Adaptation Strategies and Defence Mechanisms of Plants During Environmental Stress

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Abstract Several biotic and abiotic stresses affect plant growth, development and crop productivity. To cope up all these stresses, plant develops certain efficient strategies to avoid or tolerate the stresses which allow them to adapt and defense themselves from stress situations. Such adaptation strategies are at morphological, anatomical, biochemical and molecular levels. Molecular crosstalk, epigenetic memories, reactive oxygen species (ROS) signaling, accumulation of plant hormones such as salicylic acid, ethylene, jasmonic acid and abscisic acid, change in redox status and inorganic ion fluxes, R-gene resistance and systemic acquired resistance (SAR) are some of the modifications/mechanisms adopted by plants to adapt and defense themselves from the environmental stress. The novel "omics" technologies allow the researchers to identify the genetics behind plant stress

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response and adaptation and provide unbiased data that can be precisely used to investigate the complex interplay between the plants, its metabolism and the stress environment.

Keywords Stress · Adaptation strategies · Molecular crosstalk · Epigenetic memories

Introduction

Stress can be defined as a stimulus or influence that is outside the normal range of homeostatic control in a given organism. If a plant is exposed to extreme stress conditions that are beyond the tolerance level, mechanisms are activated at morphological, physiological, biochemical and molecular levels. Once the stress is released, a new physiological state is established and the plant may return to the original state thereby, reestablishing the homeostasis (Fraire-Velazquez et al. 2011). Plants being a sessile organism faced many environmental challenges including biotic and abiotic factors. In the course of evolution, plants have developed highly sophisticated and efficient strategies to cope up environmental stress imposed by the nature. Even though plant species vary in their sensitivity and response to various environmental stresses, they have developed various adaptation mechanisms to encode stress perception, signaling cascades and gene transcription networks in response to environmental cues (Fu and Dong 2013; Sanghera et al. 2011; Joshi et al. 2016) and developed a multitude of defense mechanisms to adapt and survive during the harsh environmental conditions.

Major mechanisms underlying environmental stress adaptation and defense include reactive oxygen species (ROS) signaling; accumulation of plant hormones such as salicylic acid, ethylene, jasmonic acid and abscisic acid (Wani and Kumar 2015); change in redox status and inorganic ion fluxes; molecular crosstalks; epigenetic modifications; R-gene resistance and systemic acquired resistance (SAR), etc. (Kissoudis et al. 2014). Physical barriers such as the cuticle, stomata and cell walls are also important for timely pathogen recognition and interception (Asselbergh et al. 2007). Biochemical studies revealed processes induced by stress that lead to accumulation of metabolites like nitrogen-containing compounds such as proline, quaternary amino compounds and polyamines; hydroxyl compounds like sucrose, polyols and oligosaccharides (McCue and Hanson 1990). Through "omics" technologies, it has been found out that plant responses to stress are mediated through profound changes in gene expression resulting in changes in composition of plant transcriptome, proteome and metabolome (Perez-Alfocea et al. 2011; Shriram et al. 2016). These novel techniques allow researchers to identify the genetics behind plant stress responses and enable to investigate the complex interplay between the plant, its metabolism and the stresses imposed to them (Perez-Clemente et al. 2013).

In this article, we will elaborate on the morphological, anatomical, biochemical and molecular adaptation strategies against biotic and abiotic stresses. We will also discuss molecular crosstalks and epigenetic modifications for stress response and adaptations in plants. Passive and active defense mechanisms of plant [including hypersensitive response, systemic acquired resistance (SAR) etc.] against pathogen and mechanisms against insect pests like antixenosis, antibiosis and tolerance are also highlighted in the work.

Adaptation Strategies of Plants During Environmental Stress

Morphological Adaptation Strategies

The productivity of crop is reducing due to harmful effects of various biotic and abiotic stresses. Minimizing these losses is a major area of concern to ensure food security under changing climate condition of the growing global population. Somehow, the plant changes their morphological structure to adopt the adverse climate condition to sustain in the existing environment. Abiotic stresses, such as drought, extreme temperature, cold, heavy metals, or high salinity, severely impair in plant growth, development and eventually in the productivity of the crops (Wani et al. 2016a; Hossain et al. 2016; Sah et al. 2016). These stresses reduced the yield of crops, depending on the type of crop and stress period. Drought, being an important environmental stress as it severely impairs plant growth and development (Shao et al. 2008a, b; Wani et al. 2016b; Telem et al. 2016; Dar et al. 2016). Drought impaired in germination and poor crop establishment. It also induced reduction in leaf area is ascribed to suppression of leaf expansion through reduction in photosynthesis (Rucker et al. 1995). Drought related reduction in yield and yield components of plants could be ascribed to stomata closure in response to low soil water content, which decreased the intake of CO_2 and as a result, photosynthesis decreased (Cornic 2000; Flexas et al. 2004). The presence of small roots to the lateral roots is considered as an adaptive strategy to increase water uptake by providing more absorptive surface. However, the growth of lateral roots is significantly reduced, mainly by suppression of the activation of the lateral root meristems (Deak and Malamy 2005). Plants growing in dry areas have developed xeromorphic traits such as leaf shedding as well as decrease in leaf number, leaf size, and branching. Presence of specialized tissues like rhizodermis, with a thickened outer cell wall or suberized exodermis, or reduction in the number of cortical layers is considered an adaptive advantage for drought stress survival. Another adaptation to counter drought stress is sclerophylly, where plants form hard leaves that will not suffer permanent damage due to wilting and can be restored to full functionality when normal conditions resume (Micco and Aronne 2002). Recent research has shown that decreased stomatal conductance in response to drought stress is related not only to reduced expression of aquaporin genes but also to anatomical traits leading to reduction of chloroplast surface area exposed to intercellular space per unit leaf area (Miyazawa et al. 2008; Howe and Jander 2008).

Heavy metal contamination may rigorously hinder the plant growth and its productivity, and also increase the risk of harm in animal and human health via the bio magnification process (Sharma 2012). Arsenic (As) has great potential to block various metabolic processes in cell, and may interact with sulphydryl groups of the proteins, and also replace the phosphate group of ATP. Similar to As, Mercury (Hg) also interacts with the -SH groups and form the S-Hg-S bridge, disrupting the stability of the group and consequently affects seed's germination and embryo's growth. The replacement of central atom (Mg) of chlorophyll molecule with As leading to the dismantle of chlorophyll resulting in the breakdown of photosynthesis and growth of the sunflower seedlings under As stress (Patra et al. 2004; Yadav et al. 2014). Chromium (Cr) stress declined biomass accumulation in barley plants by causing ultrastructure disorders in leaves such as uneven thickening and swelling of chloroplast, increased amount of plastoglobuli and disintegrated thylakoid membrane which resulted into decline in net stomatal conductance, cellular CO_2 concentration, transpiration rate, photochemical efficiency and net photosynthetic rate (Ali et al. 2013). In another finding, Hg reduces the plant growth due to its higher affinity to bind with-SH groups of proteins and causes uneven modification in protein structure (Cui et al. 2014). It has been observed that brinjal plant when exposed to Cadmium (Cd) showed restricted growth, leaf area, photosynthetic pigment and chlorophyll fluorescence (Singh and Prasad 2014). Similarly, Shahid et al. (2014) have reported that the growth and photosynthetic pigments of *Vicia faba* got inhibited under lead (Pb) stress.

The plant that grow in the cold region modified their morphological structure and escape the action of free air circulating by reducing plant size and adopting compact growth forms, thereby engineering their own microclimate in addition to sheltered habitat selection. In addition, some tropical alpine taxa have been found to perform what is called leaf super-cooling, which is retaining water in a gel-like metastable state below freezing point (to avoid nucleation). When the super-cooling capacity is exhausted around −12 °C, tissues freeze immediately and died. There is evidence that the xylem water of trees of cold regions can supercool down to −40 °C reported (Korner 2016).

Plants, animal and insects have been living together since million years ago. In co-evolution, they have evolved strategies to avoid each other's defence systems, resulted in the development of an elegant defence system in plants that has the ability to recognize the nonself molecules or signals from damaged cells, much like the animals, and activates the plant immune response against the herbivores (Howe and Jander 2008; Verhage et al. 2010; Hare 2011). Plant structures are the first line of defence against herbivory, and play an important role in host plant resistant to insects. The first line of plant defence against insect pests is the erection of a physical barrier either through the formation of a waxy cuticle, development of spines, setae, and trichomes (Hanley et al. 2007; Agrawal et al. 2009; Sharma et al.

2009; He et al. 2011). Development of structural traits such as spines and thorns, trichomes, toughened and sclerophylly nature of leaves reducing the palatability and digestibility of the tissues, incorporation of granular minerals into plant tissues, and divaricated branching play a leading role in plant protection thereby, reducing the herbivore damage (Handley et al. 2005; Hanley et al. 2007; Chamarthi et al. 2010; He et al. 2011).

Anatomical Adaptation Strategies

Physical Factors (Temperature, Light, Water, Oxygen Deprivation)

Plants are frequently exposed to many stress conditions, such as low temperature, salt, drought, flooding, heat, oxidative stress, and heavy-metal toxicity. In which, water stress is the major problem in agriculture. The ability of plants to withstand such stress is of immense economic importance (Shao et al. 2008a, b). Water stress is the state of moderate loss of water, which leads to closure of stomata and limits gas exchange. Water-stress tolerance involves subtle changes in cellular biochemistry. It appears to be the result of the accumulation of compatible solutes and of specific proteins that can be rapidly induced by osmotic stress (Shao et al. 2005). Water stress influences plant growth at various levels, from cell to community (Blumwald et al. 2004; Colom and Vazzana 2001). The quantity and quality of plant growth depend on cell-division enlargement, and differentiation, and all of these events are affected by water stress (Correia et al. 2001; Cabuslay 2002). There is evidence that stiff, leathery leaves are widespread in species adapted to drought occurring in various environments throughout the world. Sclerophyllous leaves are characterised by reinforcing tissues (e.g. thick-walled epidermal cells, sclereids, etc.) which prevent the collapse of the whole structure when water availability is scarce, thus reducing the risk for mechanical damage. Under drought conditions, a sclerophyllous leaf slightly reduces its volume thanks to thick cuticle and thick-walled epidermal cells, but thin-walled mesophyll cells severely shrink resulting in an increase of intercellular spaces. This allows photosynthesis to remain active also in conditions of severe water stress when other leaf types wilt (Shields 1950).

Heat Stress

Plants are frequently subjected to heat stress, which can severely hinder the growth and development and may even cause plant death. Heat stress is another serious threat to crop production and quality worldwide. Moreover extensive agricultural losses have been attributed to hindrance to growth due to heat (Wahid et al. 2007; Kotak et al. 2007). In general, it is evident that high temperature considerably affects anatomical structures not only at the tissue and cellular levels but also at the sub-cellular level which results in poor plant growth and productivity. The anatomical changes under high ambient temperatures are generally similar to those under drought stress (Wahid et al. 2007). Under heat stress, there is a general tendency of reduced cell size, closure of stomata and curtailed water loss, increased stomatal and trichomatous densities, and greater xylem vessels of both root and shoot (Anon et al. 2004), severely damages mesophyll cells and increased permeability of plasma membrane in grapes (Zhang et al. 2005). Different plants show varying responses under high temperature regime such as production of polymorphic leaves to reduce transpiration by showing bimodal stomatal behavior (Sayed 1996), changing the structural organization of thylakoids reducing photosynthesis (Karim et al. 1997). In grapes, chloroplasts in the mesophyll cells became round in shape, the stroma lamellae became swollen, and the contents of vacuoles formed clumps, whilst the cristae were disrupted and mitochondria became empty (Zhang et al. 2005). Lima et al. (2013) observed that coffee plants subjected to heat stress (37 °C) changed structural cell anatomy. Structural variations in the lignin contents of cell walls under abiotic stress may be the result of increased oxidative stress in wall-modifying plant tissues. Structural variations in the lignin contents of cell walls under abiotic stress may be the result of increased oxidative stress in wall-modifying plant tissues (Moura et al. 2010).

