



Review

Individual and Interactive Effects of Elevated Ozone and Temperature on Plant Responses

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Abstract: From the preindustrial era to the present day, the tropospheric ozone (O_3) concentration has increased dramatically in much of the industrialized world due to anthropogenic activities. O_3 is the most harmful air pollutant to plants. Global surface temperatures are expected to increase with rising O_3 concentration. Plants are directly affected by temperature and O_3 . Elevated O_3 can impair physiological processes, as well as cause the accumulation of reactive oxygen species (ROS), leading to decreased plant growth. Temperature is another important factor influencing plant development. Here, we summarize how O_3 and temperature elevation can affect plant physiological and biochemical characteristics, and discuss results from studies investigating plant responses to these factors. In this review, we focused on the interactions between elevated O_3 and temperature on plant responses, because neither factor acts independently. Temperature has great potential to significantly influence stomatal movement and O_3 uptake. For this reason, the combined influence of both factors can yield significantly different results than those of a single factor. Plant responses to the combined effects of elevated temperature and O_3 are still controversial. We attribute the substantial uncertainty of these combined effects primarily to differences in methodological approaches.

Keywords: biochemical characteristics; ozone; physiological characteristics; plant response; temperature



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1. Introduction

Climate change, including global warming, has been occurring over the last several decades, and is now recognized as the most significant threat to human and ecosystem health [1]. Recent studies have reported that future global surface temperatures are expected to increase steadily due to increasing atmospheric carbon dioxide (CO_2) levels from greenhouse gas emissions. According to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change (IPCC), the global mean surface temperature is projected to increase relative to current conditions by 1.5 °C to 4.8 °C by the end of the 21st century [2].

Additionally, the tropospheric ozone (O_3) concentration is expected to rise along with the global surface temperature, because the emissions that cause O_3 formation are also predicted to increase [3]. These increases may be especially significant in East Asia, where the average O_3 concentration is projected to increase by 20% from the current level by the end of this century [4]. Ambient O_3 concentrations have risen from less than 10 nL L⁻¹ before the industrial revolution to midday summer concentration in many parts of the Northern Hemisphere, exceeding 60 nL L⁻¹ [5]. O_3 is one of the most widespread atmospheric pollutants and is a subject of considerable concern worldwide [6,7]. As an atmospheric secondary pollutant, O_3 is formed by photochemical reactions involving nitrogen dioxide (NO_2) and volatile organic compounds (VOCs) [8,9]. The main chemical reaction in O_3 formation is NO_2 photolysis, which generates nitrogen oxide (NO) and a single oxygen atom (O). Under atmospheric conditions, the O atoms react with oxygen

(O_2) molecules to generate O_3 . In other reactions, VOCs combine with hydroxyl radicals (OH) to form water vapor (H_2O). Subsequently, the organic radical (R^\cdot) from the previous chemical reaction is chemically converted to a peroxy radical (RO_2), which can generate NO_2 by combining with O_2 . O_3 formation generally occurs via NO_2 photolysis, as shown in Figure 1 [10].

