A review of heat stress signaling in plants

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Abstract. The threat and crisis of food due to global warming has a related with global climate change. The threat of a decline in food production in various countries by climate change triggers a long drought. The increase in temperature causes heat stress on the plant which has an impact on the decline in land quality and others. Heat stress is often defined as an increase in temperature for a certain period of time, which can cause irreversible damage to plants, which generally occurs at temperatures of 10-15˚C above the threshold. Heat stress is a major factor limiting crop productivity and adaptation, especially when extreme temperatures coincide with the critical stage of plant growth.. Heat stress is a major factor limiting crop productivity and adaptation, especially when extreme temperatures coincide with the critical stage of plant growth. Excessive heat can disrupt by denaturing enzymes and damaging metabolism so that changes occur in the morphological structure, phenology, physiology and molecular plants.The response to increase in temperature of 5-10^{o} C, makes plants produce a unique protein called heat shock proteins (HSP). An increase in HSP production occurs when plants experience a sudden or gradual increase in temperature. HSP is released when the stress of exposed to plant . Under these conditions HSP is useful for protecting proteins and causes resistant plants. The resistance obtained by heat shock is different from the resistance obtained by rapid growth at moderately high temperatures. This difference is presumed by the presence of HSP responses induced by heat stress. The HSP can play a role as a sign of gene stress and activation and in regulating oxidation reactions in cells. Utilization of heat stress signaling in food crops breeding can be used to development Wheat Tolerance to High Temperature.

1. Introduction

Temperature as an environmental factor can affect crop yields both physically and physiologically. Physically, the temperature is the part that is affected by solar radiation and can be estimated based on heat balance. Physiologically, the temperature can affect plant growth, photosynthesis, stomata opening, and respiration. In addition, the temperature is one of the inhibitors in the physiological process for crop production systems when the temperature of the plant is outside the lowest and highest optimal temperatures.

Heat stress is often defined as an increase heat for a certain period of time, which can cause irreversible damage to plants, which generally occurs at temperatures of 10-15˚C above the threshold [38]. The stress factor is usually not only single but is a complex process because it involves several determinants of growth. One example in the summer can cause damage tochlorophyll, low humidity, dry soil and high temperatures. In addition, stress responses are very complex, involving various parts of plants and involving stress hormones such as abscisic acid (ABA) and ethylene which are transported to all parts of plants [42].

Heat stress is a major factor limiting crop productivity and adaptation, especially when extreme temperatures coincide with the critical stage of plant growth. The rate of change in temperature and the degree of resistance of plants to temperature is important to remember that plants have different levels of heat resistance to survive periods of stress. In addition, the response of plants to heat stress depends on adaptation to temperature, exposure interval and stage of tissue growth [5]. The response to a sudden increase in temperature of 5-10 ^{o}C, makes plants produce a unique protein called heat shock proteins (HSP). Increased HSP production occurs when plants experience an increase in temperature either suddenly or gradually [34][44]. HSP, known as a heat shock protein, is released when the plants body experiencing heat stress. Under these conditions, HSP is useful for protecting proteins.

High temperatures disrupt and kill plants by denaturing their enzymes and damaging their metabolism in various ways. Hot and dry weather also tends to cause water shortages in many plants and stomata closure. Most plants have a reserve response that allows them to survive heat stress Above a certain temperature of around 40° C in most plants that occupy four seasons, plant cells begin to synthesize a special protein in sufficient quantities called protein heat shock (heat shock protein). This heat shock protein may flank enzymes and other proteins and help prevent denaturation [4].

In tropical climate areas, excess radiation and high temperatures are often the limiting factors that greatly affect plant growth and plant yields. High temperatures can cause crop damage, including leaves, branches and stems. In addition there is an inhibition of ageing and root growth, fruit discolouration and reduced production [15][22][50]. Likewise in temperate regions, heat stress has been reported as one of the most important causes of reducing yields and production of dry matter in plants, for example corn [14].

2. Signalling Morphology of Heat Stress

Heat killing temperature for plants is the temperature at 50% of plants die. This temperature varies according to the type of plant. For higher plants that are growing, the highest temperature is reported to be 60-120°C. However, this temperature lasts only for a very short period of time, namely during the day, when growth is stopped temporarily [48].