Chemical Factors (Salinity, Heavy Metals, Pollution)

Salinity

Salinity is one of the most significant environmental challenges limiting plant productivity, particularly in arid and semi-arid climates (Ashraf and Harris 2004; Hussain et al. 2009; Wani and Gosal 2011; Wani and Hossain 2015). A soil is considered to be saline when the electric conductivity (EC) of the soil solution reaches 4 dS m^{-1} (equivalent to 40 mM NaCl), generating an osmotic pressure of about 0.2 MPa and significantly reducing the yields of most crops (Munns and Tester 2008; Wani et al. 2010). As a consequence of soil salinity, ion toxicity leads to chlorosis and necrosis, mainly due to Na^+ accumulation that interferes with many physiological processes in plants (Munns 2002). Most salinity adaptive mechanisms in plants are accompanied by certain morphological and anatomical changes (Larcher 2003). Plants tolerant to NaCl implement a series of adaptations to acclimate to salinity, including morphological, physiological and biochemical changes (Pathak and Wani 2015). These changes include increase in the root/canopy ratio and in the chlorophyll content in addition to changes in the leaf anatomy that ultimately lead to preventing leaf ion toxicity, thus maintaining the water status in order to limit water loss and protect the photosynthesis process (Acosta-Motos et al. 2017). It is reported that, the cell walls of root cells of salinized plants are often unevenly thickened and convoluted (Shannon et al. 1994). Salts often promote the suberisation of the hypodermis and endodermis in woody tree roots, resulting in the formation of a well-developed casparian strip closer to the root apex, different to that found in non-salinized roots (Walker et al. 1984). Irrigation with saline water has also been found to increase root diameter and root density of *Callistemon citrinus* plants (Álvarez and Sánchez-Blanco 2014). In other plant species (*Picea* sp., *Pinus banksiana*, *Portulaca oleracea*), root diameter (hypertrophy) is found to increase in response to salinity (Franco et al. 2011; Croser et al. 2001). Under saline conditions cell wall properties change and leaf turgor and photosynthesis rates decrease, leading to a reduction in total leaf area (Franco et al. 1997; Rodríguez et al. 2005). Romero-Aranda et al. (1998) studied anatomical disturbances produced by KCl, $CaCl_2$, NaCl in Carrizo citrange and Cleopatra mandarin citrus varieties. The salt-induced declines in photosynthesis rates were linked to changes in leaf anatomical properties, such as the increase in leaf thickness and the lower area/volume ratio of mesophyll cells, increased leaf succulence and reduced intercellular air spaces, the surface/volume ratio of cells and tissue density. The increase in leaf thickness, in combination with several metabolic components such as Cl^- overloading, low Mg^{2+}, stomatal closure and chlorophyll loss, may contribute to the decline in photosynthesis. Anatomical modifications in leaves also include an increase in palisade parenchyma and intercellular spaces and a decrease in spongy parenchyma, serving to facilitate CO_2 diffusion in a situation of reduced stomatal aperture (Acosta-Motos et al. 2017).

Heavy Metals Stress

Other than moisture and salinity stress, the stress given by the heavy metals due to ever increasing population is also becoming a matter of concern. The phytotoxic effect of heavy metals in plants manifests itself through visual symptoms such as chlorosis, necrosis and wilting, and through reduced growth and biomass accumulation (Marques et al. 2000; Sanitá di Troppi and Gabbrielli 1999). Gupta and Chakrabarti (2013) observed that heavy metal (Hg, Pb and Cd) accumulations in *Bruguiera sexangula* indicated deformed vascular bundle with Hg treatment. Xylem and phloem deformation was also recorded in treatment of Cd which finally led to visible toxicity. The stem transverse section (T.S.) for Cd treatment showed diffusion of that heavy metal up to the pith region. When stem T.S. of Hg was compared with control, it was observed that the stelar region was greatly affected. Penetration of Pb inside the stem starting from epidermis to pith region was noticed. Kasim (2006) found that *Sorghum bicolor* treated with copper or cadmium applied alone or in combination caused significant reduction in root diameter, width and thickness of leaf midrib, diameter of xylem vessels of all seedling organs, parenchyma cell area in the stem, leaf midrib and pith and cortex of root, dimensions of stem vascular bundles, number of xylem arms in root, and frequency of stomata on abaxial leaf surface. The effect was more pronounced when Cu and Cd were applied together. The molecular-physiological mechanism responsible for observed changes in anatomy is not yet clear. It is also reported that exposure to heavy metals leads to a reduction in the size of mesophyll cells (Sridhar et al. 2005; Zhao et al. 2000) and the collapse of palisade and spongy parenchyma cells (Sridhar et al. 2005). According to Pasternak et al. (2005) architectural changes imposed by Cu and other abiotic stresses on roots of *Arabidopsis thaliana* may be attributed to alteration in phytohormone metabolism and local auxin accumulation near the root pericycle. Gomes et al. (2011) observed the thickened the cell layers of endodermis and exodermis in the root tissues and the cell walls of the xylem and cortical parenchyma of *Brachiaria decumbens* Stapf. due to heavy metals (Cd, Pb, Zn, Cu). In the leaf tissues, the adaxial and abaxial epidermis presented increased thickness while the leaf blade presented reduced thickness as contamination increased with consequent change in the root growth rate. In general, the effects of heavy metal increased with the metal concentration. The ill effect of moisture stress may be enhanced by other abiotic stresses like heavy metals. Similarly, Greco et al. (2012) observed additive effects of the stresses on growth and the increase in the early mortality of plants by metal contamination in combination with drought.

Pollution

Studies regarding the anatomy of the vegetative organs under polluted conditions have been carried out by various workers (Alves et al. 2008; Silva et al. 2006; Verma et al. 2006). Epidermis structures like trichomes have been reported to bear the brunt of damage from cement dust in *Cajanus cajanus* exposed to foliar dust application (Baralabai and Vivekanandan 1996). Similarly, Gostin (2009) observed the presence of the phenolic compounds (dark deposits from the epidermis, assimilatory and vascular tissues) in *Trifolium* spp. indicate that long-term exposure to air pollutants leads to enhanced accumulation of these compounds. The stomata decrease in size and increase in density in leaves from high polluted sites. In the mesophyll cells (both in palisade and in spongy parenchyma) dark phenolic deposits could be observed. The enhanced accumulation of phenolics and lignin is considered to be one of the most common reactions of plants to stress (Wild and Schmitt 1995). Verma et al. (2006) found a significant decrease of stomatal density and stomatal index in *Ipomea pes-tigridis* grown under various degrees coal-smoke pollutants. However, Ogunkunle et al. (2013) observed no anatomical modifications to the cement factory pollution in *Pennisetum purpureum*, which may indicate that the dose-response level of the pollutants has not been reached in the grass. But there were significant reduction in stomatal size and increased stomatal index in the leaves of *Sida acuta.* This could be favourable anatomical adaptations to a polluted environment. In another study, plane tree growing in urban area reduced the size and lower stomata density, thin cuticle but the other anatomical properties were unaffected suggesting that plane trees can cope with traffic exhaust in megacities (Pourkhabbaz et al. 2010).

Biological Factors (Pests, Diseases and Allelopathy)

Plants respond to herbivore attack through an intricate and dynamic defense system that includes structural barriers, toxic chemicals, and attraction of natural enemies of the target pests (War et al. 2012). Rodrigues et al. (2015) observed various anatomical changes in young coffee leaves infected by *P. syringae* pv. *garcae* which are similar to the tabtoxin damage induced by bacteria, which causes degradation of the thylakoid membrane and chloroplasts (Bender et al. 1999), and favors the tissue colonization and its use as substrate. Palmer and Bender (1995) observed that, tomato inoculated with *P. syringae* observed plasmolysis of the cellular content in tomato mesophyll, as well as cell hypertrophy and hyperplasia. Similar changes were here observed in the mesophyll of coffee leaves. Sclerophylly (reinforcing tissues) has been interpreted as a phenomenon linked to functions as protection against pathogens or as response to scarce nutrient availability (Salleo and Nardini 2000). Sclerophylls are widely distributed in arid and semiarid environments together with seasonally dimorphic species.

Molecular and Biochemical Adaptation Strategies

The understanding of the mechanism of complex nature of stress signaling and plant adaptive strategies would require the analysis of the function of enormous amount of genes involved in stress response (Wani et al. 2012). According to the result of several investigations, plant defense response genes are transcriptionally activated by pathogens as well as by different types of abiotic stress. The activation of specific defense genes against certain pathogens depend on specific environmental conditions and this suggest the interplay of certain complex signaling network that enable the plant to recognize and protect itself against different stresses including pathogens (Bansal et al. 2012; Wani et al. 2013a; Khan and Wani 2014; Jaspers and Kangasjarvi 2010).

The APETALA2 (AP2)/ethylene-responsive-element-binding factors (ERFs) proteins function as either activators or repressors of transcription of gene as response to both biotic and abiotic factors to adapt under adverse environmental conditions (Fujimoto et al. 2000). The RNA levels of specific AP2/ERF genes are known to be regulated by factors like drought, cold, pathogenic infection, salinity, wounding or treatment with salicylic acid or jasmonic acid (Onate-Sanchez and Singh 2002). These genes resulted in improved tolerance against osmotic stress and pathogen attack in tobacco (Park et al. 2001), and were also identified in rice (Datta et al. 2012), grape (Licausi et al. 2010), Arabidopsis (Kang et al. 2011), Wheat (Zhuang et al. 2011), apple (Zhao et al. 2012) and potato (Bouaziz et al. 2015). Nakano et al. (2006) and Wu et al. (2007) reported that the AP2/ERF proteins from one plant species are functional in another plant species. Therefore, these can be potentially utilized in increasing the adaptation and stress tolerance of plants.

Another important family of transcription factors is the “basic-domain leucine-zipper (bZIP) which are regulators of many important plant processes including defense against pathogen, hormone and sugar signaling, osmotic control, abiotic stress signaling, energy metabolism, etc. (Perez-Clemente et al. 2013; dos Reis et al. 2016). TGA/*octopine synthase* (ocs)-element-binding factor (OBF), a class of bZIP proteins bind to the *activation sequence-1* (*as-1*)/*ocs* element and regulates the expression of some stress- responsive genes and are thus, linked to stress responses (Fode et al. 2008a, b). Overexpressions of *AREB2/ABF4* or *ABF3* gene in *Arabidopsis* result in hypersensitivity of ABA, reduced transpiration and elevate drought tolerance (Kang et al. 2002). *OsABF1* gene from rice (Amir et al. 2010) and *SlAREB* gene from tomato (Hsieh et al. 2010) are known to enhance tolerance to drought and salt stress and can be utilized as potential candidates for improving stress tolerance in crops.

MYB and MYC proteins are key factors involved in the response of plants to harsh environmental conditions. *AtMYB30* encodes an activator of the hypersensitive cell death program in response to pathogen attack (Raffaele et al. 2008); *AtMYB33* and *AtMYB101* are involved in ABA-mediated responses to environmental signals (Perez-Clemente et al. 2013); *AtMYB96* regulate water stress and disease resistance by acting through the ABA signaling cascade (Seo and Park 2010); *AtMYB15* is involved in cold stress tolerance (Agarwal et al. 2006). The rice gene *OsMYB4* imparts cold and drought tolerance in transgenic plants of *Arabidopsis* (Vannini et al. 2004), tomato (Vannini et al. 2007) and apple (Pasquali et al. 2008). Yang et al. 2012 reported the regulatory role of *OsMYB2* gene in salt, cold and dehydration tolerance in rice. MYC protein family play a role in the induction of apoptosis and hypersensitive cell death program in response to pathogen attack and also activate the major ABA-dependent stress response (Agarwal and Jha 2010). For instance, *MYC2* is an ABA- and drought-responsive gene and is also capable of activating the expression of the ABA response gene *Responsive to Dessication22* (*RD22*) (Abe et al. 2003).

The NAC family are plant-specific transcriptional factors (TFs) identified in *Arabidopsis* (Ooka et al. 2003), rice (Fang et al. 2008), soybean (Le et al. 2011) and poplar (Hu et al. 2010). These TFs are involved in responses to various biotic and abiotic stresses including drought, cold, salinity, pathogenic bacteria and fungi and low-oxygen stress (Nuruzzaman et al. 2013). In Arabidopsis, the expression of NAC genes viz. *ANAC019*, *ANAC055* and *ANAC072* was induced by drought, salinity and ABA respectively. Moreover, overexpression of these genes also enhances tolerance to drought (Tran et al. 2004). *MlNAC5* gene, when overexpressed, impart hypersensitivity to exogenous ABA and confers enhanced drought and cold tolerance in Arabidopsis (Yang et al. 2015). In transgenic rice, the *Os01g66120/OsNAC2/6* (Nakashima et al. 2009) and *Os11g03300/OsNAC10* genes (Jeong et al. 2010) were reported to enhance drought and salt tolerance, and *Os03g60080/SNAC1* increased grain yield by 21–34% under drought stress condition (Hu et al. 2006).

Recently, the identification and characterization of an assortment of Al resistance genes in plants allow us to understand in depth about the expression of these genes in response to Al stress. Al resistance gene expression is localized to the root tip, where Al toxicity is prevalent. This gene expression is often increased by Al stress. For e.g., Al-induced increase in expression of *ALMT* and *MATE* gene has been documented for *AtALMT*, *BnALMT1*, *SbMATE*, *ScALMT*, *ScFRDL2*, *OsFRDL4*, *VuMATE1* and *ZmMATE1* (Kochian et al. 2015). Transposon-mediated alteration of *TaMATE1B* expression in wheat is known to confer constitutive citrate efflux from root apices (Tovkach et al. 2013). OsART1 in rice (Yamaji et al. 2009) and AtSTOP1 in Arabidopsis (Iuchi et al. 2007) are the transcription factor family involved in the positive regulation of Al-induced expression of Al resistance genes. AtSTOP1 is reported to involve in the Al-induced expression of several *Arabidopsis* Al resistance genes, including *AtALMT1*, *AtMATE1* (Liu et al. 2009) and *AtALS3* (Sawaki et al. 2009).