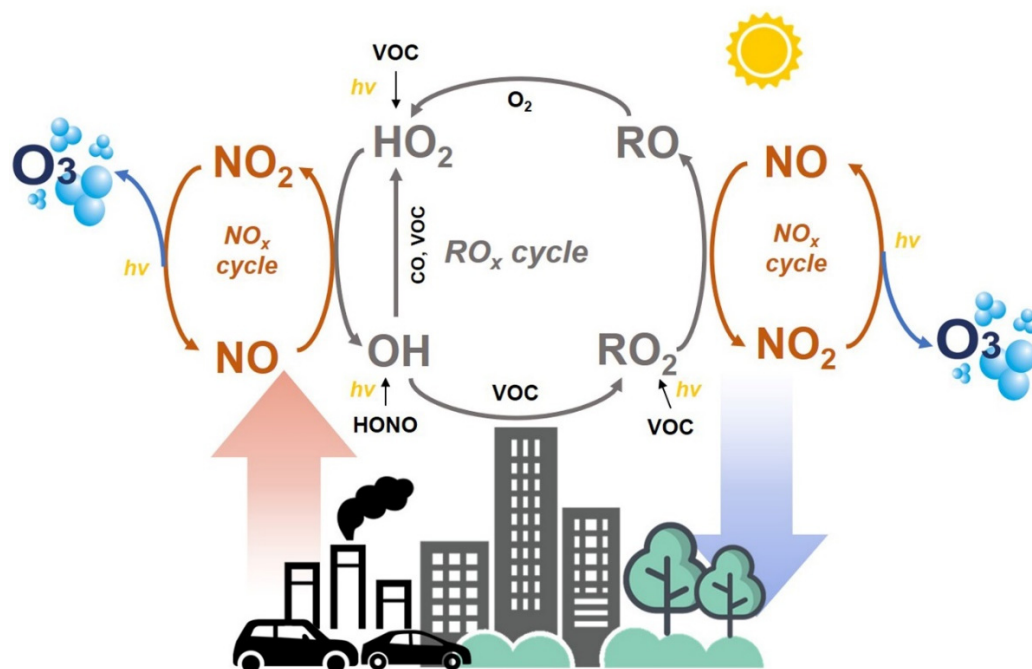


Figure 1. Diagram of the main chemical reactions involved in O_3 formation. Adapted from Fitzky et al. [10].

Plants are directly affected by elevated temperatures and O_3 concentrations. Thus, future vegetation will be sensitive to changes in both environmental parameters [11]. Temperature is among the major factors controlling plant development; it affects photosynthesis and respiration rates, which in turn, affect plant growth [12]. Depending on the species, plants have different optimal temperature ranges. A temperature rise above the tolerance threshold of a plant species can cause irreversible damage to the plant and its growth. O_3 is regarded as a phytotoxic gas that negatively affects plant development [13,14]. The plant leaf surface is the point of first contact by air pollutants such as O_3 . O_3 usually enters the leaf via the stomata and then produces reactive oxygen species (ROS), which can cause considerable damage to the plant [15].

2. Plant Responses to Ozone

2.1. Visible Symptoms of Sensitive Plants to Ozone

Recent studies have demonstrated that high O_3 concentrations may cause visible foliar symptoms in plants. Visible symptoms are generally caused by abiotic and biotic stresses that may include air pollutants, drought, insects, and fungal infections. The observation of visible symptoms in leaves is considered a particularly important tool for assessing O_3 effects on vegetation [15]. The presence and extent of O_3 -induced visible foliar symptoms may signify biological damage to vegetation, although it is less biologically significant than changes in growth and biomass [16]. The effects of O_3 on crops and tree seedlings have been studied in experiments using open-top chambers [17]. The older leaves of O_3 -sensitive plants tend to show more symptoms of injury than younger leaves. Additionally, it is well known that O_3 -induced visible symptoms can generally occur on sunlit foliage because light-induced oxidative stress leads to visible injury. Nevertheless, visible symptoms can also appear on shaded leaves [18]. When exposure to elevated O_3 levels occurs during leaf formation, leaves may be less affected and acclimate to later O_3 exposure [19,20]. Visible

foliar O_3 injury symptoms can be observed as whitish or light-green coloring, and bleaching, reddening, or bronzing patterns in interveinal patches [21]. Wan et al. [22] studied visible symptoms indicating O_3 injury on O_3 -sensitive trees and shrubs around Beijing (Figure 2). Wilting occurred in *Medicago truncatula* under an O_3 concentration of 70 nL L^{-1} , and necrotic spots appeared on the leaves within 6 days [23]. Lee et al. [24] studied *Brassica juncea* L., which is widely cultivated in East Asia. Under an O_3 concentration of 100 nL L^{-1} in an O_3 fumigated growth chamber, *B. juncea* L. displayed visible symptoms (bleaching) indicating O_3 injury (Figure 3). This study demonstrated that exposure to elevated O_3 causes foliar injury. Visible injury to O_3 -sensitive leafy vegetables may negatively affect the quality of agricultural products and may, therefore, have financial impacts on farm markets [25].