Table 1. Heat-Killing Temperatures for Plants [24]

Plant	Heat Killing Temperature (C^o)	Exposure
Nicotiana rustica (Wild Tobacco)	49-51	10 min
Cucurbita pepo (Squash	49-51	10 min
Zea mays (corn)	49-51	10 min
Brassica napus (rape)	49-51	10 min
Citrus aurantium (Sour orange)	50.5	15 – 30 min
Opuntia (Cactus)	>65	-
Sempervivum arachnoideum (Succulent)	57 -61	-
Potato Leaves	42.5	1 hour
Pine and Spruce seedlings	54 -55	5 min
Medicago seeds (alfalfa)	120	30 min
Grape (Ripe Fruit)	63	-
Tomato Fruits	45	-
Red Pine pollen	70	1 hour
Hydrated (Various mosses)	42 - 51	-
Dehydrated (Various mosses)	85 - 110	-

Most plant tissues cannot survive long exposure at temperatures above 45^{o} C. Cells that are not growing or dehydrated tissue (such as seeds and pollen) can survive at temperatures much higher than vegetative cells. Tissues that are actively growing rarely survive at temperatures above 45^{o}C, but dry seeds can withstand temperatures up to 120^{o}C, and pollen grains from some species up to 70^{o}C.

Generally, only a single cell eukaryote can complete its life at temperatures above 50^{o}C and only prokaryotes can divide and grow above 60^{o}C [48].

Responses due to high temperatures in plants can occur directly as in physiological processes while the extreme damage is the death of cells, tissues, organs, and finally plants. This may differ from one stage of phenology to another. For example, the effect of long-term heat stress causes delayed germination or loss of vigour, resulting in reduced seed formation. Growth of coleoptiles in corn decreases at 40°C and stops at 45°C. In seedlings also symptoms of burns occur at organ surface temperatures reaching 48-50^{o}C [56].

Burns or burn tissue generally occur on the trunk, branches and fruit due to the weak influence of cooling by transpiration. High temperatures in shoot growth cause a reduction in weight at the length of the first segment resulting in the premature death of plants [18]. High temperatures cause a decrease in dry weight in shoots, relative growth rates and net assimilation rates of corn, barley and sugar cane, although leaf area has minimal effect [1][53]. In wheat both seed weight and the number of seeds appear to be very sensitive to heat stress with increasing temperature [11]. In environments with intense sun radiation and high temperatures, plants avoid excessive heating of the leaves by reducing the absorption of solar radiation. This adaptation is important in hot environments. Leaves transpire near the upper limit of temperature tolerance. In such conditions, heat stress will cause evaporation of distressed water and increased absorption energy so that it can damage the leaves.

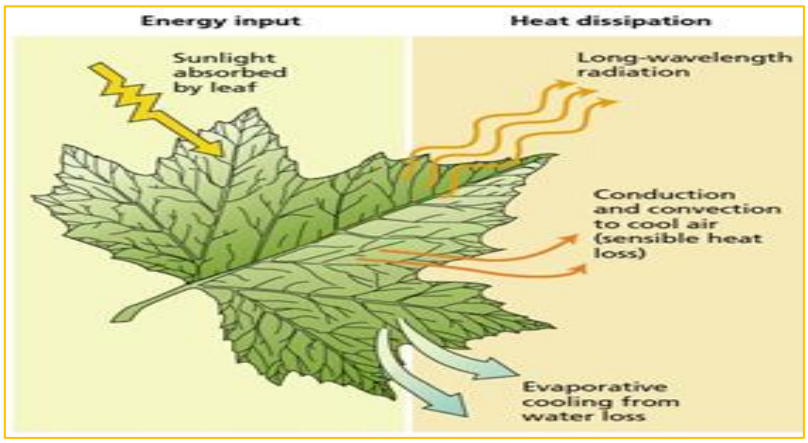

Figure 1. The absorption and dissipation of energy from sunlight by the leaf [47]

The phenology of plants is greatly affected by heat, which is generally accelerated by temperatures which cumulatively decrease yields. The heat accelerates phenology (plant development), the acceleration of the process (phenology) will accelerate the transition from a certain phase to the next phase, which results in reducing the size of plants or plant organs. Sugarcane plants that grow at high temperatures show smaller segments, tillers increase, cook quickly and biomass decreases [8]. In temperate and tropical lowlands, heat stress causes yield losses in Phaseolus vulgaris [15] and peanuts [49] If the economic yield is part of the vegetative (biomass), heat will reduce the yield. If the harvest is in the form of generative parts (seeds/fruit) then the negative impact of heat during the initial growth phase will greatly reduce the time (shortening) of seed filling in cereals, followed by a reduction in the number of seeds per grain, reducing the length of the seed filling period.

