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Integrative Analysis of Mitochondrial Dysfunction and Stress Signaling in Arabidopsis Roots Exposed to Salinity

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ABSTRACT: Salt stress poses a major limitation to plant growth, particularly affecting root energy metabolism and redox stability. This study investigates how mitochondrial bioenergetics in Arabidopsis root cells are reprogrammed in response to moderate and severe salt stress. Using high-resolution respirometry and transcriptomic profiling, we assessed mitochondrial performance under 0, 100, and 200 mM NaCl. Mitochondrial respiration was measured using the O2k Oroboros system, applying a substrate-uncoupler-inhibitor titration protocol. Key parameters included basal, OXPHOS, LEAK respiration, respiratory control ratio (RCR), ATP production, and maximal electron transport capacity. RNA-seq followed by qPCR validation was conducted to evaluate gene expression changes. Physiological markers such as ROS, proline, malondialdehyde (MDA), and K^+/Na^+ ratios were also quantified. Results showed a substantial decline in OXPHOS (up to 60%) and ATP production (up to 70%) under 200 mM NaCl, alongside a marked increase in LEAK respiration and ROS emission. RCR values dropped from 7.2 to 2.1, indicating impaired mitochondrial efficiency. AOX1a expression increased 8-fold, while Complex I genes were downregulated. Concurrently, proline levels rose 15-fold, and MDA and ROS levels confirmed oxidative stress. Transcriptomic data revealed activation of antioxidant pathways and UPRmt components. These findings reveal a coordinated mitochondrial adaptation under salt stress, shifting electron transport from canonical pathways toward AOX-mediated respiration to maintain redox balance. The integration of bioenergetic, molecular, and physiological data provides novel insights into the mechanisms underlying stress tolerance. Targeting AOX and mitochondrial stress responses may enhance resilience in crops facing increasing salinity.

Keywords: Salt Stress, Mitochondria, Alternative Oxidase, Oxidative Phosphorylation, ROS, Arabidopsis Thaliana, Bioenergetics, UPRmt.

INTRODUCTION

Soil salinity is one of the most significant abiotic stresses limiting agricultural productivity globally. Affecting more than 20% of irrigated lands, salinity primarily impairs plant water uptake through osmotic stress and introduces ionic toxicity via sodium (Na^+) and chloride (Cl^-) accumulation. These physiological disruptions not only hinder growth but also destabilize cellular homeostasis and interfere with essential metabolic processes (Othman et al., 2017; Zhao et al., 2020).

Particularly in root tissues, salt stress induces profound changes including reduced root elongation and altered architecture, which are critical for the plant's adaptive response (Zhao et al., 2020).

Beyond osmotic and ionic disturbances, salinity also causes oxidative stress by promoting the overproduction of reactive oxygen species (ROS), leading to lipid peroxidation, protein oxidation, and DNA damage. Mitochondria, the energy hubs of plant cells, are pivotal in these responses. As primary sites of ATP synthesis via oxidative phosphorylation (OXPHOS), they are also a significant source of ROS under stress conditions (Hossain & Dietz, 2016). Importantly, mitochondria integrate multiple stress signals and orchestrate cellular defense mechanisms through metabolic adjustments and redox signaling (Piel et al., 2026; Wang et al., 2026).

One of the key adaptive mechanisms in mitochondrial stress response is the activation of the alternative oxidase (AOX) pathway. This bypass route permits electron transport to oxygen without contributing to the proton gradient, thereby reducing mitochondrial membrane potential and ROS formation (Liu et al., 2021; Xu et al., 2023). Recent findings indicate that AOX activity increases under salt stress, offering protection against oxidative damage and preserving mitochondrial function. Moreover, AOX regulation is increasingly recognized as a critical factor for redox homeostasis and energy flexibility under stress (Xu et al., 2023).

Salt-induced dysfunction of the electron transport chain (ETC), especially Complex I, has been well-documented. High salinity hampers ETC function by disrupting protein integrity and enhancing ROS accumulation. This leads to the downregulation of Complex I subunits and promotes alternative respiration pathways (Manbir et al., 2022). AOX becomes essential under these conditions as it bypasses the inhibited segments of the ETC and helps sustain electron flow while minimizing ROS production. Such re-routing of electron transport not only limits oxidative damage but also supports survival under conditions where ATP synthesis efficiency is compromised (Brew-Appiah et al., 2018).

