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„GOOD" AND „BAD" OZONE - EVALUATION ON THE BASIS OF PLANT REACTION TO OZONE

„DOBRY" I „ZŁY" OZON - OCENA NA PODSTAWIE REAKCJI ROŚLIN NA OZON

Abstract: Ozone is a natural and artificial chemical compound of Earth's atmosphere. O_3 is an absorbent of ultraviolet and infrared radiation and has strong oxidative properties. In the stratosphere the ozone layer protects the planet's surface from dangerous UV radiation, its indirect effect on plant organisms is positive so stratospheric ozone can be called "good" ozone. The depletion of the ozone layer, as a result of atmosphere pollution, described as an ozone "hole" is causing UV_B radiation enhanced level on Earth's surface. The genetic, cytological, physiological and morphological reaction of prolonged UV_B exposure in plants is twofold: it damages plants and simultaneously plants protect themselves and repair their injuries. The ozone in the troposphere originates from natural sources and is also a secondary pollutant, formed in photochemical reactions, leading to "smog" and ozone "spots" occurrence. As a strong oxidant, O_3 is directly toxic to plants and can be recognized as "bad" ozone. Ozone is also classified as a "greenhouse" gas, participating in global warming. It is difficult to value the impact of O_3 as a "greenhouse" gas on plants. The combined effect of O_3 changes in the stratosphere and troposphere on plants can be estimated as loss in crop yield and in productivity of natural ecosystems.

Keywords: tropospheric and stratospheric ozone, ozone "hole", excess of ultraviolet radaition, „greenhouse" effect, "greenhouse" gas, excess of ozone, photochemical „smog", ozone "spots"

Ozon - O_3, trioxygen, as an allotrope of oxygen, is itself neither good or bad. But the impact of O_3 on the environment evidently can be evaluated. O_3 influences living organisms directly and indirectly. Organisms react directly to the actual level of O_3 in the atmosphere. The indirect impact means that actual O_3 concentration changes other environmental factors: the ultraviolet - UV strong irradiation and infrared - IR weak irradiation. As a result organisms react to the excess of UV radiation and to global warming.

When complex ozone influence on terrestrial higher plants is considered, the complicated reaction of organisms to this gas becomes clearer. The better understanding of the problem of changes in O_3 concentration helps to interpret correctly such environmental pollution phenomena as „greenhouse" effect or ozone "hole" and its impact on plants.

All living organisms react to atmospheric O_3 concentration, but plants are especially in danger. They rely on the absorption of sun radiation to assimilate carbon dioxide in

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photosynthesis but at the same time the UV frequency of the sun's radiation spectrum is harmful. Plants can not move and the local tropospheric O_3 at abnormally high concentrations in the photochemical "smog" or ozone "spots" acts as a secondary air pollutant and the future increase in global background ozone concentrations is predicted.

It is difficult to estimate the global financial loss in plant biomass production and ecological damage caused by changes in the concentration of stratospheric and tropospheric O_3. It depends to a great extend on O_3 pollutant doses and UV absorption by plants and on plant tolerance to stress of O_3 and UV excess. The current risk assessment of O_3 plant damage is taking into consideration the complex effects of O_3 from stomatal flux, detoxification and repair processes, to carbon assimilation and allocation in individual plant. The rough estimation of crops yielding or natural ecosystems productivity, including forest growth and species composition in O_3 and UV polluted environment, could be considered as a feasible indicator of O_3 direct and indirect damage, in the specific context of a changing global environment.

Ozone as an atmospheric compound

Ozone as a compound of different atmospheric layers causes mutually connected phenomena (Fig. 1), which affect plants in different ways. O_3 is a double radical and has strong oxidative features, which are responsible for its direct toxic effect on living organisms. At the same time O_3, as an atmospheric gas, prevents highly energetic UV sun radiation from passing through the atmosphere. O_3 is absorbing, together with other atmospheric gases, 99% of UV_C, about 50% of the most harmful UV_B (290-320 nm) and small quantities of UV_A.

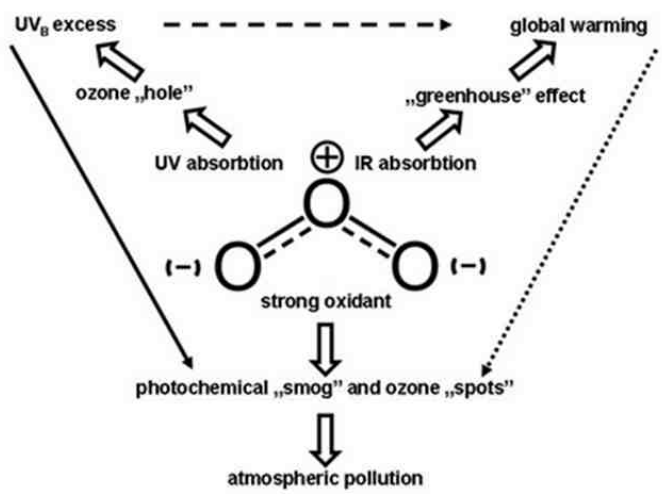

Fig. 1. Ozone properties and its impact on the phenomena occuring in the Earth's atmosphere. O_3 absorbs UV_B in the stratosphere. Ozone „hole" transmits excess of ultraviolet radaition UV_B to the troposphere. Excess of UV_B stimulates the formation of O_3 in the troposphere (present in photochemical „smog" and ozone "spots"). O_3 in the troposphere absorbs UV_B and infrared radiation (IR), contributing to the „greenhouse" effect and global warming. Excess UV_B in troposphere also stimulates global warming. Global warming stimulates the formation of O_3 in troposphere

O_3 shows uneven vertical arrangement in the Earth's atmosphere. There is 90% of O_3 in the stratospheric ozone layer and up to 10% of O_3 in the tropospheric layer. The atmosphere layer at the altitude of between 10-20 and 45-55 kilometers is called stratosphere. Troposphere is the lowest and the thinnest atmospheric layer. Its thickness, 5-9 km above the poles and 13-18 km above the equator, depends on season and geographical coordinates. O_3 concentrations in both layers vary essentially in time and in space [1].

O_3 concentration is increasing from the middle to the upper part of the *stratosphere*, where simultaneously the temperature is rising. O_3 is formed there from dioxygen by the action of UV light of 180-240 nm and it breaks down by the action of UV 200-320 nm, in reaction reverse to synthesis. O_3 instability causes dynamic balance between O_3 and O_2 concentration in the air and the absorption of a significant part of solar UV radiation by the Earth`s atmosphere. O_3 in the stratospheric ozone layer acts as anti UV sunscreen, indispensable for the life of terrestrial organisms [2].

The disturbance of dynamic balance between O_3 and O_2 concentration and stratospheric O_3 degradation, called ozone "hole", is the result of chemical reactions between man-made gas pollutants. Stratospheric O_3 depletion is mainly the effect of catalytic destruction of ozone by atomic halogens, comprising fluorine (F), chlorine (Cl), bromine (Br), iodine (I), and also by nitric oxide NO and nitrogen dioxide NO_2 [3].