Heat stress affect plant growth and development, physiological processes, and most importantly, yield (Hasanuzzaman et al. 2013). Heat stress generates excess reactive oxygen species (ROS), which leads to oxidative stress (Hasanuzzaman et al. 2012). At the molecular level, heat stress causes alterations in expression of genes for osmoprotectants, detoxifying enzymes, transporters, and regulatory proteins (Krasensky and Jonak 2012) and lead to modification of physiological and biochemical processes. During heat stress there is up-regulation of several heat inducible genes; commonly referred as “heat shock genes” (HSGs) which encode HSPs (Chang et al. 2007). This is the basis for the development of heat tolerance for acclimatization or adaptation against heat stress (Moreno and Orellana 2011). In tomato, *HSFA1* act as the ‘master regulator’ of the heat shock response and if this gene is suppressed, normal HSP production does not occur and the plant show extremely susceptible to heat stress (Mishra et al. 2002). In rice plants, overexpression of *sHSP17.7* gene confers tolerance to heat stress (Murakami et al. 2004).

The role of Late Embryogenesis Abundant (LEA) proteins in generating drought tolerant plants has been reported by many researchers. Xu et al. (1996) confirmed the role of LEA protein under water stress by overexpressing the *HVA1* gene from barley into rice plants. The *OsLEA3-1* gene in rice, *BnLEA4-1* and *JcLEA* gene in transgenic *Arabidopsis*, *TaLEA* gene in transgenic poplar, *SmLEA* gene in transgenic *Salvia miltiorrhiza* and *SiLEA14* gene in foxtail millet play a big role in activating the plant defense and adaptation under dehydration conditions (dos Reis et al. 2016).

Aquaporins are protein family that regulates the movement of water through intracellular and plasma membranes of plants and animals (dos Reis et al. 2016) and contributes to the maintenance of cell turgidity (Gomes et al. 2009). Several studies reported the role of aquaporins in response to stress in plants. For e.g. *TaTIP2;2* act as a negative regulator of drought and salinity and its response is independent of ABA (Xu et al. 2013). Khan et al. (2015) reported that the overexpression of *JcPIP2;7* gene help in faster water uptake through outer water channels and allow faster imbibition and thus, accelerate germination. Zhou et al. (2012) showed that *TaAQP7* gene enhance drought tolerance in transgenic tobacco by improving the water retention ability, reduce reactive oxygen species accumulation and membrane damage, and increase the antioxidants' activities. In *Arabidopsis thaliana*, MaPIP1;1 increased drought tolerance associated with decreased membrane injury and improved osmotic adjustment (Xu et al. 2014).

Submergence/water-logging is one of the major harmful abiotic stress for low-lying areas and crop losses due to waterlogging are considerably high (Ahmed et al. 2012). The stem elongation in deepwater rice varieties is mainly controlled by three QTLs. Hattori et al. 2009 found out that ethylene response factors *SNORKEL1* and *SNORKEL2* allow rice to adapt to deep water. The *Submergence-1* (*SUB1*) locus on chromosome 9 of rice (Xu and Mackill 1996) contains *SUB1A*, *SUB1B*, and *SUB1C*, all of which encode ethylene response factors and are upregulated under submergence, but only *SUB1A* is responsible for the water- logging tolerance (Xu et al. 2006). *SUB1A* expression is induced by drought and oxidative stress upon desubmergence and positively regulates the expressions of genes involved in ABA-mediated acclimation to drought conditions. Moreover, under oxidative stress, *SUB1A* promotes the genes expression related to the detoxification of reactive oxygen species (ROS) and reduces the accumulation of ROS (Fukao et al. 2011).

A large number of genes and transcription factors are upregulated in response to salinity in different plant species. The SOS gene family (e.g. *AtNHX1* and *H+-ATPase* gene) is believed to play a very intriguing role in ion homeostasis, thereby conferring salt tolerance (Liu et al. 2000). It is also found out that some ROS-scavenging and osmotic-regulating genes are also upregulated by salinity in some plant species. For instance, a continuous exposure to salinity for about 24 h in rice plant resulted in upregulation of glutathione S-transferase and ascorbate peroxidase, both of which play an active role in ROS scavenging (Kawasaki et al. 2001; Gupta and Huang 2014). A rice transcription factor gene *SALT-RESPONSIVE ERF1* (*SERF1*) that showed a root-specific induction upon salt and H_2O_2 treatment were identified by Schmidt et al. (2013).

Although phosphate (Pi) is an essential macronutrient that is necessary for many metabolic processes in plants including photosynthesis and respiration, it is a frequent limiting factor for plant productivity. Pi deficit elicits a complex array of morphological, physiological, and biochemical adaptations, collectively known as the Pi-starvation response (Plaxton 2004). Biochemical adaptations of Phosphate-starved plants include increased efficiency of cellular phosphate uptake through high-affinity Pi transporters, induction of Phosphate scavenging and recycling enzymes like acid phosphatase, secreted nuclease and phosphodiesterase, replacement of membrane phospholipids with non-phosphorus galacto- and sulfonyl-lipids, induction of metabolic phosphate recycling enzymes, enhanced excretion of organic acids like malate and citrate due to PEPCase induction, induction of alternative pathways of cytosolic glycolysis and Respiratory Electron Transport and induction of tonoplast H+-Pumping Pyrophosphatase (Plaxton and Carswell 1999).

In plants, heavy metal toxicity may lead to the over production of ROS, resulting in peroxidation of many vital constituents of the cell (Kumar et al. 2017). Plant defense mechanisms against heavy metal toxicity include Antioxidant defense system and cellular homeostasis. Many enzymatic antioxidants like superoxide dismutase (SOD), peroxidase (POD), catalase (CAT) and glutathione-s-transferase (GST) may efficiently convert the superoxide radicals into hydrogen peroxide and further into water and oxygen while low molecular weight non-enzymatic antioxidants like proline, ascorbic acid and glutathione may directly detoxify the ROS (Singh et al. 2015; Singh and Prasad 2014; Yadav et al. 2014; Xu et al. 2009). The exogenous application of proline elevates the level of endogenous proline under heavy metal stress and this helps in maintaining intracellular redox homeostasis potential (Hoque et al. 2008). Proline may enhance the tolerance level of plants by chelating heavy metals in the cytoplasm, regulating the water potential and maintaining osmotic adjustment through cellular homeostasis and reduce metal uptake (Singh et al. 2015).

Molecular Crosstalk and Epigenetic Memory for Stress Response and Adaptation

Recently omics data analysis and functional characterization of individual genes showed a convergence of signaling pathways for biotic and abiotic stress adaptation. Crop resilience to combined biotic and abiotic stress can be enhanced through thorough dissection of physiological and molecular crosstalk (Kissoudis et al. 2014). Whole genome expression meta-analysis experiments under different biotic and abiotic stress treatments revealed that a significant number of genes are commonly regulated under both stress conditions (Shaik and Ramakrishna 2014). These genes include response to ABA, Salicylic acid (SA), Jasmonic acid (JA) and ethylene which are major stress hormone imparting adaptation against both biotic and abiotic stress (Ma and Bohnert 2007). Moreover, several signaling pathways members including mitogen-activated protein kinase (MAPK), Ca^{2+}, ROS, Phospholipids, vesicle trafficking, mitochondrial functions and apoptosis were also induced under both biotic and abiotic stresses (Ma and Bohnert 2007; Kissoudis et al 2014). Transcription factors like WRKY, MYB, ERF, NAC and HSF are also involved in the crosstalk across stress treatment (Shaik and Ramakrishna 2013).

It has been recently shown that plants can remember past stress conditions and can utilize these memories to aid responses when these stresses reappear. The scene behind such memories is the epigenetic mechanisms in the control of gene expression through small RNAs, histone modifications and DNA methylation. Such epigenetic mechanisms are essential for stress memories and adaptation in plants (Kinoshita and Seki 2014). One good example for stress memory is ‘defense priming’ in which the plant displays a more rapid and robust response to pathogen or herbivore in a second attack compared with the first one (Pastor et al. 2013). Priming is also found for abiotic stress like drought (Ding et al. 2012).

Defence Mechanisms of Plants During Environmental Stress

Plants are constantly confronted to various abiotic and biotic stresses that seriously reduce their productivity and growth. Plants have a number of inherent and adaptive mechanisms to cope with stresses in their environment, which include such physical conditions as water (too much as well as drought), temperature (hot and cold), saline soils and oxygen deprivation, as well coping with biotic stresses such as insect pest and pathogens. Exposure of plants to various biotic and abiotic stress induces a disruption and physiological imbalance and thus leading to a reduction in fitness and productivity (Atkinson and Urwin 2012). Abiotic stress is the primary cause of huge loss of cop plants worldwide and resulting growth reduction cane reach >50% in most plant species (Rejeb et al. 2014; Surekha et al. 2015; Wani and Sah 2014). Other than abiotic stress, biotic stresses such as insect pest and pathogens also cause huge loss in term of yield and productivity. The occurrence of simultaneous abiotic and biotic stresses in natural environment presents an added degree of complexity, as the responses to these complex stresses are largely controlled by different hormone signaling pathways that may interact and inhibit one another. Often exposure of plants to long term abiotic stress can weaken the tissue/organs and cause enhanced susceptibility (Goel et al. 2008). Long exposure of biotic stress by insect pest and pathogen also increases the effect of abiotic stress such as water deficit and others (Englishloeb 1990; Khan and Khan 1996). Evidences also suggest that the climate change will also expand the host range of pathogens with increased chances of virulent strain development (Garrett et al. 2006). The ever changing climatic factors has further increases predisposition of complex nature of biotic and abiotic. Therefore, the occurrence of combined biotic and abiotic stress is likely to be higher in future.

Abiotic Stress and Defense Mechanism in Plants

A number of abiotic stresses such as extreme temperature (drought, flooding), high light intensity, osmotic stresses, heavy metals and a number of herbicides and toxins lead to over production of reactive oxygen species (ROS) including H_2O_2 causing extensive cellular damage and inhibition of photosynthesis (Wani and Gosal 2010; Das et al 2016).

Drought

Drought stress has been reported to severely reduce germination and seedling stand (Kaya et al. 2006) in different crop plant of *rainfed* ecology. Drought stress is characterized by reduction of water content, reduced leaf water potential and turgor loss, closure of stomata and decrease in cell growth and enlargement. Acute shortage of water in tissues may result in the arrest of photosynthesis, disturbance of metabolism and finally the death of plant. The adverse effect of drought on plant structure, behavior and function such as xylem embolism, reduced carbohydrate pool size, leaf and fine root production, on the ability of plants to resist pathogen attacks, the impacts on soil microbial dynamics, decomposition and nutrient supply processes, and shifting competitive abilities between plant species could not be underestimated (Ciais et al. 2005).

Mechanism of Drought Tolerance

Plants under drought stress survive by the means of several morphological, biochemical and physiological traits (Wani et al. 2008; Gosal et al. 2009). Drought tolerance of a crop plant has inherent ability to grow, flower and display economic yield under suboptimal water status. Drought stress negatively affects the water relations of plants at cellular, tissue and organ levels, causing specific as well as unspecific reactions, damage and adaptation reactions (Beck et al. 2007). Drought stress for a prolonged period act as a limiting factor at the initial phase of plant growth and establishment.

Morphological Mechanism

Water deficit stress mostly reduced leaf size and growth and in turns the leaf areas in many species of plant like Populus (Wullschleger et al. 2005), soybean (Zhang et al. 2004) and many other species (Farooq et al. 2009). Production of ramified root system under drought is important to above ground dry mass and the plant species or varieties of a species have great differences in the production of roots. The development extensive root system enhances the water uptake and maintains the osmotic pressure through higher proline levels in Phoenix dactylifera (Djibril et al. 2005). Reduced biomass has been recorded under water stressed soybean (Specht et al. 2001), Poncirus trifoliatae seedlings (Wu et al. 2008), common bean and green gram (Webber et al. 2006). Drought stress affects the growth, fresh dry weight and harvestable index in a number of plant species, but the quantitative effect may vary from species to species or varieties to varieties.

Drought Escape

Drought escape occurs when plants develop rapidly and reproduce before drought conditions become severe. Cession of vegetative growth may or may not accompany a drought escape response. Time of flowering is an important aspect related to drought adaptation, where a short life cycle can lead to drought escape (Araus 2002). Drought escape as an adaptive strategy comes from temporally replicated series of observations examining changes in populations following drought events. The genetic base under lying the variation in drought escape has been elucidated in a number of species, although the specific genes/QTLs and their physiological aspects remain to be characterized. Breeding of short-duration varieties has been an effective and viable strategy for minimizing yield loss from reproductive phase drought (Terminal drought).

