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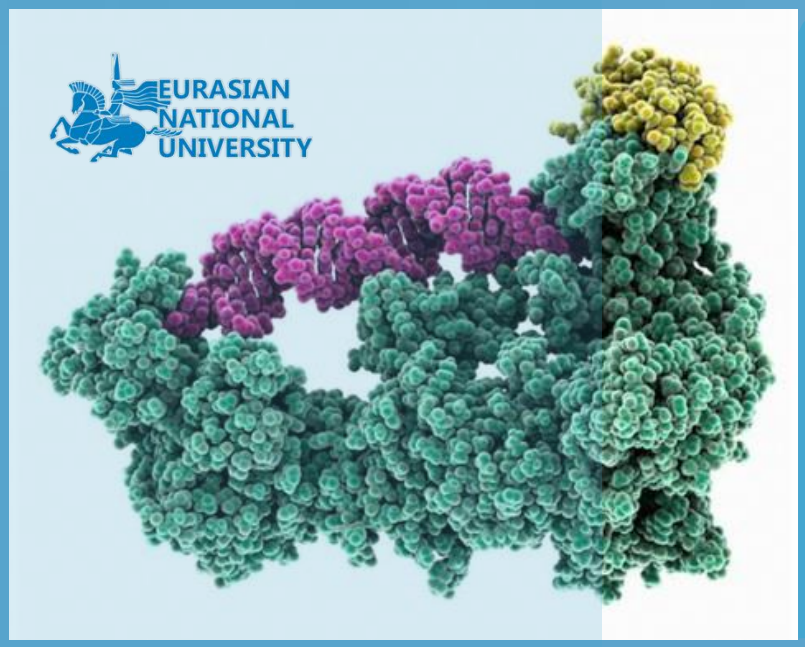

Л.Н. ГУМИЛЕВ АТЫНДАҒЫ
ЕУРАЗИЯ ҰЛТТЫҚ УНИВЕРСИТЕТІ

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"ОМАРОВ ОҚУЛАРЫ: XXI ҒАСЫРДЫҢ БИОЛОГИЯ ЖӘНЕ БИОТЕХНОЛОГИЯСЫ" АТТЫ ХАЛЫҚАРАЛЫҚ ҒЫЛЫМИ ФОРУМНЫҢ БАЯНДАМАЛАР ЖИНАҒЫ

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Жинақ «Омаров оқулары: XXI ғасыр биология және биотехнологиясы» атты халықаралық ғылыми форумына қатысушылардың баяндамаларымен құрастырылған. Бұл басылымда биология, биотехнология, молекулалық биология және генетиканың маңызды мәселелері қарастырылған. Жинақ ғылыми қызметкерлерге, PhD докторанттарға, магистранттарға, сәйкес мамандықтағы студенттерге арналған.

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"ҚАЗАҚСТАН РЕСПУБЛИКАСЫНЫҢ
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Бұл мәдениет біздің елімізде өсіріледі, өйткені ол құрғақ климатты жақсы көреді, негізінен далалық аймақтарда. Бірдей зығырдан немесе талшықтан айырмашылығы, майлы дақылдардың сорты термофильді. Сондықтан мұндай жерлерде сіз осы дақылдың ең көп өнімін ала аласыз. Көбінесе майлы зығыр алқаптары орманды дала аймағының оңтүстігінде егіледі. Бұл мәдениеттің ерекшелігі, ең алдымен, бастапқы кезеңде өте баяу дамиды. Яғни, отырғызудан кейін бірден көшеттер арамшөптерді бітеп тастауы мүмкін. Майлы зығыр өсіру технологиялары, әрине, басқа нәрселермен қатар, осы ерекшелікті ескере отырып әзірленді.

Зығыр майы тек тамақ дайындауда ғана емес, сонымен қатар халықтық медицинада да қолданылды. Оның артықшылықтары бірегей композицияда. Оның құрамында 60 пайыз полиқанықпаған май қышқылдары бар. Өсімдік майлары арасындағы біріншілік зығыр майына жатады. Артықшылықтары ғылыммен бірнеше рет расталды. Зығыр майының артықшылықтары мен кемшіліктері қандай, оны кім пайдалана алады және кім бас тартуы керек, толығырақ қарастырыңыз.

Біз өз жұмысымызда зығыр майының қасиеттерін, оны алу әдістерін қарастырдық. Сондай-ақ жеке учаскемде зығырдың бірнеше сорттарын өсірдім. Оның өсу ерекшеліктерін, морфологиялық белгілерін зерттедім. Сондай-ақ өсірілген тұқымнан май алынды. Біз тағам дайындауда тұқымдардың біразын қолдандық.

