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Genetic analyses reveal wildfire particulates as environmental pollutants rather than nutrient sources for corals

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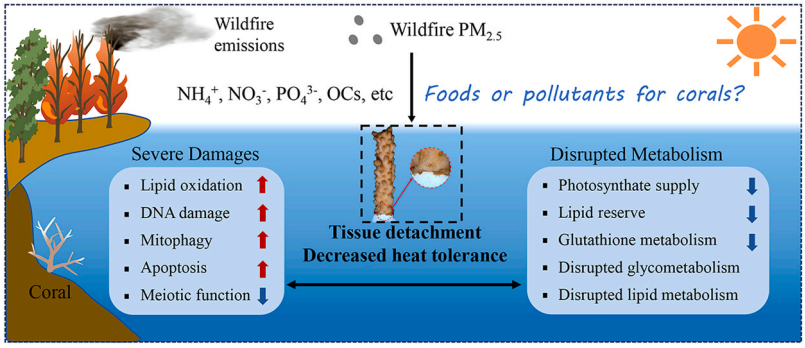
HIGHLIGHTS

- Wildfire $PM_{2.5}$ triggered severe oxidative damage to *Acropora formosa*.
- Coral promoted multicellular development via tyrosine receptors for self-repair.
- The lipid energy supply through glyoxylate cycle decreased with lipid consumption.
- Disrupted metabolism linked to cell adhesion caused coral tissue detachment.
- Coral heat tolerance decreased due to downregulation of HSP70 expression.

GRAPHICAL ABSTRACT



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ABSTRACT

Heterotrophic nutrients are crucial for coral growth and recovery from bleaching events. Although wildfire emissions are a potential source of these nutrients, their impact on corals was minimally investigated. In this microcosm experiment, *Acropora formosa* corals exhibited rapid tissue detachment upon exposure to wildfire fine particulate matter ($PM_{2.5}$). Physiological and genetic analyses revealed mechanisms associated with oxidation-reduction homeostasis and nutrient metabolism. Excessive hydrogen peroxide was generated as corals activated cytochrome P450 enzymes and the respiratory burst in phagocytic cells to metabolize $PM_{2.5}$, leading to oxidative damage, mitochondrial dysfunction, and cell apoptosis due to reduced superoxide dismutase activity and compromised glutathione antioxidant function. Subsequently, corals upregulated the transcriptions of genes which are related to tyrosine receptor proteins to regulate multicellular development for self-repair, increasing energy consumption. However, Symbiodiniaceae upregulated their metabolism and retained photosynthates, reducing nutrient supply to the coral host. Therefore, the host temporarily utilized lipid reserves via the glyoxylate cycle, but excessive consumption disrupted lipid and carbohydrate metabolism, ultimately weakening cell adhesion and causing coral tissue detachment. Additionally, the downregulation of HSP70 expression,

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potentially linked to decreased sacsin and mitochondrial apoptosis, accelerated coral heat bleaching. This study elucidates the mechanisms by which wildfire $PM_{2.5}$ at environmental concentrations poses risks to corals, particularly in a warming climate.

1. Introduction

Global warming has been precipitating a rise in extreme wildfire events, notably in tropical regions [1]. These wildfires are increasingly releasing emissions into the ocean through atmospheric deposition, posing potential effects on coral reefs that are predominantly distributed in tropical waters. While some studies suggest that wildfire emissions enhance marine productivity by promoting phytoplankton blooms [2, 3], these emissions could induce coral asphyxiation and subsequent mortality by triggering phenomena such as red tides [4]. The proximity of wildfires to coral reefs is associated with coral mortality, but the specific reasons remain unclear [5].

A recent isotopic study indicates that low concentrations of wildfire particulates (2 mg/L $PM_{2.5}$) appear to be utilized by *P. lutea* as a source of heterotrophic nutrients, sustaining total biomass, proteins, and lipids for coral growth [6]. This potential nutrient source is likely attributed to the organic carbon (57–69 %) and considerable nutrients, including nitrogen and phosphate, contained within wildfire $PM_{2.5}$ [2,7]. Numerous investigations have implicated nutrient enrichment in modulating the symbiosis between coral hosts and their symbionts (Symbiodiniaceae). In healthy corals, Symbiodiniaceae supply organic carbon (photosynthates) to meet the metabolic carbon demands of the coral host. Moderate nutrient enrichment, rich in nitrogen (N), phosphorus (P), and potassium (K), has been shown to enhance Symbiodiniaceae abundance, photosynthetic efficiency, coral tissue biomass, and total carbon translocation [8]. Nutrient loadings (e.g., ammonium) can mitigate heat stress-induced cell apoptosis and improve the physiological response of corals to temperature elevations [9]. In addition to photoautotrophy, corals also rely on heterotrophic feeding, such as consumption of zooplankton, to satisfy between 5 % and 50 % of their daily metabolic requirements [10], especially for essential nutrients like nitrogen and phosphorus that photoautotrophy does not fully provide [11]. In bleached corals, the loss of symbionts reduces the availability of photosynthates, necessitating an increased intake of heterotrophic nutrients to compensate for the diminished photosynthetic carbon [12]. Diverse heterotrophic feeding strategies, such as *Artemia* nauplii [13] and fine suspended sediment [14], could alleviate nutritional limitations and enhance coral heat tolerance. Furthermore, feeding on *Artemia* nauplii particularly facilitates the restoration of nutritional exchanges between Symbiodiniaceae and coral hosts [15]. Thus, the organic carbon in low-concentration wildfire particulates could possibly serve as a potential supplementary heterotrophic nutrient source.

In contrast to the effects of low-concentration wildfire $PM_{2.5}$, high-concentration wildfire $PM_{2.5}$ (5 mg/L) significantly reduces the autotrophy and heterotrophy of *P. lutea* [6]. These negative impacts may be analogous to the detrimental effects of nutrient enrichment or particles on corals in some cases. As reported in some investigations, nutrient enrichment is not always beneficial for corals; excessive or imbalanced nutrient levels pose a serious threat [16,17]. Nitrate enrichment and phosphate limitation damage photosynthesis and greatly reduce total carbon translocation, thereby compromising coral health. High levels of dissolved inorganic nitrogen and phosphate increase Symbiodiniaceae density, leading to resource competition with the coral host. This competition lowers photosynthetic performance and carbon translocation per algal cell [16] and accelerates coral heat bleaching [17]. External nutrients mitigate nitrogen limitation and increase the production of oxygen free radicals from Symbiodiniaceae photosynthesis, potentially triggering coral cell death pathways such as tumor necrosis factor and apoptosis mediated by caspase and bcl-2-like proteins [18]. Heterotrophic feeding exacerbates coral bleaching under heat stress in conditions of imbalanced nitrogen and phosphorus, despite the generally beneficial effect of heterotrophic feeding under ambient nutrient conditions [17]. Additionally, certain particles, such as carboxylated beads and bacteria, are phagocytosed and degraded as non-self by coral phagocytic cells [19]. The deposition of natural or artificial particles can increase turbidity and sedimentation, reducing the availability of photosynthetically active radiation for coral symbionts [20], which also occurred under high-concentration wildfire particulates exposure [6]. Artificial sediments, like silicon carbide, disrupt cilium assembly and disassembly processes in heat-stressed corals, impacting various biological functions involved in cilia motility, immune responses, and cell adhesion [21]. Microplastic exposure compromises the stress response and immune systems in the coral *Pocillopora damicornis* [22].

Heat stress is acknowledged as a primary stressor causing coral bleaching. As the frequency of massive heat-induced coral bleaching events continues to rise globally, corals with limited heterotrophic capabilities may struggle to sustain survival and recovery [10]. Despite preliminary insights into the effects of wildfire particulates on corals, a comprehensive understanding of the underlying biological mechanisms by which wildfire emissions impact corals and the effects on their heat tolerance remains elusive. To address this issue, we conducted a comprehensive assessment of the influence of wildfire $PM_{2.5}$ on *Acropora formosa* by evaluating coral physiological indices, performing transcriptome analysis, and analyzing gene expression patterns. Furthermore, we preliminarily examined the effects of wildfire $PM_{2.5}$ on coral heat tolerance through both physiological and genetic analyses.

