Chapter 4: Stress Physiology and Phenotypic Plasticity

1. Osmotic Stress (Drought/Salinity): Biosynthesis of Compatible Solutes (Proline, Glycine Betaine) and LEA Proteins

Osmotic stress, mainly caused by drought (water deficiency) and salinity (excessive salt concentrations, primarily NaCl), induces a decrease in the water potential (Ψw) of the surrounding environment. This disrupts water absorption by the roots and causes cellular wilting.

To adapt and maintain cellular functioning (osmoregulation or osmotic adjustment), plants implement two major biochemical mechanisms: the biosynthesis of compatible solutes and the synthesis of protective proteins (LEA proteins).

1.1. Biosynthesis of Compatible Solutes (Osmoprotectants)

Compatible solutes are low-molecular-weight organic molecules, either neutral or zwitterionic, that are non-toxic to cellular structures even at high concentrations. They accumulate in the cytosol and organelles to:

- Lower the osmotic potential (Ψs) of the cytosol and maintain cell turgor. By accumulating inside the cell, they increase the internal solute concentration, allowing the cell to retain water and absorb additional water from the soil through osmosis.

- Rehydrate and stabilize the structure of macromolecules (enzymes and membranes). They form a "water shield" around enzymes, proteins, and membranes, preventing them from becoming deformed or damaged under water-deficient or saline conditions.

- Scavenge reactive oxygen species (ROS) generated by stress. During stress conditions (drought, salinity, or high temperatures), normal cellular functioning is disrupted, leading to the overproduction of reactive oxygen species (ROS), such as hydrogen peroxide (H_2O_2) and free radicals. These highly unstable and toxic ROS attack and damage cellular membranes, proteins, and DNA, causing oxidative stress.

Compatible solutes (such as proline, mannitol, and glycine betaine) act as direct and indirect antioxidants:

- Direct scavenging: They react directly with free radicals to neutralize them and render them harmless before they can damage cellular structures.

- Protection of antioxidant enzymes: They stabilize the structure of plant defense enzymes (catalase, superoxide dismutase, and peroxidase), allowing them to continue efficiently eliminating ROS.

a. Proline

Proline is a multifunctional amino acid that accumulates in response to dehydration. It is synthesized from L-glutamate in the cytosol or chloroplasts through two key enzymatic steps:

- First step (P5CS) — Rate-limiting step: The enzyme P5CS (Δ^1-pyrroline-5-carboxylate synthetase) catalyzes the conversion of L-glutamate into a cyclic intermediate, P5C. This is the key regulatory step in the process. Under stress conditions, P5CS gene expression is strongly upregulated to initiate massive proline production. This step requires energy (ATP) and reducing power (NADPH).

- Second step (P5CR): The enzyme P5CR (P5C reductase) then converts P5C directly into L-proline through a reduction reaction. This step consumes an additional reducing equivalent in the form of NADPH or NADH.

Degradation pathway (during rehydration): In the mitochondria, proline is catabolized by ProDH (proline dehydrogenase) and P5CDH (P5C dehydrogenase), providing ATP and carbon to the cell after stress conditions have been alleviated.

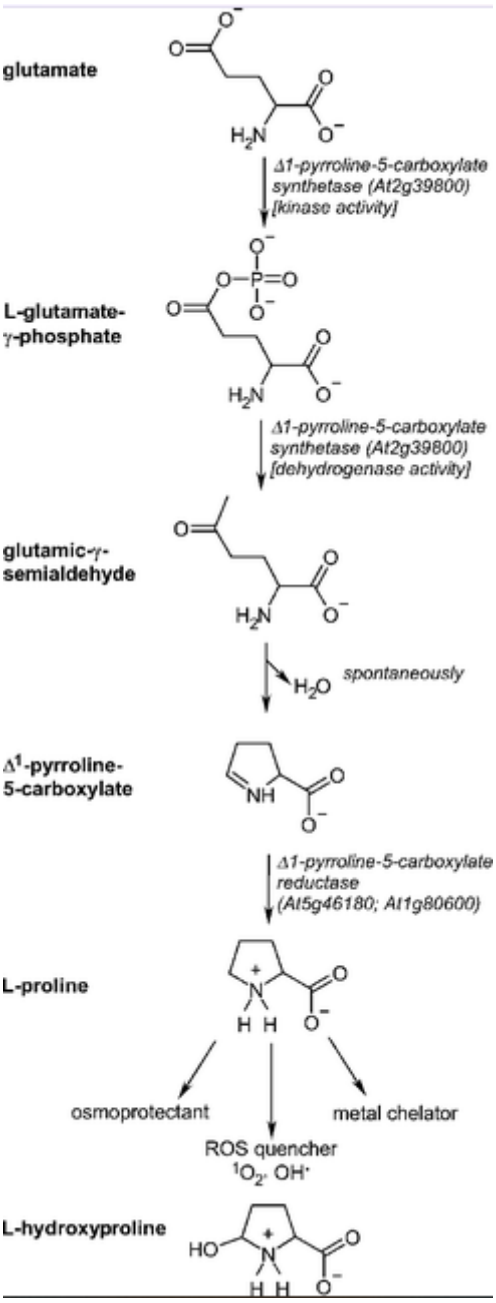


Fig. 40. Synthesis of proline and the enzymes involved.