Figure 2. Visible symptoms of O_3 damage in O_3 -sensitive trees and shrubs around Beijing. (A) *Ailanthus altissima*; (B) *Populus tomentosa*; (C) *Amygdalus triloba*; (D) *Hibiscus syriacus*; (E) *Rhus typhina*; (F) *Ulmus pumila*; (G) *Salix leucopithecia*; (H) *Kerria japonica* var. *pleniflora*. Photos from Wan et al. [22].

Controls

Ozone treatments

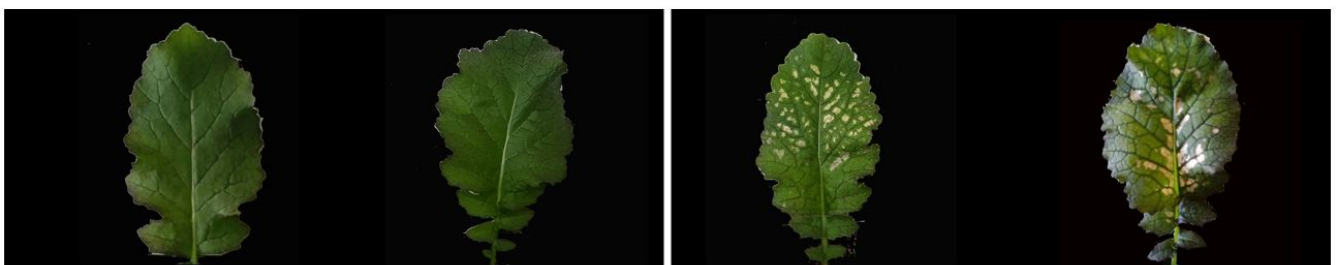


Figure 3. The visible symptoms of O_3 damage under an O_3 concentration of 100 nL L^{-1} at 14 days after exposure. Photos from Lee et al. [24].

2.2. Physiological Changes in Response to Ozone

Tropospheric O_3 has many adverse effects on plants and is the most impactful form of air pollution. Elevated O_3 concentrations can impair plant physiological processes

within plants, such as carbon assimilation. Chronic O_3 exposure reduces photosynthesis and total biomass, and accelerates senescence [26]. Several studies have reported that net photosynthesis rates in broad-leaved trees, wheat, soybean, and rice were considerably decreased by high ambient O_3 concentrations [27–29]. The degradation of photosynthesis by O_3 exposure has generally been attributed to decreased carboxylation efficiency, impacts on the photosynthetic electron transport system, and effects on the stomata [30]. Based on a study of ribulose-1,5-bisphosphate carboxylase/oxygenase (Rubisco) content and activity, O_3 -induced alterations to physiological capacity were found to correlate with decreased CO_2 fixation [5]. Moreover, O_3 can cause decreased electron transfer between photosystems (PS) I and PS II. Chlorophyll *a* fluorescence is a practical indicator of photoinhibition [31]. The F_v/F_m ratio, which indicates the maximum quantum yield of PS II, is generally reduced under elevated O_3 . The reduction in the PS II quantum yield is known to decrease photosynthetic electron transport. ATP and NADPH production are also decreased, due to reduced demand from the Calvin cycle, in plants exposed to O_3 [32]. Furthermore, the decreased photosynthetic aperture leads to the production of nonstructural carbohydrates such as sucrose and starch (Figure 4) [5].