Пайдаланылған әдебиеттер:

1. Никитин, Д.И. Масличные культуры // Д.И. Никитин / ВПК Запорожья, 1996. – 255 с. 101

2. Поляков, А.В. Состав жирных кислот семян льна // А.В. Поляков, О.Ф. Чикризова, Л.В. Никитина и др. / Интродукция нетрадиционных и редких сельскохозяйственных растений: материалы 3 Международной научно – практической конференции (14–19 мая 2000 г.). – Пенза, 2000. – С. 10-11.

3. A dry mechanical method for concentrating the lignan secoisolariciresinol diglucoside in flaxseed / B. Madhusudhan [et al.] // Lebensmittel-Wissenschaft und-Technologie. – 2000. – № 33. – P. 268–275

4. Зубцов, В. А. Льняное семя, его состав и свойства / В. А. Зубцов, Л. Л. Осипова, Т. И. Лебедева // Рос. хим. журнал. – 2002. – Т. 46, № 2. – С. 14–16.

5. Oomah, B. D. Dehulling characteristics of flaxseed / B. D. Oomah, G. Mazza, E. O. Kenashuk // Lebensmittel-Wissenschaft und- Technologie. – 1998. – № 29. – P. 245–250.

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FERROPTOSIS OF PLANTS: REGULATION OF LIPID PEROXIDATION AND REDOX STATUS

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Cells die according to a specific program scenario that can be triggered in response to various internal or external signals in a process known as programmed cell death. This process is also known as "cell death" or "apoptosis".

Programmed cell death is an important mechanism for the body's normal functioning. It can, for example, be used to eliminate damaged, infected, or excess cells. Apoptosis, necrosis, ferroptosis, and other types of programmed cell death exist. They all have distinct characteristics that are determined by the mechanisms that cause cell death.

Ferroptosis is a type of programmed cell death that is caused by oxidative stress and is dependent on the presence of iron in the body. This process is associated with a disruption in iron metabolism, which results in an increase in free radicals and cell membrane damage.

Ferroptosis is important in plants, especially when the accumulation of free iron can cause damage to plant cells and organs. Ferroptosis regulates the iron content of cells, which is important for plant metabolism, including photosynthesis, respiration, and others. As a result, ferroptosis is important in regulating iron levels in plants and protecting their cells from damage under stress conditions.

Apoptosis, or programmed cell death, occurs in response to a specific programming scenario that can be triggered by various internal or external signals. This process is active and controlled by the cell, which allows it to die in order to maintain the organism's overall health and survival. As a result, the cell shrinks and "apoptotic bodies" form, which are quickly removed from the tissue and do not cause inflammation. Accidental cell death, on the other hand, occurs by chance, usually as a result of cell damage. Mechanical damage, radiation, toxins, and other physical and chemical agents can all contribute to this process. This type of cell death is uncontrollable and can result in inflammation and tissue degradation.

Ferroptosis is a type of programmed cell death that is triggered by the presence of iron and is associated with oxidative processes. It is distinguished by lipid peroxidation and a change in plasma membrane permeability. Researchers, including Dixon, coined the term "ferroptosis" in the scientific literature in 2012. In contrast to apoptosis and necrosis, a new pathway of cell death caused by erastin in tumor cells has been described. Erastin-induced cell death results in mitochondrial atrophy and a decrease in mitochondrial crysts, while cells do not form apoptic bodies and the nucleus appears intact [1].

In the presence of oxidative-active iron and a breakdown of the cell's antioxidant system, toxic lipid hydroperoxides accumulate, resulting in ferroptosis. Lipoxygenases and reactive oxygen species can both cause lipid peroxidation. Free metal ions Fe^{2+} and Fe^{3+} continue the chain reaction. The fluidity and permeability of the plasma membrane change as the concentration of lipid hydroperoxides increases and phospholipids containing polyunsaturated fatty acids (PFA-FL) decreases, ultimately leading to cell death [2, 3].

GPX4 is the main regulator of ferroptosis and one of the many members of the GPX family. It does so by inhibiting the formation of lipid peroxides. GSH is converted to oxidized glutathione (GSSG) by GPX4 and the cytotoxicity of lipid peroxides (L-OOH) to the corresponding alcohols is reduced (L-OH). Inhibiting GPX4 activity can result in the accumulation of lipid peroxides, a sign of ferroptosis. Yang and colleagues discovered that cells with low GPX4 expression are more susceptible to ferroptosis, whereas cells with high GPX4 expression inhibit ferroptosis. RSL3, a ferroptosis inducer, directly affects GPX4 and inhibits its activity, lowering cell antioxidant capacity and accumulating ROS, resulting in ferroptosis (Figure 1) [4].