2. Material and methods

2.1. Wildfire $PM_{2.5}$ collection

$PM_{2.5}$ samples were collected using an air sampler (Laoying 2031, Qingdao Laoying Electric Inc., China) equipped with an impactor designed to capture particulates within the 0–5 μm size range. The sampler was positioned within 3 m of the burning site within a combustion simulation system to minimize the impact of other $PM_{2.5}$ sources in the air. Woody branches from subtropical trees (*Litchi chinensis*, *Dimocarpus longan*, and *Santalum album*) were burned to simulate forest fires [6]. The resulting smoke was collected upwind onto quartz filters (GE Whatman, 203 × 254 mm). The weight of wildfire particulates was ascertained by quantifying the differential dry weight of the filters before and after collection. The $PM_{2.5}$ extracts were obtained by sonicating the particulates collected on the filter membrane in 500 mL 0.45 μm-filtered seawater for 40 min. This solution was used to control the $PM_{2.5}$ input in the treatment [6]. The treatment details refer to Supporting Information Text S1.

2.2. Coral collection

The coral samples (*A. formosa*) were collected by divers from Weizhou Island in the Beibu Gulf, China. *A. formosa*, widely distributed across major coral reefs and sensitive to environmental changes, serves as an experimental subject with significant environmental indicative value. One branched coral colony was subdivided into individual twigs (4–6 cm in height) and affixed to square tiles. These samples were then acclimated to laboratory conditions in a 500 L flow-through aquarium for two weeks. Environmental parameters within the aquarium were meticulously controlled as follows: the temperature was maintained at 28 ± 0.3 ℃, salinity at 32 ± 1 PSU, pH levels at 8.04 ± 0.21, and simulated natural light under a 12:12 light-dark cycle.

2.3. Experimental design

2.3.1. Experiment 1: $PM_{2.5}$ exposure to corals

Experiment 1 comprised two groups. The control group (CG-1) received no $PM_{2.5}$ addition, with nine coral samples placed in three 5 L beakers (three corals/beaker). Twenty percent of the seawater (1 L) was replaced daily for water exchange [6,23]. The $PM_{2.5}$ group was exposed to 5 mg/L $PM_{2.5}$, with daily water changes which included the introduction of $PM_{2.5}$ extracts. Other conditions were the same as the CG-1 group, with both groups being maintained at an ambient temperature (28 ℃). The $PM_{2.5}$ input was determined from the concentration of $PM_{2.5}$ aerosols (8357 μg/m^3) near predominantly ponderosa pine forests during extreme fires [7], using the calculation method from Chance et al. [24]. For the reason that the residence times of atmospheric particulates range from approximately one month to five months in seawater [25,26], the accumulation period for deposition was set at 30–150 days. The particulates were assumed to enter a coral reef region at a depth of 5–10 m. The resulting seawater $PM_{2.5}$ concentration was calculated to be 2.17–21.66 mg/L. Calculation details are provided in Supporting Information Text S2.

Three supplemental coral twigs were collected as 0-hour standard samples (0 h SS) prior to the experiment. Based on the coral response observed during the pre-test (Supporting Information Text S3), sampling was conducted at three points: 5 h, 4 d, and 9 d. At each time point, three biological replicates were collected for both the CG-1 and $PM_{2.5}$ groups. Details are provided in Supporting Information Text S4.

2.3.2. Experiment 2: Combined stress of $PM_{2.5}$ exposure and high temperature

The increased sea surface temperature is a primary environmental stressor for reef-building corals. Therefore, 32 ℃ was employed as a simulated temperature to investigate the impact of wildfire $PM_{2.5}$ on coral heat tolerance, which is the thermal bleaching threshold of corals in the mid-northern South China Sea [27] and is a common temperature used in simulation experiments [9]. Experiment 2 was designed with four experimental groups: the control group (CG-2; 28 ℃), the high temperature stress group (HT; 32 ℃), the high temperature and high $PM_{2.5}$ stress group (HTHP; 32 ℃ + 5 mg/L $PM_{2.5}$), and the high temperature and low $PM_{2.5}$ stress group (HTLP; 32 ℃ + 1 mg/L $PM_{2.5}$). The resulting low seawater $PM_{2.5}$ concentration was calculated at 0.62–6.19 mg/L from the mean value of wildfire $PM_{2.5}$ aerosols (2390.76 μg/m^3) recorded in the literature [7], while the high $PM_{2.5}$ input was the same as in Experiment 1. Sampling was conducted at the time points corresponding to tissue damage: specifically, at 9 d for the HTHP group and at 17 d for the HTLP group. At each time point, three biological replicates were collected separately from CG-2 and HT groups for comparison. Further details are provided in Supporting Information Text S4.

2.4. Analyses of seawater nutrients and organic carbon in $PM_{2.5}$

The concentrations of seawater nutrients were determined in accordance with the seawater analysis standard of China (GB 17378.4–2007). Specifically, NH_4^+ concentrations were quantified using the indophenol blue method. The levels of NO_3^- + NO_2^- were assessed using the sulfanilamide/N-(1-naphthyl) ethylenediamine dihydrochloride method. SRP levels were measured using the phosphomolybdenum blue method. All nutrient analyses were conducted using an ultraviolet spectrophotometer (Shimadzu, UV-2700). The nutrient analyses of $PM_{2.5}$ were conducted in 500 mL seawater following the method described in Supporting Information Text S1. The organic carbon content in $PM_{2.5}$ was analyzed using an Elementar Vario EL Cube Elemental Analyzer. Prior to the test, carbonate components in $PM_{2.5}$ samples were removed by treatment with concentrated HCl, followed by drying in a 60 °C oven to eliminate the acid [6].

2.5. Photosynthesis analysis of corals

The pulse-amplitude modulated fluorometer (Monitoring-PAM, WALZ Germany) was utilized to measure the maximum quantum yield (Fv/Fm) daily at 8:30 p.m. (after a 30 min dark adaptation period). The Symbiodiniaceae density and Chlorophyll a (Chl-a) content were determined at the end of the experiment using the method recommended by Tang et al. [22].

2.6. Physiological indices

The coral samples were cut into fragments for assessing physiological indices, including total superoxide dismutase (T-SOD) activity, lipid peroxidation product malondialdehyde (MDA) content, and caspase3 activity. These measurements were conducted using commercial kits (JIANCHENG, A001–1–2, A003–1–2, and G015–1–3). Briefly, slurry samples derived from the fragments were subjected to centrifugation at 5000 rpm and 4 °C for 15 minutes, and the resulting supernatant was collected for enzyme activity assessment. All enzyme activities were normalized to the protein concentration using a total protein quantitative assay kit (JIANCHENG, A045–2–2). Details for lipid detection are provided in Supporting Information Text S5.

2.7. Transcriptome sequencing

Transcriptome sequencing was conducted on samples from the CG-1 and $PM_{2.5}$ groups collected at 9 d. Total RNA was extracted using the TRIzol method. The RNA quality and sequencing raw data were subjected to rigorous quality control, details are provided in Supporting Information Text S6. The raw data were initially aligned to the host genome of *Acropora digitifera* [28]. The remaining data were subsequently aligned to the genome of *Symbiodinium goreaui* (clade C1) (http://symbs.reefgenomics.org/download/), which is the primary Symbiodiniaceae of *A. formosa* from Weizhou Island [29].

Gene Ontology (GO) functional-enrichment analysis was conducted using Goatools on the Majorbio cloud platform to investigate the biological functions of differentially expressed genes (DEGs) in the coral host. Gene set enrichment analysis (GSEA) was applied to Symbiodiniaceae (C1) to analyze all intragene interactions. The protein-protein interaction (PPI) network of DEGs was mapped using the homologous species *Nematostella vectensis* with STRING online software (STRING: functional protein association networks (string-db.org)). Hub network modules were identified using MCODE, and important genes were screened based on betweenness centrality value in Cytoscape software (v3.9.1). Pathway enrichment analysis and visualization of omics data followed the method recommended by Reimand et al. [30]. Raw RNA-seq data are available from the NCBI SRA (BioProject: PRJNA992273).

2.8. qRT–PCR

All RNA samples underwent reverse transcription using the Prime Script™ RT Reagent Kit (TaKaRa, product number: RR047A). Gene expression was assessed using a relative quantitative method through real-time quantitative PCR (qRT-PCR) with SYBR PreMix ExTaq™ (Takara, Dalian, China). The $2^{-\Delta\Delta Ct}$ method was employed to calculate the relative mRNA expression levels of all related target genes [31]. All primer sequences are listed in Table S3.

