Understanding abiotic plant stress and its impact on plant responses and plant adaptation is a very contemporary issue in plant science, recently driven by the mounting concerns about food production under climate change. The definition of stress and how it interfaces with the plant is therefore crucial for tracing cause, effect, and adaptation in their true physiological and genomic background. Correct definitions are not just a matter of formality but an essential road map for research towards understanding the phenomenon and its consequences towards designing better research and deriving correct conclusions. The current literature indicates that definitions are not clear and not under consensus either because of lacking knowledge or of the current debate. Jacob Levitt (1972), who was the dean of plant stress physiology in his time, outlined the seminal working definitions of abiotic stress and stress resistance, which were in accordance with the known stress physiology at that time and which were accepted by his peers. This article revisits, extends, clarifies and updates his definitions in tune with modern plant science while discussing how the updated definitions should enhance contemporary plant stress research.

Plant abiotic stress is a relatively young scientific discipline within general plant physiology. Governmental and institutional concerns about climate change and global warming have pushed this discipline to the forefront of crop improvement research. A young generation of upcoming biologists is attracted by the biology and sometimes by the funding opportunities in this discipline. The result is a recent exponential rise in research on how plants respond and adapt to abiotic stress.

The most important aspect of plant stress research is the understanding of how stress interfaces with the plant and how plants respond to this interaction through their constitutive or adaptive traits. The resultant understanding not only determines how research resolves gene expressions and gene networking but also its consequence towards genetic improvement of stress resistance.

When recent publications on plant stress research are viewed it emerges that there is no consensus or even a common regard for plant stress terminology and definitions. A brief scan of research reports on abiotic stress resistance will reveal the following examples of used definitions: ‘plant defense’, ‘stress homeostasis’, ‘plant resilience’, ‘stress protection’, and so on. Do all these definitions describe ‘stress resistance’ or do they describe different physiological or genetic phenomena? For the student or the initiated researcher this would certainly cause confusion.

Correct definitions are not just a matter of formality but rather an essential road map for research. The following discussion should clarify that definitions might determine success or failure in plant stress research and that lack of consensus of definitions and their proper use in research can undermine attempts to repeat the specific research and its results. Fuzzy definitions might even bias research protocols and data interpretation. A major foundation of scientific investigation is the requirement that any research report must be sufficiently detailed and accurate so that it allows the repeating of results by peers.

Jacob Levitt (1972), who was the dean of plant stress physiology in his time, outlined the seminal working definitions of abiotic stress and stress resistance, which were in accordance with the known stress physiology at that time and which were accepted by his contemporaries. This viewpoint revisits, extends, clarifies, and updates his definitions in tune with modern plant science while discussing how the updated definitions should enhance contemporary plant stress research.

A plant subjected to stress will develop a strain. This can be explained by the analogy from civil engineering that Levitt used, and which is broadened and further developed here in tune with modern knowledge. Consider a steel beam positioned on columns at its two edges. When a heavy load (stress) is placed on the center of the beam, the latter will develop a strain. The strain can be plastic (irreversible) or elastic (reversible). The magnitude of the strain is proportional to the magnitude (weight) of the stress and its position on the beam. In other physical analogies and in biology, the magnitude of strain can also be a function of the duration of stress. The strain (not the stress) can be phenotyped by a slight bending of the beam. This bending strain can be associated with certain physical or even chemical changes in the material of which the beam is constructed and if this stress is severe enough the beam will break directly under the strain (rather than the...
stress). If the load is removed, the elastic strain will be relieved and the beam will recover its normal phenotype. In the case of a plastic strain, the beam will remain bent. The strain tolerance of the beam is a direct function of its form and material, which are the foundations of beam engineering. If we place a column under the center of the beam it will avoid being strained under stress. It will not avoid the stress since the load is still applied to the beam. If this beam is placed where there is no load at all, it escapes stress and strain altogether.

The above extended analogy deals only with the constitutive properties of beam strain tolerance. However, a biological system allows for adaptive strain tolerance, which develops in response to a signal created by the strain (not the stress). This is where biology departs from the engineering analogy. To elaborate the case for plant biology, a few examples are discussed here.

In the case of waterlogging stress for example, an important strain is plant anoxia. Certain plants can avoid the anoxia strain to some extent by obtaining oxygen through specific constitutive or adaptive modes, or tolerate anoxia strain by reverting to anaerobic metabolism. The experimental waterlogging protocol should consider the specific strain response and its possible signal(s) in a complete physiological and genomic dissection. For example, it must be clear that if in the specific case the plant is constitutively strain avoidant due to its well-developed aerenchyma, it would be trifling to search for anoxia strain-responsive genes as an explanation of this case resistance under a waterlogging stress treatment.

Salinity stress, for example, results in two primary strains, namely osmotic and toxic strains, each of which can result in secondary specific strains (e.g. pH modification etc.). The osmotic strain might signal osmotic adjustment (OA) while the excess sodium strain might signal the expression of certain membrane transporter genes. The investigation of salinity tolerance must therefore target the different strains and their possible signaling via dedicated experimental stress dissection protocols.