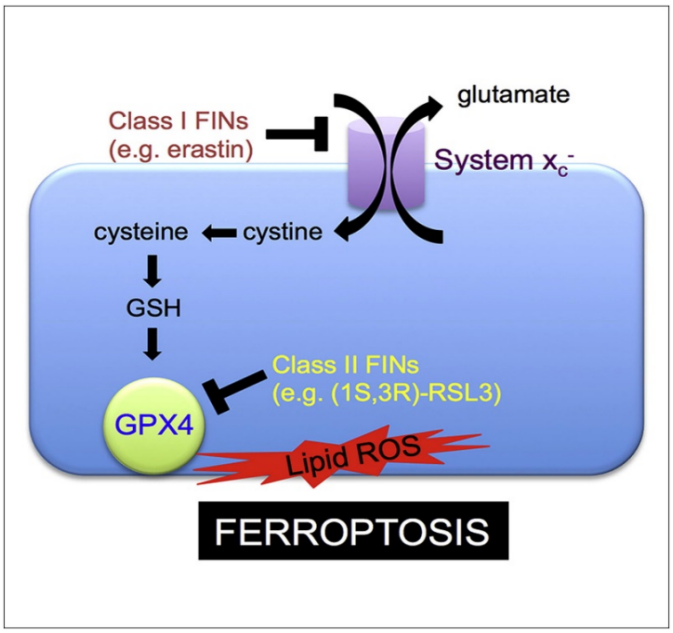

Figure 1 - Glutathione depletion causes inactivation of glutathione peroxidase (GPX) in response to one class of compounds (RSL 3)

Overexpression and knockdown of GPS 4 modulated the lethality of 12 inducers of ferroptosis, but not 11 compounds with other lethal mechanisms. GPX4 converts GSH to oxidized glutathione (GSSG) and reduces the cytotoxicity of lipid peroxides (LOOK) to the corresponding alcohols (L-OH).

Increased activity of lipoxygenases (LOX) on the other hand, causes lipid peroxidation (Figure 2). Lipoxygenases are enzymes that contain iron and catalyze the deoxygenation of lipids. They are thought to contribute to the accumulation of active oxygen, which kills the cell via ferroptosis. Although the precise subcellular location of lipid peroxide is unknown, it is assumed to occur in the plasma membrane and mitochondrial membranes because their morphology is disrupted during ferroptosis [5].

The NADPH oxidase (NOX) oxidation reaction is another source of free radical oxidation. NOX enzymes consume NADPH, producing reactive oxygen species (ROS) during processes related to body protection and signal regulation. However, when NOX activity is disrupted due to severe stress, ROS can accumulate in the cell, contributing to ferroptosis sensitivity [1].

Ferroptosis in plants is caused by heat stress and exhibits some characteristics of mammalian cells, such as ROS accumulation, lipid peroxidation, and glutathione depletion. Morphological changes such as mitochondrial shrinkage, cytoplasm retraction, the appearance of small vacuoles, and the preservation of a normal nucleus are also related to this. It was discovered that the use of canonical inhibitors of ferroptosis, such as ferrostatin-1 (Fer-1, lipophilic antioxidant) and cyclopyroxolamine, can prevent nephroptosis (CPX, intracellular iron chelator) (Figure 3) [6].

The key stage that causes ferroptosis, that is, lipid peroxidation, during which ROS accumulates, is shared by all of the species described thus far. ROS accumulation and subsequent oxidative damage occur due to a mismatch between the amount of free radicals formed and the antioxidant system's ability to neutralize or eliminate harmful effects.

