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Review

Abiotic stressors and stress responses: What commonalities appear between species across biological organization levels?

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ABSTRACT

Organisms are regularly subjected to abiotic stressors related to increasing anthropogenic activities, including chemicals and climatic changes that induce major stresses. Based on various key taxa involved in ecosystem functioning (photosynthetic microorganisms, plants, invertebrates), we review how organisms respond and adapt to chemical- and temperature-induced stresses from molecular to population level. Using field-realistic studies, our integrative analysis aims to compare i) how molecular and physiological mechanisms related to protection, repair and energy allocation can impact life history traits of stressed organisms, and ii) to what extent trait responses influence individual and population responses. Common response mechanisms are evident at molecular and cellular scales but become rather difficult to define at higher levels due to evolutionary distance and environmental complexity. We provide new insights into the understanding of the impact of molecular and cellular responses on individual and population dynamics and assess the potential related effects on communities and ecosystem functioning.

1. Introduction

Within their habitats, organisms are regularly subjected to stressors commonly defined as significant environmental deviations from optimal life conditions that decrease their fitness (Larcher, 2003; Roelofs et al., 2008). Organisms may suffer physiological effects affecting homeostasis and changing cellular metabolism and activity, which is termed as stress (Lichtenthaler, 1996; Steinberg, 2012). Stress can lead to severe damage or to specific responses that prevent or repair damage, depending on the degree of stress and the sensitivity of organisms (sensitive, tolerant, or resistant) (Lichtenthaler, 1996; Steinberg, 2012). Tolerance and resistance correspond to organisms' abilities to cope with stress with reduced or even no adverse effects, using mechanisms of stress avoidance, protection and defense (Calow, 1999). From an ecological perspective, the degree of stress sensitivity determines the edge of the ecological niche for species by exerting selective forces on the performance of life history traits (Roelofs et al., 2008). Because these traits can affect population dynamics and hence community composition, stressors (and related stresses) can result in community-scale effects and may ultimately drive species coexistence and community structures (Pierce et al., 2005; Steinberg, 2012; Moe et al., 2013). Consequently, the risk assessment of stressors must broaden its ecological base (Van Straalen, 2003; Beketov and Liess, 2012).

One challenge facing ecologists is understanding the mechanisms and extent of stress that influence biodiversity, community structure, and ecosystem functioning. The impacts of stressors are generally investigated using two, often disconnected, ways. The first way consists of analyzing molecular and trait responses to stressors of individuals of a given species, using “omics” and ecophysiological approaches. Such studies are mainly conducted under controlled conditions in order to link physiological and morphological stress outcomes to molecular mechanisms and cellular targets. High stress levels are often used to clarify mechanisms; however, studies using more environmentally realistic

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levels of stressors are increasing. The second way investigates the effects of stressors on populations and communities using mesocosms and field experiments, involving relevant stress levels and complex additional abiotic and biotic interactions. The majority of stress studies focus on certain responses of either one (model) species or a few closely related taxa. The disjunction of these two approaches results in incomplete and disconnected knowledge concerning how stress affects the diversity of taxa at the different levels of biological organization, and does not allow ecologists to develop an integrative view of the ecological and evolutionary consequences of stress.

Environmental stressors are widely diverse: they can be abiotic and/or biotic, and often vary in exposure time (acute to chronic) and intensity. Moreover, organisms must often cope simultaneously with several related or unrelated stressors that may significantly influence both the total level of stress sensed by the organisms (multi-stress conditions) and their biological responses. Given the increasing human-related alterations of ecosystems, this review focuses on chemical and thermal stressors, which represent important abiotic stressors related to anthropogenic impacts that species must deal with in both terrestrial and aquatic ecosystems. Chemicals in natural ecosystems occur in concentrations from trace to high levels, and mostly as complex mixtures of hardly predictable toxicity (Fent, 2004). All climatic scenarios predict that both temperature and its variations will increase in the forthcoming decades, as will extreme climatic events, with side-effects on the phenology and geographical range of several species and on the interactions between species (Williams et al., 2007).

Based on various taxonomic groups representing diversity key-components for ecosystem functioning in terms of primary production and food chain links (photosynthetic microorganisms, plants, and invertebrates), this review aims to analyze and discuss the current knowledge on how organisms respond and adapt to these abiotic stressors from the molecular to the community level.