The primary strain under drought stress is water loss from cells, namely dehydration. The derived cellular signals are osmotic, turgor related, and abscisic acid (ABA). Plants can avoid dehydration strain by maintaining a high plant water status under the load of stress by way of constitutively deep roots, for example. Alternatively plants can avoid dehydration strain by OA driven by an initial dehydration strain signaling. If dehydration strain cannot be avoided, then dehydration strain tolerance becomes important and it can be driven by constitutive or adaptive plant traits. The signaling of strain avoidance or tolerance can be linked to the rate of strain development and its severity (Jones and Rawson, 1979). When crop production is the issue it must be clear that certain plant adaptations to dehydration strain can be positive or negative in that respect. What evolution engineered into plants under stress might be important for survival but sometimes detrimental to productivity. For example, shoot or soil drying can signal ABA production. ABA regulates specific plant responses that may help survival but in most cases might retard productivity (Blum, 2015).

Strain and not stress constitutes a signal towards adaptation or apoptosis and it is crucial to pinpoint strain involvement when a plant is stressed experimentally. The signal, as well as its receptors, constitutes one of the very contemporary issues in molecular genetics of plant stress. The schematic Figure in Prasch and Sonnewald (2015) presents very well the various signals driven by different stresses but then one should also note the void in that Figure between ‘stresses’ and ‘signals’, which demonstrates the much needed recognition of strains as a crucial primary intermediate between stress and signaling.

OA is a major cellular strain adaptive attribute under drought, salinity, and freezing. Issues of stress, strain, signaling, and adaptation present themselves very well for OA, as an example. The dehydrating cell enters a state of osmotic strain due to cellular solute concentration. This strain constitutes a signal. At the same time turgor pressure is reduced, also creating a possible signal. The first response to the signal is very likely reduced cell expansion (growth).

In a strain-responsive genotype this signal (osmotic or turgor derived) might, for example, upregulate potassium transporter gene(s) thus increasing cellular K⁺ influx and subsequently OA. On the other hand, the signal can induce the expression of strain-responsive genes such as P5CR, TPS1, or betA. These genes regulate the accumulation of de novo produced (‘compatible’) organic solutes that can have a role in protecting cellular proteins or organelles under dehydration strain but in many cases they do not contribute towards OA because of their low cellular concentration (Shabala and Shabala, 2011). This is not a ‘drought stress protection’ mechanism as it is coined in some reports. To protect from drought stress, we need irrigation. This is a cellular dehydration strain tolerance mechanism while K⁺ accumulation towards OA is a cellular dehydration strain avoidance mechanism.

At the same time, the initial dehydration strain and/or the reduced turgor pressure strain signal a reduction in cell expansion and growth while photosynthesis is not yet reduced. The result is the shift of excess soluble carbohydrates from growth to OA. Thus, if OA is investigated (as an example), one might consider the three following important strain signals and responses: (1) enhanced cellular potassium (or other ions) uptake, which drives OA as a cellular dehydration avoidance adaptation; (2) de novo production of organic solutes which, as protectants, may enhance cellular tolerance to dehydration strain; and (3) excess soluble carbohydrates mostly resulting from arrested cell expansion, which can promote cellular dehydration avoidance or strain tolerance. In experimental terms all signals can be proportional to the level of the specific strain which in turn can be proportional to the rate and duration of dehydration stress. At the same time different strains can emit the same signal and downstream consequence, such as the accumulation of carbohydrates when growth is also retarded by cold.

The molecular genetics of plant stress deals extensively with gene expression under stress with the attempt at gene discovery towards enhancing stress resistance or towards resolving gene networks under stress. Genes are not stress-responsive but rather strain-responsive. Furthermore, a singular unique stress could elicit several different strains and different stresses could elicit the same strain, with the respective consequences towards gene expression (Fig. 1, Fig. 2). This has a huge impact on the implication or the significance of a given
‘stress-responsive’ gene and the probability of repeating the same gene expression and its function in another experiment.

For example, ABA-independent dehydration-responsive-element-binding (DREB) genes are important plant transcription factors that were reported to regulate ‘tolerance to multiple stresses’ across different experiments or within one experiment (Lata and Prasad, 2011). For example, overexpressing DREB2 in transgenic plants induced drought, salt, cold, and heat tolerance as well as disease resistance (Agarwal et al., 2006). At the physiological level a ‘universal stress resistance gene, is intellectually implausible, at least for now. Therefore, the role of DREB2 must be defined in terms of the strains and signals involved in its expression and downstream function with respect to any of these four or five stresses. These include dehydration, turgor loss, physical or chemical cell membrane disruption, osmotic concentration, Na⁺ or Cl⁻ toxicity, alkalinity, cellular ice formation, cellular freeze-thaw cycle, supercooling, chronic or acute heat strains with or without acclimation, arrested growth, and so on. Otherwise, the function of DREB2 towards resistance as determined in one study might not be possible to repeat in another.

In conclusion (Fig. 1, Fig. 2), correct consensus definitions of stress, strain, signaling, and adaptation and the understanding of their physiological and genomic consequences and interactions are the foundation of plant stress science without which research cannot achieve real progress.
and cannot be repeated or verified by peers. The terms ‘stress resistance’ or ‘stress tolerance’ are entrenched in the literature and they can be used in generalizations but when research is planned, data analyzed, and conclusions derived the correct definitions and implications of how exactly stress interfaces with the plant towards adaptation should be carefully considered.

Received 2 September 2015; Revised 26 October 2015; Accepted 27 October 2015


**Key words:** Abscisic acid, drought, gene expression, Jacob Levitt, osmotic adjustment, research, resistance, salinity, tolerance, waterlogging.

---

**References**