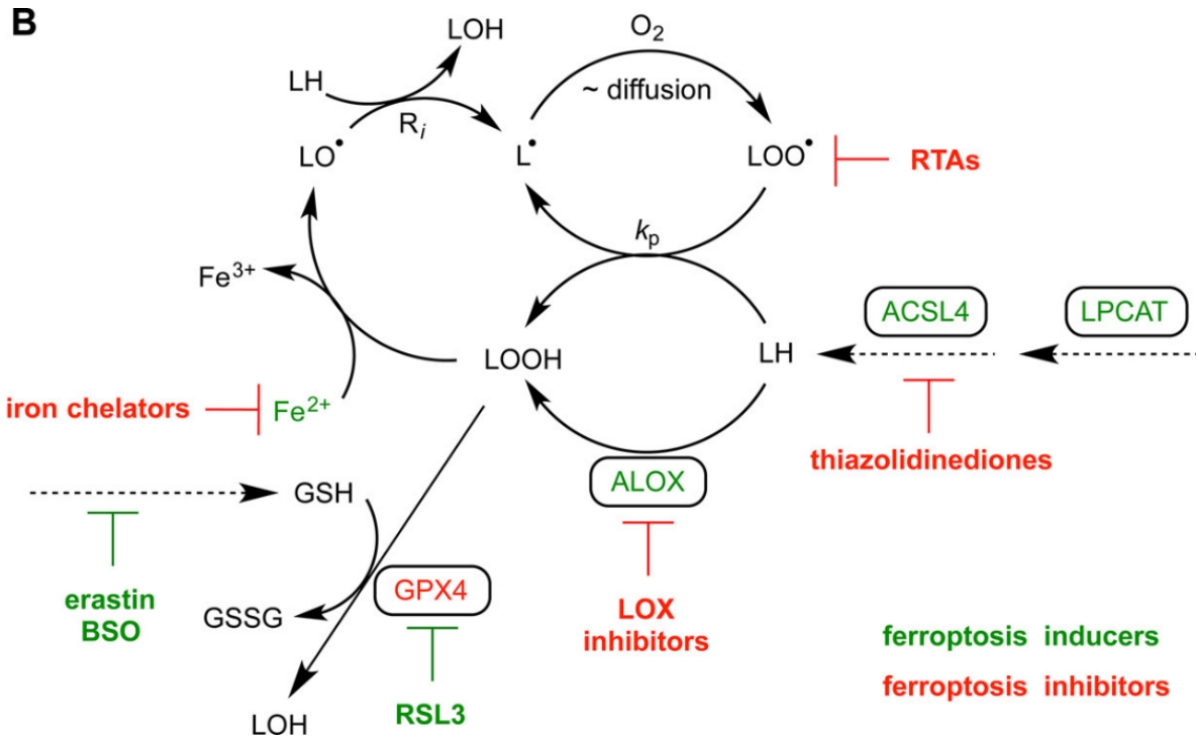

Figure 2 - A scheme demonstrating the interaction between low molecular weight and enzymatic inducers and inhibitors of ferroptotic cell death associated with the accumulation of phospholipid hydroperoxides

IAD is an essential component of life on Earth. Plants, like animals, are anaerobic organisms that require oxygen for mitochondrial respiration. Plant cells, on the other hand, are exposed to much higher oxygen concentrations than animal cells because green tissues continuously produce oxygen through photosynthesis during the day. A large amount of ROS, such as superoxide, hydrogen peroxide, and singlet oxygen, are produced as a result of the integration of the photosynthetic electron transport chain. ROS are extremely reactive and have the ability to modify basic biomolecules such as proteins, lipids, and DNA. Photosynthesis results in an increase in oxidative species, which is accompanied by a variety of mechanisms for controlling redox status and redox regulatory networks that allow plants to sense and respond to changes in redox homeostasis [7, 8].

The antioxidant system controls the buildup of ROS. This system is made up of numerous antioxidants and enzymes, such as ascorbate peroxidase (APX) and glutathione peroxidase (GPX), that regulate the body's lifespan and the specificity of the ROS signaling pathways. In addition to GSH, plants produce other antioxidants such as ascorbate and tocopherols. The balance between the formation and elimination of ROS allows cells to survive and avoids excessive damage.

ROS, on the other hand, can play an important role in plant life by acting as signaling molecules. The transmission of ROS signals can cause plant responses to various stresses, such as closing stomata, modulating root hair growth, and hormone reactions [9]. ROS are produced in a variety of subcellular compartments, including the cell wall, apoplast, chloroplasts, mitochondria, and peroxisomes. Subcellular divisions may differ in their redox state due to differences in antioxidant reserves and ROS formation sources [10]. Because different types of ROS are produced in different subcellular compartments, the outcomes and integration of such signals are highly specific. In these subcellular regions, oxidative-

reduction regulation occurs independently, and ROS outbreaks caused in specific locations will activate only the signaling route available in this space [11].

