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Priming and memory of stress responses in organisms lacking a nervous system

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ABSTRACT

Experience and memory of environmental stimuli that indicate future stress can prepare (prime) organismic stress responses even in species lacking a nervous system. The process through which such organisms prepare their phenotype for an improved response to future stress has been termed 'priming'. However, other terms are also used for this phenomenon, especially when considering priming in different types of organisms and when referring to different stressors. Here we propose a conceptual framework for priming of stress responses in bacteria, fungi and plants which allows comparison of priming with other terms, e.g. adaptation, acclimation, induction, acquired resistance and cross protection. We address spatial and temporal aspects of priming and highlight current knowledge about the mechanisms necessary for information storage which range from epigenetic marks to the accumulation of (dormant) signalling molecules. Furthermore, we outline possible patterns of primed stress responses. Finally, we link the ability of organisms to become primed for stress responses (their 'primability') with evolutionary ecology aspects and discuss which properties of an organism and its environment may favour the evolution of priming of stress responses.

Key words: priming, stress signalling, epigenetics, memory, fitness, stress tolerance, defence, bet hedging.

CONTENTS

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I. INTRODUCTION

Successful and efficient processing of information is essential for the survival of organisms. Memory of environmental stimuli may affect the response of an individual to future environmental conditions. Memory of an event experienced in a certain life stage, i.e. the capacity to retain information on this event, is not necessarily permanent, but may be short-lived or long-lasting. Evidence is growing that not only animals, but also organisms lacking a dedicated nervous system, namely bacteria, fungi and plants, can 'remember' a past experience (e.g. Casadesús & D´Ari, 2002; Bruce *et al.*, 2007; Galis *et al.*, 2009; Rensing, Koch & Becker, 2009; Thellier & Lüttge, 2013). Memory of a past event may shape or 'prime' the response to future environmental stimuli, resulting in phenotypic and stimulus-dependent plasticity of response traits. Such plasticity allows an individual to adjust its physiological or developmental phenotype to its environmental experience (Sultan, 2000; Agrawal, 2001; West-Eberhard, 2003). The term 'priming' of organismic responses by a previous experience is used in various scientific disciplines. These range from immunology, psychology and neurosciences to agricultural, soil and plant sciences, and they all use the term in a discipline-specific way.

In plant stress biology, the term priming is used to describe phenomena which have in common that the priming process positively affects future plant performance if the plant is exposed again to certain stress conditions. In parallel to the term priming, other terms are also used, e.g. systemic acquired resistance (SAR) when referring to enhanced resistance of plants against phytopathogens after pre-exposure to sublethal doses of phytopathogens (e.g. Sticher, Mauch-Mani & Métraux, 1997; Métraux, Nawrath & Genoud, 2002; Conrath, 2009, 2011; Jaskiewicz, Conrath & Peterhaensel, 2011; Dempsey & Klessig, 2012; Luna *et al.*, 2012; Kachroo & Robin, 2013; Zeier, 2013). The terms hardening or cold acclimation are used when referring to improved cold or frost tolerance of plants after pre-exposure to cold (e.g. Thomashow, 1999; Guy, 2003; Hannah, Heyer & Hincha, 2005; Stavang, Hansen & Olsen, 2008; Hincha & Zuther, 2014). However, it is not only the experience of a mild stress that 'warns' a plant of future stress that is considered as a priming stimulus which improves a plant's response to impending threats. Colonization of plant roots by beneficial rhizobacteria is also studied in the context of priming since it may result in so-called induced systemic resistance (ISR) which positively affects the response to phytopathogens infecting the leaves (e.g. Heil & Bostock, 2002; Van Loon, 2007; Van der Ent, van Wees & Pieterse, 2009). Similarly, plants can be primed by association with symbiotic fungi (e.g. Pozo *et al.*, 2009). Improved resistance against phytopathogens is also achieved by priming plants with natural or synthetic compounds. For example, treatment of plants with salicylic acid, 2,6-dichloroisonicotinic acid (INA), benzo-(1,2,3)-thiadiazole-7-carbothioic acid *S*-methyl ester (BTH) or β-aminobutyric acid (BABA) can lead to improved plant resistance against phytopathogens (e.g. Zimmerli, Métraux & Mauch-Mani, 2001; Aranega-Bou *et al.*, 2014). Priming of seeds by treatment with synthetic and biological compounds increases plant performance during later plant growth and development (Jisha, Vijayakumari & Puthur, 2013). Seed priming comprises diverse treatments, e.g. immersion of seeds in water for hydro- and osmopriming (increasing osmotic stress resistance), exposure of seeds to antioxidants for redox- and thermopriming, or so-called biopriming by coating seeds with isolates of symbiotic bacteria or fungi, thus improving seedling growth.

In microbiology, pre-exposure of bacteria or fungi to a stress may prime their responses to future stressful conditions (e.g. Zacharioudakis, Gligorls & Tzamarias, 2007; Wolf *et al.*, 2008; Mitchell *et al.*, 2009; Zakrzewska *et al.*, 2011; Dhar *et al.*, 2013; Runde *et al.*, 2014, and references therein). This phenomenon is mainly referred to as cross protection or

acquired stress tolerance. When bacteria are attacked by viruses, they can incorporate fragments of the viral nucleic acids into their genome, and thus activate a specific adaptive immune system; this phenomenon is also referred to as priming (Datsenko *et al.*, 2012; Fineran *et al.*, 2014; Li *et al.*, 2014; Richter *et al.*, 2014). However, in this case a change of the genotype due to incorporation of foreign DNA improves the bacterial immune response to future attacks by viruses. Hence, this phenomenon does not refer to a change of only the phenotype.

In summary, the common denominator of these examples is that the priming stimulus prepares an organism for improved responses to upcoming environmental challenges. Several other terms are used to describe similar phenomena (see above, e.g. SAR, ISR, hardening, acclimation, cross protection). During the last few years, excellent reviews on priming of plant responses to phytopathogen attack (e.g. Conrath, 2011; Gamir, Sánchez-Bel & Flors, 2014), herbivores (e.g. Frost *et al.*, 2008; Galis *et al.*, 2009; Arimura, Shiojiri & Karban, 2010; Heil & Karban, 2010; Holopainen & Blande, 2013) and various stresses (e.g. Bruce *et al.*, 2007; Pastor *et al.*, 2013) have been published. These reviews focus on the evidence of priming and possible mechanisms in plants and provide experimental details of individual examples.