The investigation of chemical reactions of O_3 in the atmosphere and the elucidation of its destruction mechanism by chlorofluorocarbon gases was honored by a Nobel Prize in Chemistry received by M. Molina, F.S. Rowlands and P. Crutzen (USA) in 1995.

The standard way to measure the total ozone amount in a vertical atmospheric column is by using *Dobson units* (DU) [4]. The earliest symptoms of the phenomenon of ozone layer depletion were observed over a polar region of Antarctica in the early 80s of last century. The first ozone „hole", short in duration, was manifested as the decline of total volume of O_3 to almost 70% of the normal value and O_3 concentration decrease from around 400 to 96 DU. This phenomenon was repeated with various intensity in the next years, also over the northern pole, leading to a steady decline of the total volume of O_3 in the stratosphere. It is estimated that the level of O_3 decline, from the early 90s of the last century up to the middle of this century, will drop by about 15% [5]. Stratospheric O_3 destruction is expected to cause the maximum elevation of UV_B radiation (280-315 nm), estimated for 15% in winter/spring and for 8% in summer/autumn in regions of the middle northern latitude [6]. For each 1% of stratospheric O_3 decrease UV_B radiation will increase 1.3-1.8% [7]. Terrestrial plants will experience high level of UV_B radiation for a long time after stopping air pollutants emission since such pollutants are resistant to environmental degradation and they stay in the upper atmosphere from 40 to 150 years [8].

In the *troposphere* O_3 is a secondary pollutant, originating mainly from anthropogenic sources. O_3 production in the lowest part of the Earth's atmosphere is a very complicated, nonlinear process, covering thousands of chemical reactions between primary pollutants: *volatile organic compounds* (VOC) and NO_x. NO_2 photolysis is one of numerous O_3 generating reactions, so high variability of tropospheric O_3 concentration is a function of daily and seasonally changing meteorological conditions and of latitude [9]. Tropospheric O_3 concentration is regulated not only by several synthesis and breakdown reactions but also by processes of vertical and horizontal transport. Natural O_3 concentration is low (from 0.002 to 0.3 ppm, 0.04 ppm average value). In nature O_3 is formed from gaseous hydrocarbon (isoprene and terpene) emissions by plants and animals and in small quantities

it is the effect of thunderstorms. Some O_3 is transported to the troposphere from the stratosphere [10]. The location of high tropospheric O_3 concentrations is highly correlated with the vicinity of urban and industrial areas, the main sources of primary pollutants and with climatic and meteorological cycles [9, 10]. The increase of natural O_3 concentration is estimated from 0.05 to 2.5% at an annual rate and continuous global O_3 concentration raising is observed in the troposphere [11]. This reactive and strong oxidant is locally present in the photochemical „smog" observed in industrial cities, most commonly in sunny, warm, dry, windless climates and in conditions of temperature inversion. It is a "smog" compound generated in photochemical reactions in polluted air. The products of the combustion of fossil fuels, especially NO_x, volatile hydrocarbons and carbon monoxide derived from fumes and vehicle exhaust gases are "smog" forming pollutants. O_3 is the main source of hydroxyl radicals in the troposphere, which starts almost each oxidation process in the atmosphere [10]. The mechanism of photochemical "smog" formation is mainly of a radical reaction nature and "smog" is a highly toxic mixture of air pollutants, which can include also peroxyacyl nitrates (PANs). The "smog" impact on plants can be lethal [4]. The photochemical reaction of tropospheric O_3 synthesis is in balance with O_3 breakdown reaction in darkness, dependent on NO_x and VOC concentration [12, 13]. A majority of O_3 particles is disintegrated in weeks after precursors breakdown. Nevertheless the highest O_3 concentration may be observed, as the ozone „spots", thousands of kilometers from the densely populated place of primary pollutants emission [13].

The exposure of plants to elevated O_3 concentration was initially assessed by the AOT40 index (*accumulated exposure over a threshold of 40 ppb or* 10^{-9} dm^3 dm^{-3}). The critical level for ozone effects on sensitive perennial plants (grasses) communities has been recommended as an AOT40 value of 5 ppm h^{-1} over a growing period of 6 months. However, the responses of vegetation to O_3 are evidently better related to the dose absorbed through stomata than to atmospheric O_3 concentration. In recent years the risk of O_3 damage to plants is based on the cumulative flux of O_3 through the stomata, according to the DO_3SE (*Deposition of Ozone for Stomatal Exchange*) model, which is incorporated into the EMEP (*European Monitoring and Evaluation Programme*) model. This model is capable of estimating O_3 deposition on plants as well as O_3 stomatal flux into leaf tissues and includes detailed algorithms for assessment of O_3 drawn from the air by main European species of crops and trees [14].

O_3 is one of the "greenhouse" gases. The "greenhouse" effect is a phenomenon of connected effects of the penetration of sun radiation through the atmosphere to warm a planetary surface and of the absorption by "greenhouse" gases of infrared radiation re-radiated from the surface. Since part of the thermal re-radiation goes back towards the globe surface and the lower atmosphere, it results in an increase of the average surface temperature and global climate change [3]. The high concentration of "greenhouse" gases in the Earth`s atmosphere is the reason for natural and artificial "greenhouse" effect. The artificial "greenhouse" effect results from the emissions of binary gases, mainly CO_2 of anthropogenic origin. O_3 as a trioxygen absorbs also in the upper troposphere IR radiation emitted from the planetary surface and it is very effective in the process of warming the stratosphere [5]. At the same time the absorption of part of solar UV radiation by stratospheric O_3 partly reduces the "greenhouse" effect. UV radiation is absorbed by O_3 not only in the stratosphere but also in the troposphere [15]. As a result O_3 makes up for only a few percent of the "greenhouse" effect phenomenon [3, 5]. Positive or negative impact of

the "greenhouse" effect on plants growth and development is not the subject of this article anyway.

Direct impact of tropospheric O_3 on plants

The responses of plants to O_3 include metabolic, physiological, anatomical and morphological changes [16] as well as changes in the signal transduction pathway and gene expression [17, 18].

