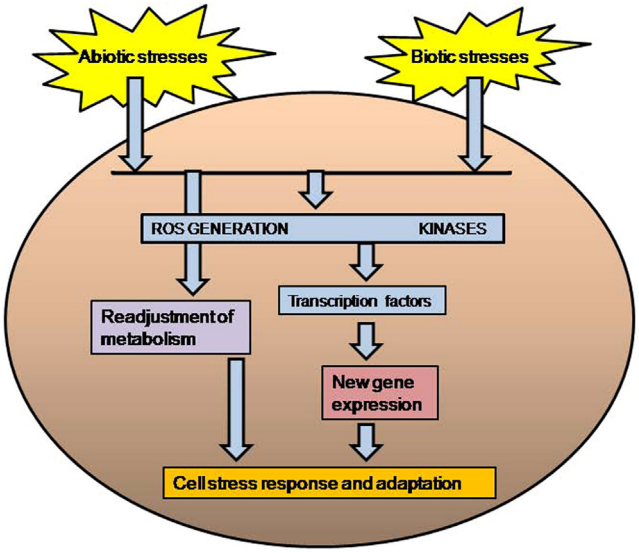

Fig. 2. Schematic representation of the different strategies involved in the cellular stress response.

environmental factors that may trigger a damage to any living organism, for instance; pH, temperature, osmotic stress, (solar) radiation, inorganic (metals, metalloids, metallic nanoparticles) and organic (xenobiotic) compounds, starvation, drought, etc. On the other hand, biotic stress involves the presence of whole cells or organisms acting as the stress source, and the interaction between the living stressor and the living receptor is the real cause of the stress, and, as they are living beings, both can be stressed. Some examples of biotic stresses are parasitical interactions (virus-, bacteria-, or protist-host), predator–prey interactions, or symbiotic interactions (Schwartzman and Ruby 2016). These types of stresses have convergent points in the signaling networks and overlapping gene clusters (Fujita et al. 2006) (Fig. 2). The generation of reactive oxygen species (ROS) has been considered as a key process present in both abiotic and biotic stress (Apel and Hirt 2004). Likewise, MAP-kinase cascades are another convergence point involved in the signaling network of abiotic and biotic stresses (Nakagami et al. 2005; Swiecilo 2016).

Cell mechanisms to respond to environmental changes are universal, so, in general, they are present in all living beings (including protists). The continuous or regular exposure to a specific stressor involves a cell acclimatization to that environmental stressor. This adaptive change can be reversible returning to the non-acclimatized cellular stage after the stressor is removed or disappears from the environment. When the stressor agent appears in the environment, a cell recognition mechanism carries out a chemical transduction by specific or unspecific receptors, indicating the cell the presence of that stressor. From this point, a complex signaling network connects the initial receptor with the molecular mechanism involved in the cell response against that specific stressor (Fig. 2). The cell response can be specific to only one stressor or general (common response) to several different stressors. In general, both cellular responses can co-exist, because cross-protection exists among different environmental stressors (Swiecilo 2016). Depending on the nature of stressor, cell adaptive responses may consist in a readjustment of metabolism or induction of new gene expression (Ruis 1997). In some cases, the new gene expression involves a cell differentiation process inducing a stressor-resistant cellular stage. All these cell alterations are focused to maintain cell survival under the stress conditions.

Both abiotic and biotic environmental stressors can modify gene activities via epigenetic mechanisms, so representing a connection between environmental change and genome response. In fact, several epigenetic control events (opening or closing gene expression) have been reported in organisms undergoing environmental stress (Meyer 2015). Three main epigenetic mechanisms seem to be involved in environmental stress acclimatization; DNA methylation, histone modifications (acetylation or methylation) and non-coding microRNAs (miRNAs). Before transcription initiation the gene expression can be regulated by the chemical

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