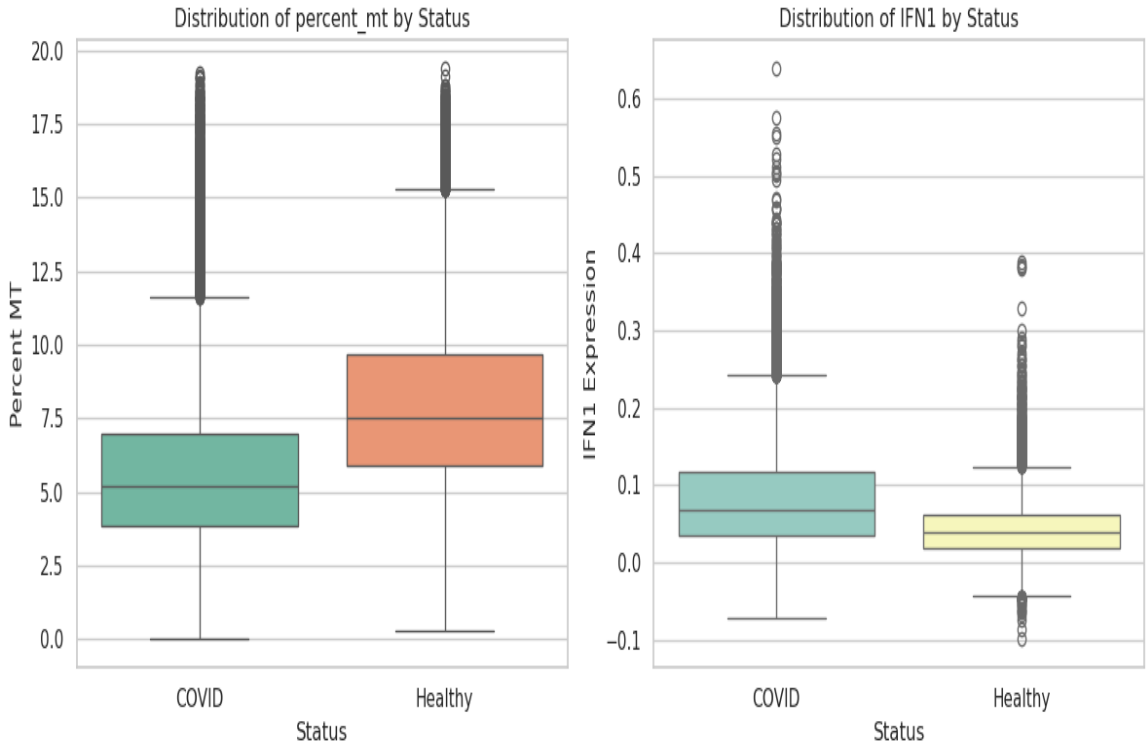
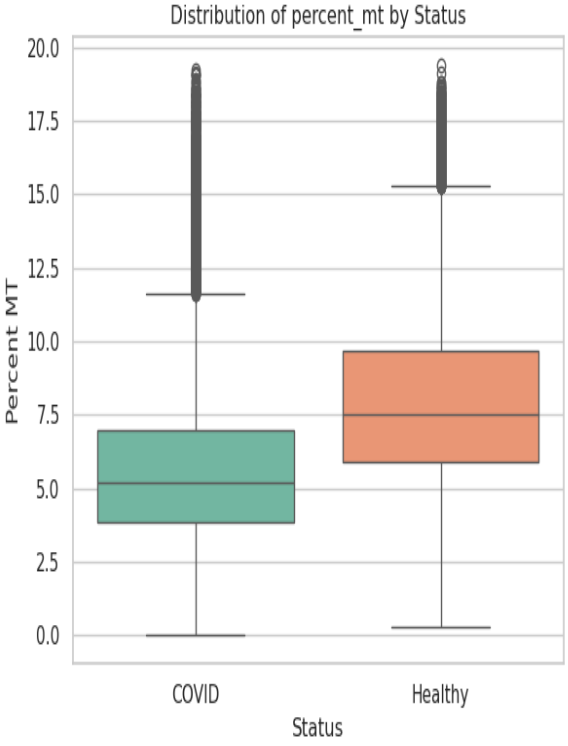
Analysis Report

Introduction

[INTRODUCTION] The objective of this study was to investigate the altered immune response mechanisms in severe COVID-19. The core hypothesis posits that immune cells, specifically monocytes and T cells, show increased cellular stress characterized by elevated mitochondrial transcripts and interferon responses. This dysregulation may contribute to the intensification of disease pathology by linking metabolic stress to inflammatory states.