Detoxification (*ie* O_3 decomposition) occurs partly on the surface of the cuticle, in a chemical reaction with waxes, and in the hot climate in reaction with volatile isoprenoids, diffusing into the air from the leaf. A large part of O_3 is deposited on the surface of the leaf cuticle and on the stems, especially when they are wet. Under natural conditions, the penetration of O_3 through the leaf cuticle is negligible, and O_3 diffuses into the leaf through open stomata [15, 19, 20]. The degree of stomatal opening, which depends on many factors, is regulated also by O_3. The increase of O_3 concentration in the atmosphere stimulates stomatal closure and reduces the penetration of O_3 into the leaf, but at the same time limits the CO_2 uptake for photosynthesis. The mechanism of stomatal closure is rather not due to the direct O_3 impact. The increase of CO_2 concentration in the leaf, which is the effect of photosynthesis being inhibited by O_3, is the probable reason for stomata closure. The impact of O_3 on stomatal closing *via* hydrogen peroxide - H_2O_2 can not be excluded. H_2O_2 is formed in the aquatic environment with O_3 participation and regulates the functioning of calcium channels in membranes of stomata cells [21]. The average hourly O_3 dose of 70 nmol mol^{-1} and short exposures causes quick stomata closure. Nevertheless in a very sensitive species, and at higher concentrations and longer exposures, stomatal closure response to O_3 becomes slow. It may be related to the induction of the *ethylene* (ET) synthesis by O_3. ET reduces the sensitivity of stomata cells to signals for closing stomata, *ie* to the increased concentration of the phytohormon - *abscisic acid* (ABA). Furthermore ET accelerates the aging of leaves. The slowdown of stomata closure leads to reduced control of water losses from leaves, because stomata are only partly closed. The degree of stomatal opening is also modulated by the "greenhouse" effect. Increase of the CO_2 concentration, the gas recognized as the most important "greenhouse" effect component, causes stomata closure. Similarly, the shortage of soil water accompanying steppe formation, and the stress of drought, which are anticipated as a result of global warming, causes stomatal closure. O_3 absorption by plants under these conditions is limited, and its phytotoxicity is reduced [15, 22].

The dosage of pollutant, that passes through open stomata into the leaf parenchyma, depends on the degree of stomata opening and on the exposure time. The quantity of O_3, further penetrating parenchyma intercellular spaces, results from the balance between the O_3 uptake and O_3 decomposition inside the leaf. In the cell walls of parenchyma the ascorbic acid is to the great extent responsible for O_3 breakdown [23].

The mechanism of O_3 toxicity is not entirely clear. Undoubtedly O_3 induces a state of oxidative stress in the leaf. Oxidative stress represents an imbalance between the predominant formation of *reactive oxygen species* (ROS) an the ability of the plant to readily detoxify the reactive oxidative intermediates and to repair the resulting damage. O_3 is therefore one of many environmental factors that trigger the formation of harmful excess of ROS. In the literature, however, there is increasing evidence indicating the positive role

of ROS in plants. ROS participates in the signal transduction in cells and regulates many physiological processes, including gene expression [24]. The signaling role is associated with small ROS concentrations. Biphasic response to the concentration of toxic substances is called hormesis in biology. It can be assumed that in case of O_3 the hormetic effect may occur. Low concentration of O_3 can cause a positive plant reaction but high O_3 concentration shows destructive effects. Only high concentrations of O_3 have been investigated to date, causing oxidative stress and damaging cellular structures [25].

O_3 concentration inside the leaf decreases rapidly after absorption, but the decomposition products of ozonolysis may influence the life processes of plants. The extent of injury depends on external O_3 concentrations and on the duration of O_3 contamination [26]. High O_3 concentration in a relatively short time (150-300 $\cdot$ 10^{-9} dm^3 dm^{-3} for 4-6 hrs) causes major changes, manifested as leaves damage and aging. Prolonged exposure to low concentrations (<100 $\cdot$ 10^{-9} dm^3 dm^{-3} for days to months) does not cause visible symptoms on leaves, but due to inhibition of photosynthesis, strongly limits plant productivity and reduces the biomass. At high doses O_3 can act directly as a powerful primary oxidant, reacting with components of cell walls (phenols, proteins, glycoproteins). After the dissolution of O_3 in the water film surrounding the cells O_3 decomposes and secondary oxidants arise spontaneously, as was mentioned before. These are ROS such as H_2O_2, superoxide anion $O_2^{-\bullet}$ and singlet oxygen [12, 27]. In the cell walls of leaf parenchyma a hydroxyl $OH^\bullet$ and perhydroxyl $HOO^\bullet$ radicals are also formed [28]. ROS production in apoplast is biphasic and can be caused by O_3 decomposition in reactions with phenols and with other compounds susceptible to oxidation. The first phase of the oxidative stress results directly from the O_3 impact on the organic compounds of the cell wall and plasma membrane surface. The second phase is the oxidative burst of secondary oxidants from plant cells, which runs initially in the apoplast. After plasma membrane injury, oxidative stress occurs in the cytoplasm and cell organelles and causes the visible symptoms on leaves. The ozone-injured leaf surface may show stippled discoloration, silvery gloss, spot necrosis and/or colored spots [27]. Beside the ROS burst from leaves O_3 treatment causes the VOC emission by plant [29]. In the internal spaces of the leaf O_3 may also react with hydrocarbons: ET or isoprene - gaseous hydrocarbon formed in the specific environmental conditions in plants. The plant exposure to O_3 polluted air induces ET emission from sensitive plants. O_3 has also the ability to oxidize ET. The role of ET and isoprene in plant response to O_3 is not clearly understood. ET can increase oxidative injury through the formation of peroxides. On the other hand ET can also act as the stabilizer of cell membranes and the “scavenger” of free radicals, thus reducing damage [27].

Destruction of cell membranes is mainly due to membrane lipid peroxidation. Ozonation of polyunsaturated fatty acids chains results in the formation of H_2O_2 and reactive peroxides of fatty acids, which intensify the oxidative stress and generate formation of subsequent reactive molecules. O_3 and ozonolysis products also react with sensitive amino acid of membrane proteins, containing thiol groups. Organic peroxides of proteins, amines and nucleic acids are also the secondary oxidants. Other plant hormones, not only ET, may also be oxidized and their membrane receptors can be oxidized too. Some secondary metabolites, such as already mentioned phenols, react with O_3 as well [25, 27, 28]. O_3 damage of the membranes causes changes in the transport of ions, the increase in membrane permeability, moreover it inhibits the activity of proton pumps, reduces the

membrane potential and causes the uncontrolled influx of Ca^{2+} from the cell walls to the protoplast [28].

Metabolic changes, caused by the contamination of O_3, are accompanied by physiological process disorders. Quick responses observed in leaves are: reduction of the intensity of *photosynthesis* - Fn, increased respiration rate, stimulation of phloem unloading, inhibition of floem loading and leaf chlorosis and necrosis [12, 30-32]. Inhibition of Fn by O_3 is due to disruption of photosynthetic electron transport, reduced carboxylic activity of the RuBiCO enzyme complex and / or stomata closure [21].

It may also be associated with changes in the structure of chloroplasts, chlorophyll degradation and reduced expression of several major nuclear genes, encoding proteins responsible for photosynthesis [12]. Changes in chlorophyll fluorescence parameters show that under the influence of O_3 the functioning of photosystem PSII and xantophyll cycle is disturbed, which may cause photosynthesis photoinhibition elevation [27].

Chronic reactions of plants appear after days or weeks of exposure to low O_3 concentrations. The serious disturbance of water and minerals uptake is an effect of cell membranes destruction during long-term exposure to O_3 and results in highly limited plant growth. There are changes in the distribution pattern of assimilates and the export reduction of assimilates from shoot to roots. In effect the increase of the sensitivity of plants to stresses, related with poor soil conditions, is evident and it is accompanied by the reduction of mycorrhiza symbiosis [12, 27, 33]. Limitation of root and shoot biomass production reduces yield, seed production and has a negative impact on plant cold hardening. Growth inhibition may be due not only to reduced intensity of photosynthesis, but also to an increase in respiration, changes in hormonal balance and reduced leaf surface [27].