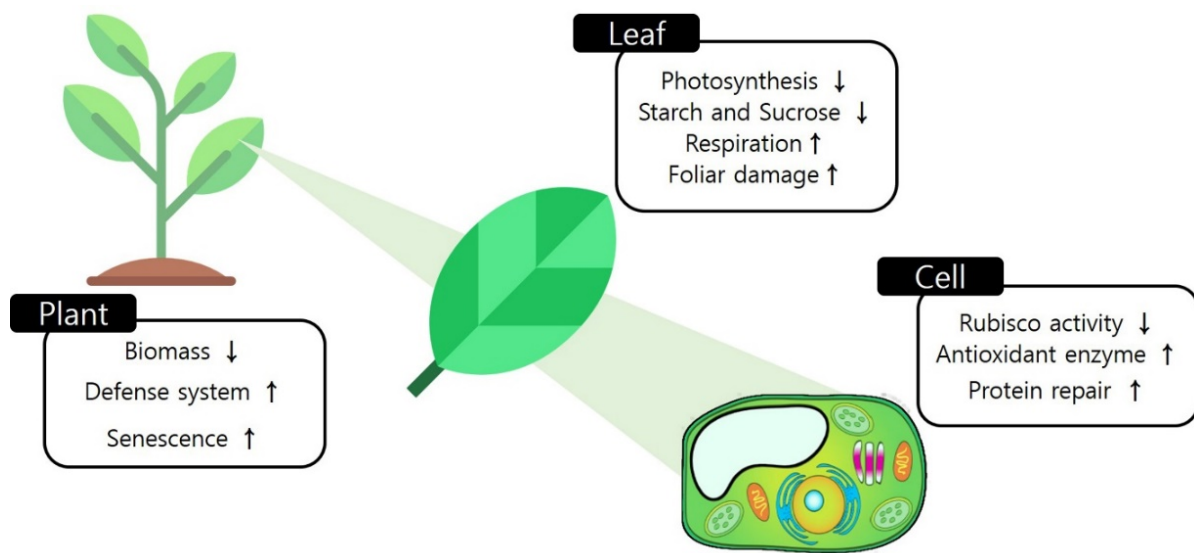


Figure 4. Plant changes caused by elevated O_3 at the plant, foliar, and cellular levels. Figure modified from Ainsworth et al. [5].

The rate of O_3 influx into leaves is controlled by the stomatal aperture. Stomatal closure is generally recognized as a response that limits O_3 uptake [30]. Acute O_3 exposure causes a notable decrease in stomatal conductance by causing reactive oxygen species (ROS) to accumulate in guard cells. In *Arabidopsis*, higher O_3 concentrations caused a rapid transient decrease in stomatal conductance within 3–6 min of exposure [33]. Several studies using open-top chamber experiments have reported that O_3 usually decreases stomatal conductance, consequently limiting CO_2 inflow to the leaves [34]. However, this process was not supported by the results of all experiments [35]. Some studies have reported that stomata were unable to close rapidly when impaired by exposure to elevated O_3 levels [36]. Regardless of the mechanisms involved, however, O_3 concentration is clearly an important determinant of plant physiological conditions. Plants can enhance their O_3 stress tolerance by regulating physiological mechanisms such as stomatal closure and an increase in antioxidants. This acclimation mechanism enables stomata to remain partly open and facilitates photosynthesis-related gas exchange, avoiding drastic plant growth decreases [37,38].

2.3. Biochemical Changes Caused by Ozone

During exposure to elevated O_3 concentrations, O_3 induces changes in plant characteristics at the biochemical and molecular levels [39,40]. O_3 enters leaves through the stomata and decomposes within the apoplastic space. This produces reactive oxygen species (ROS), such as the superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (OH^-) [41]. Although O_2^- and H_2O_2 are among the less reactive types of ROS, they are still recognized as dangerous ROSs because they can diffuse into cellular compartments [42]. OH^- has been thought to damage cell membranes because of its strong oxidation potential [43]. These apoplastic ROSs cause oxidative damage to cell membranes, proteins, and DNA molecules and can cause changes in enzyme activities, thereby leading to cell destruction [44]. ROS concentrations are mitigated by enzymatic antioxidants such as those involved in the ascorbate–glutathione cycle (AsA–GSH cycle), which include catalase (CAT), superoxide dismutase (SOD), ascorbate peroxidase (APX), dehydroascorbate reductase (DHAR), and glutathione reductase (GR) [45]. The detoxification of O_2^- by SOD produces H_2O_2 , which is then removed by CAT or APX. Peroxidase (POX) requires the phenolic compound guaiacol as an electron donor, which can then be used to reduce the amount of H_2O_2 . APX uses a reduced form of AsA to protect cells against oxidative damage caused by H_2O_2 . The oxidized AsA formed by APX activity is regenerated through other components of the AsA–GSH cycle, including dehydroascorbate reductase (DHAR) and monodehydroascorbate reductase (MDHAR). GR reduces the oxidized form of glutathione (GSSG) by reducing NADPH activity [46,47] (Figure 5).