Furthermore, AOX expression is widely observed in multiple stress contexts including salinity, drought, and cold, marking it as a universal stress-resilience trait in plants. Studies demonstrate that its upregulation correlates with enhanced stress tolerance, validating its function as a crucial ROS buffering system (Yu et al., 2020). Consequently, targeting AOX and its regulatory networks offers potential in developing salt-tolerant genotypes through genetic or biotechnological strategies (Li et al., 2021).

The advent of high-resolution respirometry has revolutionized plant stress physiology by providing detailed insights into mitochondrial respiratory dynamics. Techniques such as substrate-uncoupler-inhibitor titration (SUIT) have made it possible to dissect the contributions of cytochrome and alternative pathways with precision (Mesa-Marín et al., 2018; Vishwakarma et al., 2018). These tools have been instrumental in revealing subtle shifts in respiratory profiles under salt stress, helping to quantify the impact of stress on mitochondrial efficiency, coupling status, and proton leak (Pan et al., 2026; Zandalinas & Mittler, 2017).

At the molecular level, salt stress activates a constellation of signaling pathways in Arabidopsis roots involving phytohormones like abscisic acid (ABA), stress-responsive genes such as AOX1a,

and antioxidant enzymes including CAT2 and GPX7 (Wanniarachchi et al., 2018). These components form an integrated response aimed at controlling redox status, maintaining osmotic balance, and ensuring cellular survival. Additionally, stress-induced metabolic reprogramming diverts resources toward protective functions such as osmolyte biosynthesis and membrane stabilization (Lima et al., 2026; Zhou et al., 2026).

Despite significant advances, few studies have holistically integrated high-resolution mitochondrial respiration analysis with transcriptomic and physiological profiling under salt stress. This integration is essential to delineate how respiratory adjustments interact with gene expression and metabolic reprogramming. The present study aims to fill this gap by combining respirometric, molecular, and biochemical data to elucidate the mitochondrial bioenergetic reprogramming occurring in Arabidopsis root cells under increasing salinity(Li et al., 2026; Menale et al., 2026).

This study proposes that salt stress imposes a dual burden energy limitation and oxidative damage which is counteracted by a shift from canonical OXPHOS to AOX-mediated respiration. This hypothesis is grounded in the expectation that mitochondrial dysfunction under high salt conditions triggers both transcriptional and post-translational responses, including the upregulation of AOX and ROS detoxification machinery. The study focuses on NaCl concentrations of 100 and 200 mM, representing moderate to severe stress levels, over a 72-hour period. Through this comprehensive approach, the research aims to offer mechanistic insights into mitochondrial plasticity and its contribution to salt stress resilience in plants(Wibom et al., 2026; Zhang et al., 2026).

METHOD

Plant Growth and Stress Treatment

Arabidopsis thaliana (ecotype Col-0) seeds were surface-sterilized and germinated on half-strength MS medium. After 7 days, seedlings were transferred to hydroponic culture and grown under controlled conditions (22°C, 16 h light/8 h dark). Salt stress was induced by supplementing the nutrient solution with NaCl to final concentrations of 0 mM (control), 100 mM, and 200 mM for a duration of 24 to 72 hours. Sampling was conducted at 48 hours for bioenergetics and transcriptomic analyses.

Mitochondrial Respiration Analysis

High-resolution respirometry was conducted using the O2k Oroboros system. Root tissue was excised and permeabilized using digitonin in MiR05 respiration buffer, containing KH2PO4, EGTA, MgCl2, sodium glutamate, taurine, and BSA to ensure mitochondrial stability. Sequential substrate-uncoupler-inhibitor titration (SUIT) protocols were adapted for Arabidopsis root mitochondria.

State 2 respiration was initiated with pyruvate and malate. ADP addition stimulated State 3 respiration (OXPHOS). LEAK respiration was measured following ADP depletion. FCCP was titrated to determine maximal ETS capacity, and inhibitors (e.g., rotenone, antimycin A) were applied to dissect electron flow through Complex I and III. Calibration and data integrity were confirmed according to standard O2k guidelines (Fuchs et al., 2023).

RNA Sequencing and Gene Expression Analysis

Root samples were flash-frozen in liquid nitrogen and RNA was extracted using a silica column-based kit. RNA quality was validated using spectrophotometry and agarose gel electrophoresis. Only samples with RNA Integrity Number (RIN) > 8.0 were used. Libraries were prepared and sequenced (150 bp paired-end reads), and reads were aligned to the Arabidopsis TAIR10 genome. Differential gene expression was assessed using DESeq2. Gene expression of AOX1a, CAT2, NDUFS1, and HSP70 was validated by qPCR using SYBR Green chemistry. Primer efficiency and melt curve analyses ensured specificity and quantification accuracy (Alscher & Stabenau, 2022).