Plant growth is usually inhibited, but growth stimulation was also reported [31, 34].

The stimulating effect of small O_3 doses on plant growth, probably occurred by modifying the growth regulating mechanisms. It is unclear whether this is an effect of O_3 or H_2O_2 hormesis, as H_2O_2 is a product of O_3 dissolution in water [25]. O_3 also reduces the reproductive processes of plants due to inhibition of flowering, pollen damage, inhibition of pollen germination and pollen tube growth and as a result of flowers, pods or seeds falling and also reduced weight of seeds and fruits [11, 27, 35]. The premature aging of plants and shedding of leaves is also observed [12]. Leaf necrosis induced by O_3, known as "ozone spots", appears a few hours after exposure to high (usually above 150 10^{-9} dm^3 dm^{-3}) concentrations of O_3 [27, 36]. The appearance of local necrotic spots is due to the death of leaf parenchyma cells and to the callose deposition, which separate the healthy from necrotic cells. The thickening of palisade parenchyma and the accumulation of phenolic compounds appears in leaves of certain tree species [37]. There are several symptoms of leaf damage by O_3, so it is difficult to give their universal physiological interpretation. Symptoms belong to two categories: characteristic small necrotic spots, and surface discoloration caused by accumulation of phenolic pigments (tannins and flavonoids - including anthocyanins), acting as antioxidants [37, 38]. The injury usually appears between the conductive bundles of leaves, on the upper surface of older and middle age leaves, and in some species on both sides of the leaf blade. Type and size of damage spots depends on the duration and concentration of O_3 pollution, weather conditions and plant genotype and location [39]. The acute visible injury for many horticultural crops, with a market value dependent on their visible appearance, can cause an obvious and immediate loss of economic value [16]. Different changes can occur under the influence of O_3 on leaves in

different species, but they are not a good indicator of the species' sensitivity to O_3, as the correlation with growth limitation is low [40]. Leaf O_3 damage is a good indicator of environmental pollution anyway. For biomonitoring of O_3 contamination a very sensitive tobacco variety Bel-W3 is commonly used as an indicator plant [25]. The analysis of plants productivity changes in response to tropospheric O_3 pollution may serve as an assessment of O_3 impact on plants. There is ample evidence that current atmospheric concentrations of O_3 are sufficient, in many parts of the world, to significantly reduce the crop yields and wild plants productivity. Yield loss may result from damage of plant vegetative or reproductive organs and from disruption of several physiological processes. During the generative phase of development plants show greater sensitivity to O_3 than in the vegetative stage. The biomass loss is often accompanied by loss of crop quality. Deterioration of crops quality and changes in timing and abundance of flowering are of particular importance in the case of ornamental plants. Global crop yield loss, caused by the toxic effect of O_3, is currently estimated at 7-12% for wheat, 6-16% for soybeans 3-4% for rice and 3-5% for maize, resulting in financial loss of approximately $ 14-26 billion [41]. Tropospheric O_3 is a serious threat not only for crop yields but also for the productivity of grasslands and forests and for carbon sequestration in ecosystems [30]. Decrease in the quantity and quality of seeds and a negative effect on germination and seedling growth of susceptible species may have significant ecological consequences in natural ecosystems polluted by O_3 [11].

Tropospheric O_3 pollution is thus for sure a negative environmental factor that triggers the defense reactions of stressed plant.

Plants constitutive protection and induced defense against excess of O_3

Plants are protected against permanent damage from O_3 by: a thick cuticle layer, stomata closure and detoxification of O_3 and secondary oxidants [15]. In response to oxidative stress plants enhance reactions of enzymatic and non-enzymatic antioxidant defense system and biochemical repair mechanisms. In tissues of O_3 treated plants the accumulation of a low molecular weight antioxidants: ascorbate, α-tocopherol, glutathione and of some antioxidative enzymes: *superoxide dismutase* (SOD), catalase, peroxidase and glutathione reductase as well as secondary metabolites in the form of dyes and volatile isoprenoids is high [12, 25]. In water O_3 chemical reactions with lipids and proteins containing double bonds occur more slowly than with the antioxidants. The disintegration of O_3 and ROS in plant cells, especially with the participation of ascorbate in apoplast, and glutathione in cytoplasm (Halliwell-Asada path in chloroplasts), protects membranes from oxidation to the great extent [42]. Despite the fact, that O_3 in principle does not overcome the barrier of the cell membrane, it starts the signaling pathways and affects gene expression changes. O_3-induced transcripts of the DNA sequences encode proteins associated with changes in cell wall structure, typical of defensive response to disease [43]. Oxidative burst of H_2O_2 activates the signal transduction path leading to the induction and/or repression of the genes that trigger defensive reactions of plants to pathogens [42, 44].

The increase of H_2O_2 concentration stimulates the production of *salicylic acid* (SA) and the transient increase in the level of transcripts of many genes is observed. These are mainly genes encoding enzymatic proteins from synthesis pathways of secondary metabolites (phytoallexins, lignin, polyphenols, callosis), extensins - *hydroxyproline-rich glycoproteins* (HRGPs) and PR (*Pathogenesis Related*) proteins. Through the overlap of signaling

pathways the plant reaction to O_3 pollution is related to HR (*hypersensitivity reaction*) to pathogens and to *programmed cell death* (PCD) and to *systemic acquired resistance* (SAR) to pathogens. O_3 can increase plant resistance to pathogens then. Although the damage, caused by O_3, may increase susceptibility of plants to pathogen attack, simultaneously O_3 induces resistance to pathogens. This occurs also by the stimulation by O_3 of genes expression encoding antioxidant enzymes. O_3 treatment increases the expression of synthase and oxidase of 1-*amino-cyclopropyl*-1-*carboxylic acid* (ACC) genes. ACC is a precursor of ET, so the production of ET is stimulated. ET over-production may cause, as mentioned before, local cell death, leading to so-called ozone necrosis [27, 28]. O_3 alters the concentration of *jasmonic acid* (JA) and *nitric oxide* (NO) signalling particles and activates the metabolism of polyamine growth regulators [43]. In several molecular studies the impact of O_3 treatment on plant transcription factors was found and increased expression of genes involved in signaling and metabolic pathways associated with immune response was stated. But results of scientific research show also the decrease of the expression of photosynthesis and energy transformations related genes [17].

Indirect effect of stratospheric ozone on plants, resulting from an excess of UV_B radiation

The photoreceptors of UV_A rays are cryptochrome and phototropins [7, 45], and UV_B radiation photoreceptor is in *Arabidopsis* a dimer protein UVR8 [46]. The plant receives a UV_B stimulus by specific photoreceptor molecules and by absorbing photons by other compounds in the cell. UV_B radiation is reflected in about 10% and absorbed in 90% and practically is not transmitted through the leaf. UV_B is cytotoxic, mutagenic and cancerogenic for live tissues [47].