Heat stress combined with salinity generally has problems when the flowers bloom and the filling of seeds of cereal plants that grow in temperate climates. For example, the extension of heat stress when filling grain causes losses in wheat seeds up to 7% [16]. The same thing happened in the formation of starch, protein and oil in corn kernels [57] and the quality of cereal grains affected by heat stress[28].

3. Signaling Physiology of Heat Stress

Temperature as an environmental factor that can affect physical and physiological plant production. Physically,the temperature is the part that is affected by sunlight radiation. Physiologically, the temperature can affect plant growth, photosynthesis, stomata opening, and respiration. In addition, the temperature can be one of the inhibitors in the physiological process for crop production systems if the plant temperature is outside the optimal temperature both low and high temperatures. Heat stress affects all aspects of plant processes like germination, growth, development, reproduction and yield [19][31][30].

Heat stress differentially influences the constancy of various proteins, membranes, RNA species and cytoskeleton structures, and alters the efficiency of enzymatic reactions in the cell for which the major physiological processes obstacle and creates metabolic imbalance (Ruelland and Zachowski, 2010). It is believed by some workers that night high temperatures are major limiting factors, whereas others argued that day and night high temperatures do not affect the plant independently. The diurnal means temperature is a superior forecaster of plant response to high temperature with day temperature having a secondary role [38]

An increase in leaf temperature during the day in a dry area causes plants to experience a lack of water due to high irradiation from sunlight. Plant water status is the most important component under changes in threshold temperature [29] Temperature stress also affects plant growth in greenhouses. Low air velocity and high humidity reduce the rate of leaf cooling. In general, plants tend to maintain the stability of the water status in the tissue without regard to temperature when the humidity is sufficient. However, high temperatures are very damaging and water is a limiting factor [27]. In field conditions, heat stress is often associated with reduced water availability [45]

Air temperature or soil temperature affect plants through metabolic processes in the body of the plant, which are reflected in various properties such as growth rate, seed dormancy, germination, flowering, fruit growth maturation/maturation of plant tissues or organs. Plant responses to temperature differ depending on the type of plant, variety, stage of plant growth, type of organ /tissue [47]. Plants based on physiological functions are grouped into 3 groups, namely CAM, C3 and C4.

Plants with open stomata at night and closed during the day are called *Crassulacean Acid Metabolism* (CAM). The difference in vapour pressure or VPD (*Vapor pressure difference*) from the leaves to the air causes very low transpiration at night when the leaves and the air cools. CAM plants can get 1 g of dry weight using only 125 g of water. A ratio of 3-5 times greater than the ratio for C3 plants. CAM plants, such as Opuntia and Sempervivum can withstand tissue temperatures of 60^0-65^0C in the heat of the sun in summer. Because CAM plants keep their stomataclosed during the day, CAM plants cannot be cooled by transpiration but releases heat from solar radiation by re-emitting long-wave radiation (infra red) and losing heat by conduction and convection.

C3 and C4 plants affected by heat stress rely on transpiration to reduce leaf temperature. Leaf temperature can immediately rise 4-5^0C above the ambient air temperature in bright sunlight before noon. Lack of groundwater causes partial closure of stomata or when high relative humidity decreases the potential for transpiration. Moderate temperature stress slows overall growth. Large respiration is associated with high agronomic outcomes. Heat stress interferes with the movement of water, ions and organic solutions across the plant membrane [21].

High temperatures reduce membrane stability while the stability of various cell membranes is important at hightemperature stresses, such as when cold and freezing. Excessive exchange of substances from membranes at high temperatures is associated with loss of physiological function. In oleander (*Nerium oleander*), the impact of high temperature is related to the higher degree of saturation of the membrane fatty acids so that the membrane is less liquid. The function of a cellular membrane that is sustained under stress is important for photosynthesis and respiration [3].