2.9. Statistical analysis

Statistical analysis was conducted using SPSS Statistics version 26. An independent *t*-test was utilized to evaluate the differences in nutrient content, physiological indices, and gene expression between the CG-1 and $PM_{2.5}$ groups in Experiment 1. Tukey's multiple-comparison test was applied to evaluate physiological indices and gene expression across

different groups in Experiment 2. The significance threshold was set at $p \leq 0.05$. All data are presented as mean ± standard deviation.

3. Results and discussion

3.1. Rapid coral tissue detachment under wildfire $PM_{2.5}$ exposure

In pre-test results, the average survival time of corals (with no significant visual difference) was 8 days under 5 mg/L/Day $PM_{2.5}$, 20 days under 2 mg/L/Day $PM_{2.5}$, and 41 days under 1 mg/L/Day $PM_{2.5}$. Detailed information is provided in Supporting Information Text S3. The theoretical maximum added concentration of $PM_{2.5}$, calculated based on the daily replacement of 20 % seawater, was 20.81 mg/L for an 8 days add-exposure at 5 mg/L/Day, 9.88 mg/L for a 20 days add-exposure at 2 mg/L/Day, 5.00 mg/L for a 41 days add-exposure at 1 mg/L/Day. These results indicate that both short-term high-concentration wildfire $PM_{2.5}$ exposure and long-term low-concentration wildfire $PM_{2.5}$ exposure can induce *A. formosa* tissue detachment. The survival time of *A. formosa* corals increased as the daily added concentration of wildfire particulates decreased, indicating that a higher concentration of wildfire particulates deposition poses a greater threat to corals. This is particularly hazardous in scenarios such as shallow seawaters of coral reefs, coral reef areas in proximity to wildfires, and areas affected by particulate deposition from extreme wildfires.

The photosynthetic efficiency (Fv/Fm), Symbiodiniaceae density, and Chl-a content exhibited no significant changes (all $p > 0.05$; Fig. 1 B C). The photobiologically active radiation (PAR) variation under the same stress conditions, as monitored by Qin et al., indicated that 5 mg/L wildfire $PM_{2.5}$ stress had a significant impact on light intensity within approximately 3–4 h. The impact on light intensity led to a decline in the photosynthetic efficiency of *P. lutea* after 14 days of exposure [6]. Thus, wildfire particulates slightly impacted the symbionts' photosynthesis of coral *A. formosa* due to the short exposure time (9 d), despite causing alterations in light availability. However, coral tissue detachment was observed on Day 9 (Fig. 1 A), and the corals completely detached within the following 2 days. The rapid tissue detachment coincided with the increase in seawater nutrients derived from $PM_{2.5}$ (Tab. S2). During the first 3 days, nutrient levels changed slightly compared to initial levels (all $p > 0.05$), indicating effective nutrient uptake by corals. However, the nutrients significantly increased by day 9 (all $p < 0.05$; Fig. 1 D E F). These nutrient increases suggested a probable disruption in metabolic processes (e.g., nutrient assimilation). In the CG-1 group, the levels of seawater nutrients and the coral photosynthetic efficiency remained stable (all $p > 0.05$; Fig. 1 B D E F), with normal coral tissue status.

The $PM_{2.5}$-induced tissue detachment observed in this study is similar to coral tissue ablations reported in Pacific Costa Rica reefs [32], and similar to the coral polyp bail-out observed under insecticide chlordecone stress that involved programmed cell death (apoptosis) and degradation of extracellular matrix proteins [33]. However, as there was no significant loss of symbionts (Fig. 1 C), the corals retained their coloration despite tissue damage (Fig. 1 A), which differs from the bleached appearance caused by symbiont loss during heat bleaching events. Our findings also differ from the responses of corals (*G. pectinata* and *M. elephantotus*), which showed no apparent bleaching or mortality under sediment stress [20]. The impact of wildfire $PM_{2.5}$ on branching coral *A. formosa* in our study, which resulted in rapid tissue detachment within 9 days, is much more severe than that observed on massive coral *P. lutea* under the same level of wildfire $PM_{2.5}$ stress [6], with *P. lutea* surviving for over 35 days.

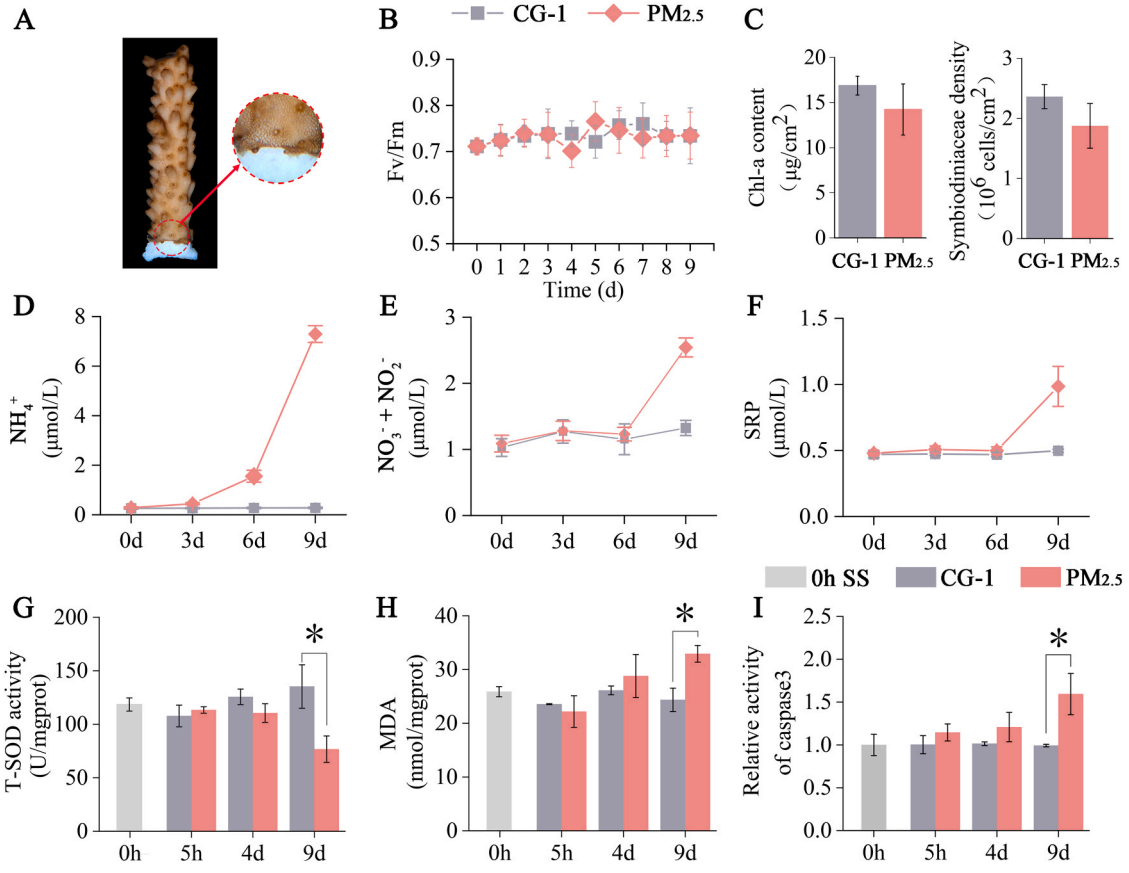

Fig. 1. Changes in *A. formosa* and seawater under 5 mg/L wildfire $PM_{2.5}$ exposure. A: Tissue detachment; B: Fv/Fm; C: Symbiodiniaceae density and Chl-a concentration on 9 d; D, E, F: seawater nutrients (NH_4^+; $NO_3^- + NO_2^-$; soluble reactive phosphorus, SRP). G, H, I: Physiological indicators (total superoxide dismutase, T-SOD; malondialdehyde, MDA; caspase3). The asterisks represent the statistical difference ($p < 0.05$).