Drought Avoidance

Drought avoidance consists of a mechanism that maintains relatively high tissue water potential despite a shortage of moisture status in soil. It is performed by maintenance of turgor through deep root system, stomatal control of transpiration and by reduction of water loss by reduced epidermal layers (Kavar et al. 2007). Drought avoidance adaptations can occur through a variety of morphological and physiological traits. Genotypes that adapted to xeric conditions may have lower specific leaf area, greater succulence (Eggli and Nyffeler 2009), increased leaf reflectance (Ehleringer et al. 1976), accentuated leaf lobing (Talbert and Holch 1957) with altered stomatal size and density (Masle et al. 2005) The root character such as root biomass, root length, root angle, root density and root depth are the major drought avoidance traits that contribute to final yield under terminal drought environments. Drought avoidance can evolve a constitute change in water use efficiency or as plastic response under moisture stress or other environment cues. Presently much research has been focused on transpiration efficiency despite the existence of other mechanism of drought avoidance. Therefore it is need to emphasize more on the all possible mechanism for drought avoidance.

Phenotypic Flexibility/Plasticity

Phenotypic plasticity is unique quality of genotype that change their phenotype expressed by a single genotype in different environments. At morphological level, the shoot and root biomass are the most affected and both are the key traits for plant adaptation under drought. Certain morphological traits such as leaf pubescence, root length, trichome and cuticle which help to protect the plants from drought stress. Selection for a deep and extensive root system has been practiced to enhance productivity of food legumes under moisture stress conditions as it can optimize the capacity to acquire water (Subbarao et al. 1995). Intra-specific and inter-specific variation reported in different crop plant for specific morphological traits contributing in drought stress.

Physiological Mechanism

Inadequate water availability in drought prone environment often affects growth and productivity of crops by reducing its water potential and turgor (Kiani et al. 2007; Gosal et al. 2010). Drought tolerant plants have several physiological mechanisms such as Osmotic adjustment, osmo-protection, antioxidation and scavenging defense system which operate at different stage of growth and development. Osmotic adjustment is the main component of plant at cellular level to minimize the damage caused by drought stress in crop plants (Blum 2005). Under drought, excess accumulation of compatible solutes as proline, amino acid and glycine betaine occur (Farooq et al. 2008; Wani et al. 2013b). Drought alters the endogenous growth substances and creates imbalance of hormonal level and ultimately lead to change in metabolic system in stress induced plants.

Antioxidant Defense Mechanism

Under drought stress conditions cell or tissue has exposed for oxidative stress with production of reactive oxygen species. Stress lead to decline in CO_2 influx and stomatal closure. Reactive oxygen species (ROS) such as H_2O_2 (hydrogen peroxide), O_2^- (superoxide) and OH (hydroxyl) radical produced under stress through enhanced leakage of electrons to molecular oxygen (Arora et al. 2002). Oxidative stress damages the plants by oxidizing photosynthetic pigments, membrane lipids, proteins and nucleic acids (Yordanov et al. 2000). To keep the levels of active oxygen species under control, plants have non-enzymatic and enzymatic antioxidant systems to protect cells from oxidative damage (Mittler 2002). Non-enzymatic antioxidants include β-carotenes, ascorbic acid (AA), α-tocopherol. (α-toc), reduced glutathione (GSH) and enzymes including: superoxide dismutase (SOD), guaiacol peroxidase (POD), ascorbate peroxidase (APX), catalase (CAT), polyphenol oxidase (PPO) and glutathione reductase (GR).

Defense Mechanism for Water Logging in Plants

Waterlogging is a serious problem, which affects plant growth and development in low lying rainfed areas in large part of globe (Lone et al. 2016). The prime cause of plant damage under waterlogging is oxygen deprivation, which leads to problem in nutrient and water uptake and ultimately plants die when flooding prevails for longer duration. The reduction of oxygen content below optimal levels, termed hypoxia, is the most common form of stress in wet soils and occurs during short-term flooding when the roots are submerged. Oxygen deficiency generally leads to the substantial decline in net photosynthetic rate (Ashraf et al. 2011). Ethylene accumulates in flooded soils and in submerged plant parts as an adaptive mechanism under hypoxic root and shoot. Ethylene initiates act as regulatory mechanism for many adaptive molecular, chemical and morphological responses that allow the plant to avoid anaerobiosis by enhancing oxygen availability to the roots in a flooded or waterlogged soil conditions (Sairam et al 2008). Aerenchyma formation under submerged or waterlogged condition have been reported in a range of crop species such as, Trifolium subterraneum (Aschi-Smiti et al. 2004), soybean (Bacanamwo and Purcell 1999), wheat (Watkin et al. 1998), rice (Justin and Armstrong 1991), maize (Gunawardena et al. 2001), *Carex* spp. (Visser et al. 2000). Due to shift in energy production metabolism from aerobic to anaerobic mode the energy requirements of tissue is greatly restricted because of very few ATPs generated per molecule of glucose. This necessitates availability of comparatively higher amount of readily metabolizable sugar pool. At the DNA level, low oxygen induced genes have been identified and characterized by the presence of an anaerobic response element (ARE) in the promoter (Walker et al. 1984). Manipulation of gene expression under low oxygen stress in different crop species provides a new insight in order to develop high tolerant crop plants. In future, generation of genetically improved flood tolerant crop plants thought insertion, regulation and manipulation of different genes conferring different metabolic and physiological pathway is one of the most sustainable and viable straggles for low lyaing area or flood prone area.

Defense Mechanism for Heat

Presently global climate change has risen the temperature and it severely affects the growth and production of crops. Heat stress affects plant growth throughout its ontogeny however heat-threshold level varies considerably at different developmental stages. Heat stress also induces indirect injuries that include inactivation of enzymes, inhibition of protein synthesis, protein degradation and loss of membrane integrity (Howrath 2005). Major impact of high temperatures on shoot growth is a severe reduction in the first internode length resulting in premature death of plants (Hall 1992). The most noticeable effect of high temperatures on reproductive processes in tomato is the production of an exerted style (i.e., stigma is elongated beyond the anther cone), which may prevent self-pollination. Poor fruit set at high temperature has also been associated with low levels of carbohydrates and growth regulators released in plant sink tissues. Heat tolerance is the ability of the plants to maintain normal metabolic process and produces economic yields under high temperature. Heat stress causes an abrupt increase in the expression of stress-associated low molecular weight proteins which provide tolerance by stimulating the defense response in plants (Roy et al. 1993). Heat-shock proteins (Hsps) osmoprotectants and antioxidant enzymes are important defence mechanism in encountering heat stress in plants. Hsps play a role in stress signal transduction, protecting and repairing damaged proteins and membranes, protecting photosynthesis as well as regulating cellular redox state (Asthir 2015). Three classes of proteins, as distinguished by molecular weight, account for most HSPs, viz., HSP90, HSP70 and low molecular weight proteins of 15–30 kDa. HSP70 and HSP90 mRNAs can increase ten-fold, while low molecular weight (LMW) HSPs can increase as much as 200-fold under heat stress conditions (Wahid et al. 2007). The major sites of thermal damage are the oxygen-evolving complex (OEC) along with other associated cofactors in photosystem II, carbon fixation by Rubisco and the ATP-generating system. Heat stress also negatively interferes with electron transport and consequently leading to excessive production of reactive oxygen species (ROS) in plant cells. Apart from HSPs, there are a number of other ubiquitin proteins including cytosolic Cu/Zn-SOD and Mn-POD whose expressions are stimulated upon heat stress. The ability of the plant to cope with or adjust to the heat stress varies across and within species as well as at different developmental stages. Traditional breeding has greatly exploited the variability among the species for development of heat tolerance variety. Manipulations of the heat shock protein gene in transgenic plants have the potential to improve common abiotic stress tolerance and this may have a significant impact on the exploitation of the inherent genetic potential of agronomically important plants.

Defense Mechanism for Salinity/Alkalinity

Salinity is a major abiotic stress limiting the increase in the demand for food crops (Gupta and Huang 2014). Salinity stress involves changes in various, morphological, physiological, biochemical and metabolic processes, depending on severity and duration of the stress, and ultimately limits the crop production. Salinity in initial stage induces, osmotic stress in the which causes various physiological changes, such as interruption of membranes, nutrient imbalance, impairs the ability to detoxify reactive oxygen species (ROS), differences in the antioxidant enzymes and decreased photosynthetic activity, and decrease in stomatal aperture (). Under saline conditions, plants accumulate high Na+ concentration which interfere with uptake of K+ ions which is an essential element for plant growth and development (Dang et al. 2010). Under high salt concentration, plant develops different physiological and biochemical mechanisms for survival and growth. Principal mechanism includes ion homeostasis, compartmentalization, ion transport and uptake, biosysyntesis and accumulation of osmoprotectant, activation of antioxidant enzyme, generation of nitric oxide, synthesis of polyamines and hormone modulation (Pathak et al. 2014). Plants have inherent mechanisms to mitigate osmotic stress by reducing water loss while maximizing water uptake. plants generally minimize the adverse effects of Na+ stress by exclusion of Na+ from leaf tissues and by compartmentalization of Na+, mainly into vacuoles. Genetic transformation of plants for enhancing the salinity tolerance can be successfully executed by focusing on genes controlling ion transport, as regulation of Na+ uptake and compartmentalization is a critically important mechanism of plant survival under salinity stress. Several studies on model plant have identified allelic variation for sodium transporters genes viz., HKT1 (High-Affinity K+ Transporter1), SOS1 (Salt Overly Sensitive1) and NHX (Na+/H+ Exchanger) as crucial determinants of cellular Na+ homeostasis.

Defense Mechanism for Metal Toxicity

Heavy metals include the transition-metal elements essential to plant nutrition, iron (Fe), zinc (Zn), manganese (Mn), copper (Cu), nickel (Ni) and molybdenum (Mo), cobalt (Co), which is required for nitrogen fixation in legumes, and the non-essential elements, chromium (Cr), cadmium (Cd), mercury (Hg) and lead (Pb). All these elements if exceed its threshold level cause toxic to crop plants. The presence of toxic heavy metals (HMs), is one important factor that can cause damage to plants by altering metabolic and plant physiological and metabolic processes (Farid et al. 2013). Symptoms associated with metal toxicity includes, reduction in plant growth including leaf chlorosis, necrosis, turgor loss, a decrease in the rate of seed germination, and a dysfunctional photosynthetic apparatus, often correlated with plant death. Some crops plants appear to tolerate metals either by excluding them from the shoot or by accumulating metals in older leaves as similar in case of salinity tolerance. Some plant tolerates high concentration of metals even up to four times than normal termed as hyperaccumulators. Hyperaccumulators are plant are the actual metal tolerant plant and frequently occurs in at least 500 plant species (or about 0.2% of taxa), and accumulate metals that include aluminium, cadmium, copper, lead, nickel and zinc (Hodson 2012). Antioxidant defenses components including SOD, ascorbate peroxidase (APX), peroxiredoxins (PRXs) and catalase (CAT). Glutathione (GSH) and ascorbate have a major role in defense against oxidative damage caused by reactive oxygen species that generated as a result of metal toxicity. Besides ROS and antioxidants, the other mechanisms of metal toxicity tolerance include, complexion, chelation and compartmentation. Classic genetic demonstrated many genes/QTLs are involved in HM uptake, translocation, sequestration, chemical modification, and tolerance. Role of regulatory genes other than structural genes are more important for simultaneously induction of many HM-related genes, in order to enhance heavy metal (HM) phytoremediation capacity (Hossain et al. 2012) Overexpression of single or in combination of genes are a possible strategy for genetic engineering. Genetic engineering would act as an efficient mechanism or tools for phytoremediation of crop plants by engineering of transporter, uptake and regulatory genes.

Biotic Stress

Pathogens

Biotic stresses result from an array of potential pathogens: fungi, bacteria, nematodes and insects intercept the photosynthate produced by plants, and viruses use replication machinery at the host's expense. Plants, in turn, have evolved sophisticated mechanisms to perceive such attacks, and to translate that perception into an adaptive response (Jeffery et al. 2001).

Passive Defenses

Potential plant pathogens must overcome the physical barrier presented by healthy plants to gain access to the nutrients or replication machinery available within the host cell. These barriers may be physical (the cuticle, cell wall, stomatal aperture or lenticel) or chemical (including inhibitory compounds or the absence of stimulatory compounds needed for pathogen development). Saprophytes lack the ability to penetrate these natural barriers (Guest and Brown 1997).

Physical Barriers

The importance of the cuticle as a barrier to penetration has been demonstrated by the dependence of many pathogens on adhesion and the subsequent release of cutin-degrading enzymes at the time of penetration. Although cutin-degrading enzymes are also secreted by many saprophytic fungi and bacteria, their primary activity is to allow access to cellulose in plant cell walls as a nutritional substrate. Different forms of cutin-degrading enzymes are used by pathogens to puncture the cell wall. The activity of this type of cutinolytic enzyme in isolates of *Fusarrum solani*, F. sp. *pisi* is directly related to their aggressiveness on pea stems, indicating that pathogens unable to dissolve the cuticle at the point of penetration are excluded (David and John 1997).