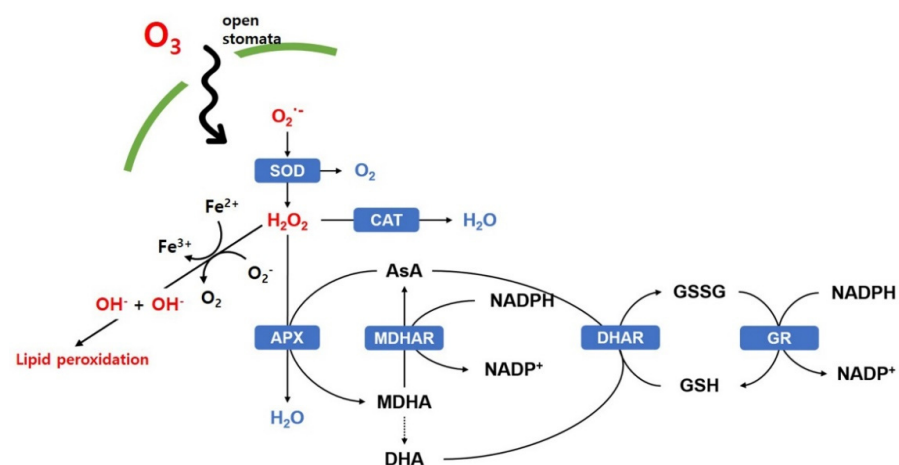


Figure 5. Reactive oxygen species detoxification systems in plants under ozone stress conditions. APX: ascorbate peroxidase; AsA: ascorbate; CAT: catalase; DHAR: dehydroascorbate reductase; DHA: dehydroascorbate; GR: glutathione reductase; GSH: reduced glutathione; GSSG: oxidized glutathione; H_2O_2 : hydrogen peroxide; MDHA: monodehydroascorbate; MDHAR: monodehydroascorbate reductase; O_2^- : superoxide anion; OH^- : hydroxyl radical; SOD: superoxide dismutase. Adapted from Foyer and Halliwell [46].

The accumulation of ROS limits stomatal movements and abscisic acid (ABA) levels independently of CO_2 [48]. Plant responses to O_3 involve the regulation of guard-cell ion channels. The accumulation of ROS in the extracellular space activates a still-unknown calcium channel protein and increases cytosolic calcium accumulation, thus activating calcium-dependent protein kinases (CPKs) [49]. Calcium is a second messenger that contributes to diverse signaling cascades that regulate plant stress responses. The activation of two types of anion channels—the slow anion channel 1 (SLAC1) and the quickly activating anion channel 1 (QUAC1)—then causes stomatal closure [50,51]. ROSs activate the protein kinase open stomata 1 (OST 1), which controls the outward rectifying SLAC1 and QUAC1 channels, as well as the inward rectifying K^+ channel KAT1 [52,53]. OST1 activity is limited by ABA-insensitive 1 (ABI 1) and ABI 2 protein phosphatase 2Cs, which are inactivated by

the abscisic acid receptor PYR/PYL. SLAC1 is also activated by CPKs and the ROS-induced guard cell hydrogen peroxide-resistant 1 (GHR1) protein [48] (Figure 6). ROSs are thus involved in the regulation of stomatal movements, as they affect various signaling pathways.