3.2. Redox responses in corals triggered by wildfire $PM_{2.5}$

3.2.1. Oxidative stress resulted from $PM_{2.5}$ defense

The top 20 GO terms (upregulated DEGs) included those related to redox reactions (e.g., monooxygenase activity and oxidoreductase activity), heme cofactors (e.g., heme binding and tetrapyrrole binding), and iron binding (Fig. 2 A). Heme, with its tetrapyrrole structure containing a central ferrous ion, is essential in aerobic reactions and is primarily found in proteins such as catalase (CAT) and cytochrome P450 enzymes (CYPs) [34]. The monooxygenase activity GO term revealed multiple upregulated CYPs (Tab. S1; Fig. S1 B), potentially involved in metabolizing organic chemicals, as wildfire $PM_{2.5}$ contained 49.39 $\pm$ 5.66 % organic carbon (Tab. S2). The metabolism and detoxification of xenobiotic organics typically involve monooxygenases, especially CYPs [35], with enhanced efficiency through cytochrome b5 [36], which was also suggested by our findings (Fig. S1 C). Moreover, coral phagocytic cells could metabolize particles through a respiratory burst produced by the nicotinamide adenine dinucleotide phosphate (NADPH) oxidase complex [19], which was upregulated in response to wildfire $PM_{2.5}$ (Tab. S1). Hydrogen peroxide is substantially released during the respiratory burst of phagocytic cells [37] and via metabolic processes involving CYPs [35]. CAT (CAT_1, adig_s0028.g266), a key enzyme responsible for hydrogen peroxide breakdown, was identified as a key gene by the PPI network (Fig. 2 G). Hydrogen peroxide, a reactant in the Fenton reaction, interacts with ferrous ions to generate highly oxidative and damaging hydroxyl radicals (HO •). To mitigate this damage, organisms activate CAT to neutralize hydrogen peroxide, thus inhibiting the Fenton reaction [38]. The continuous and significant upregulation of CAT under $PM_{2.5}$ stress (Fig. S2 A) suggests the presence of excessive oxidative stress caused by hydrogen peroxide accumulation.

3.2.2. Severe oxidative damage to coral lipids and DNA

Lipids are key targets of oxidative damage [35], the significant increase in MDA (lipid peroxidation product) levels (Fig. 1 H) indicated that wildfire $PM_{2.5}$ induced severe oxidative stress in *A. Formosa*. MDA in corals typically increases under certain stressors such as ultraviolet stress [39] and polyethylene microplastics [40]. However, MDA levels in corals under thermal stress are not always elevated, which may be attributed to regulation by antioxidants [41]. Therefore, the significant increase in MDA implied that the action of antioxidants may be weakened. PPI submodule 1 revealed alterations in the cellular response to DNA damage (Fig. 2 C), suggesting that corals experienced DNA damage under wildfire $PM_{2.5}$ stress. Within PPI submodule 1, an upregulation of mutS protein homolog 5 (adig_s0002.g33) was observed, which is crucial for protecting against oxidative DNA damage [42]. Concurrently, there was a downregulation of genes involved in double-strand DNA repair, including RAD51 (adig_s0164.g25), RAD50 (adig_s0179.g37, adig_s0179.g39), and MRE11 (adig_s0005.g166). This downregulation may be associated with their roles in meiosis [43]. The upregulation of MEIOB (adig_s0049.g167) supports this interpretation, as MEIOB protein levels are associated with the severity of meiotic defects and regulate meiotic recombination in a dosage-dependent manner [44]. This inhibition of meiosis is consistent with observations in coral (*Pocillopora damicornis*) under glyphosate stress [45]. These findings suggest that wildfire $PM_{2.5}$ increases oxidative damage to lipids and DNA and impairs coral meiotic function.

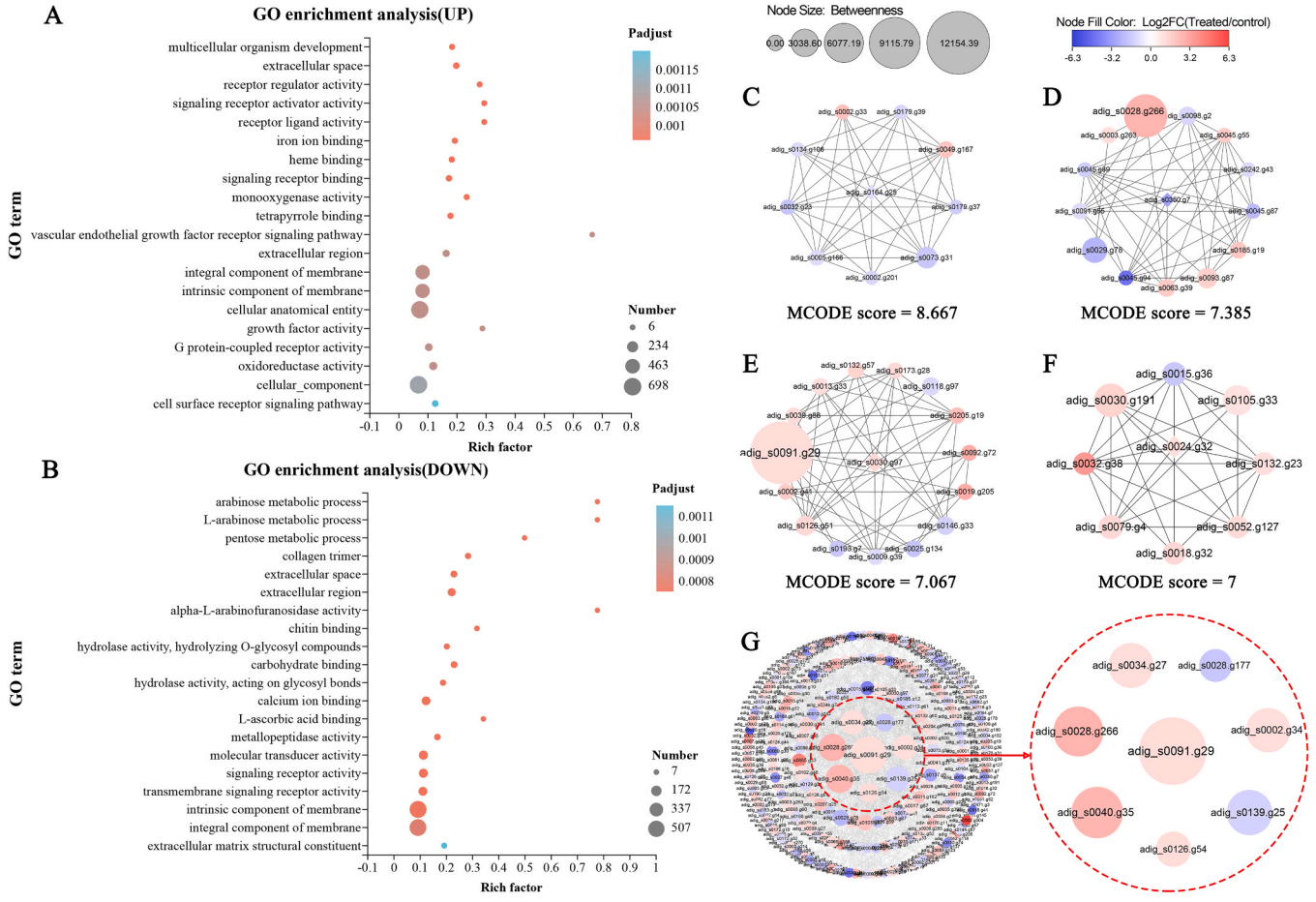

Fig. 2. GO enrichment and protein-protein interaction (PPI) networks analysis of host differentially expressed genes. GO enrichment of (A) upregulated genes and (B) downregulated genes, colors represent Padjust values, and dot size represents the number of genes. Four PPI hub network modules (C, D, E, F) and top 8 important genes (G), colors represent changes in gene expression, and betweenness centrality value is indicated by circle size.