Nucleic acids, proteins, lipids and quinones are compounds which directly absorb UV_B radiation in plant cells. UV_B absorption by the leaf may be accompanied by visual symptoms such as chlorotic or necrotic spots and discoloration [8, 48]. The response of plants to excess UV_B is associated rather with modification of activity than with typical genetic damage. Regulation of gene expression involves changes in the rhythm of development, plant appearance and production of secondary metabolites for plant protection. But the DNA absorption spectrum of UV radiation of 220-330 nm coincides with an action spectrum of such phenomena as the inactivation of cell cycle, induction of chromosomal aberrations and genes mutations. This indicates that DNA is the main cell compound absorbing UV_B radiation. Damage to DNA and RNA made by UV_B involves dimers creation by the connections between cyclobutane and pyrimidine or pirymidionine with pyrimidine. The cracks of cross-linking may happen between DNA strands or the insertion of base pairs [7, 23, 49, 50]. The shift of the active response of plants to UV toward longer wave frequency suggests the participation of proteins in the absorption of UV_B. Protein damage is due to strong absorption of UV_B by several amino acids, which residues are damaged or modified. In effect the inactivation of protein molecules, including enzymes occurs [51].

UV_B radiation causes the formation of ROS in the presence of O_2 in leaves. Oxidative damage occurs in protein molecules and lipids with double bonds. The result of lipid peroxidation is the destruction of cell membranes structure and function. The organic radicals and singlet oxygen are responsible for further cell damage. The cell membranes

destruction results in abnormal transport across membranes, the lack of selective membrane permeability and K^+ efflux from cells [50]. UV_B has an important negative effect on plant pigments. The assimilation of pigments is impaired. Chlorophyll, especially chlorophyll b, is destructed to greater extent than carotenoids. Chlorophyll content severely decreases (10 to 70%). Quinones also undergo degradation [45, 48]. The growth regulators are molecules susceptible to UV_B radiation too. The photolytic degradation of indoleacetic acid (IAA) was found in seedlings of sunflower. ABA was inactivated by photolysis in result of strong UV_B absorption. UV_B has impact on ET and gibberellins concentration [7]. Photosynthetic apparatus is particularly susceptible to UV_B damage. Limitation of photosynthesis rate comes not only from the decomposition of photosynthetic pigments, lipid peroxidation and changes in the lipid composition of membranes, resulting in the destabilization of the structure of chloroplasts. UV_B inactivates important enzymes such as RuBisCO, ATP synthase, or de-epoxydasis violaxanthin, the complex of water oxidation and the Calvin cycle enzymes. It also damages the protein subunits of PSI and PSII photosystem (proteins D1 and D2) and quinone electron transporters. Exposure of plants to UV_B reduces significantly stomatal density and degree of stomatal opening. These changes lead to a reduction in dry matter production of plants. The growing loss of leaf photosynthetic productivity is associated with the reduction of leaf area, more than with the reduction of the photosynthesis intensity, even though one of the earliest effects of UV_B radiation is to reduce the amount of mRNA transcripts encoding photosystems proteins and other chloroplast proteins [47, 52-54].

Changes in the morphology and anatomy of leaves in response to UV_B include both a reduction in leaf area and leaf thickness change. Photomorphogenetic effect of UV_B on susceptible plants results in reduced plant height and flowering disturbance [55, 56]. Low intensity UV_B stimulates the specific signaling path - the expression of genes associated with hypocotyl elongation of etiolated seedlings, the other photomorphogenetic reaction run by UV_B [50].

As a result of excess UV_B plants reduce biomass production (in susceptible plants from 10 to 30%) [48, 57]. In about 300 species tested, about 2/3 demonstrated sensitivity to elevated levels of UV_B [58]. One of the changes reported for different plant species is the reduction of the viability of pollen under UV_B radiation [50].

It is worth noting that UV_B also affects plant growth in natural ecosystems indirectly, by changing their competitive abilities and growth conditions. The direction and magnitude of change in productivity are difficult to determine in ecosystems [47]. It depends on the stress factors related to the effect of "greenhouse", accompanying UV_B excess, such as shortage of water and minerals and the increase of CO_2 concentration [59].

It should be emphasized that the effect of UV_B on plants is dependent on the intensity of the radiation. The level of radiation that causes oxidative stress, cellular damage and disruption of photosynthesis is an order of magnitude higher than the intensity of UV_B inducing the signal to the nucleus and other organelles and causing changes in the expression of genes that stimulate defense or photomorphogenetic reactions [50]. At high doses of UV_B the signaling pathways typical for response to stress are activated, similar to those initiated by the attack of pathogens, and including second messengers such as Ca^{2+} signals, kinase, NO and ROS. UV_B induces therefore at least two different signaling pathways [53].

The protection and defense of plants against excess UV_B

The balance of the damage caused by UV_B and the defense and the repair process in crop plants has a high degree of variability between species and varieties, which allows plants adaptation to elevated levels of UV_B.

Structural protection against UV_B includes a presence of hairs on the epidermis, more numerous on upper leaf side. Epidermal cells contain ingredients which absorb UV_B. Increased thickness of the leaf wax layer increases the reflection of radiation by leaf. Reduction in leaf area under the influence of UV_B can be regarded as an adaptation to reduce the absorption of destructive radiation [60]. Epidermal thickening is of similar importance, twisting of leaves and changing composition of the layer of wax on the cuticle of leaves as well. Elongation of cells of palisade parenchyma layer and increased leaf thickness may increase the dispersion of UV_B radiation before it reaches the spongy mesophyll - the main site of photosynthesis in the leaf [7].

Compounds selectively absorbing radiation of UV_B, which content increases from 10 to 300% under high irradiation of this range, are responsible for biochemical protection against excessive UV_B [48]. Phenolic compounds soluble in water, such as flavonoids, mainly anthocyanins, are anti - UV protective filter. They accumulate mainly in the upper leaf epidermis and stop the majority of the UV_B, proportionally to their concentration and to thickness of the leaf. In dicotyledonous plants the protective pigments are present in the hairs and epidermal cells, and in monocot also in mesophyll as glycosides accumulated in the vacuole. Due to the presence of phenols epiderma absorbs most of UV_B radiation and the mesophyll of needles reaches 0% of UV_B irradiation, in grasses and leaves of dicotyledonous trees 3-12% only and herbaceous dicots 18-41% of UV_B radiation incident on the leaf.

The synthesis of flavonoid pigments is the primary plant immune response, resulting from the stimulation of gene expression. The major enzymes of flavonoid synthesis pathway (PAL-L-phenylalanine ammonia-lyase and CHS-synthase chalcones) are induced by UV_B [7, 53, 60]. Flavonoids and other phenols are an important protection against both UV_B radiation and against oxidative stress generated by UV_B [61]. UV_B induces a powerful antioxidant defense system, consisting of low molecular weight compounds and enzymes [47].