Protective role

- Osmoregulation: They accumulate in the cytosol without interfering with cellular metabolism, thereby reducing the cell's osmotic potential. This allows the cell to absorb water through osmosis and maintain its turgor under drought or saline stress.

- Neutralization of free radicals (•OH): The hydroxyl radical (•OH) is the most destructive reactive oxygen species (ROS). Osmoprotectants (such as mannitol and proline) directly

scavenge this radical by donating electrons, thereby preventing lipid peroxidation of cellular membranes.

- Stabilization of the PSII complex: Under stress conditions, the reaction center of Photosystem II (PSII) and its key proteins (such as the D1 protein) are at risk of denaturation. Osmoprotectants preserve the supramolecular assembly of PSII and its water-oxidizing complex, thereby maintaining photosynthetic activity.

- Maintenance of the $NAD(P)^+$/NAD(P)H ratio: By protecting the photosynthetic and respiratory electron transport chains, they prevent excessive accumulation of reduced NAD(P)H. This maintains a sufficient supply of electron acceptors ($NAD(P)^+$), helping to prevent ROS overproduction and preserve cellular redox balance.

b. Glycine Betaine (GB)

Glycine betaine is a quaternary ammonium compound that accumulates in many stress-tolerant plant species.

Stress-tolerant plants are organisms capable of withstanding environmental stress (abiotic or biotic) while maintaining a sufficient level of physiological functioning to ensure their survival, growth, and reproduction, without necessarily avoiding the stress. Examples include halophytes, grasses (Poaceae), and plants belonging to the Chenopodiaceae family.

Glycine betaine is synthesized in the chloroplast stroma (the internal fluid where the Calvin cycle, or light-independent phase of photosynthesis, takes place), where it directly protects the photosynthetic machinery.

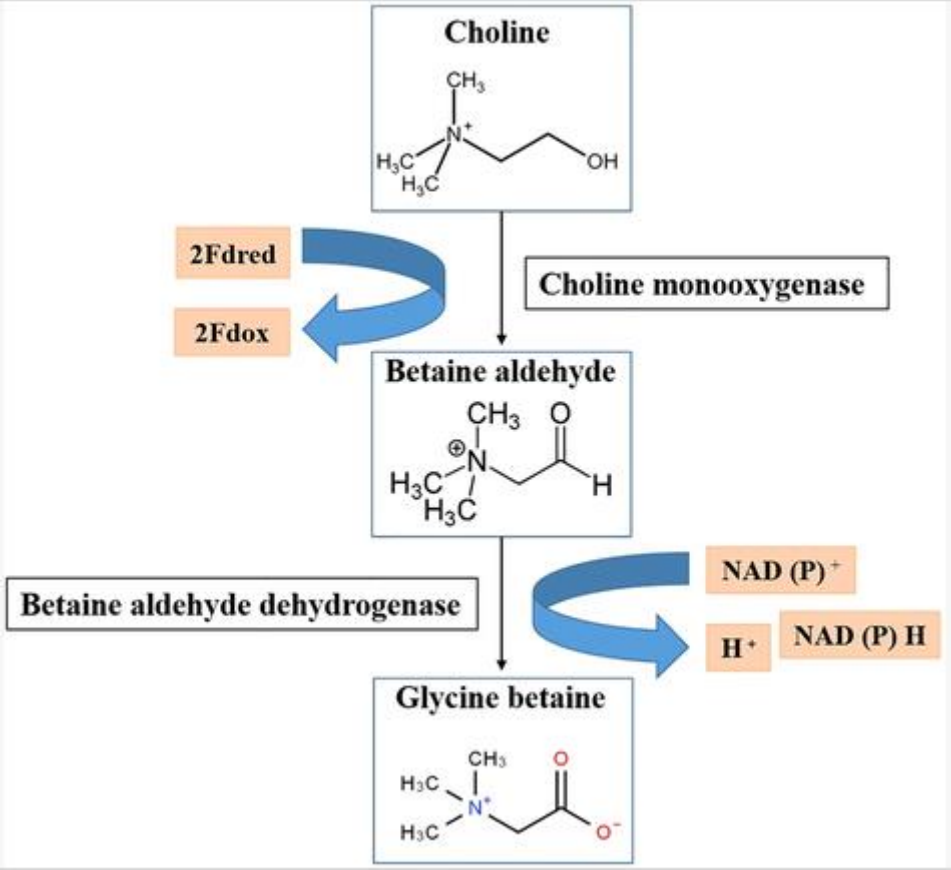
Its synthesis involves the conversion of the precursor choline through two successive oxidation steps:

- First step (CMO):

The enzyme Choline Monooxygenase (CMO) uses molecular oxygen (O_2) and reduced ferredoxin (generated during the light-dependent reactions of photosynthesis) to introduce an oxygen atom into choline, converting it into an intermediate compound called betaine aldehyde.

- Second step (BADH):

The enzyme Betaine Aldehyde Dehydrogenase (BADH) oxidizes this potentially toxic intermediate into the final, non-toxic product: glycine betaine. This reaction uses $NAD(P)^+$ as a cofactor and produces NAD(P)H.