3.2.3. Mitochondrial dysfunction and cell apoptosis

The upregulation of mitochondrial inner-membrane-related genes (PPI submodule 4, Fig. 2 F) indicated increased mitochondrial activity, potentially elevating the production of mitochondrial superoxide anions. SOD acts as the primary antioxidant defense against superoxide anions and is typically upregulated in corals in response to various stresses, including microplastic exposure [22], *Vibrio coralliilyticus* challenge [46], herbicide glufosinate exposure [45], and thermal pressure [47]. Despite this, SOD expression did not increase (Fig. S2 A), and its activity decreased (Fig. 1 G) in our results. Concurrently, PPI submodule 2, related to glutathione metabolism (Fig. 2 D), exhibited downregulated gene expression (Tab. S5), indicating a decreased antioxidant capacity. This likely resulted from reduced pentose metabolism, limiting the availability of the critical cofactor NADPH for glutathione regeneration. Given the critical role of SOD and glutathione in redox homeostasis, these disruptions increased mitochondrial susceptibility to superoxide anion damage [35]. However, glutamate synthesis, a precursor for glutathione synthesis, was significantly upregulated (Tab. S5; PPI submodule 2), possibly due to the elevated ammonium levels from wildfire $PM_{2.5}$ (Tab. S2), as coral glutamate metabolism is associated with ammonium assimilation [31]. We observed a significant increase in mitochondrial mitophagy and the cell death marker gene BCL-2/adenovirus E1B-interacting protein-3 (BNIP3) at all time points (Fig. S2 A), indicating rapid induction of mitochondrial autophagy due to oxidative stress. BNIP3 overexpression is known to promote apoptosis [48], as reflected by the increased caspase3 activity and expression (Fig. 1 I, Fig. S2 A). This oxidative stress scenario (Fig. 6 a, f), characterized by excessive production of ROS (superoxide anions, hydrogen peroxide) and reduced antioxidant capacity (SOD activity and glutathione metabolism), leads to mitophagy and cell apoptosis.

3.2.4. Self-repair mechanisms in response to coral damage

One key upregulated gene, catenin beta (adig_s0091.g29), identified in the PPI network (Fig. 2 G), was involved in PPI submodule 3, which was related to multicellular organism development (Fig. 2 E). Catenin beta plays a crucial role in cell adhesion and is essential in multicellular development [49]. This finding was supported by pathway enrichment analysis (Fig. 3 A), indicating that wildfire $PM_{2.5}$ stress activated the transcriptions of genes involving various tyrosine receptor proteins that regulate biological processes in multicellular development. These genes were enriched in diverse GO terms, including enzyme-linked receptor protein signaling, cell surface receptor signaling, transmembrane receptor protein tyrosine kinase signaling, fibroblast growth factor (FGF) receptor signaling, and vascular endothelial growth factor (VEGF) receptor signaling. These results were consistent with several of the top 20 GO terms (upregulated DEGs), including multicellular organism development, vascular endothelial growth factor receptor signaling, and other signaling GO terms (Fig. 2 A). The activity of receptor protein tyrosine kinases influences cell growth and division [50], and the roles of FGF and VEGF signaling in the repair and regeneration of coelenterates, such as in *Hydra*, have been documented [51]. Furthermore, network analysis identified multiple genes involved in coelenterate development and repair (Tab. S4). These findings suggest that corals activate self-repair mechanisms by regulating multicellular development processes in response to extensive damage caused by wildfire $PM_{2.5}$.

3.3. Metabolism disruption linked with cell adhesion network disorder

3.3.1. Symbiodiniaceae reduced energy supply to coral host

GSEA analysis indicated a general upregulation in processes related to photosynthesis and the metabolism of Symbiodiniaceae, including the pentose phosphate pathway, C5-Branched dibasic acid metabolism, and oxidative phosphorylation, among others (Tab. S1). This upregulation may be attributed to the presence of ammonium and nitrate from wildfire $PM_{2.5}$ (Tab. S2), which alleviates nitrogen limitations and facilitates the adaptation of Symbiodiniaceae to adverse conditions. This hypothesis is supported by the study showing that C5-Branched dibasic acid metabolism plays a crucial role in plant responses to excessive nitrogen [52]. Symbiodiniaceae tended to retain more photosynthates to support their increased metabolism, leading to reduced carbon translocation to coral hosts. The presence of nitrate may exacerbate the nutrient retention effect [53]. Consequently, wildfire $PM_{2.5}$ tends to reduce the nutrient supply of symbionts to coral hosts (Fig. 6 d).

3.3.2. Coral hosts utilized stored energy for self-consumption

The amino-acid betaine metabolic process exhibited upregulation (p value < 0.05, Tab. S1), suggesting an increased demand for protein synthesis in response to wildfire $PM_{2.5}$ stress. This aligns with the coral's response to heat stress [54]. To meet the increased energy demands for wildfire $PM_{2.5}$ removal, oxidative stress response, and self-repair, corals might utilize resources from their carbohydrates and lipids when the host receives insufficient nutrients from Symbiodiniaceae. However, the pentose metabolic process, hydrolase activity targeting O-glycosyl compounds and glycosyl bonds, and monosaccharide metabolic process were downregulated (Fig. 2 B, Tab. S1), indicating a reduction in energy

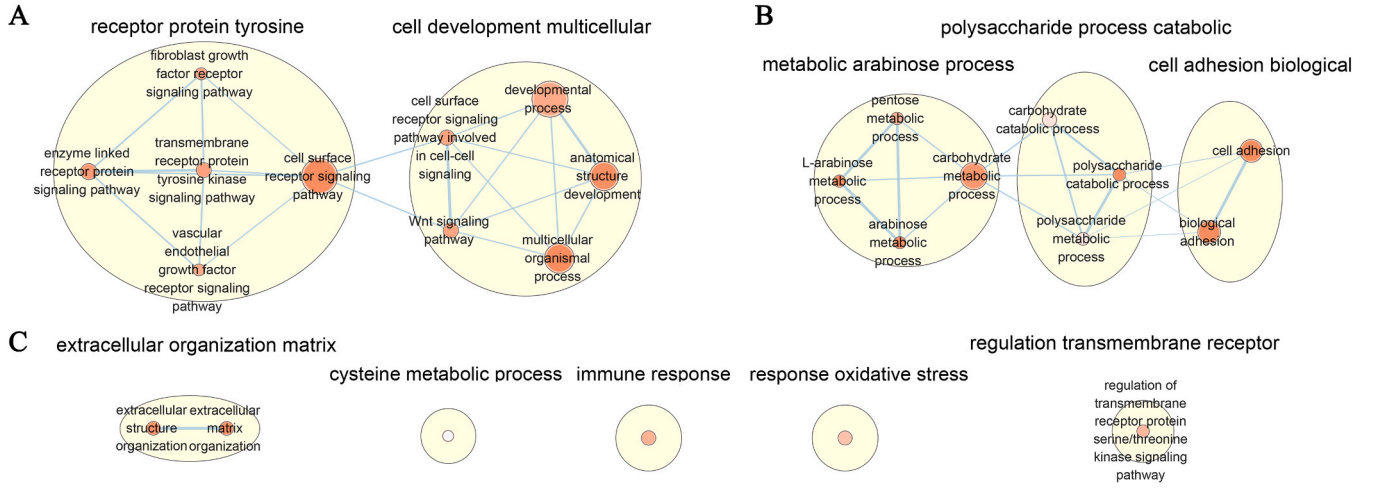

Fig. 3. Pairwise pathway enrichment analysis of all significantly different GO biological process terms. Pathway enrichment between receptor protein tyrosine and cell development multicellular (A); Pathway enrichment among cell adhesion biological and polysaccharide process catabolic and metabolic arabinose process (B); Other unenriched themes (C); Blue lines depict intra- and inter-pathway relationships according to the number of genes shared between each pathway. Black circles group related pathways into themes. Colored circles (nodes) represent pathways, color deepened by p-value, sized by the number of differential genes.

supply from carbohydrates. Additionally, very long-chain fatty acids can be converted into medium-chain fatty acids through peroxisomal beta-oxidation [55]. The increased expression of very long-chain specific acyl-CoA dehydrogenase (Tab. S6) suggested substantial fatty acid consumption, but the downregulation of carboxylic ester hydrolase activity, involved in lipid hydrolysis metabolism (Tab. S1), along with the decreased lipid content (Fig. S4), suggested an insufficient lipid supply. Corals could generate glucose/glycogen (gluconeogenic-type pathway) from aliphatic acid breakdown products (e.g., acetyl-CoA) through the glyoxylate cycle, a bypass of the tricarboxylic acid (TCA) cycle, as demonstrated when processing natural particles or artificial silicon carbide mixture [20]. Transcriptome analysis revealed that, between the two key enzymes of the glyoxylate cycle (Tab. S6), only isocitrate lyase (IL) showed a significant increase, while malate synthesis (MS) did not change significantly. However, both IL and MS exhibited significant upregulation during the early resistance phase (5 h, 4 d; Fig. S2 D). It appears that corals initially utilize their carbohydrates and lipids for protein synthesis and energy supply (Fig. 6 b, c, e), but this energy source likely diminishes over time due to excessive consumption.