The cuticle provides a physical barrier against water loss and protects against irradiation, xenobiotics, and pathogens. Components of the cuticle are perceived by invading fungi and activate developmental processes during pathogenesis. In addition, cuticle alterations of various types induce a syndrome of reactions that often results in resistance to necrotrophs (Serrano et al. 2014). These are important factors in the resistance of some plants to certain pathogens by making direct penetration fungal pathogens difficult or impossible. Many pathogenic fungi and bacteria enter plants only through stomata. The structure of stomata e.g. a very narrow entrance and broad, elevated guard cells, may confer resistance to some varieties against certain bacterial pathogens (Tom Schultz 2006). Thicker, tougher cell walls could be associated with adult plant resistance. Some pathogens such as *Puccinia graminis* only infect young barberry leaves with thin cuticles and the germ tubes emerging from basidiospores do not penetrate thicker cuticles on mature leaves. Similarly, the ability of *Taphrina dejorrnans* to infect only young, newly unfolded leaves has been attributed to the inability of germ tubes to penetrate the thicker cuticles of older leaves. The presence of secondary cell walls in sclerenchyma, xylem or older plant tissue often retards pathogen development, leading, for example, to angular leaf spots where pathogen spread is restricted by leaf veins. Thick cuticles may physically prevent the eruption of sporophores and release of spores. However, most experimental evidence suggests that toughened cuticles and cell walls are just one of the many factors that contribute to resistance (David and John 1997).

Waxy cuticles and vertically oriented leaves may prevent the formation of moisture films on leaf surfaces. Dry leaf surfaces inhibit infection by pathogens such as bacteria, nematodes and fungal zoospores that require a film of water for motility. Fungal spores might also be inhibited because most require moisture for germination. This must be balanced with the fact that vertically oriented leaves are more prone to impaction by wind-borne pathogen propagules and are likely to face higher inoculum levels compared with those that are horizontally oriented (Guest and Brown 1997).

Pathogens enter the host by direct penetration through natural openings or are introduced by vectors. Bacteria and fungi are capable of triggering stomatal closure through pathogen-associated molecular patterns (PAMPs), which prevents penetration through these pores. Therefore, the stomata can be considered part of the plant innate immune response. Some pathogens have evolved mechanisms to evade stomatal defense. The bacterial pathogen *Xanthomonas campestris* pv. *campestris* (Xcc), which infects plants of the Brassicaceae family mainly through hydathodes, has also been reported to infect plants through stomata. A recent report shows that penetration of Xcc in Arabidopsis leaves through stomata depends on a secreted small molecule whose synthesis is under control of the rpf/diffusible signal factor (DSF) cell-to-cell signaling system, which also controls genes involved in biofilm formation and pathogenesis. The same reports shows that Arabidopsis ROS- and PAMP-activated MAP kinase 3 (MPK3) is essential for stomatal innate response (Gudesblat et al. 2009). The black pod pathogen, *Phytophthora palmiuora*, enters cocoa pods through stomata. Cocoa genotypes that produce pods with few, relatively smaller stomata, allow fewer lesions to establish than genotypes with more numerous, larger stomata. Not surprisingly, as the pathogen enters through stomatal pores, there is no correlation between cuticle thickness or pod case hardness and resistance to black pod. The bacterium that causes citrus canker, *Xanthomonas campestris* pv. *citri*, enters grapefruit through open stomata. Mandarins are resistant because their stomata are too small to allow entry of the bacterium. Similarly, lenticels that suberise rapidly so that their size is reduced may physically exclude pathogens such as Streptomyces scabies, the cause of common scab of potato (David and John 1997).

Lignifications: Lignified cell wall provide effective barrier to hyphal penetration. Lignin is a highly branched heterogeneous polymer found principally in the secondary cell walls of plants, although primary walls can also become lignified. It consists of hundreds or thousands of phenolic monomers and is a primary component of wood. Because it is insoluble, rigid, and virtually indigestible, lignin provides an excellent physical barrier against pathogen attack. Lignin acts as impermeable barrier for free movement of nutrient causing starvation of pathogen. Some examples of lignin containing crops and fungi inhibited include *Peronospora parasitica*, *Alternaria japonica* Potato: *Phytophtora infestans* Wheat: *Septoria nodorum* Cucumber: *Cladosporium cucumerium*, *Colletorichum lagenarium* Carrot: *Botrytis cineria* (Jones and Dangl 2006).

Suberization: In several plants the infected cells are surrounded by suberized cells, thus, isolating them from healthy tissue. Corky layer formation is a part of natural healing system of plants. e.g. common scab of potato and rot of sweet potato are good examples (Van Baarlen et al. 2007).

Abscission layers: It is a gap between host cell layers and devices for dropping–off older leaves and mature fruits. Plant may use this for defence mechanism also. i.e., To drop-off infected or invaded plant tissue or parts, along with pathogen. Shot holes in leaves of fruit trees is a common feature.

Tyloses: The tyloses are formed by protrusion of xylem parachymatous cell walls, through pits, into xylem vessels. The size and number of tyloses physically block the vessel. The tyloses are inductively formed much ahead of infection, thus blocking the spread of pathogen. It suggests biochemical elicitors and movement of tyloses inducing facto (TIF) up the stem. e.g. Sweet potato: Fusarium oxysporum f. sp. Batatas (Jan et al. 2011; Dou and Zhou 2012).

Gum deposition: The gums and vascular gels quickly accumulate and fill the intercellular spaces or within the cell surroundings the infection thread and haustoria, which may starve or die.

Chemical Barriers

The pathogen may not be provided with the required nutrients by the plants and thus resist infection. The compounds in plant cells may stimulate or inhibit the development of pathogens. Some fungal resting spores such as *Spongospora subterranea* (powdery scab of potato), *Urocystis agropyri* (flag or leaf smut of wheat) and *Plasmodiophora brassicae* (club root of crucifers) and eggs of the potato cyst nematode, *Globodera rostochiensis*, require specific substances to stimulate germination or hatching. These are provided in secretions from certain plants, including potential hosts. Plants that fail to secrete these stimulators are resistant by default (David and John 1997).

Plant chemicals can be divided into two major categories: primary metabolites and secondary metabolites. Primary metabolites are substances produced by all plant cells that are directly involved in growth, development, or reproduction. Examples include sugars, proteins, amino acids, and nucleic acids. Secondary metabolites are not directly involved in growth or reproduction but they are often involved with plant defense. These compounds usually belong to one of three large chemical classes: terpenoids, phenolics, and alkaloids (Brian and Gwyn 2008).

The name phytoanticipins was coined by J.W. Mansfield. "Phytoanticipins are low molecular weight, antimicrobial compounds that are present in plants before challenge by microorganisms or are produced after infection solely from preexisting constituents" (VanEtten et al. 1994). Phytoanticipins may be excreted into the external environment (e.g. rhizosphere or phylloplane), accumulate in dead cells or they may be sequestered in vacuoles in an inactive form. The dead cells of brown onion skins contain the quinones, catechol and protocatechuic acid, which inhibit germination of spores of the smudge pathogen, *Colletotrichum circinans*, and the neck rot pathogen, *Botrytis cinerea*; white onions do not produce these compounds and are susceptible to smudge. *Aspergillus niger* is insensitive to these inhibitors and attacks both white and brown onions. Avocado rootstocks resistant to root rot caused by *Phytophthora cinnamomi* secrete borbinol, an antimicrobial phenolic compound, into the rhizosphere. The secretion of nematode-inhibiting substances into the rhizosphere surrounding asparagus and marigold roots has already been mentioned. Symptoms of anthracnose of avocado, caused by *Colletotrichum gloeosporiordes*, only develop on ripe fruit. The peel of unripe avocado fruit contains antifungal lipids called dienes that prevent appressorial germination. As these dienes are gradually metabolised during fruit ripening to less toxic compounds, quiescent appressoria germinate and susceptibility to anthracnose increases. In anthracnose-resistant cultivars, diene breakdown is blocked following infection, so that antifungal levels are sustained for longer periods. The resistance of immature apples and pears to scab, caused by *Venturia inaequalis* and *V. pirina* respectively, correlates with the presence of the phenolic compounds chlorogenic acid, phloridzin, arbutin and iso-chlorogenic acid in the outer layers of the fruit. These compounds also contribute to the bitter taste of unripe apples and pears and, as the fruit ripens and sweetens, it also becomes more susceptible to scab (David and John 1997).

One group of phytoanticipins, the saponins, are plant glycosides with surfactant (wetting agent) properties. Saponins bind sterols in pathogen cell membranes, destroying membrane integrity and function. In this way saponins are toxic to organisms containing sterols in their membranes (e.g. plants and fungi, but not Oomycota). Inactive saponin precursor molecules appear to be stored in vacuoles of intact plant cells, but hydrolase enzymes released following wounding or infection convert these precursors to active, antimicrobial forms. Several lines of evidence suggest that saponins are involved in disease resistance and host range determination. It appears that the ability of some pathogens to detoxiff specific saponins matches their host range. For example, a strain of the take-all pathogen that attacks oats as well as wheat and barley (*Gaeumannomyces graminis* var. *auenae*), releases the enzyme avenacinase. Avenacinase detoxifies the triterpenoid saponin, avenacin, found in epidermal cells of the roots of oat plants. Mutants in which the gene for avenacinase production has been deleted are sensitive to avenacin in vitro and are not pathogenic on oats, but remain pathogenic to wheat and barley. *Gaeumannomyces graminis* var. *tritici* lacks avenacinase and attacks wheat and barley, but not oat species containing avenacin. An oat species that does not produce avenacin, *Avena longiglumisis* susceptible to *Gaeumannomyces graminis* var. *tritici* (Osbourn 1994; David and John 1997). Another saponin, tomatine, contributes to the resistance of tomato leaves to *Botrytis cinerea* (Osbourn 1996). Some plant peptides also inhibit the development of fungi, bacteria, viruses and insects. They act as proteinase and polygalacturonase-inhibitors, as ribosome inhibitors or lectins. These inhibitors interfere with pathogen nutrition and retard their development, thus contributing to disease resistance. Because of their similarity to peptides called defensins found in insects and mammals, they have been termed plant defensins.

The first plant defensins were isolated from wheat *T. aestivum* and barley *Hordeum vulgare* and initially classified as γ-thionins. Plant defensins are small (ca.

5 kDa), basic, cysteine-rich peptides ranging from 45 to 54 amino acids, and are positively charged. Biological activities reported for plant defensins include anti-fungal, antibacterial, proteinase, and insect amylase inhibitor activities (Wijaya et al. 2000; Stotz et al. 2009). The plant defensins have quite diverse amino acid composition and conserved three-dimensional structure, which comprises a triple-stranded β-sheet with an α-helix in parallel stabilized by four disulfide bridges. Plant defensins are very similar to defense peptides of mammals and insects what suggest their ancient and conserved origin. Generally, plant defensins are composed by one subunit, being found in monomeric forms. On the other hand, the defensins from *Pachyrrhizus erosus* and other from *Vigna unguiculata* showed the ability to dimerism (Pelegrini and Franco 2005). The mode of action of plant defensins is still unclear and not all plant defensins have the same mode of action. Probable defensins used glucosylceramides as receptors for fungi cell membrane insertion. Then, repulsion of defensins into cell membrane by their positive charges leads to membrane disruption, membrane destabilization, and ion efflux (Pelegrini and Franco 2005). Plant defensins can be divided in two groups: (1) plant defensins that inhibit fungal growth through morphological distortions of the fungal hyphae and (2) plant defensins that inhibit fungal growth without morphological distortion (Hegedus and Marx 2013). Most plant defensins were isolated from seeds. In radish, defensin RS-AFPs represents 0.5% of total protein in seeds. Defensins were also isolated from leaves, pods, tubers, fruits, roots, bark, and floral organs of such plants as *Heuchera sanguinea* (Hs-AFp1), *Raphanus sativus* (Rs-AFP1), *Aesculus hippocastanum* (Ah-AMP1), Dahlia merckii (Dm-AMP1), and *Clitoria ternatea* (Ct-AMP1; De Lucca et al. 2005). Defensins are expressed during normal plant growth and development and induced by environmental factors and biotic and abiotic stress (Pestana-Calsa and Calsa 2011). The defensins gene induced upon pathogen infection has been identified in pea, tobacco, Arabidopsis, and spruce (Lay and Anderson 2005). Defensins may constitute up to 10% of the total proteins in cereal, legume and solanaceous seeds. Similar studies have shown defensins are also present in the outer cell layers of other plant organs such as flowers, leaves and tubers. While many defensins accumulate during normal plant development, others are induced, or their accumulation is enhanced, after wounding. Defensins, because of their anti-feeding activity against insects, provide a defence against insect-transmitted viruses (David and John 1997).