Protective Role

- Stabilizes the photosystem II (PSII) reaction center and Rubisco under salt or heat stress.
- Maintains thylakoid membrane integrity and prevents potassium (K^+) loss.

c. Late Embryogenesis Abundant (LEA) Proteins

LEA proteins are hydrophilic, highly heat-stable proteins (intrinsically disordered in their hydrated state) that are abundantly synthesized during seed maturation and in response to dehydration stress.

Structural Characteristics

- Rich in hydrophilic amino acids and poor in hydrophobic amino acids:

- Under normal hydration conditions: Due to their abundance of charged or polar amino acids (glycine, lysine, and glutamate) and their low content of hydrophobic/aromatic amino acids (valine, leucine, and tryptophan), these proteins lack a hydrophobic core.
- Intrinsically Disordered Proteins (IDPs): They remain flexible, unfolded, and highly soluble. Their high hydrophilicity allows them to retain water and act as a molecular "sponge" in the cytosol.

- Adoption of a secondary structure (alpha-helix) during dehydration:

- Stress-dependent conformation: As water evaporates, solvent loss induces changes in the spatial conformation of these proteins. They organize into amphipathic α-helices (possessing both a hydrophilic face and a hydrophobic face).
- Physical stabilization role:
    - The hydrophobic face interacts with membrane lipids, stabilizing lipid bilayers and preventing them from breaking under dehydration.
    - The hydrophilic face interacts with enzymes, metabolites, and the cellular framework to form a molecular glass (vitrification), preventing the aggregation and denaturation of macromolecules.

Main Physiological and Cellular Functions

- Prevent the denaturation and precipitation of enzymes and cytosolic proteins caused by water loss.
- Amphipathic α-helical regions bind to the polar heads of membrane phospholipids, maintaining membrane fluidity and preventing membrane fusion or rupture.
- Bind metal cations (Fe^{2+}, Cu^{2+}), whose concentrations may increase dangerously during water loss, thereby preventing Fenton reactions and the formation of reactive oxygen species (ROS).
- In combination with non-reducing sugars (such as trehalose or raffinose), form a glass-like matrix that immobilizes molecules and stops degradation reactions without damaging cellular structures.

2. Oxidative Stress and the Redox System: Production of Reactive Oxygen Species (ROS) and Antioxidant Defense Systems (Enzymatic and Non-Enzymatic)

Chapter 4: Stress Physiology and Phenotypic Plasticity

Oxidative stress refers to a disruption of the physiological balance between the production of harmful oxidizing molecules, known as reactive oxygen species (ROS) or free radicals, and the body's ability to neutralize them using antioxidants.

During normal cellular metabolism, particularly during mitochondrial respiration, cells naturally produce oxidizing compounds. Certain external factors, such as UV radiation, pollution, smoking, toxic substances, and inflammation, can considerably increase their production.

The body has an antioxidant defense network consisting of:

- Endogenous enzymes (superoxide dismutase, catalase, and glutathione peroxidase).
- Dietary antioxidants (vitamins C and E, polyphenols, selenium, and zinc).

When ROS are produced in excessive amounts or antioxidant reserves become depleted, the defense system is overwhelmed. ROS are highly reactive and unstable molecules that directly attack cellular components:

- Lipids: Lipid peroxidation of cell membranes, altering their permeability.
- Proteins: Oxidation of amino acids, leading to loss of function or denaturation of enzymes and structural proteins.
- DNA: DNA strand breaks or mutations in nitrogenous bases, increasing the risk of tumor transformation or cellular dysfunction.

2.1. Production of Reactive Oxygen Species (ROS)

ROS include free radicals (which possess an unpaired electron) and non-radical molecules with strong oxidizing properties.

Reactive oxygen species are also produced in various subcellular structures, including the endoplasmic reticulum, peroxisomes, and cellular membranes. They are sometimes referred to as reactive oxygen derivatives. The English abbreviation is ROS (Reactive Oxygen Species).

ROS are unstable oxygen-containing molecules produced as by-products of normal oxygen metabolism. These highly reactive molecules can exist in several forms, including nitric oxide (NO), peroxynitrite ($ONOO^-$), hydrogen peroxide (H_2O_2), the hydroxyl radical (•OH), and hypochlorous acid (HOCl).

Environmental factors, particularly UV radiation, air pollutants, and certain dietary compounds, can increase the production of reactive oxygen species.

a. Main Reactive Oxygen Species

- Superoxide anion ($O_2^{\bullet -}$): Formed by the addition of an electron to molecular oxygen. It is the precursor of most other ROS.
- Hydrogen peroxide (H_2O_2): A non-radical ROS produced by the dismutation of superoxide ($O_2^{\bullet -}$). It is relatively stable and can cross biological membranes.
- Hydroxyl radical ($\bullet OH$): Generated from H_2O_2 in the presence of transition metals (Fe^{2+}, Cu^{+}) through the Fenton reaction. It is the most reactive and destructive ROS.
- Peroxynitrite ($ONOO^-$): Formed through the rapid reaction between superoxide ($O_2^{\bullet -}$) and nitric oxide ($NO\bullet$).

b. Cellular Sources of ROS

The production of reactive oxygen species (ROS) originates from both endogenous sources (generated within cells) and exogenous sources (derived from the environment).