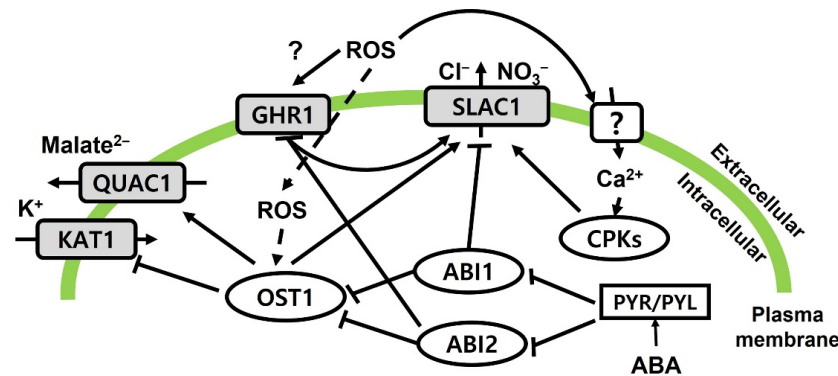


Figure 6. Diagram showing the regulation of guard cell ion channels in response to ozone. ABA: abscisic acid; ABI1: ABA-insensitive 1; ABI2: ABA-insensitive 2; CPKs: calcium-dependent protein kinases; GHR1: guard cell hydrogen peroxide-resistant 1; KAT1: inward rectifying K⁺ channel; OST1: open stomata 1; PYR/PYL: pyrabactin resistance/PYR-like; QUAC1: quickly activating anion channel. Figure from [47].

3. Plant Responses to Elevated Temperatures

3.1. Physiological Changes Caused by Elevated Temperatures

Heat stress due to prolonged exposure to elevated temperatures is a critical threat to vegetation and crop production worldwide [54]. Elevated ambient temperatures can positively influence plant growth and metabolism. Plants respond differently to elevated temperatures depending on their developmental stages and species characteristics [55]. However, temperatures above the upper tolerance threshold of a plant species decrease their net photosynthetic rate and total biomass [56]. Constantly elevated temperatures also cause physiological changes in plants. The plant characteristic most susceptible to elevated temperatures is water status [57]. Elevated temperatures increase transpiration and stomatal conductance. Therefore, elevated temperature stress is usually related to reduced water availability [58]. Under elevated temperatures, plants usually grow smaller leaves, and extend their root systems to increase water uptake and decrease water loss from leaves through transpiration [59]. Wahid et al. [57] showed that increased temperatures greatly reduced the leaf-water potential in treated plants, compared to the control group. Elevated temperatures also negatively impacted plant height, leaf area, and total biomass of *Brassica napus* due to reduced CO₂ assimilation [59]. Changes in CO₂ assimilation under elevated temperatures are good indicators of plant responses to high temperatures. The photochemical reactions in the thylakoid lamellae have been regarded as the prime sites of injury at elevated temperatures [60]. The functioning of PS II is highly decreased or partly stopped at elevated temperatures due to it being thermolabile [61]. Additionally, the photosynthetic ability of plants at elevated temperatures is limited by the decreased activity of Rubisco, an enzyme involved in carbon fixation [12]. Further, an imbalance between photosynthesis and respiration impairs plant growth under elevated temperature conditions, because high temperatures generally decrease the rate of photosynthesis in plants while increasing the rates of photo- and dark respiration [57]. These physiological changes in plants at higher temperatures result in decreased growth and development, thus reducing total biomass and crop production.

3.2. Biochemical Changes in Response to Elevated Temperatures

Temperature is the primary environmental factor affecting plant growth and development. Generally, the biochemical characteristics of plants are more negatively affected by air temperatures above 5 °C than the optimum [55]. Elevated temperature stress may cause

the excess production of reactive oxygen species (ROS), leading to oxidative stress [62]. ROSs, including singlet oxygen ($^1\text{O}_2$), the superoxide radical (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (OH^-), can cause lipid peroxidation, protein oxidation, changes in enzyme activities, and oxidative damage to membranes, all of which lead to cell death [62,63]. Plants have a variety of enzymatic and non-enzymatic antioxidant systems to diminish ROS levels in their tissues, thereby protecting their cell membranes from oxidative damage [64].

Elevated environmental temperatures increased leaf temperatures, which degraded the activity of enzymatic antioxidant systems that are responsible for the malondialdehyde (MDA) content in rice leaves in [65]. Measuring MDA content usually indicates the occurrence level of lipid peroxidation, which is a sign of damage in living organisms [66]. In a study with wheat kept at 33 °C, oxidative stress significantly increased membrane peroxidation by 28%, greatly increasing solute leakage [67].