By providing a conceptual framework for priming of stress responses in plants, fungi and bacteria, this review aims to facilitate discussion and interactions across biological subdisciplines. Special attention is paid to providing a unifying definition of priming which (*i*) allows inclusion of other terms used in this context, but also makes distinctions between them, and (*ii*) places priming abilities into an evolutionary ecology context. In addition to mechanistic aspects of priming that tackle the question of how the process of priming of stress responses works in organisms lacking a nervous system, we will also consider ecological aspects and discuss the environmental conditions as well as organismic properties that favour the evolution of priming. Throughout this review, we refer to this 'ability to be primed' as 'primability'.

II. A GENERAL CONCEPT OF PRIMING OF STRESS RESPONSES IN ORGANISMS WITHOUT A NERVOUS SYSTEM

We here define priming of organismic responses to stress as the phenomenon whereby a temporally limited environmental (priming) stimulus prepares and modifies the response to a future stress incident (the triggering stimulus); priming acts on the phenotype of individuals, leaving the genetic information provided by the DNA sequence unchanged, but including epigenetic, cellular, hormonal and other phenotypic changes. Priming allows for reversion to the naïve state.

The priming signal occurs first, and the triggering signal is perceived subsequently. They can be of the same or different nature. Both the priming and triggering stimuli are sporadic, transient events, rather than permanent properties of a stressful, extreme habitat. A primable organism is able to perceive the priming stimulus and store information about it. This information can be retrieved to shape the response to a subsequent stressful environmental event, the triggering stimulus.

The priming stimulus may be a stress itself and/or may be indicative of future stress. It prepares the organism for an improved response to a future stress (Karban, 2008). According to this definition, the naturally occurring priming stimuli vary enormously with the various threats that bacteria, fungi and plants may face in their habitats. Table 1 illustrates this variety by listing examples of experimental studies and/or review articles focusing on distinct stimuli that prime the organism for an impending stress.

In many cases, priming and triggering stimuli are of the same nature. For example, pathogen infection primes plant resistance against future pathogen infection, as is the case for SAR (see Section I and Table 1 for references). We call this effect *cis*-priming. By contrast, we refer to *trans*-priming, when priming and triggering stimuli are of different nature. For example, drought primes grasses for frost tolerance (Kreyling *et al.*, 2012), or exposure to high temperatures primes *Escherichia coli* against low oxygen levels (Tagkopoulos, Liu & Tavazoie, 2008).

The terms *cis*- and *trans*-priming refer to the type of priming and triggering stimuli rather than to the type of signalling and mechanism elicited by the stimuli. For example, even though both necrotrophic fungi and chewing herbivorous insects elicit the jasmonate defence pathway (Walling, 2000; Kunkel & Brooks, 2002; Wasternack, 2007; Kliebenstein & Rowe, 2008), we refer to *trans*-priming if one of these stressors primes resistance to the other (e.g. Felton *et al.*, 1999; Rostás, Simon & Hilker, 2003).

A primed response to stress is a response that has been modified by the experience of a preceding priming stimulus. The function of priming is the improvement of future individual fitness with preferably minimal investment of resources. Hence, priming is expected to reduce the costs of responding to a future stressful event. The response to a priming event itself is expected to be associated with some costs since it requires changes in a regulatory network that is kept in a vigilant state until activated by a triggering stress event; the maintenance of a primed state means maintenance of a reversible 'standby' mode (Fig. 1).

During the priming process of a stress response, organisms pass through successive states referred to as naïve (control, C) state, primed (P) state and primed-and-triggered (P + T) state (Fig. 1, Table 2). The primed (P) state is also referred to as 'condition of readiness' (Conrath, Pieterse & Mauch-Mani, 2002; Frost *et al.*, 2008). A full-factorial experimental design studying naïve, primed, primed-and-triggered organisms, and organisms that experienced no priming stimulus but just a stress stimulus, allows for the elucidation of mechanisms, costs and benefits of the priming process. While it is possible under laboratory conditions to work with organisms that are naïve with respect to a distinct priming and triggering

Table 1. The varied nature of naturally occurring priming stimuli that may indicate impending stress and improve stress responses by plants, fungi and bacteria

Priming stimulus (P)	Triggering stress (T)	Organism	Reference
Abiotic stimulus			
High temperature	High temperature	Plants	Charng *et al.* (2007) and Saidi, Finka & Goloubinoff (2011)*
		Fungi	Berry & Gasch (2008)
		Archaea bacteria	Trent (1996)* and Runde *et al.* (2014)
Low temperature	Low temperature	Plants	Thomashow (1999)*, Hincha & Zuther (2014)* and Trischuk *et al.* (2014)
		Fungi	Kandror *et al.* (2004)
		Bacteria	Harrison (1955) and Lee (2004)
Salt	Salt	Plants	Sani *et al.* (2013)
		Fungi	Guan *et al.* (2012)
		Bacteria	Hernandez *et al.* (2012)
Drought	Drought	Plants	Ding *et al.* (2012)
Biotic stimulus			
Phytopathogen	Phytopathogen	Plants	Maleck *et al.* (2000), Conrath *et al.* (2002)* and Attaran *et al.* (2009)
Volatiles of pathogen- infected plants	Phytopathogen	Plants	Shulaev, Silverman & Raskin (1997)
Herbivory	Herbivory	Plants	Frost *et al.* (2008)* and Rasmann *et al.* (2012)
Volatiles of herbivore-infested plants	Herbivory	Plants	Dicke, Agrawal & Bruin (2003)*, Baldwin *et al.* (2006)* and Engelberth *et al.* (2004, 2013)
Insect egg deposition	Herbivory	Plants	Hilker & Meiners (2011)* and Hilker & Fatouros (2015)*
Insect pheromones†	Herbivory	Plants	Helms *et al.* (2013, 2014)
Noise (leaf munching larvae)†	Herbivory	Plants	Appel & Cocroft (2014)
Fungivory†	Fungivory	Fungi	Caballero Ortiz, Trienens & Rohlfs (2013) and Döll *et al.* (2013)

*Review article.
†Suggested as priming stimulus.

stimulus, assessment of the naïve state of organisms obtained from their natural (field) habitats may be difficult, especially when studying long-lived organisms such as trees.

The analysis of priming of microorganisms has long been hard to address experimentally. The advent of microfluidic systems, however, will make the study of priming and phenotypic plasticity in microorganisms much more powerful and will enable the study of individuals rather than microbial populations. These devices allow the study of single cells or hyphae and can be coupled to the monitoring of fluorescent markers that report up- or down-regulation of individual genes (Zaslaver *et al.*, 2006). Microfluidics has already provided a breakthrough in understanding non-heritable antibiotic resistance (Balaban *et al.*, 2004) and is ideally suited to investigate gene expression in individual cells during priming and triggering of stress responses.