Photosynthesis and respiration are inhibited at high temperatures, but when the temperature rises, the rate of photosynthesis decreases sharply before the respiration rate. Treshow (1970) reports that photosynthesis is an important reaction that is affected by heat. Photosynthesis occurs in a temperature

range of 10-30°C depending on the climate of the plant. Above the temperature of 10˚C photosynthesis will increase with increasing temperature, and will experience a decrease above 30˚C. Respiration generally occurs in the temperature range of 5-25˚C, increases at 30-35˚C and slows above 35˚C [20]. Heat stress reduces chemical reactions, gas solubility, mineral absorption and water uptake, disrupts the electron transfer system in photosynthesis and increases oxidative damage to membrane lipids[6].

4. Signalling Biochemical of Heat Stress

Heat stress causes oxidative stress, which is a condition when the amount of free radicals in the body exceeds the capacity to neutralize it and consequently the intensity of the oxidation process of normal body cells becomes higher and causes more damage. Reaction of activated oxygen species (AOS) including singlet oxygen ($^1O_2$), superoxide radical (O^{2-})hydrogen peroxide (H_2O_2) and hydroxyl radical (OH^-) are symptoms of cell damage due to high temperatures [26]. AOS causes lipid membranes to lose the ability to regulate the exchange of substances in cells [58]

The Energy needed for biological functions in aerobic organisms is produced in the mitochondria through the electron transfer chain. In addition to AOS reactions, Reactive Oxygen Species (ROS) also have the potential to cause cellular damage. ROS can damage DNA, RNA and protein, which contribute to the physiology of ageing. ROS are produced as normal products of cell metabolism. Specifically, one of the main causes of oxidative damage is hydrogen peroxide (H_2O_2) which is converted from superoxide leaked from mitochondria. A study shows that accumulation of ROS can reduce the resistance of organisms due to oxidative damage. Specifically, oxidative damage can affect mitochondrial efficiency and increase the rate of ROS production. Oxidative damage and its effect on ageing depend on the type of tissue in which the damage occurs [26]

Decreased antioxidant activity in the tense tissue shows a higher amount of AOS when the organism is gripped [10]. Protection against oxidative stress is an important component in determining the survival of plants in heat stress. Available data shows. thatseveral molecules can cause an increase in the antioxidant capacity of cells [12]

SOD is a preventive anti oxidant made by the body to prevent excessive free radicals in the body. The enzyme catalase and superoxide dismutase (SOD) repair the damage effects that function as a catalyst to change the dismutation reaction from superoxide anions to hydrogen peroxide and oxygen molecules. However, peroxide can survive in cells. While ROS is produced as a product of normal cellular function. Excessive amounts can cause damage. The body produces free radical compounds through the process of phosphorylation. During the oxidative phosphorylation process, O2 is reduced to H2O by the addition of 4 electrons, so that superoxide anion radicals are formed which then become hydrogen peroxide (H2O2) by the SOD enzyme.

Plants have developed a series of detoxification systems to fight AOS both enzymatically and non-enzymatically, thus protecting cells from oxidative damage [41]. SOD enzymes in plants affect a number of physiological phenomena including the elimination of H202, toxic oxidation of reducers, biosynthesis and degradation of lignin in cell walls, enzyme catabolism, defense against injury, defense against pathogenic or insect attack and some respiratory processes [43] More specifically, APX activation is related to the appearance of physiological damage caused by temperature stress [29]

The process of phosphorylation in the mitochondria causes 1 O2 molecule to be reduced by 4 electrons together with H + ions to form 2 H2O molecules. If the electrons reducing O2 are less than 4, the phosphorylation process is incomplete and free radicals are formed. SOD enzymes work can be seen in the number of lipid peroxidation products per organelle. If the activity of the enzyme increases it will cause lipid oxidation products to decrease [13].