Endogenous Sources (Enzymatic and Metabolic)

These ROS are generated within the organism, either as accidental by-products of energy metabolism or deliberately to perform specific biological functions.

- Mitochondrial Electron Transport Chain (Complexes I and III): During cellular respiration, electrons pass through protein complexes in the inner mitochondrial membrane to produce ATP. Approximately 1–2% of these electrons prematurely leak from the electron transport chain and react directly with molecular oxygen (O_2), forming the superoxide anion radical ($O_2^{\bullet -}$). This is one of the main endogenous sources of ROS in most cells.

- NADPH Oxidases (NOX): Unlike mitochondrial ROS production, which occurs mainly as a by-product of respiration, NOX enzymes are membrane-bound enzymes whose primary function is to deliberately produce superoxide ($O_2^{\bullet -}$). This mechanism is essential to the immune system. During phagocytosis, neutrophils and macrophages release an oxidative burst of ROS to destroy pathogenic microorganisms.

- Xanthine Oxidase (XO) and Peroxidases: Xanthine oxidase participates in purine catabolism by catalyzing the conversion of hypoxanthine into xanthine and subsequently into uric acid,

releasing superoxide and hydrogen peroxide (H_2O_2). Peroxidases, such as myeloperoxidase, use H_2O_2 to generate even more reactive compounds (e.g., hypochlorous acid, HOCl) for antimicrobial defense.

Exogenous Sources (Environmental Factors)

These external agents enter the organism or interact with exposed tissues, inducing excessive ROS production.

- UV and Ionizing Radiation: UV rays and X-rays/gamma rays cause the photolysis or radiolysis of cellular water (H_2O), directly generating hydroxyl radicals (•OH), among the most destructive ROS.

- Air Pollutants and Tobacco Smoke: Cigarette smoke and fine particulate matter in air pollution contain stable free radicals and organic compounds (e.g., quinones) that trigger continuous redox cycles in lung tissue.

- Toxic Substances and Heavy Metals: Certain chemical compounds (pesticides and solvents) and transition metals (iron and copper) can promote Fenton-type reactions, converting relatively less reactive hydrogen peroxide (H_2O_2) into highly toxic hydroxyl radicals (•OH).

2.2. The Antioxidant Defense System

The antioxidant defense system refers to the set of biochemical and molecular mechanisms available to the organism to neutralize reactive oxygen species (ROS), prevent chain oxidation reactions, and repair damage to macromolecules.

Its fundamental objective is to maintain redox homeostasis, meaning a dynamic balance in which ROS concentrations remain sufficiently low to prevent toxicity while still allowing ROS to perform their normal physiological functions, such as cellular signaling and immune defense.

To achieve this, the system relies on the synergistic action of two complementary categories: enzymatic and non-enzymatic antioxidants.

a. Enzymatic Antioxidants (Primary Line of Defense)

These are proteins produced directly by cells. They act sequentially and with high specificity to catalyze the conversion of ROS into less harmful compounds, such as water (H_2O) and molecular oxygen (O_2).

- Superoxide Dismutase (SOD): Represents the first enzymatic line of defense. It converts the highly reactive superoxide radical ($O_2^{\bullet -}$) into hydrogen peroxide (H_2O_2). Several forms exist depending on their cellular localization and metal cofactors (Cu/Zn-SOD in the cytosol and Mn-SOD in the mitochondrial matrix).

- Catalase (CAT): Mainly located in peroxisomes, catalase breaks down two molecules of H_2O_2 into two molecules of water (H_2O) and one molecule of oxygen (O_2). It is particularly active when H_2O_2 concentrations are high.

- Glutathione Peroxidase (GPx): A selenium-dependent enzyme found in the cytosol and mitochondria. It neutralizes H_2O_2 and lipid hydroperoxides (ROOH) by using reduced glutathione (GSH) as an electron donor, producing oxidized glutathione (GSSG).

- Glutathione Reductase (GR) and Thioredoxin Reductase (TrxR): These are antioxidant regeneration enzymes. They consume NADPH to recycle GSSG into GSH, thereby maintaining the pool of active antioxidants.

b. Non-Enzymatic Antioxidants (Free Radical Scavengers)

This category includes low-molecular-weight molecules capable of directly scavenging ROS by donating an electron or a hydrogen atom. This neutralizes the primary radical while producing a secondary radical that is less reactive and more stable.

They can be classified according to their origin and solubility:

- Water-soluble antioxidants (aqueous environments): These act in the cytosol, plasma, and cellular stroma. The main examples are:

  - Glutathione (GSH): A tripeptide synthesized by cells and a major component of intracellular redox homeostasis.
  - Vitamin C (ascorbic acid).

- Lipid-soluble antioxidants (lipid environments): These are incorporated into cell membranes and lipoproteins to prevent lipid peroxidation. The main example is Vitamin E (alpha-tocopherol), supported by carotenoids (beta-carotene and lycopene) and coenzyme Q10.

3. Heat Stress: Thermoperiodism and the Role of Molecular Chaperones (HSPs)

Chapter 4: Stress Physiology and Phenotypic Plasticity

Heat stress induces major cellular changes to which organisms respond through thermoperiodism and the rapid mobilization of chaperone proteins, such as heat shock proteins (HSPs).