III. SPATIAL ASPECTS OF PRIMING: SPREADING THE INFORMATION

Distribution of information about the priming stimulus within a multicellular organism is a crucial aspect of the priming process. In plants and multicellular fungi, the tissue that experiences the priming stimulus may differ from tissue that is exposed to the triggering stressful event. In this case, not only does a temporal gap between the onset of the priming and triggering stimulus need to be bridged, but also a spatial gap. Bridging this spatial gap requires information transfer. Hydraulic, electric and chemical signalling is known in plants (Frantisek, 2013). So far, studies on spatial distribution of information in the context of priming have focussed on chemical signalling (see Section V) that can be conveyed in plants *via* different transfer paths (Table 3).

IV. TEMPORAL ASPECTS OF PRIMING: MEMORY OF THE PRIMED STATE

When a time gap separates the end of the priming stimulus and the beginning of the triggering stimulus, information about the priming experience is stored during the lag phase until the triggering stress starts. The term 'memory' refers here to the storage and maintenance of this information (Bruce *et al.*, 2007; Galis *et al.*, 2009; Jaskiewicz *et al.*, 2011; Ding *et al.*, 2013; Pastor *et al.*, 2013; Walter *et al.*, 2013; Kinoshita & Seki, 2014). In some cases, the priming stimulus may still be present when the triggering stress occurs, relieving the need for active memory. For example, herbivory-induced leaf volatiles can prime the anti-herbivore

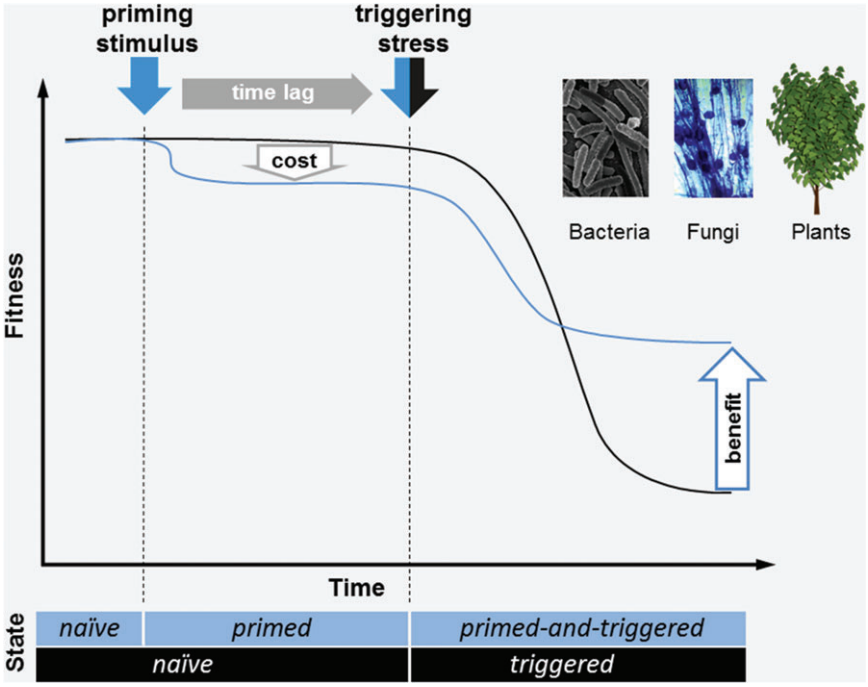

Fig. 1. Generalised scheme of the relationship between fitness and priming of a stress response by bacteria, fungi or plants over time. The priming stimulus acts on a naïve organism and precedes the stress response induced by a triggering stimulus. An organism which has experienced a priming stimulus shows a primed response to the triggering (stressful) stimulus, i.e. a primed stress response. An organism that is exposed to a stress is expected to benefit from priming. In this generalised scenario, establishing and (possibly) maintaining the primed state is expected to have modest costs. If sufficient resources are available, long-lived organisms may recover from a stress experience, and then a fitness loss will no longer be detectable. Priming and triggering stimuli may be different or the same. Photographs: bacteria, *Escherichia coli*, changed after Mattosaurus – Wikimedia Commons; fungi: root colonization by arbuscular mycorrhizal fungi, Trypan blue staining, Matthias Rillig. Plant drawing: *Ulmus*, Elisabeth Eilers.

Table 2. Successive stages through which an organism passes during priming of a stress response

State	Abbreviation	Definition
Naïve (control)	C	The non-induced organismic state prior to the first exposure to a stress or a stress-indicating priming event
Primed	P	The state an organism enters after it has experienced a priming stimulus The primed organism is able to store information on the priming event and to retrieve this information in response to a subsequent stressful event The establishment and maintenance of P is expected to have costs Typically, the primed state persists after the priming stimulus has subsided
Primed and triggered	P + T	The state an organism enters after it has experienced priming and triggering stimuli. The P + T organism shows a primed response to the triggering stressful event. It benefits from having been primed when experiencing the triggering stress as compared to a non-primed (T) organism

defence of as yet non-infested neighbouring plants (see Table 1); these volatiles may still being released from an infested plant when the herbivores start to attack the primed neighbouring plant. However, studies by Karban & Shiojiri (2009) indicate that plants can also remember these volatiles several months after exposure to them. Memory that lasts for several months is also known in trees which are primed for improved anti-herbivore defence by feeding damage in the preceding year (Haukioja, 1990; Nykänen & Koricheva, 2004). Shorter memory that lasts for a couple of days has been shown, for example, in *Arabidopsis thaliana* in response to drought. Plants that had very briefly (2 h) been primed by a dehydration period produced higher transcript levels of drought-inducible genes in response to recurring drought. The 'transcriptional memory' was maintained for 5 days, but lost after 7 days (Ding, Fromm & Avramova, 2012). Hence, the phases of memory of priming stimuli have different durations ranging from long periods to short-term reversal to the naïve state. How long a priming stimulus is kept in memory may depend on several parameters, including

Table 3. Long- and short-distance signalling routes used to distribute informative signals within a plant and between plants

Distance	Route of transmission	References
Long (within a plant)	Vascular tissue, especially phloem for chemical signalling	Ursache, Heo & Helariutta (2014), and references therein
Long (within a plant; between plants)	Gaseous phase	Shulaev *et al.* (1997) and Heil & Adame-Alvarez (2010)
Long/medium (within a plant, between plants)	Mycorrhizal hyphae interconnecting roots	Barto *et al.* (2011) and Babikova *et al.* (2013)
Short (within a plant, cell to cell)	Plasmodesmata (symplast)	Faulkner (2013) and Benitez-Alfonso (2014)
Short (within a plant, cell to cell)	Plasma membrane - Protein kinase-dependent calcium/ROS generation - WASP (wound-activated surface potential charges)	Dubiella *et al.* (2013), Romeis & Herde (2014) and Mousavi *et al.* (2013)

ROS, reactive oxygen species.

e.g. the exposure time to the priming stimulus, the stimulus intensity or the organismic internal state (age, nutritional state) (see Section VIII).