In response to direct DNA damage an important defense system against UV_B made of different repair mechanisms of nuclear DNA is activated. A constitutive or UV_B induced repair system is created mainly by photo-lyases or endonucleases [62, 50]. Enzymatic repair processes include also repairing of damage to photosynthetic apparatus - D1 and D2 proteins and chloroplast DNA. Damage to the photosynthetic apparatus by excess UV_B is possibly prevented by zeaxanthin cycle. It gives back the environment the excess of excitation energy of assimilation pigments in the form of heat dissipation from leaves [47].

Summary and conclusions

Tropospheric O_3, in concentrations classified as atmospheric pollution, has a direct negative impact on plants. It can therefore be specified as "bad" ozone.

Similarly, the stress of excess UV_B radiation, which is an indirect result of phenomenon of increased O_3 degradation in the stratosphere, has a negative impact on plants.

Stratospheric O_3, protecting against photo-destructive UV_B, can be defined as "good" ozone.

There is a similarity of the effects induced in plants by O_3 and by UV_B. Both factors generate ROS formation and oxidative stress. O_3 and UV_B activates signaling pathways, typical of the response to the attack of pathogenes. O_3 and UV_B penetration into leaves results in reduced stomatal conductance and reduced intensity of photosynthesis. Under the influence of O_3 and UV_B the WUE (*water use efficiency*) indicator is reduced. In result the reduction of the stomatal CO_2 uptake for photosynthesis against water loss from leaves in the form of water vapor in the process of transpiration is observed. Each of the factors, acting alone, leads to a reduction of leaf area, and increased thickness of leaves, thus reducing the sensitivity to drought stress. At the same time, however, an excess of O_3 and excess UV_B, causing tissue damage and destruction of vital processes, results in loss of plant biomass in a specific way.

Tropospheric O_3 causes damage associated mainly with primary and secondary oxidative stress. UV_B causes mainly genetic mutations and photomorphognetic changes in plants. The effect of UV_B seems to be more widespread as its harmful effects were found in many plant species. Harmful effects of O_3 are observed more locally, mainly in sensitive species. The specificity of action of both factors can also be seen in the way they affect the flowering and ripening of crops. O_3 significantly reduces reproductive processes and impairs the maturation of crops. UV_B can stimulate or inhibit flowering and does not affect the ripening process [63]. Fundamentals of varietal and species differences in susceptibility of plants to both stress factors, especially working simultaneously, are not yet understood. Significant genetic variations in the sensitivity of species to O_3 contamination have however been noted. The existence of genotypes with high tolerance to O_3 provides a basis for breeding O_3 resistant forms to O_3 stress [41]. In *Arabidopsis* mutants with increased nuclear DNA content a new mechanism for high UV_B tolerance was found. Tetraploid forms have therefore a much higher tolerance to UV_B than diploid and it can be used for breeding forms with increased tolerance to UV_B in other plant species [64].

High UV_B tolerance in the mutant cells of *Bupleurum scorzonerifolium* correlates with a high content of polysaccharides as antioxidants, as stated by recent reports [65].

Significant risk of crop yield loss and diminished growth of forest trees, caused by an excess of O_3 and UV_B, can be reduced by breeding processes leading to increase in plant stress tolerance. The processes of adaptation and acclimatization to both factors represent the chance of survival for plants in natural ecosystems. The next steps to the reduction of plants injury by O_3 and UV_B is the inhibition of pollutants emissions, which cause changes in the natural concentration of O_3 in the atmosphere.

A full understanding of the impact of excess O_3 and UV_B on plants requires an examination of the simultaneous action of both factors, especially due to the oxidative stress induced by them. The effect of hormesis, which is mentioned in the literature for both factors, needs to be deeply analyzed. Plant response to both stressors may be further modified by accompanying unfavorable environmental conditions such as nitrogen deposition and drought. This issue is the proposal for further research, from molecular level to the level of ecosystems. The problem of O_3 and UV_B excess is also important in the context of long-term elevation of CO_2 concentration and the predicted global climate change scenario.

References

[1] Sivasakthivel T, Siva Kumar Reddy KK. Ozone layer depletion and its effects: a review. Int J Environ Sci Dev. 2011;2(1):30-37.

[2] Rowland FS. Stratospheric ozone depletion. Phil Trans R Soc B. 2006;361(1469):769-790. DOI: 10.1098/rstb.2005.1783.

[3] Andrews JE, Brimblecombe P, Jickells TD, Liss PS. Wprowadzenie do chemii środowiska. Warszawa; Wyd Nauk-Tech. 1999.

[4] Makles Z, Galwas-Zakrzewska M. Ozon - bezpieczeństwo ludzi i środowiska. Bezpieczeństwo Pracy. 2004;6:25-28.

[5] Mackenzie A, Ball AS, Virdee SR. Instant Notes in Ecology. BIOS Scientific Publishers Ltd.1998:291-294.

[6] Robinson SA, Wilson SR. Environmental effects of ozone depletion and its interactions with climate change: 2010 Assessment Executive Summary. Photochem Photobiol Sci. 2011;10:178-181. DOI: 10.1039/C0PP90043E.

[7] Hollòsy F. Effects of ultraviolet radiation on plant cells. Micron. 2002;33:179-197. DOI: 10.1016/S0968-4328(01)00011-7.

[8] Reddy KR, Kakani VG, Zhao D, et al. Cotton responses to ultraviolet-B radiation: experimentation and algorithm development. Agr Forest Meteorol. 2003;120:249-265. DOI: 10.1016/j.agrformet.2003.08.029.

[9] Tong NYO, Leung DYC, Liu CH. A review on ozone evolution and its relationship with boundary layer characteristics in urban environments. Water Air Soil Pollut. 2011;214:13-36. DOI: 10.1007/s11270-010-0438-5.

[10] Cristofanelli P, Bonasoni P, Collins W, et al. Stratosphere-to-troposphere transport: A model and method evaluation. J Geophys Res. 2003;108(12):8525-8548. DOI:10.1029/2002JD002600.

[11] Black VJ, Black CR, Roberts JA, Steward CA. Impact of ozone on the reproductive development of plants. New Phytol. 2000;147:421-447. DOI: 10.1046/j.1469-8137.2000.00721.x.

[12] Murkowski A. Oddziaływanie czynników stresowych na luminiscencję chlorofilu w aparacie fotosyntetycznym roślin uprawnych - monografia. Lublin: Acta Agrophysica (PAN); 2002.

[13] Schmieman EC, Van Ierland EC, Hordijk L. Dynamic efficiency with multipollutants and multi-targets: The case of acidification and tropospheric ozone formation in Europe. Environ Resour Econ. 2002;23:133-148. DOI: 10.1023/A:1021234423189.

[14] Ashmore MR, Büker P, Emberson LD, et al. Modelling stomatal ozone flux and deposition to grassland communities across Europe. Environ Pollut. 2007;146:659-670. DOI: 10.1016/j.envpol.2006.06.021.