3.1. Thermoperiodism: Response to Temperature Rhythms

Thermoperiodism is the physiological and morphological response of plants to alternating temperature variations between day (thermophase) and night (cryophase).

It refers to the cyclical variation in plant growth and development in response to temperature, following a principle similar to photoperiodism (which depends on light) and thermoregulation. In addition to plants, all ectothermic animals are highly sensitive to ambient temperatures.

Thermoperiodism is defined as the ability of plants to distinguish between daytime temperatures (thermophase) and nighttime temperatures (cryophase).

The thermoperiod is the overall response, particularly in plants, to appropriate fluctuating temperature conditions.

This ability of plants to respond differently to specific temperatures is exploited in vernalization, which involves the artificial exposure of plants (or seeds) to low temperatures to stimulate flowering or improve seed production.

DIF (Tday − Tnight): A key parameter measuring the difference between daytime and nighttime temperatures.

- Positive DIF (Tday − Tnight): Stimulates internode elongation and stem growth.
- Negative DIF (Tday − Tnight): Restricts cell elongation, resulting in more compact plants. This technique is used in horticulture to control plant height without chemical growth regulators.

Role in Plant Development

- Vegetative growth: Alternating temperatures regulate cell division and elongation through the modulation of gibberellin activity and phytochromes, particularly phytochrome B, which acts as a thermosensor.
- Flowering induction and dormancy: Temperature fluctuations regulate the expression of key genes involved in the circadian clock (PIF4, FT), coordinating flowering with favorable seasonal conditions.

3.2. Cellular Heat Stress

A sudden increase in temperature beyond the critical threshold causes major cellular disturbances:

- Protein denaturation: Loss of the tertiary and quaternary structures of functional enzymes, leading to toxic protein aggregation.

- Excessive membrane fluidization: Loss of the selective permeability of the plasma membrane and thylakoid and mitochondrial membranes.

- Photosynthetic dysfunction: Rapid inactivation of the oxygen-evolving complex of Photosystem II (PSII) and reduced Rubisco activity.

The PSII reaction centers ($P680^{+}$) remain in a highly oxidizing state and cannot obtain the electrons they require. As a result, they generate reactive oxygen species (ROS), which damage the PSII D1 protein.

Rubisco (Ribulose-1,5-bisphosphate carboxylase/oxygenase) is the key enzyme of the Calvin cycle, responsible for fixing CO_2 onto RuBP.

It requires a regulatory enzyme, Rubisco Activase (RCA), to release its active site when it becomes blocked by sugar-phosphate inhibitors. RCA is extremely heat-sensitive (thermolabile): it loses its activity when the temperature exceeds a certain threshold, leaving Rubisco trapped in an inactive state.

- Oxidative stress: Accumulation of reactive oxygen species (ROS), resulting in lipid peroxidation and DNA damage.

3.3. Molecular Chaperones (HSPs) and the Survival Response

Molecular chaperones, mainly represented by heat shock proteins (HSPs), are key components of the cellular stress response, known as the Heat Shock Response (HSR).

Their fundamental role is to maintain a functional proteome (protein homeostasis or proteostasis), ensuring cell survival in response to environmental stresses such as heat, drought, oxidative stress, and heavy metals.

Mechanisms of Action and Main Functions

Under stress conditions, increased temperature or the presence of ROS (Reactive Oxygen Species) causes structural proteins and enzymes to unfold and become denatured.

HSPs intervene through three major mechanisms:

- Prevention of toxic protein aggregation: HSPs recognize and bind to exposed hydrophobic regions of denatured proteins, preventing their irreversible aggregation.

- Assistance with protein refolding: Through ATP-dependent cycles, chaperones such as HSP70 and HSP60/HSP10 (GroEL/GroES) help damaged proteins recover their native three-dimensional conformation.

- Targeting proteins for degradation: If a protein cannot be repaired, chaperones facilitate its delivery to the ubiquitin–proteasome system or the autophagic pathway for elimination without damaging the cell.

Main HSP Families

HSPs are classified according to their molecular weight, expressed in kilodaltons (kDa).

- Small HSPs (sHSPs, less than 30 kDa): ATP-independent chaperones that sequester unfolded proteins in a holding state (holdases), preventing their immediate aggregation.

- HSP70 (70 kDa): Central ATP-dependent chaperones that assist de novo protein folding, refolding of damaged proteins, and protein targeting to specific cellular compartments.

- HSP90 (90 kDa): Stabilize key signaling proteins, including kinases and transcription factors, and participate in the regulation of stress responses.

- HSP100 (100+ kDa): Act as protein disaggregases (e.g., ClpB), capable of solubilizing already-formed protein aggregates and providing proteins with another opportunity to refold with the assistance of HSP70.

Regulation of the Survival Response (HSF)

The massive synthesis of HSPs in response to stress is controlled at the transcriptional level by heat shock transcription factors (HSFs).

- Under normal conditions: HSFs are maintained in an inactive monomeric form in the cytosol through interactions with HSP70/HSP90.
- During stress: The accumulation of denatured proteins in the cytoplasm recruits HSP70/HSP90, causing them to dissociate from HSFs.

- Activation: The released HSFs form trimers, translocate into the nucleus, and bind to heat shock elements (HSEs), inducing the expression of genes encoding HSPs.