Several studies have investigated whether and how information about a stress experience could be transgenerationally maintained (e.g. Agrawal, 2002; Blödner *et al.*, 2007; Rasmann *et al.*, 2012; Slaughter *et al.*, 2012), and subsequent vigorous debate is available in several reviews (e.g. Jablonka & Raz, 2009; Boyko & Kovalchuk, 2011; Hauser *et al.*, 2011; Holeski, Jander & Agrawal, 2012). Pecinka & Mittelsten-Scheid (2012) argue that unambiguous evidence for stable transgenerational inheritance of exclusively chromatin-controlled stress effects is currently lacking. On the other hand, stress-induced genetic changes due to transposon mobility are clearly heritable. Paszkowski & Grossniklaus (2011) emphasize that it is important to distinguish transgenerational inheritance from a direct parental transfer of information, for example through seed properties. Spoel & Dong (2012) discuss that phytopathogen-promoted epigenetic changes may allow for DNA rearrangements in resistance (R) genes, thereby establishing a genetically fixed transgenerational memory of plant stress responses. Transgenerational stress memory is also known in plants and bacteria in the form of reactivation of transposons. While most transposable elements in the genome are epigenetically silenced, stress can lead to changes in their epigenetic state associated with transcriptional reactivation and transposition (Takeda *et al.*, 1998; Ito *et al.*, 2011). Transposition may be elicited in both plants and bacteria by experience of harsh environmental conditions and typically results in genetic alterations such as DNA rearrangements (Walbot, 1988; Brettell & Dennis, 1991; reviewed in McClintock, 1984; Casacuberta & Gonzales, 2013; Dragosits *et al.*, 2013). However, according to our definition the mechanisms of priming and stress memory act at the phenotypic level of individuals and include epigenetic modifications, changes in gene expression, physiology and metabolism, but not changes of DNA sequence. A number of studies have started to unveil the mechanisms regulating stress priming in different kingdoms, including bacteria, fungi (especially the yeast *Saccharomyces cerevisiae*) and plants, as will be outlined below (see Section V).

While memory consolidation – a prerequisite of long-term memory – has been studied intensively for organisms with nervous systems (McGaugh, 2000; Rosenberg *et al.*, 2014, and references therein), the current state of knowledge of stress memory in organisms without nervous systems only provides hints on the mechanisms involved; so far, generalisation regarding long- and short-term memory in this latter type of organisms is premature.

V. MOLECULAR MECHANISMS OF PRIMING AND MEMORY

Mechanisms of epigenetic memory in plants have been extensively reviewed, most recently by Iwasaki & Paszkowski (2014, and references therein). Chromatin changes including histone modifications and DNA methylation are known to occur in response to stress (e.g. Dowen *et al.*, 2012). Histone modifications as potential memory marks of stress were detected in several studies (e.g. Jaskiewicz *et al.*, 2011; Sani *et al.*, 2013, and references therein). Liu, Fromm & Avramova (2014) distinguish between histone modifications which persist longer ('memory marks') and short-lived, dynamic modifications which are quickly removed again ('chromatin marks'). In addition to histone modifications, stalled RNA polymerase II was proposed as a memory mark following drought stress in *A. thaliana* (Ding *et al.*, 2012). According to Wu & Snyder (2008, p. 1), RNA polymerase II stalling 'could help to provide an active chromatin environment and prepare developmental and stimulus-responsive genes for timely expression'. Furthermore, several studies implicated DNA methylation in transgenerational priming of plant responses to phytopathogens (Boyko *et al.*, 2007; Kathiria *et al.*, 2010; Luna *et al.*, 2012). Priming of anti-herbivore defence of a plant by exposure to odour from a

feeding-damaged plant resulted in enhanced expression of a trypsin inhibitor (*TI*) gene, and this correlated with demethylation of the *TI* promoter. Trypsin inhibitors can impair digestion enzymes of larvae, and thus, larval performance (Ali *et al.*, 2013).

Epigenetic regulation of the bacterial cell cycle has been reviewed by e.g. Collier (2009), however, its role in priming of stress responses is unclear as yet. Instead, there is growing evidence that DNA methylation is a factor controlling initiation of replication and other cellular processes (Donczew, Zakrzewska-Zerwinska & Zawilak-Pawlik, 2014). Several recent reviews provide an overview of the effects of so-called bacterial histone-like proteins - or nucleoid-associated proteins (NAPs) - which are critical for chromosome topology and are involved in the regulation of cell metabolism in response to environmental changes (e.g. Browning, Grainger & Busby, 2010; Dillon & Dorman, 2010; Dorman, 2013).

Parallel transcriptomic (RNA-seq) and epigenomic (ChIP-seq) studies allow analysis of the direct impact of epigenetic modifications on transcript levels of stress-relevant genes in plants (e.g. Sani *et al.*, 2013). Ding *et al.* (2012) define 'transcriptional memory' as a transcriptional response following a recurring stress that differs from the transcriptional response to a primary stress. Pastor *et al.* (2013) review studies which show faster and stronger transcriptional activation of defence-regulating genes in primed and pathogen-stress-triggered (P + T) plants; they also highlight studies showing cellular accumulation of mitogen-activated protein (MAP) kinases that phosphorylate transcription factors in primed (P) plants which enhance transcriptional activation in P + T plants.

Accumulation of inactive transcription (co)factors in plants after experiencing a priming stimulus has previously been suggested to occur, e.g. by Conrath *et al.* (2006) and Bruce *et al.* (2007). The multitude of factors that regulate the dynamics of transcription (co)factor activation in plant stress responses was reviewed by Moore, Loake & Spoel (2011). The *A. thaliana* transcription factor HSF A2 (heat shock factor A2) is required for thermomemory and is highly inducible by a priming stress (Charng *et al.*, 2007); this may involve activation *via* the JUNGBRUNNEN1 transcription factor (Shahnejat-Bushehri, Mueller-Roeber & Balazadeh, 2012). However, the gene regulatory networks through which these transcription factors affect thermomemory remain largely unknown at present.