Several molecular mechanisms and physiological responses (e.g. chaperone proteins, energy allocation) are often highlighted in stress studies. In particular, the existence of a "minimal stress response proteome" across species (Kültz, 2005) suggests a potential uniformity in the stress responses of different species at the cellular level. The commonalities of stress responses at higher levels of organization (from individual to community), however, have not yet been clearly addressed. Evaluating environmentally realistic experimental and field studies, here, we develop a wider integrative view, taking into account ecosystem biodiversity and the diversity of anthropogenic-related stressors in order to i) compare how molecular and physiological mechanisms related to protection and repairing functions and energy allocation can impact the life history traits of organisms under exposure to environmental stressors, and ii) determine to what extent trait responses influence individual and population responses, and with what repercussions on communities and ecosystems (Fig. 1). Commonalities in stress responses become less obvious at higher levels of biological organization as consequences of evolutionary distance and environmental complexity (particularly involving stress interactions as well as species/biotic interactions) but still remain connected *via* their energetic based origins.

2. Molecular and cellular levels: how analogous are responses to abiotic stressors?

2.1. Do abiotic stressors induce similar mechanisms across taxa to protect cell integrity?

At the cellular level, abiotic stressors cause metabolic imbalances, cascading effects on biochemical and physiological processes, and in severe cases, cellular death. Stress-induced injuries can occur directly by altering the biochemical structures and associated functions of biological molecules, or indirectly through the variation of osmotic pressure, the production of toxic metabolites and free radicals, the generation of oxidative stress, or the modification of signaling pathways (Couée et al., 2006; Guy et al., 2008; Srivastava et al., 2013; Teets and Denlinger, 2013). Chemicals such as pesticides strongly affect enzyme activities, the balance of reactive oxygen species (ROS) and cell communication, independently of their specific molecular targets (Couée et al., 2006; Brulle et al., 2009; Ramel et al., 2009a; Bouetard et al., 2013). Thermal stresses have considerable effects on the stability of nucleic acids and proteins, enzyme activities, and the fluidity of biological membranes (Kültz, 2005; Pierce et al., 2005).

Comparing various taxa, Kültz (2005) demonstrated that photosynthetic microorganisms, plants, and invertebrates share approximately three hundred proteins that are involved in cellular stress responses, highlighting a high degree of uniformity among species. This "minimal stress response proteome" seems to reflect the limited number of stress responses that have evolved among taxa to efficiently counteract the adverse endogenous effects of environmental stressors. Indeed, those proteins are involved in the protection (detoxification and excretion, damage sensing and repairing) of the same cellular functions (cell integrity, cell cycle or apoptosis, nucleic acid-related processes, metabolism and energy homeostasis, and redox status) across taxa, suggesting that similarity of responses at the cellular level may thus be connected to similarity of cellular organization and functions at the molecular level. This "minimal stress response proteome" constituted a first basis on which to analyze the commonality of stress responses among key taxa of ecosystem functioning in a context of anthropogenic-related stressors.

Ubiquitous mechanisms of stress response have evolved in order to limit or to repair abiotic stress-related damage to DNA, protein, and phospholipid native conformation (Kültz, 2005; Moller et al., 2007). The constitutive or induced expression of chaperone proteins, such as heat shock proteins (HSPs, HSCs), is one of the most ubiquitous and evolutionarily conserved mechanisms among organisms. Chaperone proteins facilitate the correct folding of proteins in *statu nascendi* or the refolding of damaged ones upon exposure to a wide range of environmental stressors (Hightower, 1991; Timperio et al., 2008; Zhao and Jones, 2012; Srivastava et al., 2013). HSP induction has thus been reported in diverse organisms exposed to a large variety of stressful conditions (e.g. pesticides, metals, salts, drought, heat, cold, UV radiation, and diseases), all causing protein denaturation, which is the genuine inducer (e.g. Slabas et al., 2006; Kim et al., 2007; Timperio et al., 2008; Bouetard et al., 2013; Srivastava et al., 2013; Peñuelas et al., 2013).

Detection of DNA damages triggers DNA repair mechanisms through the transcriptional and/or post-transcriptional induction of a variety of DNA repair and recombination (DRR) genes (Kimura and Sakaguchi, 2006). Completed genome sequences from prokaryotic and eukaryotic organisms reveal a large proportion of DRR genes to be conserved, although phylum-specific mechanisms of DNA repair have also been proposed (Kimura and Sakaguchi, 2006). Thus, whereas plant genomes contain DRR eukaryotic homologs (closer to human than to yeast counterparts) and some prokaryotic-specific genes (e.g., *RadA* and *FPG* homologues), they also exhibit several plant-specific DRR genes (e.g. *Rad2* family nucleases) (Kimura and Sakaguchi, 2006). Nevertheless, in species as different as cyanobacteria, plants, and invertebrates, DNA damage due to temperature and chemical stressors generally increases DNA repair mechanisms (base or nucleotide excision repair, non-homologous end joining and homologous recombination), with tolerance