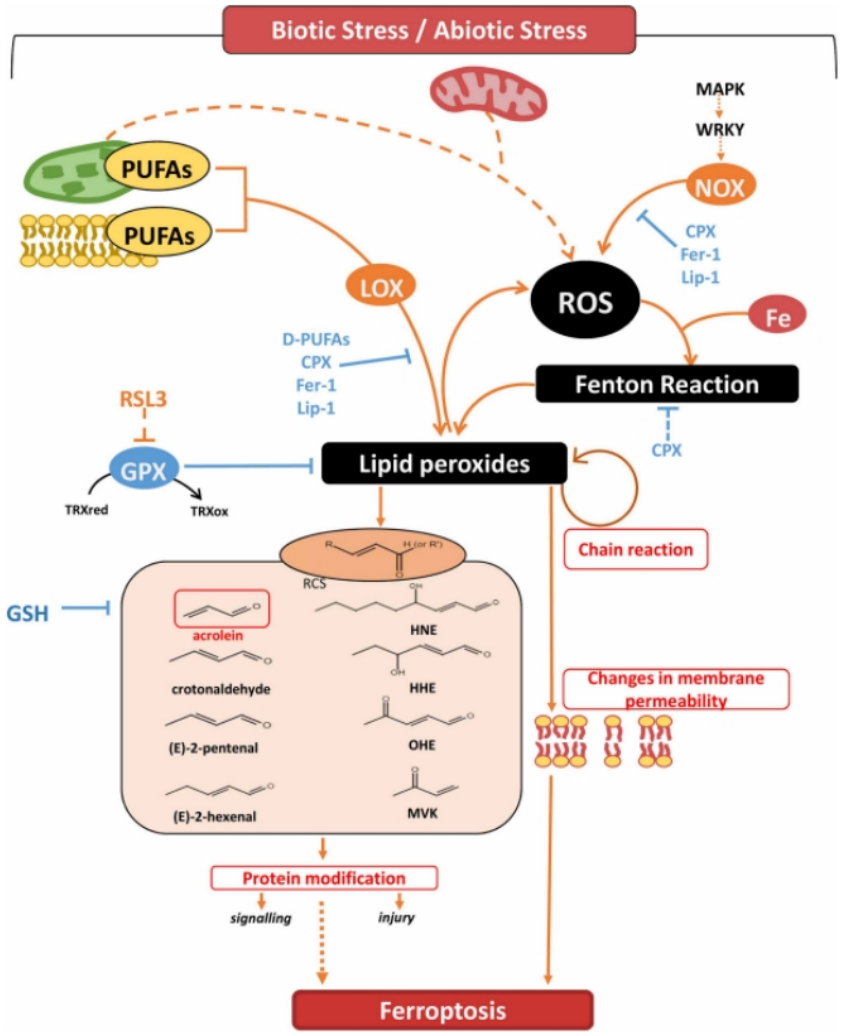

Figure 3 - The main mechanisms of oxidative damage and antioxidant protection

Ferroptosis can be caused by biotic or abiotic stresses. ROS can be produced by three main sources: (1) ROS produced by membrane NOX enzymes (2) ROS produced in mitochondria and (3) Fenton reaction. Peroxidation The oxidation of PUFA lipids (PUFAs) can occur through enzymatic or non-enzymatic processes. Mitogen-activated protein kinases (MAPK) can phosphorylate WRKY transcription factors, which, in turn, induce NOX expression, leading to the accumulation of ROS. GPX detoxifies lipid peroxides with thioredoxin (TRX) as a reducing agent. RSL3 inhibits GPX, leading to the accumulation of lipid peroxides. Lipid ROS can break down into reactive carbonyl species (RCS), such as acrolein, which are associated with cell death. The addition of acrolein causes cell death, which can be prevented by glutathione (GSH). Treatment of CPX, Fer-1, DPI, LIP-1 (liproxtin-1) and D-PUFA inhibits ferroptosis. Pro-ferroptotic pathways are shown in orange, anti-ferroptotic pathways are shown in blue. Dotted lines indicate circumstantial evidence.

Although the formation of ROS and signaling remain unknown, these facts may explain why ROS can regulate various types of RCD [12–15].

Certain types of interactions between a plant and a pathogen during biotic stresses can cause a hypersensitive response (HR), which is a type of regulated cell death that occurs at the pathogen entry site and prevents its spread. According to the available data, ROS formation occurs after NOX activation in HR observed in various plant-pathogen systems, which is a key event in causing cell death [16, 17].

The accumulation of ROS is one of the first biochemical events that occurs after the induction of regulated cell death during ferroptosis in plants. Pretreatment with dipphenyleniodonium (DPI), a NOX inhibitor, can prevent this pathway of cell death caused by biotic or abiotic stresses. When exposed to heat, the level of cytosolic ROS rises immediately after treatment and can be measured for 15 minutes to 3 hours afterwards. DPI pretreatment not only prevents cytosolic ROS accumulation, but also cell death [6].

The majority of ROS is produced during the oxidative phosphorylation process in the electron transport chain on the inner membrane. Incomplete oxygen reduction caused by electron leakage from complexes I and III results in the formation of a superoxide ion (O_2), which quickly converts to hydrogen peroxide (H_2O_2). The accumulation of mitochondrial ROS results in a decrease in transmembrane potential, which is observed in both animal and plant cell death [18, 19]. However, because the mitochondrial superoxide-sensitive fluorescent probe could not detect the role of mitochondria in ROS production, there is still no clear evidence.