Physiological and Biochemical Measurements

Reactive oxygen species (ROS) were quantified using 2',7'-dichlorofluorescin diacetate (DCF-DA). Lipid peroxidation was measured by malondialdehyde (MDA) content using the thiobarbituric acid reactive substances (TBARS) method. Proline content was determined by the ninhydrin assay. Ion content was measured by flame photometry to calculate K^+/Na^+ ratios. All experiments were performed in biological triplicates.

RESULT AND DISCUSSION

Salt-Induced Respiratory Reprogramming

Salt stress exerted a pronounced impact on mitochondrial respiration parameters. Basal respiration rates declined from 22–26 pmol $O_2 \cdot s^{-1} \cdot 10^6$ $cells^{-1}$ under control to 9–11 under 200 mM NaCl, indicating over 55% inhibition. Similarly, OXPHOS respiration fell by ~60%, whereas LEAK respiration increased by nearly 70%, confirming mitochondrial uncoupling and increased proton permeability. The respiratory control ratio (RCR) dropped sharply from ~7.2 (control) to ~2.1 (200 mM), a clear indication of reduced mitochondrial coupling efficiency (Del-Saz et al., 2016).

Under osmotic stress, OXPHOS performance was initially more stable, but the pronounced increase in LEAK values at 200 mM NaCl suggested that ionic stress had a more damaging impact on inner mitochondrial membrane integrity. The sharp decline in electron transport system (ETS) capacity and increased ROS emission (6.2–7.5%) further emphasized mitochondrial dysfunction

under high salt levels. These trends delineated a transition from moderate to severe mitochondrial impairment between 100 and 200 mM NaCl treatments.

Mitochondrial Coupling Efficiency and Energy Yield

Mitochondrial energy coupling was significantly compromised. RCR values above 6.7 under control conditions declined to ~3.9 and ~2.1 under 100 and 200 mM NaCl, respectively, marking a clear reduction in OXPHOS efficiency (Del-Saz et al., 2016). ATP production, measured at 12–14 nmol·min^{-1}·mg^{-1} protein under control, declined to 3–4 nmol·min^{-1}·mg^{-1} at 200 mM NaCl, indicating a 70% energy deficit.

Quantification via O2k Oroboros and ATP bioluminescence assays validated the energetic decline. Elevated LEAK respiration diverted electron flow from ATP synthesis, exacerbating energy loss. This metabolic inefficiency underscores mitochondrial vulnerability under ionic stress and supports the use of RCR as a diagnostic tool for energy coupling breakdown.

Transcriptomic Remodeling

RNA-seq analysis revealed robust transcriptomic shifts. AOX1a expression was upregulated 8-fold under 200 mM NaCl, serving as a biomarker for mitochondrial oxidative bypass activation (Del-Saz et al., 2016). In contrast, Complex I genes such as NDUFS1 were downregulated by over 60%, confirming electron transport inhibition.

Concomitant upregulation of CAT2, GPX7, and HSP70 indicated enhanced ROS detoxification and proteostasis support. Transcriptional regulators, including MYB and NAC factors, may underlie AOX1a induction. Bioinformatics (e.g., WGCNA, GSEA) supported co-expression of these gene clusters, revealing a mitochondrial stress module enriched in ROS-responsive elements.

Physiological Stress Indicators

Physiological markers aligned with bioenergetic and transcriptomic data. Proline levels surged from 1.2 μmol·g^{-1} FW to 20 μmol·g^{-1} FW under 200 mM NaCl, indicating osmotic adjustment (Del-Saz et al., 2016). MDA concentrations rose from 2–3 to 9–12 nmol·g^{-1} FW, reflecting lipid peroxidation.

ROS accumulation, measured via DCF fluorescence, increased over fourfold, particularly in salt-sensitive phenotypes. The K^+/Na^+ ratio dropped from ~20 to <2 under 200 mM NaCl, surpassing the critical threshold for ionic stress. This ion imbalance correlated with impaired potassium-dependent processes, contributing to overall growth inhibition.

The results of this study reveal a clear reprogramming of mitochondrial bioenergetics in Arabidopsis root cells under salt stress, characterized by a decline in oxidative phosphorylation (OXPHOS) capacity and a shift toward alternative respiration pathways. Notably, the observed

increase in LEAK respiration and the drop in respiratory control ratio (RCR) underscore a substantial reduction in mitochondrial coupling efficiency and energy output. These changes highlight the mitochondria's role as both targets and mediators of salt-induced stress.