3.4. Gene Induction Mechanism (HSF–HSP Axis)

1. Basal state (absence of stress): Heat shock transcription factors (HSFs) are maintained in an inactive monomeric form through association with molecular chaperones, particularly HSP70/HSP90.

2. Activation phase: When temperature increases, the accumulation of denatured proteins recruits HSP70/HSP90 chaperones, releasing the HSFs.

3. Trimerization and transactivation: The free HSFs assemble into trimers, migrate into the nucleus, and specifically bind to promoter sequences known as heat shock elements (HSEs).

4. Massive transcription: This triggers the rapid and amplified expression of genes encoding the different HSP families.

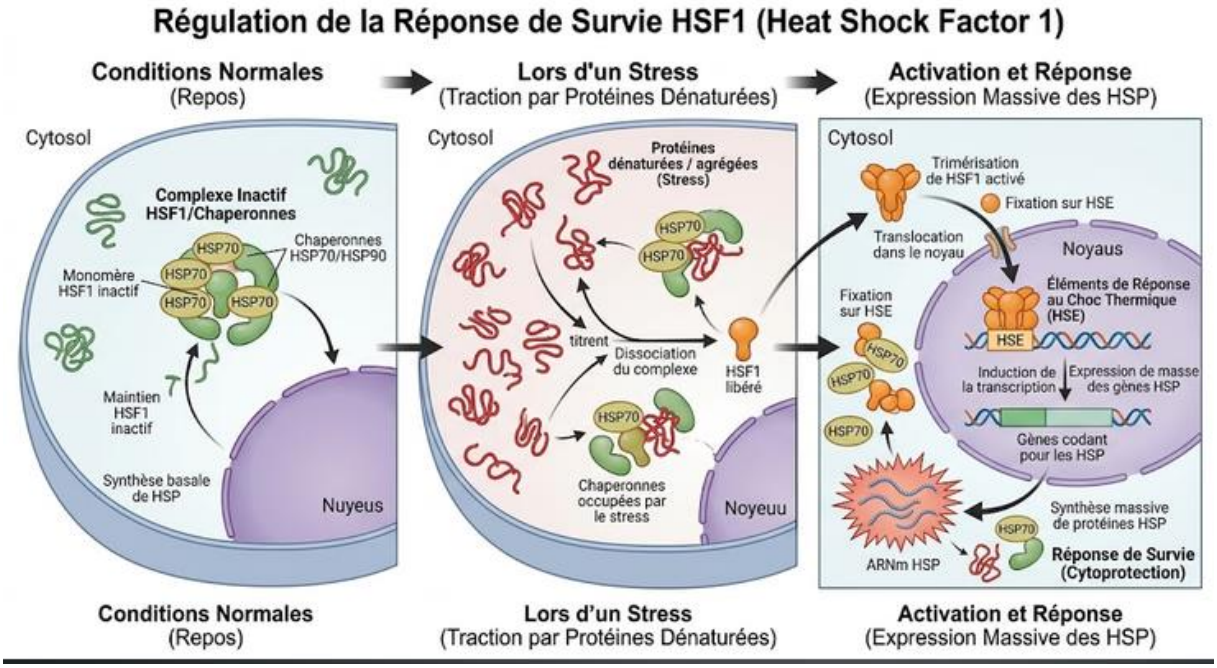


Fig. 36. Gene Induction Mechanism (HSF–HSP Axis)

3.5. Acclimation and Heat Memory (Thermotolerance)

HSPs are not only involved in immediate heat tolerance (basal thermotolerance), but also play a decisive role in acquired thermotolerance:

- Primary priming: Prior exposure to moderate heat stress induces the preliminary synthesis of HSPs and the establishment of epigenetic modifications (e.g., DNA methylation and histone modifications).

- Short- to medium-term memory: During a second, more severe stress event, the plant responds more rapidly and intensely due to residual HSP levels and a "primed" chromatin state (priming), thereby preventing irreversible damage.

4. Biotic Interactions: Recognition of Pathogen-Associated Molecular Patterns (PAMPs) and Salicylic Acid and Jasmonic Acid Signaling

Since plants do not possess an adaptive immune system (such as antibodies in animals), they rely on a sophisticated innate immune system to detect pathogens and coordinate their defense responses.

4.1. PAMP Recognition and PTI Immunity

The recognition of PAMPs and the activation of PTI immunity constitute the first line of plant defense, relying on the detection of conserved molecules found in microorganisms.

a. PAMP Recognition

PAMPs (Pathogen-Associated Molecular Patterns) are molecular structures that act as microbial "identity markers." They are essential molecules for the survival of microorganisms and are absent from plants.

Examples include:

- Flagellin: A protein that forms the tail-like structure (flagellum) of bacteria.
- Chitin: A polysaccharide that forms a major structural component of fungal cell walls.
- LPS (Lipopolysaccharide): A component of the outer membrane of Gram-negative bacteria.

PRRs (Pattern Recognition Receptors): These are receptor proteins located on the plant plasma membrane. Each PRR is specialized in recognizing and binding to a specific PAMP. For example, the FLS2 receptor specifically recognizes bacterial flagellin.