Rapid Active Defenses

Plant responses to infection are complex and there is no universal model or sequence of events that accurately describes the dynamics of resistance in the few interactions studied, let alone the vast majority of undescribed interactions. Almost every host-parasite interaction is unique in the details of the activation, localization, timing and magnitude of each component of the defense response. As previously stated, resistance is rarely absolute and whether a plant ends up being resistant or susceptible depends on the sum of many individual responses.

Changes in Membrane Function

Most studies on the earliest stages of the host-parasite interaction conclude that the host membrane is involved in pathogen recognition and signal transduction. Electrolyte leakage accompanies plant response to stresses, such as salinity, pathogen attack, drought, heavy metals, hyperthermia, and hypothermia; however, the mechanism and physiological role of this phenomenon have only recently been clarified. Accumulating evidence shows that electrolyte leakage is mainly related to K^+ efflux from plant cells, which is mediated by plasma membrane cation conductances (Demidchik et al. 2014). Membrane permeability changes rapidly following the exposure of plant cell suspension cultures to fungal and bacterial elicitors, usually leading to a loss of cellular electrolytes such as K+ and an uptake of H+. Most probably they are encoded by GORK, SKOR, and annexin genes. Hypothetically, cyclic nucleotide-gated channels and ionotropic glutamate receptors can also be involved. The stress-induced electrolyte leakage is usually accompanied by accumulation of reactive oxygen species (ROS) and often results in programmed cell death (PCD). Recent data strongly suggest that these reactions are linked to each other. ROS have been shown to activate GORK, SKOR, and annexins. ROS activated K+ efflux through GORK channels results in dramatic K+ loss from plant cells, which stimulates proteases and endonucleases, and promotes PCD. This mechanism is likely to trigger plant PCD under severe stress. However, in moderate stress conditions, K+ efflux could play an essential role as a 'metabolic switch' in anabolic reactions, stimulating catabolic processes and saving 'metabolic' energy for adaptation and repair needs. At the same time, there is often an influx of Ca^{2+}, a key intracellular signal in plants that is involved in the activation of enzymes and gene expression. Ca^{2+} signaling in plants functions in local and systemic communication. Rapid systemic signaling is activated in response to different stimuli including mechanical force, pathogen infection, and abiotic stresses and results in systemic propagation of Ca^{2+} and reactive oxygen species (ROS) waves. This allows plant cells to transmit long-distance signals via cell-to-cell communication (Steinhorst and Kudla 2013; Gilroy et al. 2014). The experimental blocking of Ca^{2+} transport across membranes in inoculated bean cells also inhibits gene activation and subsequent defence responses.

The Oxidative Burst

Rapid generation of superoxide and accumulation of H_2O_2 is a characteristic early feature of the hypersensitive response following perception of pathogen avirulence signals. Emerging data indicate that the oxidative burst reflects activation of a membrane-bound NADPH oxidase closely resembling that operating in activated neutrophils. The oxidants are not only direct protective agents, but H_2O_2 also functions as a substrate for oxidative cross-linking in the cell wall, as a threshold trigger for hypersensitive cell death, and as a diffusible signal for induction of cellular protectant genes in surrounding cells. Activation of the oxidative burst is a central component of a highly amplified and integrated signal system, also involving salicylic acid and perturbations of cytosolic Ca^{2+}, which underlies the expression of disease-resistance mechanisms (Lamb and Dixon 1997).

Reactive oxygen species (ROS) are produced as a normal product of plant cellular metabolism. Various environmental stresses lead to excessive production of ROS causing progressive oxidative damage and ultimately cell death. Despite their destructive activity, they are well-described second messengers in a variety of cellular processes, including conferment of tolerance to various environmental stresses. Whether ROS would serve as signaling molecules or could cause oxidative damage to the tissues depends on the delicate equilibrium between ROS production, and their scavenging. Efficient scavenging of ROS produced during various environmental stresses requires the action of several nonenzymatic as well as enzymatic antioxidants present in the tissues (Sharma et al. 2012).

Doke a plant pathologist at Nagoya University (Nagoya, Japan), first reported on the involvement of ROS in the plant–pathogen interaction, after observing that infection by *Phytophthora infestans* in potato tubers causes the generation of O_2^- at the host cells' plasma membrane (PM), only in the incompatible interactions. *P. infestans* is a typical pseudofungal species which is now classified within the class of Oomycetes (Subclass, Peronosporomycetidae; Order, Pythiales) that causes the serious potato disease known as late blight or potato blight. A series of Doke's works demonstrated for the first time that ROS generation occurs in plants upon attacks by a pathogenic microorganism and that the members of ROS possibly function as the chemical signals required for induction of hypersensitive response (HR) as typified by host cell death, now often referred to as plant apoptosis. Doke also demonstrated that the treatment of potato tuber protoplasts with the cell wall preparation from *P. infestans* effectively induces the oxidative burst, suggesting that chemical components derived from pathogenic microorganisms (elicitors) trigger the burst of ROS production in order to stimulate the plant defense mechanisms (Kawano and Bouteau 2013).

The work of Doke and his colleagues undergo a two-step oxidative burst. The first burst rapidly follows wounding and inoculation, while a much larger burst in incompatible interactions immediately precedes hypersensitive cell death. Since then, an oxidative burst has been described in a range of plant-fungal and plant bacterial interactions. The rapid oxidative burst generates levels of reactive oxygen species that initiate membrane lipid peroxidation and cell death. The oxidative burst in plants is associated with the release of local and systemic signals that trigger gene expression and the oxidative cross-linking of host cell wall components. Levels of reactive oxygen species accumulate at the infection courts that are sufficient to kill micro-organisms in vitro. Experimental suppression of the oxidative burst shows that it is involved in initiating later defence responses. On the other hand, colonisation of avocado fruit by the necrotroph, *Botrytis cinerea*, apparently exploits the oxidative burst to kill host cells in advance of invasion (David and John 1997).

Cell Wall Reinforcement

Prospective plant pathogens must overcome the physical barrier presented by the plant cell wall. In addition to being a preformed, passive barrier limiting access of pathogens to plant cells, the cell wall is actively remodeled and reinforced specifically at discrete sites of interaction with potentially pathogenic microbes. Active reinforcement of the cell wall through the deposition of cell wall appositions, referred to as papillae, is an early response to perception of numerous categories of pathogens including fungi and bacteria. Rapid deposition of papillae is generally correlated with resistance to fungal pathogens that attempt to penetrate plant cell walls for the establishment of feeding structures. Despite the ubiquity and apparent importance of this early defense response, relatively little is known about the underlying molecular mechanisms and cellular processes involved in the targeting and assembly of papillae (Underwood 2012). The rapid deposition of papillae is a common response of cereals to attempted penetration of epidermal cells by the powdery mildew fungus (*Blumeria graminis*). Papillae in resistant cultivars form more rapidly and are more difficult to penetrate, than those formed by susceptible cultivars. As a result, haustorial development is prevented. Lignitubers are lignified callose deposits that ensheath invading hyphal tips. Lignitubers have been observed in both resistant and susceptible cereals following challenge by the take-all pathogen, *Gaeumannomyces graminis*, demonstrating again the importance of timing-the more rapid the response, the more likely it is to succeed (David and John 1997).

The most abundant structural proteins in plant cell walls are the hydroxyproline rich glycoproteins (HRGPs) which involved in the organization of secondary cell wall thickening. They are induced as defense responses, specifically in incompatible plant-pathogen interactions (Davis et al. 1997). Functionally, there is evidence that HRGPs act as impenetrable physical barriers against pathogen ingress and that they immobilize the pathogens by binding to their negatively charged surfaces, making cell walls tougher (Leach et al. 1982; Mazau et al. 1987; Cassab and Varner 1988). HRGPs include extensins, arabinogalactan proteins (AGPs), proline/hydroxyproline-rich proteins (P/HRGPs), and solanaceous lectins (Sommer-Knudsen et al. 1998). Among these, the P/HRGPs and extensins are known to be insoluble proteins whereas AGPs are soluble. Most of the earlier studies on infection-induced accumulation of HRGPs were carried out in dicotyledons, e.g. French bean infected with *Colletotrichum lindemuthianum* (Templeton et al. 1990; Millar et al. 1992; Bindschedler et al. 2006), lettuce infected with *Pseudomonas syringae* (Bestwick et al. 1995), French bean infected with *Xanthomonas campestris* (Brown et al. 1998) and tobacco leaves infected by *Erysiphe cichoracearum* (Raggi 2000). The HRGPs accumulation in monocotyledons as a response to pathogens has also been reported, e.g. in pearl millet with *Sclerospora graminicola* (Shailasree et al. 2004), in wheat infected by *Fusarium culmorum* (Kang and Buchenauer 2003) and in maize treated with a *Fusarium monilifore*-derived elicitor (García Muniz et al. 1998).

The plant cell wall constitutes one of the first lines of defense against pathogen invasion, and peroxidases are key enzymes in the wall-building processes. These processes include peroxidase-mediated oxidation of hydroxycinnamyl alcohols into free radical intermediates (Gross 1980), phenol oxidation (Schmid and Feucht 1980), polysaccharide cross-linking (Fry 1986), crosslinking of extensin monomers (Everdeen et al. 1988), lignification (Grisebach 1981; Walter 1992), and suberization (Espelie and Kolattukudy 1985; Espelie et al. 1986). The rapid deposition of lignin and suberin following infection is associated with resistance to non-pathogens and to avirulent pathogens in many plants, including cereals, Solanaceae, brassicas, melons and carrots. Hydrogen peroxide, released during the oxidative burst following pathogen challenge, causes extensive cross-linking between hydroxyproline-rich glycoproteins and other cell wall components. The oxidative cross-linking of cell wall structural proteins is thought to be a rapid defense response to strengthen the cell wall against the invading pathogen prior to the activation of other post-transcription dependent defense responses such as accumulation of pathogenesis-related proteins (PR-proteins) and defense genes expression (Bradley et al. 1992; Brisson et al. 1994). This oxidative cross-linking at the cell surfaces following fungal infection is known to be driven by peroxidases and H_2O_2, which rapidly accumulates from an oxidative burst (Brisson et al. 1994; Ribeiro et al. 2006). HRGPs cause cell wall strengthening by the formation of intra and inter-molecular cross-links resulting in their rapid insolubilization in cell walls. The cross-linking process is reported to involve isodityrosine (IDT) links (Cooper and Varner 1984; Epstein and Lamport 1984; Fry 1986; Smallhood et al. 1995). The whole process strongly decreases solubility of HRGPs meaning that they lose their extractability by salts and SDS (Bradley et al. 1992). Finally, this leads to the formation of a cross-linked mesh of defined porosity interpenetrated by a cellulose microfibrilar wrap (Epstein and Lamport 1984).

Lignification renders the cell wall more resistant to mechanical pressure applied during penetration by fungal appressoria as well as more water resistant and thus less accessible to cell wall-degrading enzymes. Lignin also binds to hyphal tips and bacterial cells, preventing further growth and movement and restricting the diffusion of pathogen enzymes and toxins and the uptake of water and nutrients by the pathogen. For examples resistance of cucumber to *Cladosporium cucumerinum* and *Colletotrichum lagenarium* is due to deposition of lignin in cell wall (Hammerschmidt and Kuc 1995). It is mentioned previously that Trichoderma GT3-2, Fusarium GF18-3 and Phoma GS8-2 were able to induce resistance in melon plants against *Colletotrichum orbiculare*, the causal agent of cucumber anthracnose and found lignin deposition at the point of pathogen infection in the epidermal tissues of cucumber hypocotyls (Koike et al. 2001).

Furthermore, precursor molecules and free radicals produced during lignin biosynthesis are toxic to pathogens and inactivate pathogen enzymes, toxins, elicitors or suppressors. The effect of lignin can be further enhanced by the release of reactive oxygen species and the activation of phenol oxidase enzymes that convert phenolic compounds to more toxic complex polymerised phenolics and quinones during the defence response. The evidence that cell wall reinforcements are important components of plant disease resistance can be summarised as follows:

- Their deposition often coincides with failed penetration and sometimes precedes the cessation of pathogen growth.
- Reinforcements in resistant hosts are larger, form more quickly (often before penetration) and are more dense than those formed by susceptible hosts.
- Experimental attempts to re-penetrate induced reinforcements usually fail.
- Inhibition of lignin or callose biosynthesis enhances penetration efficiency.

However, the deposition of cell wall reinforcements is not always associated with disease resistance. Clearly, cell wall reinforcements contribute to resistance and cell repair but are not always sufficient on their own to prevent infection (David and John 1997).