3.3.3. *Metabolism disruption linked with adhesion led to coral tissue detachment*

The coral tissue detachment observed in our study represents a stress response known as "polyp bail-out", characterized by the dissociation of coral colonies from their calcareous skeletons through coenosarc degradation and detachment of zooxanthellate polyps. The correlation enrichment network indicated the link between disruptions in glycometabolism and cell adhesion functions (Fig. 3 B; Tab. S1). In *Montastraea faveolata*, adhesion network disorders under heat stress induced the detachment of gastrodermal cells containing Symbiodiniaceae [56]. In our study, we speculated that coral tissue detachment under wildfire $PM_{2.5}$ stress may be attributed to disordered cell adhesion networks. The phenomenon of "polyp bail-out" has been observed under various stressors, including reduced pH conditions [57], hyperosmotic treatment [58], insecticide chlordecone [33], and thermal stress [59]. The mechanisms underlying "polyp bail-out" vary depending on the stressor. "Polyp bail-out" caused by reduced pH conditions is attributed to triggering tissue-specific apoptosis [57], hyperosmotic treatment is associated with extracellular matrix degradation induced through FGF signaling [58], and exposure to insecticide chlordecone leads to apoptosis and degradation of cellular matrix proteins [33]. Similarly, our results showed significant differences in apoptosis (Fig. 1 I), extracellular matrix, FGF signaling, etc. (Tab. S1). The parallelism of adhesion network disorders was also documented in *Pocillopora damicornis* exposed to chlordecone [33]. Chlordecone is an organic pollutant, and wildfire particulates contain a large amount of organic carbon; both activate the metabolic responses through the cytochrome P450 family and similarly result in adhesion network disorders and tissue detachment. These findings support the possibility that the disordered cell adhesion network leads to coral tissue detachment.

Furthermore, the adhesion network was influenced by disruptions in polysaccharide and arabinose metabolism (Fig. 3 B). Arabinose, previously detected at enriched levels in the coral skeletal organic matrix [60], plays a role in cell adhesion through its participation in polysaccharides [61]. Consequently, the decrease of L-arabinose (Fig. 2 B) impaired adhesion function. Moreover, the downregulation of monosaccharide metabolic process (Tab. S1) indicated a reduced energy supply, leading to a decrease in collagen trimers (Fig. 2 B). Collagen-derived proline can be metabolized to support the TCA cycle, thereby aiding cell survival in nutrient-limited conditions [62]. Collagen provides cell-adhesion sites that are specifically recognized by cell-surface receptors [63]. Thus, declines in collagen affected adhesion function. Additionally, there were upregulations of polysaccharide catabolic and metabolic processes, with a predominance of fibrocystin-L transcripts (Tab. S1). Cells with elevated fibrocystin-L levels exhibited reduced attachment to collagen matrices compared to those with lower expression [64]. The chitin binding was downregulated (Fig. 2 B), likely related to disrupted glycometabolism, given chitin's polysaccharide nature. Chitin forms a resilient matrix that interacts with adhesive proteins on barnacle larvae, securing their attachment to substrates [65]. In corals, chitin in the skeletal matrix likely enhances structural integrity and indirectly supports tissue adhesion [60]. Moreover, calcium ion binding was downregulated (Fig. 2 B), potentially impacting cell adhesion mechanisms by affecting components such as the cadherin-catenin complex [66]. Meanwhile, PPI analysis identified the upregulation of key genes integrin beta-1 (adig_s0126.g54) and catenin beta (Fig. 2 G), which were likely involved in regulating disrupted cell adhesion networks. Integrin beta and catenin beta are known to be important in cell adhesion processes [49,67]. These findings further confirm the contribution of adhesion dysfunction to coral tissue detachment. Collectively, polysaccharide and L-arabinose metabolism, along with collagen, affect coral adhesion during wildfire $PM_{2.5}$ exposure. Insufficient lipid reserves exacerbate disruptions in the adhesion network and contribute to tissue detachment in corals (Fig. 6 e).

3.4. *Weakened HSP function reduced coral heat tolerance*

PPI network analysis identified heat shock protein (HSP) 70 (adig_s0028.g177) as an important gene, which was downregulated under wildfire $PM_{2.5}$ stress (Fig. 2 G, Fig. S2 C). Further analysis of DEGs in the HSP family indicated the downregulation of three HSP70s, an overall upregulation of HSP20s, and no significant changes in HSP90s (Fig. S3 A B C). The induction and regulation of HSPs serve as a defense mechanism, promoting recovery from stress events, including elevated temperatures and pollutants [68,69]. Typically, HSPs increase under oxidative stress to protect normal protein function, with HSP70 playing a critical role [70]. Due to their dependence on ATP, high molecular weight HSPs (40–105 kDa), such as HSP70s and HSP90s, are likely to decrease when ATP supply is insufficient [71]. Conversely, HSP20s, being small HSPs, may respond differently under varying conditions. ATP synthase expression remained relatively stable (Fig. S5), likely because corals utilized reserved substances (e.g., carbohydrates and lipids) to temporarily sustain ATP production rates under adverse conditions [72]. However, the increased energy demand during wildfire $PM_{2.5}$ stress may deplete these reserves, thereby impacting ATP production. Moreover, the middle domain of HSP90 and the SIPRT domains of sacsin exhibit structural similarities, facilitating ATP recruitment for binding [73]. In contrast, HSP70 lacks this structural feature and depends on sacsin for ATP recruitment [71]. Sacsin levels decrease under conditions of severe oxidative stress and mitochondrial dysfunction [74]. We hypothesized that the downregulation of the sacsin gene (Fig. S3 D) was linked to oxidative stress and mitochondrial dysfunction induced by wildfire $PM_{2.5}$ (Fig. S2 A), potentially contributing to the decreased expression of HSP70.

The reduced expression of HSP70 induced by wildfire $PM_{2.5}$ increased coral susceptibility to heat bleaching, as demonstrated in our subsequent experiment (Fig. 4 A). In the HT group, corals experienced bleaching and mortality within 28–31 days. Under combined stress, coral survival decreased to 9–12 days in the HTHP group, with apparent changes that were similar to coral gradual bleaching and death under heat stress (Fig. 4 A). Coral survival decreased to 17–20 days in the HTLP group, with apparent changes that were similar to tissue detachment (Fig. 4 A). In contrast, no visible signs of bleaching or tissue detachment were observed in the CG-2 group by Day 32. We speculated that the reason for HP accelerating the thermal bleaching process might be related to its higher concentration of nutrients, while LP mitigated the harm of thermal stress to symbionts to a certain extent by providing nutrients at a more appropriate concentration, but also led to tissue detachment in corals under long-term low-concentration wildfire particulates stress. Further research is needed to elucidate the detailed mechanisms in the future. The Fv/Fm of HT, HTLP, and HTHP groups showed a synchronous decrease compared to the CG-2 group (Fig. 4 B).