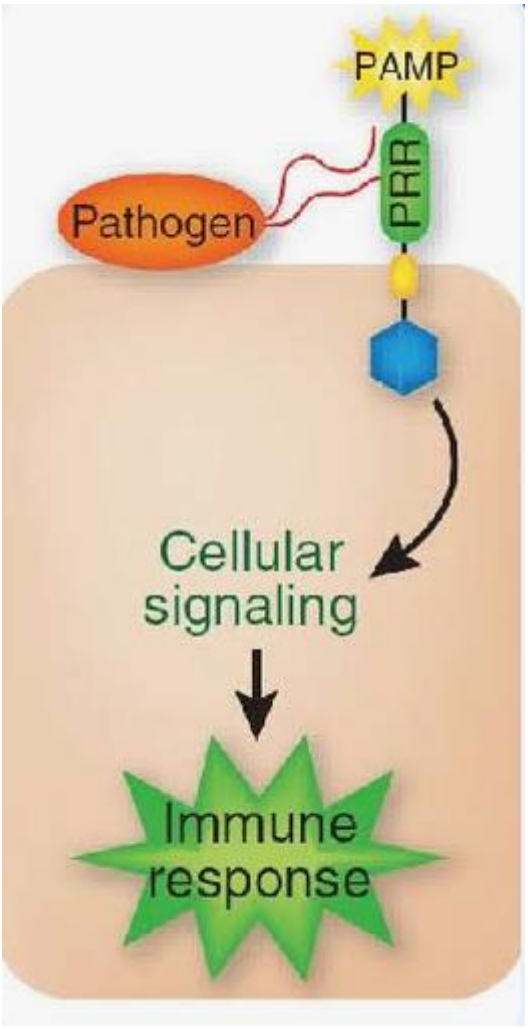


Fig. 40. Recognition of PAMPs

b. PTI Immunity (PAMP-Triggered Immunity)

As soon as a PRR detects a PAMP, a defense alarm is triggered. The plant immediately activates PTI immunity, a rapid defense response organized into four major steps:

1. Calcium Influx (Ion Fluxes): PAMP binding to the receptor triggers the opening of membrane ion channels. Calcium ions (Ca^{2+}) rapidly enter the cell, acting as an internal warning signal.

2. Oxidative Burst (ROS Production): The cell suddenly produces reactive oxygen species (ROS), such as hydrogen peroxide (H_2O_2). These toxic molecules directly attack the pathogen and also act as chemical signals to alert neighboring cells.

3. Activation of MAPK Cascades: A series of protein kinases, known as MAPKs (Mitogen-Activated Protein Kinases), are sequentially activated like falling dominoes. They transmit the signal to the cell nucleus, where defense genes are activated.

MAPKs are enzymes distributed throughout the cytoplasm. When a receptor detects a pathogen, the first MAPK activates the next one, which in turn activates another, creating a domino-like signaling cascade. This rapid signaling network transmits information from the plasma membrane to the nucleus, triggering the activation of defense-related genes.

4. Cell Wall Reinforcement (Callose Deposition): To physically block pathogen progression, the plant produces and deposits a complex carbohydrate called callose between the plasma membrane and the cell wall, creating a physical barrier.

More specifically, the plant rapidly synthesizes callose and deposits it beneath the cell wall, precisely at the site of pathogen attack. This deposition strengthens the cell wall and seals potential entry points, preventing the pathogen from penetrating further into plant tissues.

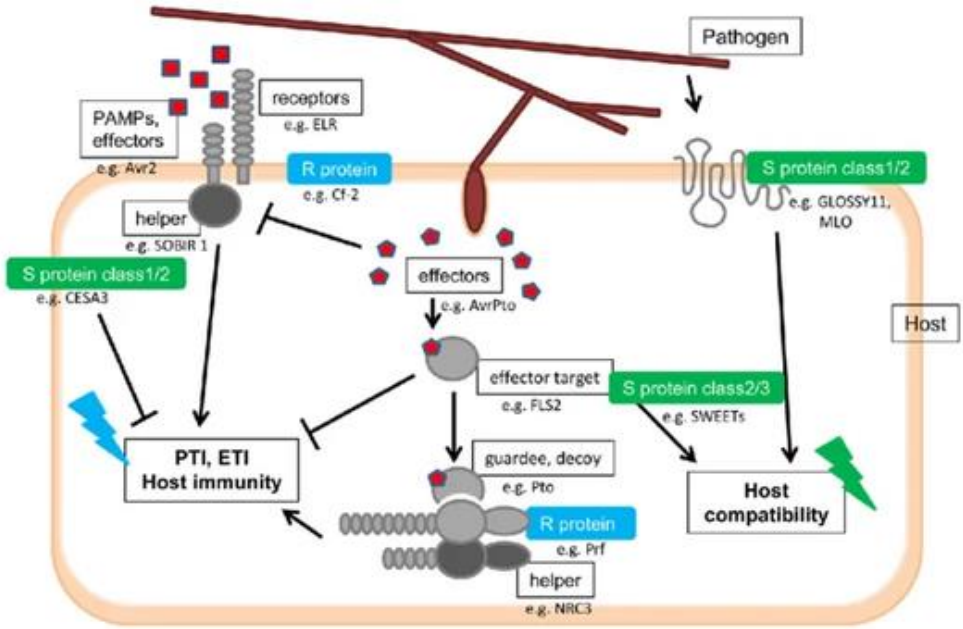


Fig. 50. Plant Innate Immunity: PAMP-Triggered Immunity (PTI) and Effector-Triggered Immunity (ETI). Apoplastic pathogen-associated molecular patterns (PAMPs/apoplastic effectors), intracellular effectors, or modified effector targets are actively recognized by receptors located in the plasma membrane or by resistance (R) proteins in the cytoplasm, resulting in the activation of PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI). Helper proteins, guard proteins, and decoy proteins are involved in the co-recognition of pathogen-derived molecules.