Heat stress due to elevated temperatures induces the expression of stress proteins that are not produced under normal conditions [68]. Heat-shock proteins (HSPs) are exclusively involved in the heat-stress response. HSPs prevent heat-stress-induced protein denaturation and mediate protein homeostasis [69]. HSPs are classified into five families based on their molecular masses: HSP100, HSP90, HSP70, HSP60, and sHSPs [70]. The diversity and abundance of HSPs that can be expressed, indicates the heat tolerance of a plant species. Heat-stress factors (HSFs) and HSPs play important roles in the plant heat-stress response [71] (Figure 7). HSFs located in the cytoplasm mainly control the transcription of HSP genes [72]. At least 21 HSF members cloned in plants have been found to cooperate in all steps of their heat-stress responses. Therefore, HSFs are regarded as transcriptional activators of heat-shock responses [73]. HSF proteins are grouped into three conserved classes (A, B, and C) based on the structural features of their oligomeric domains. HSFA1a is the master regulator for the heat-stress-induced synthesis of HSFA2, a major heat-stress factor [74]. Heat-induced gene expression resulting in HSP synthesis is initiated by mechanisms that can sense and transduce signals of heat stress to HSFs [75,76]. HSFs then bind to a heat-shock promoter element (HSE) in the promoter region of the HSP gene and initiate transcription [75]. HSPs mainly stabilize partially unfolded proteins to help maintain assembly and reduce protein degradation. However, while these proteins contain no specific information about the correct folding of any specific proteins, they do prevent unproductive interactions that result in unfolding and loss of structure and function [77].

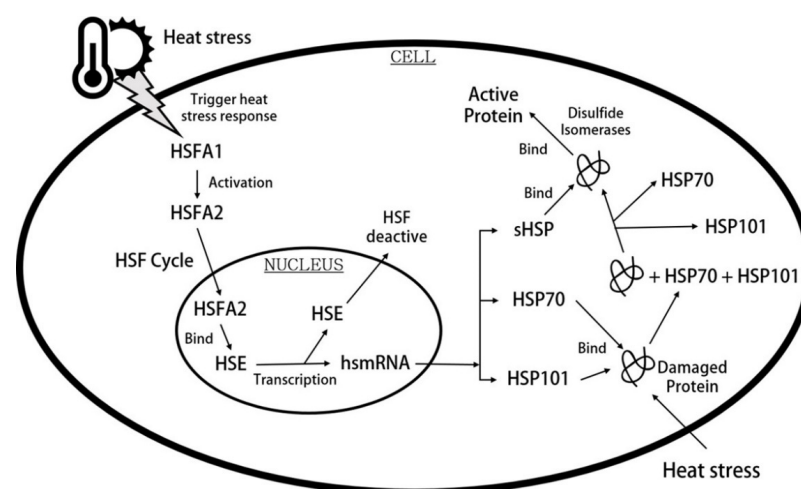


Figure 7. Diagram of the heat-shock protein pathway. HSFA1, one of the heat-stress factors (HSFs), triggers a heat-stress response by inducing the expression of HSFA2, which forms co-activators. HSFA2 binds to a heat-shock promoter element (HSE), which then induces the expression of various heat-shock proteins (HSPs). HSP101, HSP70, and sHSP help to repair damaged proteins. Adapted from Asthir [71].

4. Plant Responses to Ozone under Elevated Temperatures

To date, researchers have studied the effects of single abiotic stress parameters on plants. However, the interactive effects of stress factors on plants remain unclear, because different plant responses can be deduced from combinations of single factors [78]. In particular, the considerable variety of plant responses to elevated O₃ is most likely related to the interactive effects of other co-occurring environmental variables including temperature, solar radiation, drought, and increased NO_x in the atmosphere [79]. In this review, we focus on the plant response on the interactive effect of elevated temperature and O₃. Because temperature directly affects chemical kinetics and VOC emissions, both of which are associated with O₃ production, temperature is known to be a strong predictor of tropospheric O₃ concentration levels. Furthermore, elevated temperatures may promote O₃ accumulation by influencing sunny, dry, and stagnant atmospheres [80]. For these reasons, high temperature and elevated O₃ conditions generally co-occur [11].