Hypersensitive Cell Death

Cell death has a central role in innate immune responses in both plants and animals. Besides sharing striking convergences and similarities in the overall evolutionary organization of their innate immune systems, both plants and animals can respond to infection and pathogen recognition with programmed cell death. The fact that plant and animal pathogens have evolved strategies to subvert specific cell death modalities emphasizes the essential role of cell death during immune responses. The hypersensitive response (HR) cell death in plants displays morphological features, molecular architectures and mechanisms reminiscent of different inflammatory cell death types in animals (pyroptosis and necroptosis) (Coll et al. 2011). The term hypersensitivity indicates that the host cells are ‘over- (hyper-) sensitive’ to the presence of the pathogen. Host cells suicide in the presence of the pathogen, preventing further spread of the infection. The first observations of HR date back to 1902 in the wheat-*Puccinia glumarum* pathosystem (Ward 1902), and the counter-intuitive term ‘hypersensitiveness’ was coined in 1915 (Stakman 1915), to describe a pathogen-triggered cell death reaction that correlated with disease resistance in wheat infected with *Puccinia graminis*. Morphologically, HR is a specific and unique type of cell death. Its hallmarks, some of which are typical for different forms of animal cell death, include cytoplasmic shrinkage, chromatin condensation, mitochondrial swelling, combined with other characteristics that are plant specific, such as vacuolization and chloroplast disruption during the final stages (Mur et al. 2008).

The chloroplast has a central role in defense responses and HR in plants. First, it constitutes a very important source of defense signaling molecules such as reactive oxygen species (ROS), reactive nitrogen oxide intermediates (NOI) and the defense hormones salicylic acid (SA) and jasmonic acid (JA). Second, in many cases, light is required for HR development. Third, several pathogen effectors have chloroplast localization signals (Guttman et al. 2002), and in some cases they have been shown to suppress immunity (Fu et al. 2007; Jelenska et al. 2007).

In plants, the molecular events that lead to HR during effector-triggered immunity (ETI) are partly overlapping with those associated with (Microbial-associated molecular patterns) MAMP-triggered immunity (MTI), including accumulation of SA, ROS and NOI, activation of mitogen-activated protein kinase(MAPK) cascades, changes in intracellular calcium levels, transcriptional reprogramming and synthesis of antimicrobial compounds (Mur et al. 2008). Compared with MTI, ETI is typically an accelerated and amplified response, suggesting that quantitative rather than qualitative differences account for HR induction (Jones and Dangl 2006).

Increased ROS levels are a hallmark of necroptosis and may be one of the main causes of necroptotic cell death. Enhanced ROS production during necroptosis can be mediated by mitochondria, due to a RIP3-dependent increase in energy metabolism (Zhang et al. 2009), and/or by the NADPH oxidase NOX1, which is recruited to the plasma membrane by RIP1 (Kim et al. 2007). Hypersensitive cell death in plant cells shares many features in common with apoptosis, or programmed cell death, observed during development of defense against disease in animals. In plants, apoplastic ROS (superoxide) generated by the plasma membrane NADPH oxidases are essential for HR development and activation of systemic immunity (Torres and Dangl 2005), drawing a possible mechanistic connection between these two types of cell death. ROS produced in other plant organelles as the chloroplast, mitochondria and peroxisomes also contribute to the HR response and, in fact, compartmentalization might be essential for ROS signaling functions during defense (Torres 2010).

Necroptosis has a pivotal role in inflammation and immunity. Similar to pyroptotic cells, necroptotic cells secrete a broad array of pro-inflammatory molecules that signal through PRRs (Zitvogel et al. 2010). Necroptosis has been reported to occur in response to infection by certain viruses that block apoptosis in the host cell as a colonization strategy (Challa and Chan 2010). Because of the pro-inflammatory nature of necroptosis, it may constitute not only a backup mechanism for virus clearance when apoptosis is inhibited, but also a way to engage the immune system leading to a systemic response.

The emerging similarities between hypersensitive cell death in plants and apoptosis in animal cells suggest that cell suicide is an ancient defense response. It is not always easy to conclude from research data whether host cell death is a consequence of murder or suicide. Recent experiments have shown that in many host-parasite interactions hypersensitive cell death precedes pathogen death, regardless of whether biotrophic or necrotrophic pathogens were involved. In some interactions however, disease resistance does not depend on hypersensitive cell death. The success of hypersensitive cell death as a resistance mechanism in individual host-parasite interactions depends on the nutritional requirements of the pathogen and on the timing, location and magnitude of the host response in relation to pathogen development. In some interactions the rapid suicide of challenged host cells undoubtedly restricts pathogen development, contributing to the overall defence response (David and John 1997).

Phytoalexins

Phytoalexins are low molecular weight antimicrobial compounds that are produced by plants as a response to biotic and abiotic stresses. As such they take part in an intricate defense system which enables plants to control invading microorganisms. In the 1950s, research on phytoalexins started with progress in their biochemistry and bio-organic chemistry, resulting in the determination of their structure, their biological activity, as well as mechanisms of their synthesis and catabolism by microorganisms. Elucidation of the biosynthesis of numerous phytoalexins also permitted the use of molecular biology tools for the exploration of the genes encoding enzymes of their synthesis pathways and their regulators. This has led to potential applications for increasing plant resistance to diseases. Phytoalexins display an enormous diversity belonging to various chemical families such as for instance, phenolics, terpenoids, furanoacetylenes, steroid glycoalkaloids, sulfur-containing compounds and indoles (Jeandet 2015). An astonishing range of structures may be formed, as many as 25 substances in a single interaction. They are formed consistently within a given plant group; in a survey of 60 species of the legume tribe Vicieae, every member tested responded. Although phytoalexins have been most widely studied in the Leguminosae, they have been recorded in over 20 other families. These plants range from trees, through shrubs to herbs and from monocots to dicots. There may be limits to the distribution of this defense mechanism and results of exploring this response in further families will be presented (Harborne 1986). Between 1986, when the first cruciferous phytoalexins were reported, and 2011, the chemical structures, syntheses and antimicrobial activities of 44 cruciferous phytoalexins were reported (Pedras et al. 2011).

Phytoalexins are synthesized by either the cells adjacent to the infection site, the infected host cells or by the invading pathogen. It is thought that such infected cells produce some sort of signals which induces the adjacent cells to produce the phytoalexins, which are packaged in lipid vesicles and exported to the infected cell (Mogensen 2009). Consequently, the infected cell becomes a toxic micro-environment for the invading pathogen. Phytoalexin accumulation is often associated with hypersensitive cell death. Examples include medicarpin (alfalfa, Medicago sativa), rishitin (tomatoes and potatoes (the Solanaceae family), and camalexin, (*Arabidopsis thaliana*) (Jones and Dangl 2006; Cai et al. 2009).

Some plants, such as soybean and chickpea, synthesize phytoalexins upon infection, but convert a proportion into inactive sugar conjugates held in reserve in vacuoles. If the initial defense response fails to check pathogen growth, enzymes that cleave the sugar molecule are activated and the phytoalexin reserves are rapidly releases. Like other active defense responses, the success of phytoalexin accumulation depends on the speed, location and magnitude of the response. There is a good experimental correlation between resistance and rapid, localized phytoalexin accumulation in many host-parasite interactions. There is evidence that:

- Phytoalexins accumulate faster and to higher concentrations in resistant cultivars. In resistant plants, gene transcription begins within one hour of recognition, phytoalexins appear within four hours and concentrations peak to fungitoxic levels about 18–24 h after challenge. These events are delayed and more diffuse in susceptible plants.
- Phytoalexin biosynthesis is localized in cells immediately surrounding the infection court. There is no evidence that they disperse in the plant. Experiments using laser microprobe analysis, radioimmunoassay, hybridisation histochemistry and immunocytochemistry of the phytoalexin biosynthesis pathway have confirmed this in several host pathogen interactions.
- In a number of interactions, resistance is lost if phytoalexin biosynthesis is blocked by inhibitors of enzymes involved in the process of phytoalexin biosynthesis and is reduced in mutants that are slow to accumulate phytoalexins.
- Resistance is increased in plants transformed to express novel phytoalexins or if exogenous phytoalexins are applied. For example, although the biochemical precursor of resveratrol is widely distributed in the plant kingdom, only grapevine and peanut have the en4rme required to complete its synthesis. When the genes encoding this enzyme are transformed into tobacco, resveratrol is synthesized in response to infection.
- Phytoalexin synthesis is not universal among plants. Wheat and cucumber apparently do not produce phytoalexins, yet effectively resist most pathogenic fungi and bacteria. Nevertheless, in many interactions the rapid accumulation of toxic concentrations of phytoalexins at the infection court plays a decisive role in the expression of resistance (David and John 1997).

Delayed Active Defences

Pathogen containment and wound repair

While earlier responses retard the development of pathogens, later responses restrict their spread and contain the damage to host tissues. The ability of a plant to repair tissue damage may contribute to its ability to fight off-secondary infections by opportunistic pathogens. Infected areas of fleshy tissues, roots, fruits and bark are sealed by layers of cork cells with thick, suberised walls. Wound cork is produced by a secondary meristem, the cork cambium, formed from mature parenchyma tissue in response to the damage caused by infection. In some cases, such as in the response of potato tuber tissue to the powdery scab pathogen (*Spongospora subterranea*), cork barriers appear to seal the infected area and prevent further colonization by the pathogen. However in other interactions, including the response of brassicas to the leaf spot pathogen, *Alternaria brassicae*, cork layers do not restrict infection. Some pathogens induce plants to form abscission layers in which cork cambium develops around the infected area and extends from the upper to lower surface of the infected leaf. The infected areas fall out, leaving the classical 'shothole' symptom. Such pathogens include *Stigmina carpophila* and *Pseudomonas syringae* pv. *morsprunorum* on plum and Cercospora *beticola* on silverbeet. Wounded tree trunks often secrete gums that effectively seal the wound from opportunistic pathogens (David and John 1997).

Tyloses are outgrowths of vessel-associated parenchyma cells which protrude into the xylem vessel through pits and block the spread of pathogens (Beckman 1964; Talboys 1972; Grimault et al. 1994; Agrios 2005). They are formed during both compatible and incompatible interactions between the host and vascular wilt pathogens, although the time and extent of tylose formation significantly differs. Tyloses form much faster and more extensively in resistant plants when compared to susceptible plants (Grimault et al. 1994; Fradin and Thomma 2006). The formation of tyloses involves a cost to the plant, as they not only block the spread of the pathogen, but reduce the translocation of water, possibly causing wilt symptoms.

Pathogenesis-Related Proteins

During the massive shift in cellular metabolism and gene expression referred to earlier, plants synthesize many novel proteins following infection. Some of these novel proteins may be enzymes involved in phytoalexin biosynthesis and some may have no role in disease resistance at all. However, the 'pathogenesis-related proteins have β-glucanase, chitinase or lysozyme activity. Some are related to plant defensins while others are proteinase inhibitors that disrupt pathogen nutrition. Pathogenesis-related proteins are sometimes present in low levels before infection and are induced following stress, wounding or flowering, indicating that they may have a wider function in plant growth and development than just disease resistance (David and John 1997).

PR proteins are categorised into structurally homologous families. Some of these PR-protein families have direct antimicrobial activities, whereas for others, no intrinsic antimicrobial effects have been found yet, suggesting that those without intrinsic antimicrobial effects might have different functions. An important common feature of most antimicrobial PR proteins is their antifungal activity, although some of them also have antibacterial, insecticidal and antiviral properties. Originally, five main groups of PR proteins (PR-1 to PR-5) were characterised in tobacco. Since then, the number of PR protein groups has increased up to PR-17 across many plant species (Table 1) (Buonaurio et al. 2009). It has also been reported that several genes could be induced after AMF colonisation in host plants and are involved in plant defence against pathogens. These genes encode pathogenesis related proteins

Table 1 Families of pathogenesis related proteins (PRPs)

S. No.	Protein family	Protein activity	Targeted pathogen sites
1.	PR-1	Unknown	Active against oomycetes
2.	PR-2	1,3 β-glucanase	Cell wall glucan of fungi
3.	PR-3	Chitinases	Cell wall chitin of fungi
4.	PR-4	Chitinase type I, II	Active against oomycetes
5.	PR-5	Thaumatin	
6.	PR-6	Proteinase inhibitor	Active on nematodes and insects
7.	PR-7	endoproteinase	Microbial cell wall dissolution
8.	PR-8	Endochitinase with lysozyme activity	Cell wall chitin of fungi and mucopeptide cell wall of bacteria
9.	PR-9	Peroxidase	Strengthening of plant cell wall
10.	PR-10		
11.	PR-11	Endochitinase	Cell wall chitin of fungi
12.	PR-12	Defensin	Antifungal and antibacterial activity
13.	PR-13	Thionin	Antifungal and antibacterial activity
14.	PR-14	Lipid transfer proteins	Antifungal and antibacterial activity
15.	PR-15	Oxalate-oxidase	Produce H_2O_2 that inhibits microbes and also stimulates host defence
16.	PR-16	Oxalate-oxidase-like with super dismutase activity	Produce H_2O_2
17.	PR-17	Uncharacterized	Unknown

Source Niderman et al. (1995), Van Loon et al. (1998), Sels et al. (2008), Okushima et al. (2000)

such as PR-1a, β-1,3 glucanase and PR-10 in tomato, pea and parsley (Haneef Khan et al. 2010).