The role of certain transcription factors in priming future stress responses has also been pointed out in bacteria and fungi. For example, in *Bacillus subtilis*, protection from severe heat stress by prior exposure to a mild heat stress is mediated by a transcription factor (Spx, suppressor of clpP and clpX, clp = caseinolytic protease) (Runde *et al.*, 2014). A key regulator of general stress resistance in *E. coli* and other γ-proteobacteria is σ^S, a subunit of RNA polymerase, which positively controls expression of hundreds of genes. Accumulation, (in)activation and degradation of σ^S are regulated by a highly complex network (Hengge, 2009). The role of σ^S accumulation has also been discussed with respect to cross protection of bacteria from consecutive stresses (Nyström, 2002; Hengge, 2009). Indeed, transcriptome analyses revealed that *E. coli* can adjust its transcriptional profile to future conditions after having experienced a stimulus that is predictive of the future environment (Tagkopoulos *et al.*, 2008). In *S. cerevisiae*, two stress-activated transcription factors (zink finger proteins Msn2p, Msn4p, multicopy suppressor of SNF1 mutation; SNF = sucrose non-fermenting) which regulate many stress-response genes are crucial for future stress protection, but not for survival after the initial stress (Berry & Gasch, 2008). Kelley & Ideker (2009) exposed yeast to mild pre-treatment with hydrogen peroxide as a priming stimulus and subsequently to strong hydrogen peroxide stress. Expression profiling revealed that the transcription factor Mga2 plays a crucial role in the primed stress response since *mga2*Δ mutants lost their primability for an improved response to severe hydrogen peroxide stress; Mga2 is involved in the regulation of ergosterol, fatty acid and zinc metabolic pathways.

At the interface between nucleus and cytosol, the nuclear pore component Nup42p is involved in priming of a stress response in yeast. Exposure of yeast to salt results in increased resistance of daughter cells to hydrogen peroxide and faster gene expression to subsequent stresses. The hydrogen peroxide resistance persists for four to five generations. The faster gene activation in salt-primed and salt-triggered cells (time lag 4 h) is dependent on Nup42p which might mediate the exchange between the nucleus and the cytosol required for priming (Guan *et al.*, 2012).

At the post-transcriptional level, microRNAs (miRNAs) which can cause mRNA degradation or translational inhibition are also involved in modulating priming of plant stress responses (Sunkar, Li & Jagadeeswaran, 2012; Stief *et al.*, 2014*b*). For example, induction of *miR156* by a priming heat stress in *A. thaliana* is important for the maintenance of acquired thermotolerance. The induced miRNA targets and represses transcripts of *SPL* genes, which encode transcription factors regulating plant growth (Stief *et al.*, 2014*a*). A study by Rasmann *et al.* (2012) revealed that small interfering RNAs (siRNAs) play a role in transgenerational priming of anti-herbivore defence of *A. thaliana;* mutants deficient in biogenesis of siRNA (i.e. lacking nuclear RNA polymerases, *nrpd2a*, *nrpd2b*, and dicer-like enzymes, *dcl2*, *dcl3*, *dcl4*, for siRNA production and processing) were no longer transgenerationally primed for anti-herbivore defence. Khraiwesh, Zhu & Zhu (2012) provide an overview of recent studies on the biogenesis and function of miRNA and siRNA in plant responses to biotic and abiotic stress.

With respect to regulation at the post-translational level, it is acknowledged that post-translational modifications of proteins, such as the methylation and acetylation of histones or the phosphorylation of transcription factors, are a prerequisite to some of the mechanisms of information storage discussed above. However, it is unclear whether the experience of a priming stimulus is directly stored in a distinct post-translational modification pattern *per se*,

for example, in the form of an enzyme that has already been differentially expressed but is kept 'on hold' with respect to its activity. Such modification patterns may rely on differential phosphorylation, ADP-ribosylation, ubiquitinylation, sumoylation or other modifications and may not only determine the activity state of a protein, but also its stability and half-life.

At the physiological level, plants can maintain information about experience of a priming stimulus by storage of (inactive) chemical signalling factors. Precursors or conjugated forms of signalling factors such as phytohormones or certain plant metabolites may accumulate in response to a priming stimulus (Bruce *et al.*, 2007; Kaplan *et al.*, 2007; Galis *et al.*, 2009; Dervinis *et al.*, 2010; Pastor *et al.*, 2013; Schulz, Herde & Romeis, 2013; Zeier, 2013; Dethloff *et al.*, 2014). Plant amino acid profiles that have been established in response to a priming stimulus may mediate – in concert with phytohormones – amplification of a defence response to a triggering stress stimulus; for example, the lysine catabolite pipecolic acid can act as key regulator of SAR (Navarova *et al.*, 2012; Zeier, 2013). Furthermore, several other SAR markers have been identified, including e.g. a lipid transfer protein (defective in induced resistance, DIR1), a glycerol-3-phosphate dependent signal, the dicarboxylic acid azelaic acid, the diterpenoid dehydroabietinal, jasmonic acid and methyl salicylate (Dempsey & Klessig, 2012). Accumulation of heat shock proteins is suggested to play a key role in thermopriming of plants and other organisms (Kotak *et al.*, 2007). In fungi, information on exposure to a mild priming stimulus (salt stress) is maintained by production of high catalase levels which are transferred to daughter cells and thus, mediate enhanced hydrogen peroxide resistance upon a subsequent triggering salt stress stimulus (Guan *et al.*, 2012).

While there is growing information about the mechanisms of memory of environmental stimuli in microorganisms and plants, less is known about how the information is 'forgotten' and the primed state is reset to the naïve state. The processes will certainly depend on the half-life of signalling metabolites, enzymes, phytohormones, transcription factors and RNAs involved in establishing and maintaining a primed state. Furthermore, forgetting depends on the stability of the individual epigenome (Paszkowski & Grossniklaus, 2011; Iwasaki & Paszkowski, 2014). The multitude of implicated mechanisms is reflected by the variable duration of the memory phase. It is tempting to speculate that the length of the memory phase may be correlated with the stability of the regulatory molecules involved. However, this may be an oversimplification as it does not take into account auto-regulatory loops that may provide a long-lived memory with short-lived molecules.

VI. PATTERNS OF PRIMED STRESS RESPONSES

The molecular, biochemical and physiological response parameters of a primed and stress-triggered (P + T) organism are expected to differ from those of a non-primed only stress-triggered (T) organism. However, for which subset of parameters and to what extent and in which direction this occurs is still difficult to predict. The highly complex network of signalling, regulating and defensive factors that are involved in a priming process is expected to show up- and down-regulated parameters at the different systems levels outlined above (Section V). However, on the ecological level priming involves performance of the whole organism, and by definition, improved performance is expected in a P + T organism when compared to a T organism.