4.2. Hormonal Signaling: Salicylic Acid (SA) vs. Jasmonic Acid (JA)

Once a threat has been detected, the plant activates specialized hormonal signaling pathways depending on the lifestyle of the attacker.

a. Salicylic Acid (SA): Defense Strategy Against Pathogens That Feed on Living Tissues

Salicylic acid (SA) primarily mediates defense responses against biotrophic pathogens, which require living host cells to survive.

The plant deliberately sacrifices infected cells through programmed cell death (the hypersensitive response, HR) to deprive the pathogen of nutrients. It then spreads the defense signal throughout the foliage (systemic acquired resistance, SAR) and produces defense-related proteins (PR proteins).

- Local Sacrifice (Hypersensitive Response – HR): As soon as the plant detects a pathogen in a leaf, it immediately triggers the destruction of cells surrounding the infection site. This process is known as programmed cell death. By sacrificing its own cells, the plant deprives the pathogen of nutrients and isolates it within a localized area of dead tissue, thereby restricting its spread.

- Systemic Alarm (Systemic Acquired Resistance – SAR): During this localized defense response, the plant activates salicylic acid signaling, which contributes to the transmission of defense signals throughout the foliage. This acts as an alarm message to healthy leaves: "Warning! We are under attack. Prepare your defenses!"

- Chemical Weapons (Pathogenesis-Related Proteins – PR Proteins): In response to this alarm, plant cells increase the production of PR (Pathogenesis-Related) proteins. These proteins include enzymes capable of attacking and degrading microbial cell walls, helping protect other leaves against subsequent infections.

b. Jasmonic Acid (JA): Defense Strategy Against Destructive Pathogens and Herbivores

Jasmonic acid (JA) primarily regulates defense against necrotrophic pathogens, which kill host cells before feeding on the dead tissue, as well as herbivorous insects.

Programmed cell death would be ineffective against necrotrophic pathogens because they feed on dead organic matter. Instead, the plant produces toxins (secondary metabolites) and digestive inhibitors (protease inhibitors) to poison or repel the attacker while strengthening its overall defense system through induced systemic resistance (ISR).

- Chemical Warfare (Toxins / Secondary Metabolites): The plant produces toxic or bitter compounds, such as alkaloids and terpenes, making its leaves unpalatable or directly poisoning the attacker.

- Blocking Digestion (Protease Inhibitors): Against insects, the plant produces proteins that inhibit their digestive enzymes. The insect continues feeding on the leaf but can no longer digest its food properly, causing digestive disturbances and potentially reducing or stopping its feeding activity.

- Systemic Defense Reinforcement (ISR – Induced Systemic Resistance): In parallel, jasmonic acid-related signaling contributes to the activation of defense responses throughout the plant, preparing its tissues to resist future attacks.

4.3. Antagonism Between SA and JA

In general, activation of the SA signaling pathway suppresses the JA pathway, and vice versa. This interaction prevents the plant from wasting resources by simultaneously activating two potentially incompatible defense responses.

SA and JA signaling pathways therefore often function antagonistically, creating a regulatory switching mechanism.

1. Resource Optimization (SA/JA Antagonism)

The plant has a limited energy budget. Activating both defense pathways simultaneously may lead to unnecessary energy expenditure and reduced defense efficiency.

- The switching principle: When a biotrophic pathogen attacks, the plant strongly activates the SA signaling pathway. To avoid unnecessary energy expenditure, SA signaling can suppress the JA pathway.

- The objective: To allocate resources preferentially to the most appropriate defense mechanism. For example, the plant may prioritize the production of PR proteins rather than digestive inhibitors when responding to a biotrophic bacterial pathogen.

2. Pathogen Manipulation (Immune Hijacking)

This represents a chemical strategy used by pathogens to manipulate the plant's immune system.

Because pathogens can exploit the antagonistic relationship between SA and JA pathways, some have evolved mechanisms to interfere with hormonal signaling and activate inappropriate defense responses, thereby weakening plant resistance.

- The pathogen's strategy: A necrotrophic pathogen, which is normally counteracted by JA-dependent defenses, may produce a toxin or effector that interferes with hormonal signaling, potentially activating the SA pathway or suppressing JA-mediated defenses.

- The plant's misleading response: The plant activates an inappropriate defense pathway, which may suppress the JA-dependent response needed to resist the necrotrophic pathogen.

- The outcome: The plant's JA-mediated defense is weakened, allowing the necrotrophic pathogen to establish and spread more easily.

Famous example:

The bacterium Pseudomonas syringae produces a toxin called coronatine, which mimics the biological activity of jasmonic acid (JA). Coronatine activates JA signaling and promotes the suppression of SA-mediated defenses, allowing the bacterium to overcome part of the plant's immune response.