[15] Mauzerall DL, Wang X. Protecting agricultural crops from the effects of tropospheric ozone exposure: reconciling Science and Standard Setting in the United States, Europe, and Asia. Annu Rev Energ Env. 2001;26:237-68. DOI: 10.1146/annurev.energy.26.1.237.

[16] Ashmore MR. Assessing the future global impacts of ozone on vegetation. Plant Cell Environ. 2005;28:949-964. DOI: 10.1111/j.1365-3040.2005.01341.x.

[17] Ludwikow A, Sadowski J. Gene networks in plant ozone stress response and tolerance. J Integr Plant Biol. 2008;50(10):1256-1267. DOI: 10.1111/j.1744-7909.2008.00738.x.

[18] Tamaoki M. The role of phytohormone signaling in ozone-induced cell death in plants. Plant Signal Behav. 2008;3(3):166-174. DOI: 10.4161/psb.3.3.5538.

[19] Fares S, Goldstein A, Loreto F. Determinants of ozone fluxes and metrics for ozone risk assessment in plants. J Exp Bot. 2010;61(3):629-633. DOI: 10.1093/jxb/erp336.

[20] Kerstiens G, Lendzian KJ. Interactions between ozone and plant cuticules.1. Ozone deposition and permeability. New Phytol. 1989;112:13-19. DOI: 10.1111/j.1469-8137.1989.tb00303.x.

[21] Kivimäenpää M, Selledén G, Sutinen S. Ozone-induced changes in the chloroplast structure of conifer needles, and their use in ozone diagnostics. Environ Pollut. 2005;137:466-475. DOI: 10.1016/j.envpol.2005.01.033.

[22] Paoletti E, Grulke NE. Does living in elevated CO_2 ameliorate tree response to ozone? A review on stomatal responses. Environ Pollut. 2005;137:483-493. DOI:10.1016/j.envpol.2005.01.035.

[23] Chen Z, Gallie DR. Increasing tolerance to ozone by elevating foliar ascorbic acid confers greater protection against ozone than increasing avoidance. Plant Physiol. 2005;138:1673-1689. DOI: 0.1104/pp.105.062000.

[24] Thirupathi K, Jun-Cheol M, Changsoo K, et al. Reactive oxygen species in plants: their generation, signal transduction, and scavenging mechanisms. Aust J Crop Sci. 2011;5:709-725.

[25] Roshchina VV, Roshchina VD. Ozone and Plant Cell. Kluwer Academic Publishers, United States; 2003.

[26] Percy KE, Ferretti M. Air pollution and forest health: toward new monitoring concepts. Environ Pollut. 2004;130:113-126. DOI: 10.1016/j.envpol.2003.10.034.
[27] Fiscus EL, Booker FL, Burkey KO. Crop responses to ozone: uptake, modes of action, carbon assimilation and partitioning. Plant Cell Environ. 2005;28:997-1011. DOI:10.1111/j.1365-3040.2005.01349.x.
[28] Buchanan B, Gruissem W, Jones R. Biochemistry & Molecular Biology of Plants. ASPP. 2000:1158.
[29] Beauchamp J, Wisthaler A, Hansel A, et al. Ozone induced emissions of biogenic VOC from tobacco: relationships between ozone uptake and emission of LOX products. Plant Cell Environ. 2005;28(10):1334-1343. DOI:10.1111/j.1365-3040.2005.01383.x.
[30] Augustaitis A, Bytnerowicz A. Contribution of ambient ozone to Scots pine defoliation and reduced growth in the Central European forests: A Lithuanian case study. Environ Pollut. 2008;155:436-445. DOI: 10.1016/j.envpol.2008.01.042.
[31] Hayes F, Jones MLM, Mills G, Ashmore M. Meta-analysis of the relative sensitivity of semi-natural vegetation species to ozone. Environ Pollut. 2007;146:754-762. DOI:10.1016/j.envpol.2006.06.011.
[32] Fuhrer J, Booker F. Ecological issues related to ozone: agricultural issues. Environ Int. 2003;29(2-3):141-154. DOI: 10.1016/S0160-4120(02)00157-5.
[33] Hoshika Y, Omasa K, Paoletti E. Both ozone exposure and soil water stress are able to induce stomatal sluggishness. Environ Exp Bot. 2011 (in press). DOI: 10.1016/j.envexpbot.2011.12.004.
[34] Ainsworth EA, Yendrek CR, Sitch S, et al. The effects of tropospheric ozone on net primary productivity and implications for climate change. Ann Rev Plant Biol. 2012;63:637-661. DOI: 10.1146/annurev-arplant-042110-103829.
[35] Leisner CP, Ainsworth EA. Quantifying the effects of ozone on plant reproductive growth and development. Global Change Biol. 2012;18(2):606-616. DOI: 10.1111/j.1365-2486.2011.02535.x.
[36] Matoušková L, Novotný R, Hůnová I, Buriánek V. Visible foliar injury as a tool for the assessment of surface ozone impact on native vegetation: a case study from the Jizerské hory Mts. J Forest Sci. 2010;56(4):177-182.
[37] Bussotti F, Agati G, Desotgiu R, et al. Ozone foliar symptoms in woody plant species assessed with ultrastructural and fluorescence analysis. New Phytol. 2005;166(3):941-955. DOI: 10.1111/j.1469-8137.2005.01385.x.
[38] Gravano E, Bussotti F, Strasser RJ, et al. Ozone symptoms in leaves of woody plants in open-top chambers: ultrastructural and physiological characteristics. Physiol Plant. 2004;121: 620-633. DOI: 10.1111/j.1399-3054.2004.00363.x.
[39] Klumpp A, Ansel W, Klumpp G, et al. Ozone pollution and ozone biomonitoring in European cities Part II. Ozone-induced plant injury and its relationship with descriptors of ozone pollution. Atmos Environ. 2006;40(38):7437-7448. DOI: 10.1016/j.atmosenv.2006.07.001.
[40] Griffiths H. Effects of air pollution on agricultural crops. Factsheet OMAFRA 2003; 85-002. http://www.omafra.gov.on.ca/english/contact.html.
[41] Comis D. Breeding plants for a high-ozone world. Agric Res Service, USDA News and Events 2011:14-16. http://www.ars.usda.gov/is/AR/2011/jul11/plants0711.htm.
[42] Noormets A, Krishna G, Podila Z, Karnosky D. Rapid response of antioxidant enzymes to O_3 induced oxidative stress in Populus tremuloides clones varying in O_3 tolerance. Forest Genet. 2000;7(4):335-338.
[43] Olbrich M, Gerstner E, Welzl G, Winkler JB, Ernst D. Transcript responses in leaves of ozone-treated beech saplings seasons at an outdoor free air model fumigation site over two growing seasons. Plant Soil. 2009;323:61-74. DOI:10.1007/s11104-009-0129-4.
[44] Bhattacharjee S. Reactive oxygen species and oxidative burst: Roles in stress, senescence and signal transduction in plants. Curr Sci. 2005;89(7):1113-1121.
[45] Ulm R, Nagy F. Signalling and gene regulation in response to ultraviolet light. Curr Opin Plant Biol. 2005;8:477-482. DOI:10.1016/j.pbi.2005.07.004.
[46] Rizzini L, Favory JJ, Cloix C, et al. Perception of UV-B by the Arabidopsis UVR8. Protein Sci. 2011;332(6025):103-106. DOI:10.1126/science.1200660.
[47] Mpoloka SM. Effects of prolonged UV-B exposure in plants. Afr J Biotechnol. 2008;7(25):4874-4883.
[48] Kakani VG, Reddy KR, Zhao D, Sailaja K. Field crop responses to ultraviolet-B radiation: a review. Agr Forest Meteorol. 2003;120:191-218. DOI:10.1016/j.agrformet.2003.08.015.
[49] Ishibashi T, Kimura S, Furukawa T, Sakaguchi K. DNA repair mechanisms in UV-B tolerant plants - review. Japan Agric Res Quar. 2006;40(2):107-113.
[50] Frohnmeyer H, Staiger D. Ultraviolet-B radiation-mediated responses in plants. Balancing Damage and Protection. Plant Physiol. 2003;133:1420-1428.