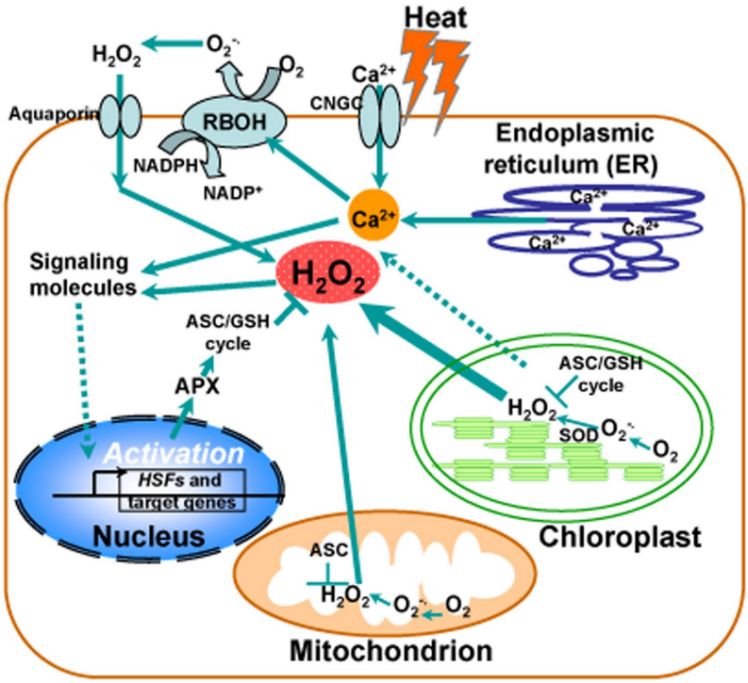

Figure 4 - Calcium and ROS homeostasis in HR

Schematic representation of the main sites of ROS generation and transient increase in calcium content from various intracellular stores and the influx of extracellular calcium into the cell caused by the opening of channels controlled by cyclic nucleotides (CNGC) in the plasma membrane in response to heat stress. Heat stress causes activation of calcium channels in EPR membranes, which leads to the release of calcium into the cytosol. Hydrogen peroxide (H2O2) and Ca2+ serve as secondary messengers involved in the heat-response activation of genes with heat shock elements in their promoters, such as heat shock transcription factors (HSF), heat shock proteins (HSP) and cytosolic ascorbate peroxidase (APX). Under thermal stress, redox enzymes and metabolites, such as superoxide dismutase

(SOD) and the ascorbate-glutathione cycle (ASC-GSH), functioning in different cellular compartments, participate in maintaining ROS homeostasis. NADPH oxidase in the plasma membrane is activated by thermal stress due to an increase in membrane fluidity and/or due to a subsequent increase in cytosolic Ca2+ levels controlled by the Ca2+ permeable channel (CNGC). The influx of Ca2+ activates RBOH, contributing to its phosphorylation, which leads to an increase in ROS.

During heat stress, chloroplasts can also contribute to an increase in ROS. In chloroplasts, antioxidant defense mechanisms are constantly evolving. This ability to provide antioxidant protection is required for heat stress adaptation and the development of thermal tolerance. Furthermore, ROS produced by chloroplasts can act as nucleus signals, activating genes that provide effective adaptation to environmental stresses (Figure 4) [20].

It's also possible that ROS produced in chloroplasts are involved in the oxidative surge that occurs after heat stress in ferroptotic plants. When the aboveground parts of arabidopsis seedlings were exposed to high temperatures, it was discovered that plants exposed to 43°C died faster in light than in darkness, indicating the involvement of active chloroplasts in cell death in leaves [6]. After studying the effect of ferroptosis inhibitors on the pathway of cell death caused by heat stress, it is reasonable to assume that chloroplasts are involved in this process, though the mechanism of this participation has not yet been thoroughly investigated. It has also been proposed that mitochondria and chloroplasts can interact with one another during plant cell death induction [21–24]. Furthermore, plants with a mutation in the MOSAIC DEATH 1 (MOD1) gene, which encodes enoyl-ACP reductase, accumulate ROS and die, which can be prevented by mutations in the mitochondrial complex I. In the absence of mod1, chloroplastic dicarboxylic acid transporter 1 (DiT1) and mitochondrial malate dehydrogenase 1 (mMDH1) can prevent ROS accumulation and the PHC phenotype, indicating a possible interaction of chloroplasts and mitochondria via the malate shuttle during cell death [25].