Because O₃ and temperature can each independently affect the physiological processes of plants, the interplay of factors must be investigated. Many studies have focused on the interaction of these factors to study possible ways in which temperature affects plants exposed to O₃. Plants exposed to modest temperature elevation (1–5 °C) showed increased photosynthesis, growth, and biomass in various experiments [24,81,82]. Increased O₃ concentration, however, can have a variety of detrimental effects on plants, including reduced photosynthesis, stomatal conductance, and growth [83]. Elevated O₃ conditions are thought to suppress plant development and growth. At the same time, elevated ambient temperatures are expected to enhance plant development and growth. Nevertheless, the final effect of elevated ambient temperature and O₃ on plant responses may be determined by how these factors interact with the plant's physio-biochemical processes. Increased temperatures above the optimum may decrease the uptake of ozone by reducing stomatal conductance [55,84]. However, due to increased stomatal conductance, moderately elevated temperatures (+5 °C) can enhance ozone uptake [24,85]. Lee et al. [24,85] reported that +5 °C above an optimal temperature increased O₃-induced foliar damage and reduced photosynthesis in chamber experiments with constant humidity. Conversely, O₃ accelerated leaf senescence in silver birch (*Betula pendula*), while a +1.2 °C-temperature increase delayed leaf senescence in field experiments [86]. Elevated temperatures in natural conditions are frequently related to increased vapor pressure deficits (VPD), which significantly affect stomatal movement [79,87] as well as stomatal O₃ flux [88]. Many studies have shown that stomatal O₃ uptake, rather than the O₃ level, is related to O₃ deposition and damage to plants [88–90]. Pea cultivars with slower stomatal closure under elevated O₃, and thus higher O₃ flux in the interior leaf tissue, had more O₃ damage [91]. Stomatal O₃ flux can be determined by leaf boundary layer resistance and stomatal resistance; both factors are affected by heat flux and VPD [79]. Without confounding changes in humidity, the stomatal response to higher ambient temperatures obviously showed increased stomatal opening in relation to O₃ flux [92,93]. Presently, the plant physiological characteristics resulting from the combined effects and interacting mechanisms of elevated O₃ and temperature are debatable [24,85,87,88,94,95].

5. Conclusions

In this paper we summarized the plant responses to the combined and individual effects of elevated O₃ and temperature, including physiological and biochemical changes. Plant responses to abiotic stressors, such as O₃ and temperature, are dynamic and complicated. The inevitable impacts of O₃ and temperature on terrestrial vegetation may exceed those of any other abiotic stress factors.

As demonstrated in Section 2, Plant Responses to Ozone, tropospheric O₃ is a critical air pollutant with many negative consequences for plants. At the physiological, biochemical, and anatomical levels, the mechanisms of O₃ effects on plants are relatively well characterized. Elevated O₃ has been correlated with photosynthesis degradation due to lower carboxylation efficiency, effects on photosynthetic electron transport, and effects on

stomata. Because O₃ enters the leaf through the stomata, differences in stomatal opening may potentially cause O₃ sensitivity. Because O₃ can decompose within the apoplastic space in the cell after penetrating through stomatal pores, it can form reactive oxygen species (ROS).

The examples discussed in Section 3 clearly show that extremely and moderately high temperatures can cause physiological and biochemical changes in plants in different ways. Moderately elevated temperatures may positively impact plant growth and metabolism. Extremely high temperatures, however, may decrease plant growth due to an imbalance between photosynthesis and respiration. Extremely high temperatures also increase leaf temperature, thereby reducing antioxidant system function.

Increased temperature and O₃ conditions frequently co-occur because elevated temperatures may increase O₃ concentrations in nature. The combined influence of both factors can produce results that are remarkably different from those produced by a single factor. Stomatal movement, which can be highly determined by temperature, may be an important factor in the plant response to O₃ exposure. Stomatal O₃ flux can be determined by the leaf boundary layer, vapor pressure deficit (VPD), and relative humidity, which can be changed by temperature. The leaf boundary layer, vapor pressure deficit (VPD), and relative humidity are also factors that influence stomatal O₃ flux. As a result, the plant response to a combination of elevated O₃ and temperature is still a subject of debate.

To obtain a comprehensive understanding of how plants respond to both parameters, more extensive methodological approaches, such as long-term eddy-covariance, open top chambers, and free-air enrichment systems, are needed. Plant response assessments at the organ, tissue, and cellular levels must also be extended to studies of proteomics and enzyme activity.

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