Indeed, defence-related genes that are activated by AMF are key players in the defence against several root pathogens. Previous studies have identified a large number of defence-related genes in both compatible and incompatible plant–pathogen interactions (Liu et al. 2007; Lehtonen et al. 2008). However, in potato plants challenged with *Rhizoctonia solani*, 24 induced genes related to cell defence were identified by microarray analysis (Lehtonen et al. 2008). These genes encode chitin-hydrolysing enzymes such as acidic chitinases of classes II, III and IV, members of the pathogenesis-related (PR) protein groups (including 1,3-bglucanase and lignin-catalysing peroxidases), osmotin-like proteins, defence-associated signalling kinases, host protein protecting substances and enzymes leading to phytoalexin accumulation (Van Loon et al. 1998; Lehtonen et al. 2008). In tobacco roots, AMF-induced defence genes encode isozymes catalysing to peroxidation and production of phytoalexins and phenolic compounds such as phenylalanine ammonia lyase (PAL) and peroxidase (Blilou et al. 2000). Recently, Liang et al. (2005) reported that foliar applied Si only produced physical barrier and osmotic effect, but root applied Si leaded to systemic acquired resistance when *Cucumis sativus* plants were infected by powdery mildew pathogen, *Podosphaera xanthii* (syn. *Sphaerotheca fuliginea*) and the production of pathogenesis-related proteins (PRs) (Ismail and Hijri 2012).

These novel proteins are accumulated maximally 7–10 days after infection, and indicate the attainment of the systemic acquired resistance by the plant. The PRPs accumulate in the intercellular spaces and in the vacuole. PRPs are recognized as markers of the systemic acquired resistance (SAR), and PR genes are involved in the list of the so-called SAR-genes. The term “SAR-genes” is used to collectively designate this family of nine genes whose expression is correlated with the onset of SAR. Seventeen families of PRPs have been officially recognized (Van Loon et al. 1998), most of which have enzymatic activities.

Systemic Acquired Resistance

Systemic acquired resistance (SAR) refers to a distinct signal transduction pathway that plays an important role in the ability of plants to defend themselves against pathogens. After the formation of a necrotic lesion, either as a part of the hypersensitive response (HR) or as a symptom of disease, the SAR pathway is activated. SAR activation results in the development of a broad-spectrum, systemic resistance (Hunt and Ryals 1996; Neuenschwander et al. 1996). Although SAR is interesting as a paradigm for signal transduction, it may have practical value as well. An understanding of the biochemical changes leading to the resistance state could enable the development of either genetically engineered plants with enhanced disease resistance or novel mode-of-action plant protection chemicals that act by stimulating the plant’s inherent disease resistance mechanisms.

There are three steps involved in the development of systemic acquired resistance:

- The induction of systemic acquired resistance usually requires the development of a slowly expanding necrotic lesion. Induction of systemic resistance may be associated with other localised responses such as hypersensitive cell death, phytoalexin accumulation, papilla deposition and lignification.
- Two or three days after the inducing lesion first appears, a signal is released that is systemically translocated in the phloem. This signal is graft transmissible and is not cultivar, species or genus specific, but is not active once plants have begun flowering. All the signals originate from the induction site.
- The systemic signal primes the rest of the plant against further pathogen challenge. Defence responses such as the rapid release of reactive oxygen species, hypersensitive cell death, phytoalexin accumulation, and enhanced levels of pathogenesis-related proteins are expressed more rapidly and intensely than in uninduced plants.

The identity of the signal that triggers systemic acquired resistance is the subject of intense study, but remains unresolved. There are several molecules that can induce features characteristic of systemic acquired resistance, including salicylic acid, β-ionone and jasmonic acid. The entire response is, however, apparently mediated by a complex signal transduction pathway regulated by a number of stress signals.

Salicylic acid, a precursor of aspirin widely distributed in the plant kingdom, plays a key role in systemic acquired resistance. Salicylic acid binds to at least two proteins found in plant cell membranes. One salicylic acid-binding protein has catalase activity that is inhibited upon binding, causing a localised build-up of hydrogen peroxide. This form of reactive o4ygen, as previously mentioned, causes a number of changes in plant cells that increase their resistance to pathogens. A second, high affinity, salicylic acid-binding protein appears to directly activate gene expression. Levels of salicylic acid rise rapidly around necrotic lesions in plants and remain high in plants that have acquired resistance. However, a series of experiments show that it is a local, rather than a systemically translocated, signal. Although it must be present for systemic acquired resistance to be expressed, salicylic acid is not translocated over long distances in plants and presumably interacts with another systemic signal. Synthetic analogues of salicylic acid, such as dichloroisonicotinic acid (INA) and the benzothiazoles, induce similar responses to those induced by salicylic acid and have potential use as practical disease-protectants. Although INA induces resistance in field and glasshouse trials, the effective dose is sometimes phytotoxic and this risk will probably prevent its commercialization. A more promising benzothiazole, benzo(1,2,3) thiadiazole-7-carbothioic acid S-methyl ester (BTH), is similarly effective but less phytotoxic (David and John 1997).

Insect Pests

Any organism that consumes plant tissue is an herbivore. Insect and plants have coevolved for millions of years. Plants respond to herbivory through various morphological, biochemicals, and molecular mechanisms to counter the effects of herbivore attack. These defence strategies against herbivores are wide-ranging, highly dynamic, and could be direct and/or indirect. Host plant defence against herbivores is a complex array of structural, chemical, and physiological traits intended to perceive the attacking organisms, and restrain them before they are able to cause extensive damage. Since plants lack the physical mobility, they have evolved a number of strategies which enable them to withstand insect pressure. Plant defence against insect pests is mediated through morphological (toughness, thorns, thickness, and hairiness) and biochemical (nutritional composition of the plant tissue, and the nature and amounts of secondary metabolites) factors. The defensive compounds are produced constitutively or in response to plant damage, and affect feeding, growth, and survival of herbivores. In addition, plants also release volatile organic compounds that attract the natural enemies of the herbivores. These strategies either act independently or in conjunction with each other. Direct defences are mediated by plant characteristics that affect the herbivore's biology such as mechanical protection on the surface of the plants (e.g., hairs, trichrome, thorns, spines, and thicker leaves) or production of toxic chemicals such as terpenoids, alkaloids, anthocyanins, phenols, and quinones) that either kill or retard the development of the herbivores. Indirect defences against insects are mediated by the release of a blend of volatiles that specifically attract natural enemies of the herbivores and/or by providing food (e.g., extra floral nectar) and housing to enhance the effectiveness of the natural enemies. Plants defence against herbivory or host-plant resistance (HPR) describes a range of adaptations evolved by plants which improve their survival and reproduction by reducing the impact of herbivores. Host plant resistance is one of the most effective tools for reducing insect damage. Phytophagous insects are often suppressed by plant defence mechanisms. Their mating, oviposition feeding, feeding ingestion, fertility and growth are interrupted.

Host plant resistance is the result of interactions between two biological entities, the plant and the insect under influence of various environmental factors (Dhaliwal and Singh 2004). Plants use several strategies to defend against damage caused by herbivores. Painter (1951) grouped mechanism of resistance into three main categories, Viz. Antixenosis (also called as non-preference), antibiosis and tolerance. Antixenosis is employed by the plant to deter or reduce colonization by insects. Plants that exhibit antixenotic resistance should have a reduced initial number of colonies early in the season. Antibiosis operates after the insect have colonized and have started utilizing the plant. Tolerance of the plant does not affect the rate of population increase of the target pest but does raise the threshold level.

Mechanism of Resistance

Antixenosis (Non Preference)

Antixenosis refers to the resistance mechanism employed by host plant to deter or reduce colonization by insects. It refers to plant characteristics that lead insects away from a particular host; it includes activities of both plant and insect. It is mainly the response of insect to the characteristics of host plant which make it unattractive to the insect for feeding, oviposition or shelter. It denotes the presence of morphological or allelochemicals in host plant factor that adversely alter insect behaviour resulting in poor establishment of the insect. Antixenosis may represent one or more breaks in the chain of responses leading to oviposition or feeding. These breaks may be due to (i) the absence of an arrestant or attractant, (ii) the presence of a repellent, or (iii) an unfavourable balance between an attractant and a repellent (Panda and Khush 1995).

Antixenosis is of 2 types: Morphological antixenosis and Allelochemic antixenosis.

Morphological antixenosis

Morphological characters of plant interfere with insect behaviour activities such as mating, oviposition, feeding and feeding ingestion. Various morphological characters are

(a) Trichomes
(b) Pubescence
(c) Surface waxes
(d) Thickness of cell wall and proliferation of plant tissues
(e) Silica content
(f) Solidness and other stem characters
(g) Anatomical adoptions of organs
(h) Colour and intensity of light.

Allelochemic nonpreference

Several chemical present in the plant affect the olfactory and gustatory stimuli in insects. These chemicals may attract or repel the insects and also inhibit feeding by the insects. Ex: In cucurbits, a compound called cucurbitacin acts as an attractant and feeding incitant to spotted cucumber beetles.

Antibiosis

Antibiosis refers to the adverse effect of the host plant on the biology (survival, development or reproduction) of the insects and their progeny infesting it. All these adverse physiological effects of permanent or temporary nature following ingestion of a plant by an insect are attributed to antibiosis. The insects feeding on resistant plants may manifest antibiotic symptoms varying from acute or lethal to sub chronic or very mild. The most commonly observed symptoms in insects include adverse effects on the nutritional physiology of the insect including consumption, assimilation, utilization and subsequent allocation for reproduction (Ananthakrishnan 1994). This is manifested by larval death in first few instars, abnormal growth rates, disruption in conversion of ingested food, decline in size and weight of larvae or nymphs, prolongation of the larval period, failure to pupate, failure of adults to emergence from pupae, abnormal adults, inability to concentrate food reserves followed by failure to hibernate, decreased fecundity, reduction in fertility, restlessness and abnormal behaviour (Panda and Khush 1995). These symptoms may appear due to various physiological processes such as presence of toxic substances, absence or insufficient amount of essential nutrients, nutrient imbalance or improper utilization of nutrients. This mechanism impairs an insect's metabolic processes and involves consumption of plant metabolites. Both insect and plant factors are involved in the antibiosis mechanism. The physiological explanations for antibiosis are

a. Presence of toxins
b. Absence/insufficiency of essential nutrients
c. Unbalanced proportion of nutrients
d. Presence of antibodies.

Tolerance

Tolerance refers to the ability of the host plant to withstand an insect population sufficient to damage severely the susceptible plants. It is generally attributable to plant vigour, regrowth of damaged tissues, resistance to lodging, ability to produce additional branches, utilization of non-vital parts by insects and compensation by growth of neighbouring plants. However, tolerance has no adverse effect on the insect infestation for longer periods without loss in yield or quality than the susceptible varieties and enables them to frequently escape insect damage through compensation by the plants. Tolerance is a plant response to an insect pest. Whereas, antibiosis, antixenosis, resistance cause an insect response when the insect attempts to use the resistant plant for food, oviposition or shelter. Tolerance is useful in pest management programmes due to certain distinct advantages (Panda and Khush 1995):

(a) Tolerant varieties have a higher economic threshold level than the susceptible varieties and hence require less insecticide application and promote biocontrol.
(b) Tolerant varieties do not depress insect populations nor do they provide any selection pressure on the insect and thus are useful in preventing the development of insect biotypes.
(c) In varieties with a combination of thee mechanisms of resistance, tolerance increases yield stability by providing at least a moderate level of resistance, when vertical genes providing a high level of resistance through antixenosis and antibiosis succumb to the new biotype.

Conclusion

Plants are challenged by different types of environmental stresses during their life cycle. In order to avoid such stresses, plants develop various adaptation strategies and defense mechanisms at morphological, anatomical, physiological, biochemical and molecular levels. Having deep knowledge about the various mechanisms by which plants tolerate environmental stresses is necessary to improve the crop performance under stress conditions. The study of plant response to stress will let us know about the metabolism, various pathways and cascades involved during the stress condition. Further, the understanding about anatomical and molecular mechanisms of plant response to stress will help in development of genetically engineered plants possessing high tolerance to biotic and abiotic stresses. This will also provide better insights of the highly complex anatomical, molecular and biochemical strategies adopted by the plants to adapt themselves under several environmental stresses (dos Reis et al. 2016).

Breeding for resistance to stress is challenging. Various novel techniques like genomics, proteomics and metabolomics can aid in identifying the genetics behind plant stress responses, enabling a direct and unbiased monitoring of the factors affecting crop growth, development and productivity. They also provide data that can be directly used to investigate the complex interplay between the plants, its metabolism and the environmental stress, both biotic and abiotic (Perez-Clemente et al. 2013). Moreover, the genome analysis of plants enable identification of DNA markers linked to stress tolerant/resistant genes, facilitating the crop improvement under climate change scenario.

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