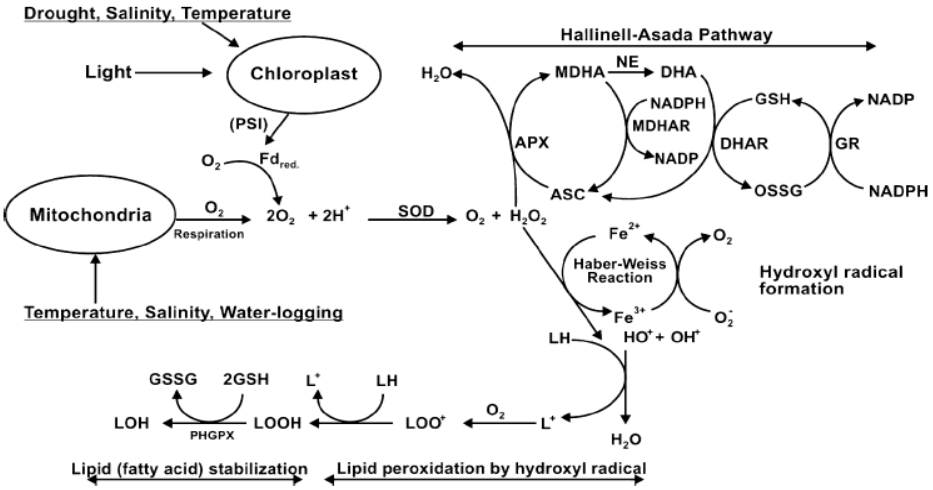

Figure 2. The mechanism of cell resistance to oxidative stress that helps stabilize fatty acids in tense environmental conditions [41]

Superoxide radical compounds are regularly synthesized in chloroplasts and mitochondria and some parts are also produced in microbodies. O2-flushing by superoxide dismutase (SOD) results in the production of H2O2 lost by APX (ascorbate peroxidase) or CAT (catalase). SOD is a class of enzymes that catalyzes oxygen and hydrogen into oxygen and hydrogen peroxide. Thus, SOD is an important antioxidant defense in cells. Almost all cells are exposed to oxygen, but both O2- and H2O2 are not OH- or toxic compounds formed by a combination of O2- and H2O2 in the presence of Fe2 + and Fe3 + traces by the Haber-Weiss reaction. OH- can damage chlorophyll, protein, DNA, fat and other important macromolecular, thus affecting plant metabolism and limiting growth and yield [41].

5. Heat Shock Protein

The response to a sudden increase in temperature of 5-10^{0}C, makes plants produce a unique protein called heat shock proteins (HSP). Increased HSP production occurs when plants experience an increase in temperature either suddenly or gradually [34]. HSP, known as a heat shock protein, is released when the body is hot. Under these conditions, HSP is useful for protecting proteins. HSP is found in all living things.

Most of the function of HSP is to help cells survive heat stress by acting as chaperons. Heat Shock Proteins are a group of proteins in living cells that can be found in all phases of the development of these living things. This protein is active when stimulated by various forms of stress such as oxidative stress, heat, cold, inflammation and oxygenation disorders in cells. Under normal conditions, HSP is also commonly found in cells as a companion. Heat stress causes many cell proteins that function as enzymes or structural components to unfold or fold folded incorrectly. This results in a loss of structure and correct enzyme activity. HSP induction is a response to the temperature observed in all organisms ranging from bacteria to humans [51].

The role of HSP as a companion in the formation of proteins. To support the action of ribosomes along with RNA molecules, an amino acid chain is built to form a new protein molecule. This chain is protected from unwanted interactions with other cytoplasmic molecules with HSP and chaperonin molecules until they have successfully completed their cycle into proteins. HSP acts as a chaperonin (molecular companion) and serves to achieve the correct folds of collected protein and to prevent incorrect folds of protein. This all facilitates proper cell function at high temperatures. Chaperons are proteins that help fold / unload noncovalent folds and attach /release proteins with other macromolecular structures. Chaperons function primarily if there is an error in protein folding and does not function if the structure of the protein formed has normal biological functions [34]

The protein binds to the chaperone molecule and starts the folding process. After folding and translating the genetic code by tRNA into the amino acid sequence, the protein is released from the chaperon and transferred to the Golgi apparatus. If the protein assembly is incomplete, then the wrong folded protein will bind to the chaperons and be in the RE. This landfill in RE serves as a signal for the formation of a new chaperon. Proteins that are wrongly folded are removed and degraded in the cytoplasm [9]