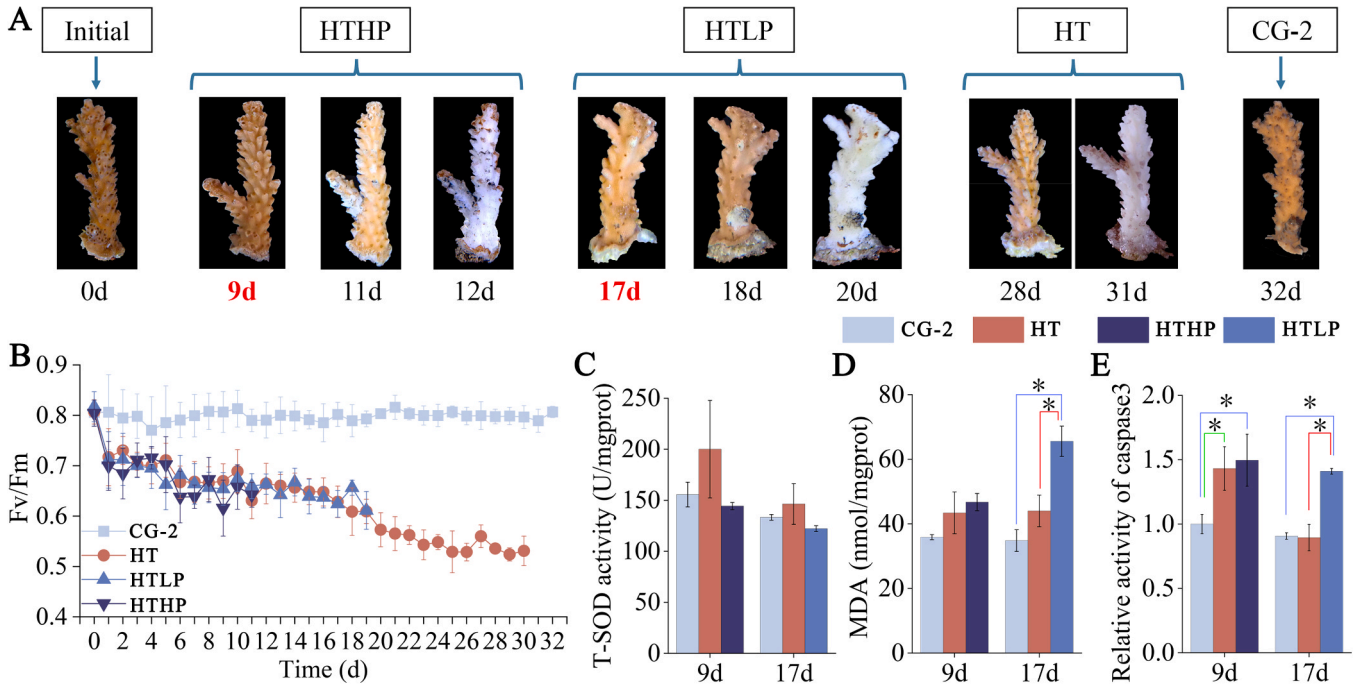

Fig. 4. Changes in *A. formosa* under combined stress of high temperature and wildfire $PM_{2.5}$ (CG-2: 28℃, HT: 32℃, HTHP: 32 ℃ + 5 mg/L $PM_{2.5}$, HTLP: 32 ℃ + 1 mg/L $PM_{2.5}$). A: Coral state; B: Fv/Fm; C, D, E: Physiological indicators (total superoxide dismutase, T-SOD; malondialdehyde, MDA; caspase3) in all collected samples. The asterisks represent the statistical difference ($p < 0.05$).

Furthermore, compared to the HT group, coral expression levels of CYP and CAT1 genes were significantly elevated in the HTHP and HTLP groups ($p < 0.05$; Fig. 5 A, B). There were no significant differences in CYP and CAT gene expression between the HT and CG-2 groups ($p > 0.05$). These results suggest that corals allocate energy to metabolize $PM_{2.5}$ and detoxify hydrogen peroxide, potentially reducing the energy available for coping with heat stress. Moreover, compared to the HT group, corals in the HTHP showed significantly lower gene expression of HSP70_1 and HSP70_3 ($p < 0.05$; Fig. 5 E) and a non-significant difference in T-SOD activity ($p > 0.05$; Fig. 4 C). There were also significantly lower gene expressions of MnSOD and CuZnSOD in the HTHP group compared to the HT group ($p < 0.05$; Fig. 5 C). These results indicate that wildfire $PM_{2.5}$ stress suppresses coral's SOD defense and HSP70 protection. Additionally, there were significantly higher values in MDA, caspase3 level and expression, and BNIP3 expression in the HTLP group compared to the CG-2 and HT groups (Fig. 4 D, E, Fig. 5 D). Although the survival times of corals were longer under low $PM_{2.5}$ stress compared to high $PM_{2.5}$ stress, prolonged exposure to low wildfire $PM_{2.5}$ stress still exacerbated oxidative stress, mitophagy, and cell apoptosis in corals under heat stress. In a warming climate, atmospheric POC is expected to supplement food availability from oceanic sources, thus offsetting coral decline caused by elevated temperatures. Recent research suggests a limited potential for wildfire particulates to serve as an alternative nutrient source for massive corals [6]. This study elucidates the underlying mechanisms of coral tissue detachment and reduced heat tolerance induced by wildfire particulates, suggesting that

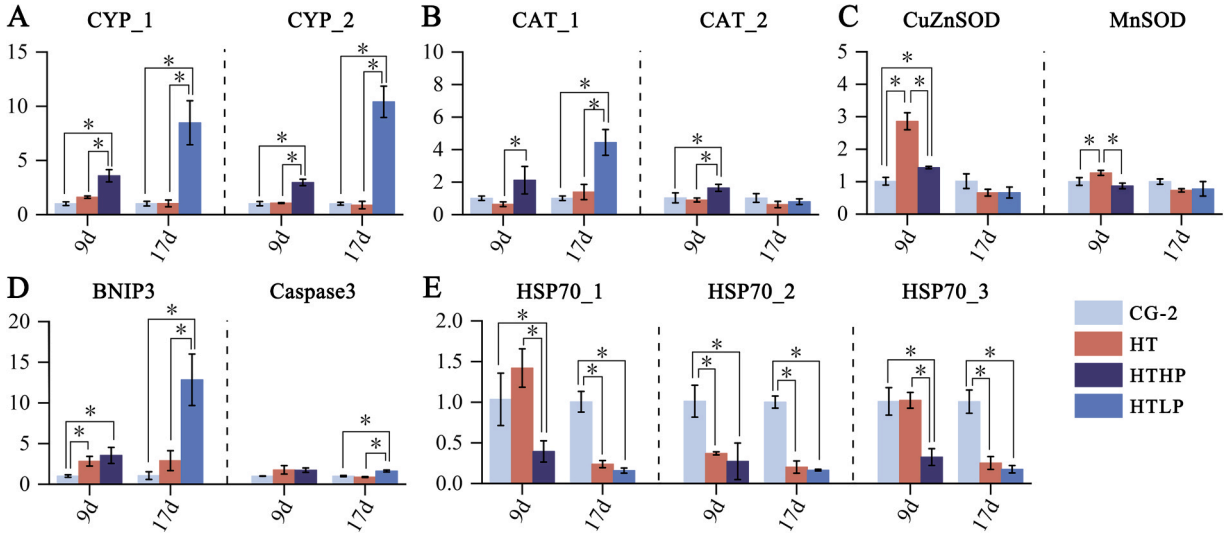

Fig. 5. Changes in gene expression under combined stress of high temperature and wildfire $PM_{2.5}$ (CG-2: 28℃, HT: 32℃, HTHP: 32 ℃ + 5 mg/L $PM_{2.5}$, HTLP: 32 ℃ + 1 mg/L $PM_{2.5}$). A: two CYP genes; B: two CAT genes; C: two SOD genes; D: BNIP3 gene and caspase3 gene; E: three HSP70 genes. The asterisks represent the statistical difference ($p < 0.05$).