Generally, the primed stress response by a P + T organism is expected to be modified compared to the naïve stress response by a T organism. Here, we suggest possible scenarios for these primed stress responses (Fig. 2). The scenarios A–D shown in Fig. 2 are not exclusive. A faster (scenario A) or an earlier primed response to a triggering stress stimulus (scenario B) may also end up in a stronger response that has been produced faster or initiated earlier. Furthermore, the detected scenario will depend on the parameter that is measured. The kinetics of different response parameters

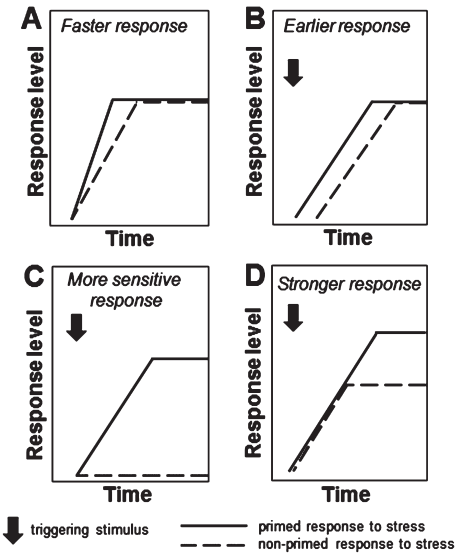

Fig. 2. Schematic response patterns of primed and non-primed organisms to a triggering stress stimulus. The velocity, onset, sensitivity and strength of the organismic response to a stressful triggering stimulus (arrow) may be shaped by the organism's experience of a preceding priming stimulus. The response parameters may vary and include e.g. expression of genes involved in the regulatory network of a primed stress response, phytohormone levels, defensive compounds. (A) The primed organism responds with faster kinetics than a non-primed one to the triggering stress. (B) The primed organism responds earlier than the non-primed one or (C) responds to a lower dose of stress to which a non-primed organism does not yet respond. (D) The primed organism responds to a triggering stress with a higher amplitude than the non-primed one. These patterns are expected for responses that e.g. directly act against a biotic stressor, whereas the complex network of signalling and regulating factors may show up- and down-regulation, activation and retardation.

measured in a P + T organism may match different scenarios. Hence, several scenarios may be detected in a single organism depending on the parameters considered.

In Fig. 2A, the primed response to a triggering stress stimulus shows faster kinetics than the non-primed one. For instance, the biosynthesis rate of defensive compounds is higher, and effective levels are reached faster. *Arabidopsis thaliana* plants primed by the non-protein amino acid β-aminobutyric acid (BABA) display faster stomatal closure when exposed to low humidity than non-primed plants; the BABA-primed plants show enhanced tolerance to drought stress (Jakab *et al.*, 2005).

In Fig. 2B, the primed response to a triggering stress has the same or similar kinetics as the non-primed one, but is initiated earlier than the non-primed response to a stress. Thus, this scenario also leads to a quicker response, but the mechanism is different from scenario A. For instance, the expression of a gene encoding an herbivory-inducible cystatin-like proteinase inhibitor in maize plants was induced earlier in plants that have been 'warned' and primed by the wound-induced odour of neighbouring plants than in non-primed plants; the primed plants showed enhanced anti-herbivore defence (Ton *et al.*, 2006).

In Fig. 2C, the primed stress response is more sensitive than the non-primed one, such that a response is triggered at a lower stress intensity after priming. As an example, defence-related PR genes in BABA-pre-treated *Arabidopsis* plants are induced by lower salt concentrations compared to control plants (Jakab *et al.*, 2005).

In Fig. 2D, the primed stress response is stronger, e.g. the amplitude of a defensive action is increased. For example, the expression of *HKT1* encoding a high-affinity K^+ transporter protein is more strongly induced in primed plants triggered with a second salt stress than in control plants; P + T plants show reduced sodium accumulation and increased salt stress tolerance (Sani *et al.*, 2013).

We emphasize that Fig. 2 only shows examples of some scenarios, and that others are possible. For instance, we cannot rule out that the primed stress response results in the biosynthesis of novel compounds that would not have been produced in response to stress in the absence of a prior priming stimulus. Furthermore, organisms which experience a mild priming stress may show a weaker response to a recurring, mild triggering stress and thus save energy that is important for growth and reproduction (e.g. Gagliano *et al.*, 2014). In this case, the organism also benefits from prior experience of a stress and the down-tuning of its response to recurring stress.

VII. PRIMING AND OTHER TERMS: ADAPTATION, ACCLIMATION, INDUCTION, ACQUIRED RESISTANCE AND CROSS PROTECTION

The definition of priming of organismic stress responses as outlined here shows parallels and differences to several other terms used to describe how organisms cope with environmental stress.

(1) Priming and adaptation

Priming of plant responses to environmental stress differs from adaptation to environmental stimuli, since adaptation is used to describe selection of adaptive genotypic changes which result in phenotypes that cope successfully with their environmental conditions (e.g. Stapley *et al.*, 2010; Elmer & Meyer, 2011). By contrast, priming refers to stimuli which affect the phenotype of individuals, but of course the mechanism of priming can be a result of the adaptation of species to their ecological niche (van Kleunen & Fischer, 2005).

(2) Priming and acclimation

Both priming and acclimation are terms used to describe the effects of external stimuli on the phenotype of individuals. Woods & Harrison (2002) define acclimation as a response that results from chronic exposure of an individual to a new environment. According to this definition, acclimation may also be considered as adjustment to a new environment. Also Smith & Dukes (2013) define acclimation as an organismic adjustment to a sustained environmental change. The changes occurring upon a switch from one environment to another may be beneficial for an acclimated organism, especially when the change of environmental stress is increased gradually from mild to severe stress, as known for example in cold acclimation (Sage, 1994; Thomashow, 1999; Woods & Harrison, 2002). Mittler (2006) emphasized the need for further studies on plant acclimation to a combination of different stressors, e.g. to drought and heat stress since these stressors frequently co-occur. In contrast to studies on acclimation to simultaneously occurring stressors (e.g. Prasch & Sonnewald, 2013, and references therein), other studies on plant acclimation investigate the responses to discrete, sequentially occurring environmental stimuli (e.g. Wu *et al.*, 2013; Cerasoli *et al.*, 2014). Hence, there are parallels between priming and acclimation with respect to (*i*) the focus on phenotypic responses to environmental changes and (*ii*) the expectation that the change of organismic responses due to acclimation and priming is beneficial. However, priming as defined above always refers to subsequently occurring, occasional stress events, whereas acclimation is also used to describe responses to co-occurring, sustained environmental properties of a new habitat.

(3) Priming and induction

In plants and microorganisms, priming generally requires induction of responses, but also requires at least two sequential environmental events, i.e. the priming and the triggering events, whereas a single stimulus is sufficient for induction. Furthermore, priming differs from induction in that information of the primed state is stored after the stimulus and modifies the response to a further

stimulus. By contrast, induced responses cease when the stress ceases. In this case, a second stress will induce the same stress responses as a preceding first stress. Hence, priming differs from induction by the dual-event condition and by the expected memory effect that maintains the primed state and as a consequence modifies the response to the second stress stimulus with benefits for the target organism.