Plants and most other organisms make HSPs of different sizes in response to rising temperatures . Most HSPs synthesized by eukaryote organisms have five different structures namely HSP100, HSP90, HSP70, HSP60, Small HSP (SmHSP) found in the nucleus, mitochondria, chloroplast, endoplasmic reticulum and cytosol [51]. The molecular weight of HSP varies from 15-104 kDa. HSP molecular weight is characterized by high sequence homologues in plants [54]

HSP 100 is formed continuously but is also regulated by environmental pressures. These proteins generally function to protect protein denaturation and protein formation [55] HSP 104 in Arabidopsis and yeast play an important role in resistance to heat stress [28]. HSP 101 in Arabidopsis influences growth during the recovery period [52]. It has been found that the homogenised Arabidopsis HSP101 is involved as a link in the plastids in the formation of thylakoids and as a temperature resistance for chloroplasts during hot strikes [32]

SmHSP is also produced under conditions of environmental stress and several stages of development (embryogenesis, germination, growth and maturation of fruit pollen). SmHSP encourages gene change. This shows the important role of proteins in abiotic stress [17]. The degree of smHSP formation depends on the temperature and duration of the stress period [46]. SmHSP shows heterogeneous structure according to its molecule, center of gravity and isoelectric stability [23]. After heat stress, smHSP is stable with half -life after 30-50 hours. This shows that smHSP might be important for recovery [46].

All cells contain companion molecules that are formed together and function like HSP. This companion is called a heat shock cognate protein. However, when cells are exposed to heat but are not damaged, the synthesis of HSPs dramatically increases while the translational continuity of other proteins is dramatically reduced or completely stopped. The response to this heat shock is preceded by a specific transcription factor (HSF) that acts on HSP mRNA transcription.

Cells that are induced to synthesize HSP exhibit better temperature resistance and can withstand gripping conditions and otherwise damage. Some HSPs are not unique to high-temperature stress. HSP is also affected by stresses or very different environmental conditions, including water shortages, ABA treatment, wounds, low temperatures, and salinity. Thus, cells that were previously exposed to one stress may get cross-protection against other stresses. The case of tomatoes given heat exposure (48 hours at 38^{o}C) supports the accumulation of HSP and protects cells for 21 days from cooling at 20^{o}C

The mechanism that occurs in HSP is still mysterious, although some roles in plant cells have been ascribed to HSP. Many studies have confirmed that the HSP chaperone molecule functions as a protein that keeps cells under heat stress. Some evidence has been shown that resistance to heat is directly related to the synthesis of HSP accumulation [2]

When there is no heat stress, the heat shock factor (HSF) is a monomer that is unable to bind DNA and direct transcription. The stress causes the associated HSF monomer to become a trimer which is then able to bind to a specific unique element in DNA called a heat shock element (HSE) or heat shock element. Once bound to trimeric HSE HSF is phosphorylated and promotes HSP mRNA transcription. HSP70 binds to HSF and causes HSF / HSE to break down. HSF is then recycled into the HSF monomer form. Thus the action of HSF causes HSP to accumulate until it becomes abundant enough to bind HSF which leads to the cessation of HSP mRNA production [39] Two alternative ways could be proposed to enhance HSP70 concentration in plant tissues. The first is subjecting plants to heat shock, so that concentration of the inducible form of HSP70 increases and harvesting the plants when protein concentration is maximum.Heat shock can be administered in the field by means of heat producing machines. The second is overexpressing HSP70 in plants by genetic transformation [36]