Non-enzymatic lipid peroxidation can also result in the formation of lipid hydroperoxides. When oxygen radicals react with double bonds in polyunsaturated fatty acids to form a lipid peroxyl radical, this is referred to as non-enzymatic lipid peroxidation. According to the following equation, the Fenton reaction produces oxygen radicals (HO•):

$$Fe^{2+} + H_2O_2 – Fe^{3+} + HO• + HO^-$$

The HO• then reacts with the PUFA, resulting in the formation of a lipid radical. These radical lipids can: I react with other lipids in a chain reaction, (ii) produce ROS, or (iii) interact with molecules like proteins or DNA [26]. Because the Fenton reaction requires the presence of labile iron, iron bound to FeS clusters, heme groups, or attached to iron storage proteins is not directly involved in the process of non-enzymatic formation of lipid ROS [27]. To maintain homeostasis, the level of labile iron must be regulated. Although iron is required for vital metabolic processes, an excess of labile iron can be dangerous because it can participate in redox reactions that produce free radicals [28, 29]. ROS have been proposed as suitable signals for modulating various physiological aspects of plants (including cell death), not only inside the cell, but also between cells, due to their greater stability compared to ROS [30].

Ferroptosis in plants uses the same basic molecular mechanisms as ferroptosis in other systems. It occurs as a result of antioxidant depletion, ROS accumulation, and iron-dependent lipid peroxide. Because many oxidative and antioxidant systems form the process of lipid peroxidation during ferroptosis, the regulation of redox homeostasis is critical in this context. The antioxidant system is activated, which limits oxidative damage, which eventually

exceeds the capabilities of cells entering the ferroptotic pathway. Because light is the determining factor for plant cells undergoing ferroptosis, active chloroplasts play an important role in this process. However, the basis of chloroplasts' contribution remains unknown, as their supposed interaction with mitochondria producing signals against cell death and ROS necessitates further investigation. Furthermore, despite the fact that lipid peroxide is required for plant ferroptosis, the final perpetrators of cell death are still unknown.

References

1. Dixon S.J. et al. Ferroptosis: An Iron-Dependent Form of Nonapoptotic Cell Death // Cell. 2012. Vol. 149, № 5. P. 1060–1072.
2. Kuhn H., Banthiya S., van Leyen K. Mammalian lipoxygenases and their biological relevance // Biochimica et Biophysica Acta (BBA) - Molecular and Cell Biology of Lipids. 2015. Vol. 1851, № 4. P. 308–330.
3. Reis A., Spickett C.M. Chemistry of phospholipid oxidation // Biochimica et Biophysica Acta (BBA) - Biomembranes. 2012. Vol. 1818, № 10. P. 2374–2387.
4. Yang W.S. et al. Regulation of Ferroptotic Cancer Cell Death by GPX4 // Cell. 2014. Vol. 156, № 1. P. 317–331.
5. Shah R., Shchepinov M.S., Pratt D.A. Resolving the Role of Lipoxygenases in the Initiation and Execution of Ferroptosis // ACS Cent. Sci. American Chemical Society, 2018. Vol. 4, № 3. P. 387–396.
6. Distéfano A.M. et al. Heat stress induces ferroptosis-like cell death in plants // Journal of Cell Biology. 2017. Vol. 216, № 2. P. 463–476.
7. Woehle C. et al. Expansion of the redox-sensitive proteome coincides with the plastid endosymbiosis // Nature Plants. 2017. Vol. 3, № 6. P. 17066.
8. Martin W.F., Sies H. Physiological evolution: Genomic redox footprints // Nature Plants. 2017. Vol. 3, № 6. P. 17071.
9. Waszczak C., Carmody M., Kangasjärvi J. Reactive Oxygen Species in Plant Signaling // Annu. Rev. Plant Biol. Annual Reviews, 2018. Vol. 69, № 1. P. 209–236.
10. Foyer C.H., Noctor G. Stress-triggered redox signalling: what's in pROSpect? // Plant, Cell & Environment. John Wiley & Sons, Ltd, 2016. Vol. 39, № 5. P. 951–964.
11. Gadjev I. et al. Transcriptomic Footprints Disclose Specificity of Reactive Oxygen Species Signaling in Arabidopsis // Plant Physiology. 2006. Vol. 141, № 2. P. 436–445.
12. De Pinto M.C., Locato V., De Gara L. Redox regulation in plant programmed cell death // Plant, Cell & Environment. John Wiley & Sons, Ltd, 2012. Vol. 35, № 2. P. 234–244.
13. Petrov V. et al. ROS-mediated abiotic stress-induced programmed cell death in plants // Frontiers in Plant Science. 2015. Vol. 6.
14. Xie D.-L. et al. Functions of Redox Signaling in Pollen Development and Stress Response // Antioxidants. 2022. Vol. 11, № 2.
15. Gechev T.S., Hille J. Hydrogen peroxide as a signal controlling plant programmed cell death // Journal of Cell Biology. 2005. Vol. 168, № 1. P. 17–20.
16. Torres M.A., Dangl J.L., Jones J.D.G. Arabidopsis gp91phox homologues AtrbohD and AtrbohF are required for accumulation of reactive oxygen intermediates in the plant defense response // Proceedings of the National Academy of Sciences. Proceedings of the National Academy of Sciences, 2002. Vol. 99, № 1. P. 517–522.
17. Yun B.-W. et al. S-nitrosylation of NADPH oxidase regulates cell death in plant immunity // Nature. 2011. Vol. 478, № 7368. P. 264–268.
18. Araniti F. et al. Rosmarinic acid induces programmed cell death in Arabidopsis seedlings through reactive oxygen species and mitochondrial dysfunction // PLOS ONE. Public Library of Science, 2018. Vol. 13, № 12. P. e0208802.