(4) Priming and acquired resistance

With the definition given above, priming of plant responses to stress applies to the term 'systemic acquired resistance' (SAR) (Sticher *et al.*, 1997; Métraux *et al.*, 2002; Conrath, 2006). SAR refers to systemically distributed increased resistance against pathogen infection after a preceding infection and requires an entire set of mobile signals which inform as yet uninfected parts of an infected plant about the imminent danger of infection (Dempsey & Klessig, 2012; Kachroo & Robin, 2013; Zeier, 2013).

Since we defined the priming stimulus above a stimulus that is either stressful in itself and/or predictive of future stress, induced systemic resistance (ISR) (Van Loon, Bakker & Pieterse, 1998; De Vleesschauwer & Höfte, 2009; Pieterse *et al.*, 2014) which refers to increased resistance against aboveground pathogen infection after exposure to beneficial soil bacteria does not fully meet our priming definition. However, since ISR results in fitness benefits, it also leads – in a broader definition – to priming of a future response to infection (Pieterse *et al.*, 2014).

(5) *Trans*-priming and cross protection, cross talk of plant defence signalling and cross stress memory

Cross protection is a term mainly used in microbiology and refers to protection of an organism from an environmental stress by prior exposure to another (stressful) stimulus (fungi: Dhar *et al.*, 2013, and references therein; bacteria: Runde *et al.*, 2014, and references therein). Hence, when used in this way, cross protection meets the definition of *trans*-priming (see Section II).

The term 'cross talk of plant defence signalling' is used when signalling pathways intersect or when two inductive processes occur simultaneously and exert synergistic or antagonistic effects (Koornneef & Pieterse, 2008). For example, when phytopathogen infection of plants induces salicylic acid (SA)-mediated signalling and simultaneous or subsequent attack by herbivorous larvae induces jasmonic acid (JA)-mediated defence signalling, the SA–JA cross talk may result in reciprocal antagonism of the signalling pathways (e.g. Thaler, Humphrey & Whiteman, 2012). The cross effects of induction may have positive, negative or no detectable effects on the performance/fitness of an organism (e.g. Rostás *et al.*, 2003). By contrast, *trans*-priming by definition always has positive fitness effects (see Section II).

Cross stress memory, a term used by e.g. Walter *et al.* (2013), refers to the memory effects occurring in *trans*-priming. For example, memory of a stress that induces oxidative stress in an organism may prime the response to another stressor that also results in oxidative stress.

VIII. EVOLUTIONARY ECOLOGY ASPECTS OF PRIMING AND MEMORY OF STRESS RESPONSES

Since organisms are exposed to numerous stressors, the question arises as to which conditions render it advantageous to be 'always prepared', i.e. to mount constitutive stress responses rather than being primed upon an environmental event or showing induced stress responses. Constitutive adaptations to stress are expected to be maintained and hardwired into the genome of a species if this stress is a constant part of the environment and if the adaptation allows the organisms to multiply within an already populated niche of an ecosystem at comparatively low or no disadvantage.

Inducibility of stress responses, i.e. the ability to respond to stress only when it occurs, is a trait that is considered as a basic cost-saving strategy since the organism invests resources only on demand (Karban & Baldwin, 1997; Zangerl, 2003). If metabolic and energy costs of a given stress response are severe, then the responses will not be constitutively expressed at the level needed for efficient defence to avoid a fitness deficit under non-stress conditions (e.g. Baldwin, 1998; Dicke & Hilker, 2003). Furthermore, on-demand responses to a biotic aggressor might be a strategy to impair adaptation of the aggressor. This 'moving target' strategy has been suggested for plants which show induced responses to stress; the phenotypic variability in itself has been claimed to be defensive (Adler & Karban, 1994). The idea that high variability of phenotypes is adaptive is also the basic idea of 'bet hedging' (e.g. Beaumont *et al.*, 2009, and references therein). However, bet hedging refers to the expression of phenotypes in response to environmental variability, but does not refer to a distinct adaptive phenotypic change in response to a distinct environmental stimulus. Bet hedging is a risk-spreading strategy, and thus adaptive in randomly and unpredictably changing environments where it might become useful to express some phenotypes that can survive an unforeseen event or can produce relatively many offspring even in variable conditions (Philippi & Seger, 1989; Veening, Smits & Kuipers, 2008; Beaumont *et al.*, 2009; Childs, Metcalf & Rees, 2010). In contrast to bet hedging, neither inducible stress responses nor primable responses are traits that spread risk across a population, but rather traits that are expected to directly improve an individual's stress response.

Mounting inducible stress responses may delay effective defence. Cipollini, Purrington & Bergelson (2003) considered this time lag a potential cost of induced plant stress responses. However, if an organism is prepared (primed) for the upcoming stress, it might respond faster or to a lower level of stress (Fig. 2). While numerous studies considered the benefit to an organism from being primed for a stress response (e.g. Baldwin *et al.*, 2006; Bruce *et al.*, 2007; Dhar *et al.*, 2013;

Pastor *et al.*, 2013; Walter *et al.*, 2013; Runde *et al.*, 2014), the question of maintenance costs of the primed state – i.e. storage of information on a priming stimulus – has hardly been studied. However, Van Hulten *et al.* (2006) showed that priming of *A. thaliana* with (low dose) β-aminobutyric acid (BABA) for improved resistance to phytopathogens had no negative impact on seed production, although a minor reduction in plant growth rate was detected. The benefit of a primed stress response that outweighs the costs of processing and memory of a priming experience is expected to favour evolution of primability of organismic stress responses.

Whether priming of organismic stress responses pays off will depend (*i*) on the organism's internal state when perceiving priming and triggering stimuli, (*ii*) on its life strategy, and (*iii*) on the environmental stimuli, including the reliability of priming stimuli and their specificity with respect to the triggering stress, the predictability of stress as well as the community in which the organism is living. In the following, we discuss the factors that may determine the costs and benefits of priming and thus, the evolution of an investment in priming and memory.

(1) Age of the organism

The age at which an organism experiences a priming stimulus may determine how much it invests in priming of stress responses. Young plants may benefit from a long-term stress memory if it helps to minimize costs of future inducible stress responses and tailor resources for growth and defence (Coley, Bryant & Chapin, 1985; Herms & Mattson, 1992; Baldwin, 2001; Huot *et al.*, 2014). For example, priming of young *A. thaliana* seedlings with a mild salt stress improved the response of adult plants to salt and drought stress (Sani *et al.*, 2013). By contrast, annual adult plants shortly before flowering are expected to invest in short-term memory of a past stress to be prepared for upcoming future stress and successful reproduction. However, so far, no information is available on possible mechanistic differences between short- and long-term memory and potentially different costs of these types of memory. An organism that expends the costs of long-term memory needs to save sufficient resources for growth and actual defence.