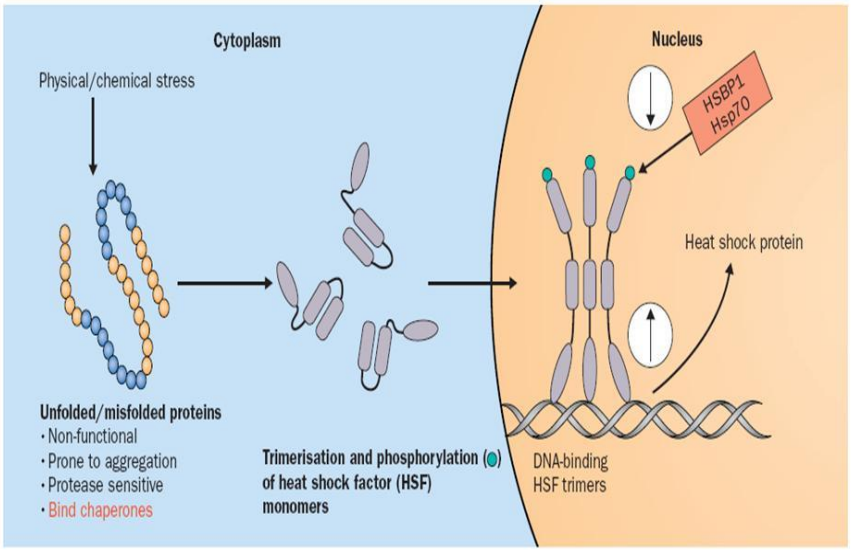

Figure 3. Induction and Regulation of Heat Shock Protein due to environmental stress [39]

6. Potential Utilization of Heat ShockProtein for Tropical Wheat Breeding

Wheat genetic improvement through breeding programs in Indonesia is initiated by introducing the elite lines from various countries which are considered suitable for tropical agroecosystems in Indonesia because Indonesia does not have local germplasm. A broad genetic diversity is needed to get wheat. varieties adaptive to tropical conditions in Indonesia. Increasing genetic diversity of wheat is conducted through cross-breeding and mutation (seeds and somaclonal variation). Not all types of wheat can be grown in Indonesia, only Triticum aestivum that can be developed at areas of > 1,000 m asl. The development of tropical wheat at < 1,000 m asl should be supported by breeding programs initiated by forming the population until releasing the varieties with shuttle breeding methods [37].

Strategies to maintain wheat plants against high temperature stress need to be studied to support genetic improvement through a tropical wheat breeding program. According to Dolferus *et al* [7], wheat crop stages that are most critical of abiotic stress are reproductive stages. Therefore, this stadium needs to be considered for studying plant turnover against abiotic stresses. Tropical wheat breeding programs that have been running and show good results are breeding through conventional crosses and mutations (seed mutationsand somaclonal variations) [36].

Information regarding the genetic basis of heat tolerance is generally scarce, though the use of traditional plant breeding protocols and contemporary molecular biological techniques, including molecular marker technology and genetic transformation, have resulted in genetic characterization and/or development of plants with improved heat stress tolerance. Constitutive expression of specific proteins has been shown to enhance heat tolerance. In addition to the knowledges concerning the expression of HSPs/ chaperones and manipulation of HSF gene expression, other transgenic plants with varying degrees of heat tolerance have been produced. Unpredictably, however, such experiments have been quite limited compared to the experiments aimed at engineering drought, salt or cold stress tolerance [20]

Small Heat Shock Proteins (sHSPs)/HSP20 are molecular chaperones that protect plants by preventing protein aggregation during abiotic stress conditions, especially heat stress. Due to global climate change, high temperature is emerging as a major threat to wheat productivity. Thus, the identification of HSP20 and analysis of HSP transcriptional regulation under different abiotic stresses in wheat would help in understanding the role of these proteins in abiotic stress tolerance.results showed that the identified TaHSP20 genes play an important role under different abiotic stress conditions. Thus, the results illustrate the complexity of the TaHSP20 gene family and its stress

regulation in wheat and suggest that SSHs as attractive breeding targets for improvement of the heat tolerance of wheat [33].

7. Conclusion

Heat stress is defined as an increase in temperature beyond the threshold level for a certain period of time, which can cause irreversible damage to plant growth and development. In general, usually ranges from 10-15˚C above the threshold. Heat stress causes changes in the morphological, phenological, physiological and molecular structure of plants. The response to a temperature rise of 5-10 ° C suddenly causes plants to produce a unique group of proteins called heat shock proteins that function to help cells survive heat stress.Utilization of heat stress signaling in food crops breeding can be used to development Wheat Tolerance to High Temperature. Knowledge relating to molecular basis and mechanism of tolerance is considered to pave the way for engineering plants that can withstand heat stress. In addition, greater emphasis on heat stress management is obligatory for heat tolerance features, molecular cloning, and characterization of genes.

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