[51] Rastogi RP, Richa, Kumar A, Tyagi MB, Sinha PR. Molecular mechanisms of ultraviolet radiation-induced DNA damage and repair- review article. J Nucleic Acids 2010: Article ID 592980, 32 pages. DOI:10.4061/2010/592980.
[52] Ghetti F, Checcucci G, Bornman JF, Environmental UV Radiation: Impact on Ecosystems and Human Health and Predictive Models: Proceedings of the NATO Advanced Study Institute on Environmental UV Radiation: Impact on Ecosystems and Human Health and Predictive Models, Pisa, June 2001, Italy: Springer; 2005.
[53] Brosche M, Strid Å. Molecular events following perception of ultraviolet-B radiation by plants. Physiol Plant. 2003;117:1-10. DOI:10.1034/j.1399-3054.2003.1170101.x.
[54] Hideg É, Rosenqvist E, Váradi G, Bornman J, Vincze É. A comparison of UV-B induced stress responses in three barley cultivars. Funct Plant Biol. 2006;33(1):77-90. DOI:10.1071/FP05085.
[55] Kakanii VG,. Reddy KR, Zhao D, Mohamed AR. Effects of Ultraviolet-B Radiation on Cotton (*Gossypium hirsutum* L.) Morphology and Anatomy. Ann Bot. 2003;91(7):817-826. DOI: 10.1093/aob/mcg086.
[56] Zuk-Golaszewska K, Upadhyaya MK, Golaszewski J. The effect of UV-B radiation on plant growth and development. Plant Soil Environ. 2003;49(3):135-140.
[57] Minorsky PV. The hot and the classic. Plant Physiol. 2004;134:16-17. DOI:10.1104/pp.900100.
[58] Zheng Y, Shimizu H, Barnes JD. Limitations to CO_2 assimilation in ozone-exposed leaves of Plantago major. New Phytol. 2002;155:67-78. DOI: 10.1046/j.1469-8137.2002.00446.x.
[59] Zepp RG, Erickson III DJ, Paul ND, Sulzberger B. Effects of solar UV radiation and climate change on biogeochemical cycling: interactions and feedbacks. Photochem Photobiol Sci. 2011;10:261-279. DOI: 10.1039/C0PP90037K.
[60] Allen DJ, Nogués S, Baker NR. Ozone depletion and increased UV-B radiation: is there a real threat to photosynthesis? J Exp Bot. 1998;49(328):1775-1788. DOI: 10.1093/jxb/49.328.1775.
[61] Aphalo PJ. Do current levels of UV-B radiation affect vegetation? The importance of longterm experiments. New Phytol. 2003;160:273-280. DOI: 10.1046/j.1469-8137.2003.00905.x.
[62] Sinha RP, Häder DP. UV-induced DNA damage and repair: a review. Photochem Photobiol Sci. 2002;1:225-236. DOI: 10.1039/b201230h.
[63] Krupa S, Jäger HJ. Adverse effects of elevated levels of ultraviolet (UV)-B radiation and ozone (O3) on crop growth and productivity. In: Global climate change and agricultural production. Direct and indirect effects of changing hydrological, pedological and plant physiological processes. Rome, Italy: FAO; 1996.
[64] Hase Y, Trung KH, Matsunga T, Tanaka A. A mutation in the uvi4 gene promotes progression of endo-reduplication and confers increased tolerance towards ultraviolet B light. Plant J. 2006;46(2):317-326. DOI: 10.1111/j.1365-313X.2006.02696.x.
[65] Li Y, Fan J, Ma H, Shen F, Zhang G, Wang J, et al. Elevated level of polysaccharides in a high level UV-B tolerant cell line of Bupleurum scorzonerifolium Willd. African J Biotechnol. 2011;10(29):5578-5586.

„DOBRY" I „ZŁY" OZON - OCENA NA PODSTAWIE REAKCJI ROŚLIN NA OZON

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Abstrakt: Ozon O_3 jest naturalnym, ale również sztucznym składnikiem chemicznym atmosfery ziemskiej. Jest absorbentem promieniowania ultrafioletowego i podczerwonego oraz ma silne właściwości utleniające. W stratosferze warstwa ozonowa chroni powierzchnię planety przed niebezpiecznym promieniowaniem UV, pośredni wpływ O_3 stratosferycznego na rośliny jest więc pozytywny i O_3 stratosferyczny można nazwać „dobrym" ozonem. Zubożenie warstwy ozonowej wynikające z zanieczyszczenia atmosfery, a opisywane jako „dziura" ozonowa, jest przyczyną zwiększonego promieniowanie UV_B na poziomie powierzchni Ziemi. Genetyczne, cytologiczne, fizjologiczne i morfologiczne reakcje roślin na długotrwałe działanie nadmiaru UV_B są dwojakie: niszczą rośliny, a jednocześnie rośliny chronią siebie i naprawiają swoje uszkodzenia ozonowe. Ozon w troposferze pochodzi ze źródeł naturalnych, a także jest wtórnym zanieczyszczeniem, które powstało w reakcjach fotochemicznych, co prowadzi do przypadków „smogu" i „plam" ozonowych. Jako silny utleniacz O_3 jest bezpośrednio toksyczny dla roślin. O_3 może powodować specyficzne ozonowe uszkodzenia roślin i zostać uznany za „zły" ozon. Ozon jest także sklasyfikowany jako jeden z gazów „cieplarnianych", biorąc udział w globalnym ociepleniu. Trudno jest jednak ocenić wpływ O_3 jako jednego z gazów „cieplarnianych" na rośliny.

Łączny wpływ zmian O_3 w stratosferze i troposferze na rośliny można ocenić jako utratę plonów i zmniejszenie produktywności naturalnych ekosystemów.

Słowa kluczowe: ozon stratosferyczny i troposferyczny, „dziura" ozonowa, nadmiar promieniowania UV, efekt „cieplarniany", gaz „cieplarniany", nadmiar ozonu, „smog" fotochemiczny, „plamy" ozonowe