Central to the stress adaptation mechanism is the upregulation of the alternative oxidase (AOX) pathway. AOX offers a critical electron transport route that bypasses complexes III and IV, allowing for continued respiratory activity without the proton pumping necessary for ATP synthesis. This mechanism helps reduce the over-reduction of electron carriers and minimizes the generation of reactive oxygen species (ROS) (Sweetman et al., 2020). The significant induction of AOX1a in our transcriptomic analysis supports its role in maintaining redox homeostasis and mitochondrial integrity under salinity.

This redox protection aligns with the increase in oxidative markers such as malondialdehyde (MDA) and H_2O_2 emission observed in salt-treated roots. Importantly, AOX activity appears to buffer the oxidative load, preserving mitochondrial structure and function even when canonical electron transport chain (ETC) components are impaired. This AOX-mediated response reinforces its potential as a genetic target for improving stress resilience in plants.

The interaction between ROS signaling and osmolyte production further illustrates the multifaceted nature of salt stress adaptation. Elevated proline levels in stressed plants not only provide osmotic adjustment but also serve as ROS scavengers, mitigating oxidative damage (Sweetman et al., 2020). This dual function highlights the plant's ability to coordinate protective metabolic pathways in response to mitochondrial and ionic stress, ensuring survival under adverse conditions.

Moreover, our data suggest activation of a mitochondrial unfolded protein response (UPRmt), evidenced by upregulation of chaperones such as HSP70 and proteostasis-related genes. The UPRmt, analogous to mechanisms described in animal systems, represents an essential quality control system for mitigating the accumulation of misfolded proteins within mitochondria during stress (Sweetman et al., 2020). Its induction reinforces the idea that mitochondrial stress responses extend beyond redox control to include protein homeostasis.

These findings underscore the value of integrating mitochondrial performance metrics with transcriptomic and physiological analyses to gain a holistic view of plant stress responses. The coordinated downregulation of Complex I components (e.g., NDUFS1) and the upregulation of AOX, ROS-scavenging enzymes, and stress-associated transcription factors indicate a tightly regulated stress adaptation network in Arabidopsis.

From a translational perspective, mitochondrial reprogramming under salt stress presents significant opportunities for plant breeding and genetic engineering. Enhancing AOX activity or stabilizing UPRmt elements may improve plant resilience under salinity, particularly in crop species with inherently low salt tolerance (Gouspillou et al., 2018). Additionally, precision genome-editing techniques such as CRISPR/Cas9 could be employed to manipulate specific mitochondrial and stress-responsive genes to optimize redox balance and energy efficiency under abiotic stress conditions.

In conclusion, this study provides strong evidence for mitochondrial bioenergetic reprogramming as a fundamental adaptive strategy under salt stress. The integration of respiratory data with molecular and physiological markers offers a comprehensive framework for understanding and potentially engineering salt tolerance in plants.

CONCLUSION

This study demonstrates that salt stress induces a profound reprogramming of mitochondrial bioenergetics in Arabidopsis root cells. Key findings reveal that increasing concentrations of NaCl progressively impair OXPHOS activity, reduce ATP production, and elevate proton leak and ROS emission. These bioenergetic shifts are accompanied by transcriptional reconfiguration, particularly the upregulation of AOX1a and stress-responsive genes, and by physiological changes such as enhanced proline accumulation and ionic imbalance.

The role of alternative oxidase (AOX) emerges as central in maintaining redox stability during mitochondrial impairment, facilitating continued electron flow while reducing ROS formation. This protective shift allows root cells to mitigate oxidative damage when canonical electron transport pathways are compromised. Furthermore, evidence for the activation of the mitochondrial unfolded protein response (UPRmt) supports the existence of an internal quality control mechanism to preserve mitochondrial proteostasis under stress.

Integrating high-resolution mitochondrial respirometry with transcriptomic and physiological data provides a comprehensive understanding of how mitochondrial metabolism supports stress adaptation. These findings highlight AOX, ROS detox enzymes, and UPRmt components as promising targets for genetic engineering or breeding strategies aimed at enhancing salt tolerance.

The implications of this study extend beyond Arabidopsis, offering a mechanistic blueprint for improving resilience in crops subjected to increasing soil salinity. Future research should explore the modulation of AOX and UPRmt pathways in diverse plant species and assess their impact on agronomic performance under saline conditions.

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