19.Yang J. et al. Prevention of Apoptosis by Bcl-2: Release of Cytochrome c from Mitochondria Blocked // Science. American Association for the Advancement of Science, 1997. Vol. 275, № 5303. P. 1129–1132.

20.Sun A.-Z., Guo F.-Q. Chloroplast Retrograde Regulation of Heat Stress Responses in Plants // Frontiers in Plant Science. 2016. Vol. 7.

21.Ambastha V. et al. Salt induced programmed cell death in rice: evidence from chloroplast proteome signature // Functional Plant Biol. 2021. Vol. 48, № 1. P. 8–27.

22.Bruggeman Q. et al. Chloroplast Activity and 3′phosphadenosine 5′phosphate Signaling Regulate Programmed Cell Death in Arabidopsis // Plant Physiology. 2016. Vol. 170, № 3. P. 1745–1756.

23.Kim C. et al. Chloroplasts of Arabidopsis Are the Source and a Primary Target of a Plant-Specific Programmed Cell Death Signaling Pathway // The Plant Cell. 2012. Vol. 24, № 7. P. 3026–3039.

24.Van Aken O., Van Breusegem F. Licensed to Kill: Mitochondria, Chloroplasts, and Cell Death // Trends in Plant Science. Elsevier, 2015. Vol. 20, № 11. P. 754–766.

25.Zhao Y. et al. Malate transported from chloroplast to mitochondrion triggers production of ROS and PCD in Arabidopsis thaliana // Cell Research. 2018. Vol. 28, № 4. P. 448–461.

26.Gaschler M.M., Stockwell B.R. Lipid peroxidation in cell death // Biochemical and Biophysical Research Communications. 2017. Vol. 482, № 3. P. 419–425.

27.Stockwell B.R. et al. Ferroptosis: A Regulated Cell Death Nexus Linking Metabolism, Redox Biology, and Disease // Cell. Elsevier, 2017. Vol. 171, № 2. P. 273–285.

28.Gao M. et al. Ferroptosis is an autophagic cell death process // Cell Research. 2016. Vol. 26, № 9. P. 1021–1032.

29.Hou W. et al. Autophagy promotes ferroptosis by degradation of ferritin // Autophagy. Taylor & Francis, 2016. Vol. 12, № 8. P. 1425–1428.

30.Riegman M. et al. Ferroptosis occurs through an osmotic mechanism and propagates independently of cell rupture // Nature Cell Biology. 2020. Vol. 22, № 9. P. 1042–1048.

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ӨСІМДІКТЕРДІҢ ЖОҒАРЫ ТЕМПЕРАТУРАНЫҢ ӘСЕРІНЕН ФИЗИОЛОГИЯЛЫҚ ӨЗГЕРІСТЕРІ

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Жоғары температура (ЖТ) стрессі бүкіл әлемдегі өсімдіктердің өсуін, метаболизмін және өнімділігін шектейтін негізгі экологиялық стресс болып табылады. Өсімдіктердің өсуі мен дамуы температураға сезімтал көптеген биохимиялық реакцияларды қамтиды [1].Температуралық өзгерістерді қабылдау қабілетіне қарай организмдерді осылай бөлінеді: +15°С-тан төмен температурада өмір сүретін және көбейетін психрофилдер, олардың кейбіреулері -20°С-қа дейінгі температурада метаболикалық белсенділікті сақтайды; +15-тен +40°С-қа дейінгі температурада өмір сүруге ыңғайлы мезофилдер; және өздерін жақсы көрсететін термофилдер +50-ден +60°С-қа дейінгі температурада (орташа термофилдер). Гипертермофилдер (немесе экстремалды термофилдер) термині +80°С-тан жоғары оңтайлы өсу қарқыны бар организмдер үшін қолданылады [2].