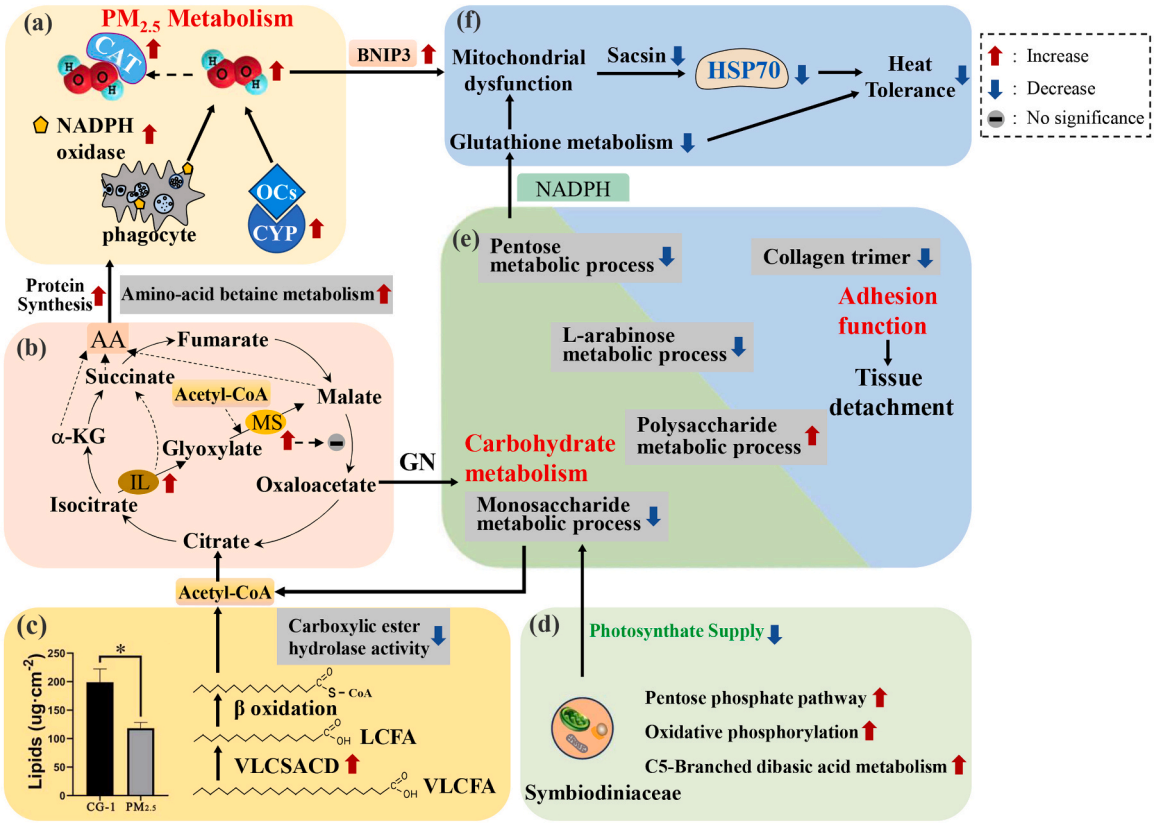

Fig. 6. Concept map illustrating the mechanisms by which wildfire $PM_{2.5}$ affects coral physiology. Coral metabolism of organic chemicals and particulates from $PM_{2.5}$ produces excessive hydrogen peroxide (a). Lipid metabolism provides energy to corals, but depletion of lipid reserves decreases this supply (b, c). Disruption of carbohydrate metabolism arises from reduced nutrient supply by Symbiodiniaceae (d) and insufficient lipid availability (c). Impaired carbohydrate metabolism compromises adhesion functions, inducing coral tissue detachment (e). Decreased pentose metabolism, combined with elevated hydrogen peroxide, impairs glutathione metabolism and mitochondrial dysfunction, reducing heat tolerance through reduced HSP70 expression (f). Abbreviations: CAT, Catalase; OCs, organic chemicals; CYP, Cytochrome P450; AA, Amino acid; MS, Malate synthesis; IL, Isocitrate lyase; HSP70, Heat Shock Protein 70; BNIP3, BCL-2/adenovirus E1B-interacting protein-3; NADPH, Nicotinamide adenine dinucleotide phosphate; GN, Gluconeogenesis; α-KG, α-Ketoglutarate; LCFA, Long-chain fatty acids; VLCFA, Very long-chain fatty acids; VLCSACD, Very long-chain specific acyl-CoA dehydrogenase.

greater risks wildfire particulates pose to branching corals, which are typically sensitive to environmental changes.

In summary, these underlying mechanisms are primarily based on rational analysis explained by existing molecular functions. Given the objective difficulties in implementing molecular functional validation in corals [75], further in-depth verification of related mechanisms is still needed in the future. Exactly as branching corals are typically more sensitive to environmental changes than massive corals, the resistance of *A. formosa* to wildfire particulates is significantly lower than that of *P. lutea*. Consequently, the significantly varied responses of different coral species to wildfire particulates may lead to alterations in the ecological structure of coral reefs. Although massive corals exhibit sustained survival [6], the heat tolerance of branched corals is reduced by wildfire particulates, an effect that warrants investigation in massive corals as well. Amid increasingly severe environmental pollution, wildfire particulates may impose superimposed stress on corals in conjunction with other pollutants, particularly organic matter pollution. Furthermore, the combined effects of upwelling and wildfire particulates on nutrients may lead to coral suffocation and death by triggering the occurrence of red tides [4,76], or further impact coral survival through the competitive threat posed by the promotion of macroalgae growth [27]. This study is based on microscopic experimental methods, and the limited amount of seawater may slightly strengthen the impact of wildfire $PM_{2.5}$ on corals. The effects of wildfire-derived particulates deposition in actual marine ecosystems deserve continued attention. Additionally, the calculated concentrations in our study primarily derive from literature-based estimations, while the actual dispersion of wildfire emissions is influenced by environmental factors such as temperature, air pressure, humidity, and wind speed [77]. Although the actual deposition of wildfire particulates cannot be fully captured by these calculations, the exposure to varying concentrations in our experimental design maximally simulates the complexity of wildfire emissions in actual environments, which are affected by environmental factors. Considering that wildfires may occur in areas near coral reefs in reality [5], more particulates may enter the coral reefs along with groundwater and rivers, leading to higher concentrations of sedimentation. Additionally, the retention effect of particulates [25] could lead to prolonged exposure to corals. Therefore, the outlook for the impact of wildfire particulates on coral is not optimistic, especially against the backdrop of global warming. This study broadens our understanding of the risks associated with oceanic deposition of wildfire emissions on marine ecosystems, underscoring the urgent need for effective wildfire management, especially in regions adjacent to coral reefs and during periods of elevated sea surface temperatures. These strategies include daily surveillance of combustible vegetation, regulation of outdoor fire sources, proactive wildfire prevention measures such as constructing isolation zones through rational strategic logging, substantial investment in emergency firefighting infrastructure in wild areas, and the dissemination of wildfire prevention knowledge to residents and tourists.

4. Conclusions

This study reveals the underlying mechanisms by which wildfire $PM_{2.5}$ triggers coral tissue detachment and coral's decreased heat

tolerance. Wildfire $PM_{2.5}$ exposure induces severe oxidative damage in corals. Corals activate defense mechanisms, including the respiratory burst of phagocytic cells and cytochrome P450 enzymes, to metabolize and detoxify $PM_{2.5}$. These processes generate excessive ROS, such as superoxide anions and hydrogen peroxide. Despite the upregulation of CAT to mitigate hydrogen peroxide levels, there is a significant decrease in SOD activity and suppression of glutathione metabolism. This compromised antioxidation system leads to oxidative damage to lipids and DNA, further exacerbating mitochondrial dysfunction and cell apoptosis. In response to $PM_{2.5}$-induced damage, coral activates the transcriptions of genes related to multicellular organism development for self-repair, increasing demands for protein synthesis and energy expenditure to support both defense and restoration processes. Concurrently, symbionts upregulate processes linked to photosynthesis and metabolism, likely retaining photosynthate and limiting nutrients transfer to the host coral. The transcriptions of coral genes related to carbohydrates and lipids metabolism are downregulated due to excessive consumption, impacting glycometabolism and adhesive molecules such as polysaccharides and collagen. Ultimately, these metabolic adjustments contribute to coral tissue detachment. Additionally, wildfire $PM_{2.5}$ exposure increases coral susceptibility to heat bleaching. Mitochondrial apoptosis and reduced sacsin expression impair HSP70 function, weakening its ability to protect against stress-induced protein damage and increasing vulnerability to heat bleaching.

Environmental Implication

Wildfire emissions were noted to potentially induce coral asphyxiation and mortality by triggering red tides (*N.J. Abram, Science, 2003*), but wildfires near coral reefs pose a direct threat to corals, which has only recently been recognized (*J. Tollefson, Nature, 2023*). This study focuses on frontier issues regarding the threat mechanism of wildfire emissions to corals and investigates the reasons for the impact of wildfire emissions on coral heat tolerance. This manuscript ascertains the pressing issue of wildfire emissions' impact on coral reef ecosystems and emphasizes the importance of wildfire management for the protection of important coral reef ecosystems.

CRediT authorship contribution statement

Kefu Yu: Writing – review & editing, Resources, Project administration, Funding acquisition. **Xiaoyan Chen:** Writing – review & editing, Project administration, Funding acquisition, Formal analysis, Conceptualization. **Yu Zhou:** Methodology. **Wenqian Zhang:** Visualization, Software. **Yichen Fu:** Visualization, Software. **Ruoxing Hao:** Visualization, Validation, Methodology, Formal analysis. **Ke Liu:** Writing – original draft, Visualization, Validation, Methodology, Formal analysis, Data curation. **Bo Qin:** Methodology.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jhazmat.2024.136840.

Data availability

Data will be made available on request.

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