In bacteria, stationary, growth-arrested cells that are gradually losing their ability to proliferate are considered ageing, senescing cells (Nyström, 2002). When these ageing cells have experienced starvation as a priming stimulus, they show increased resistance to several other secondary (triggering) stressors. Hence, their increased resistance trades off for rapid growth and multiplication (Matin, 1991).

(2) Nutritional state of the organism and resource availability

An organism that is supplied with ample energy resources has a higher capacity to invest in priming and memory of stress responses than an organism living at a nutritional minimum. As has been shown for both constitutive and induced plant resistance (Cipollini *et al.*, 2003, and references therein), the costs of priming for stress resistance may also vary with the availability of energy resources. Hence, availability of light and nutrients as well as the presence of competitors, which may limit these resources, will shape the costs and benefits of priming. In bacteria, as mentioned above, depletion of nutrients as a priming stimulus may prime a response to other subsequent stresses. For example, a much higher percentage of *E. coli* cells survive heat stress when they have been primed by starvation prior to heat exposure (Matin, 1991).

(3) Life strategy of the organism

Costs and benefits of priming of stress responses may depend on the life strategy of an organism. Adaptation of life-history traits to environmental conditions is often considered with respect to the classical conceptual frame of *r*- and *K*-selection (Dobzhansky, 1950; MacArthur & Wilson, 1967; Pianka, 1970). According to the *r*/*K* concept, genotypic adaptation to unpredictable, recurring stressful events is reflected by the *r* life strategy (rapid development, high reproduction rate), while genotypic adaptation to predictable environmental conditions is reflected by the *K* life strategy (slow development, relatively low number of offspring). The *r*/*K* categories are relative categories, i.e. the assignment '*r*-selected' or '*K*-selected' depends on the range of species that are compared and sorted within an *r*/*K* continuum. When considering *r* and *K* life strategies as relative categories, even different populations of the same species may vary along an *r*/*K* continuum, thus showing *r*/*K* strategy plasticity. Although this concept does not take into account all parameters that might act as selective pressure on life-history traits (Stearns, 1977, 2000), it is useful when trying to link life span and reproductive rates with the predictability of environmental conditions. Hence, it is useful for ecological classification. Both *r*- and *K*-selected organisms are expected to benefit from being primed for future stress. However, these organisms might differ in their primability with respect to stressor specificity and the benefits from short- and long-term memory, as will be argued below.

For *r*-selected organisms living in variable and unpredictable environments, the variability of the environment in itself may render occurrence of stress likely. Therefore, priming of stress responses may be expected to pay off even if the priming effect shows limited stressor specificity because the type of stress is not predictable in the variable habitat of an *r*-strategist. For *K*-selected organisms, priming of stress responses may be expected to be beneficial if the priming effect shows high stress specificity in a highly predictable environment.

In addition to possible differences with respect to stressor specificity in priming of *r*- and *K*-strategists, these types of organisms might also differ with respect to their benefits from short- and long-term memory. Organisms with a short life span – as typical for *r*-strategists – are expected to benefit

from remembering a specific past stress when the probability is high that this stress re-occurs soon. Hence, they are expected to invest especially in short-term memory. By contrast, long-lived organisms are expected to benefit from both short- and long-term memory of past stress.

(4) Reliability and specificity of the priming stimulus and predictability of stress

Stress events with low probability of re-occurrence have only low predictive value for future stress. It might be too costly to store information about them. If experience of a warning (priming) stimulus does not indicate future stress with a minimum degree of reliability, memory of this event would be useless (Karban, 2008). Hence, costs of priming and memory are expected to depend also on the reliability with which the priming event indicates a future stressful event. Priming is expected to evolve where a preceding stress event has a predictive power to estimate the probability and/or the intensity of a subsequent stress event. Furthermore, the type of priming stimulus may predict a specific upcoming stress and thus, act stressor specifically (typically *cis*-priming). However, a priming stimulus may also be predictive of other stresses or indicate stress in general, and thereby acting stressor unspecifically (typically *trans*-priming).

(5) Community ecology and priming

The entire species community to which an organism belongs may influence whether investment in priming of stress responses pays off. Bacteria, fungi and plants interact with other members of the resident assemblage in terms of competition or facilitation at the same trophic level. It will therefore also be highly relevant to study the effects of priming on the relative success or fitness of organisms in the community context. Microbial communities, generally useful for testing ecological principles (Jessup *et al.*, 2004), could be employed as model systems to explore priming of stress responses at the level of assemblage (Rillig *et al.*, 2015). This could ultimately lead to the propagation of the concept of priming to the community level, i.e. to the question of whether communities can be primed (which could mean priming responses of individual species or changes in species composition, or a combination), such that primed communities are better able to cope with future stresses in terms of productivity.

IX. CONCLUSIONS

(1) The term 'priming' as defined here describes the ability of organisms to respond to transient environmental (priming) stimuli in a way that prepares them for an improved response to future stress. Priming of stress responses has been shown for bacteria, fungi and plants. We define the ability to be primed for improved stress responses as primability and refer to *cis*-priming, if priming stimuli and subsequent (triggering) stress stimuli are the same, and to *trans*-priming, if they are different.

(2) Common, ancestral mechanisms of priming of stress responses in bacteria, fungi and plants are expected. The mechanisms by which an organism stores information about a priming environmental stimulus require complex networks that regulate transcription and translation of primable, stress responsive genes and the (de)activation of the numerous factors which shape functioning of the networks (e.g. transcription factors, small interfering RNAs, hormones, enzymes, metabolites). The reset of an organism from a primed to a naïve (inexperienced) state, i.e. the 'forgetting' of the priming event, may depend on the life time of the primed cellular marks that are called up on exposure to stress.

(3) The primability of an organism, the sensitivity to *cis*- and *trans*-priming and the benefits from short- and long-term memory of a priming stimulus are suggested to be shaped by the intrinsic properties of the organism (its age, nutritional state, life strategy) as well as by the environment (the presence of intra- and interspecific competitors). Among other factors, environmental conditions ranging from unpredictable variability to predictable consistency may affect the evolution of primability.

(4) Future studies will have to elucidate the ecological conditions and molecular and biochemical mechanisms that regulate and determine how long information about a priming stimulus is stored. Further knowledge will be necessary to gain insight into how bacteria, fungi and plants can 'forget' an event and are 'reset' to an inexperienced (naïve) state.

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