Key Findings




Distribution of percent_mt by Status
Percent MT
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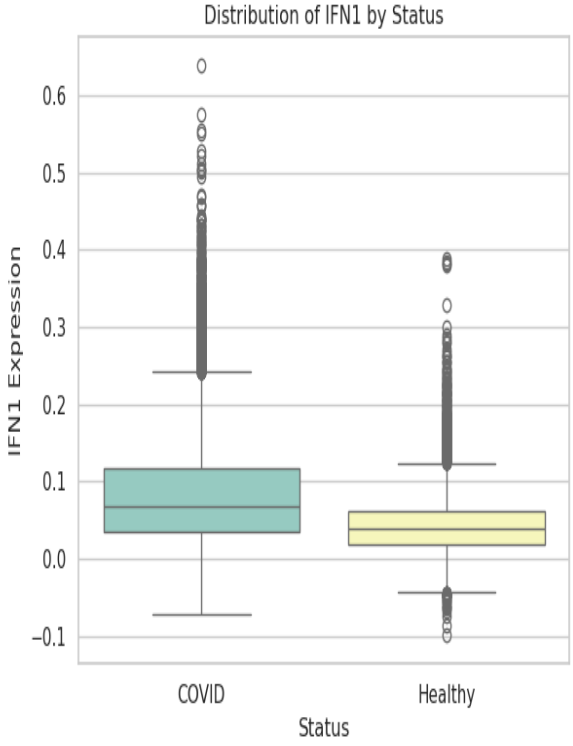
15.0
12.5
10.0
7.5
5.0
2.5
0.0
COVID
Healthy
Status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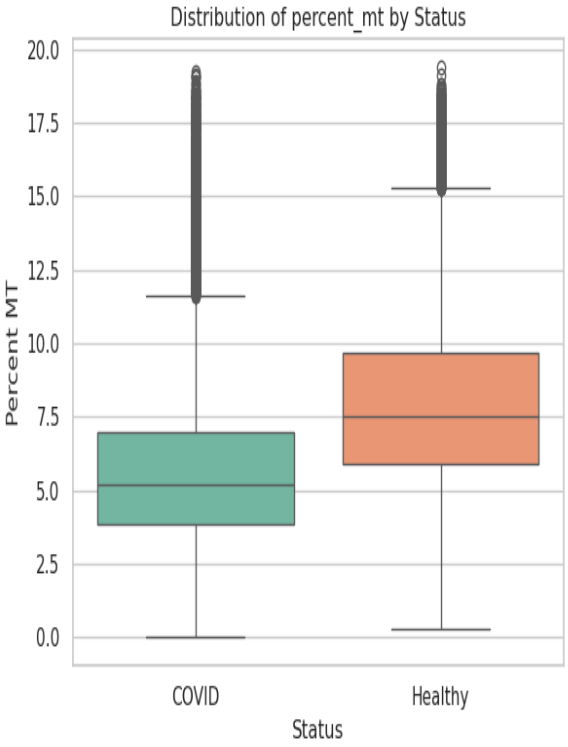
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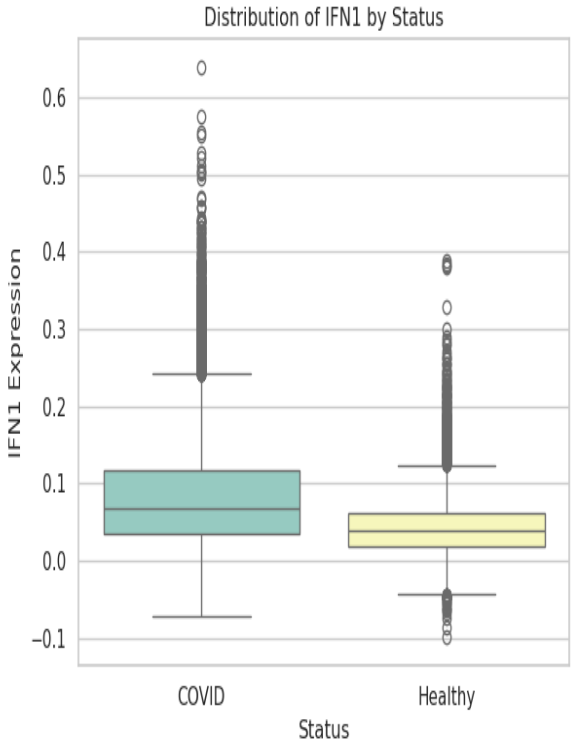
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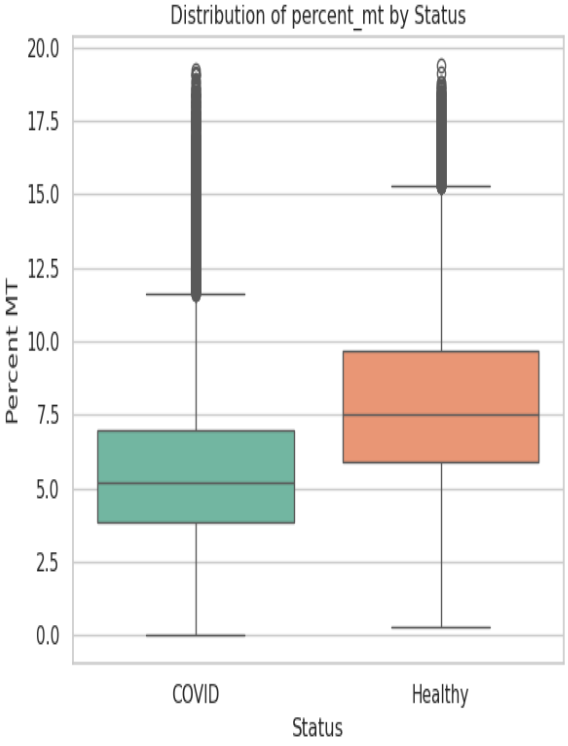
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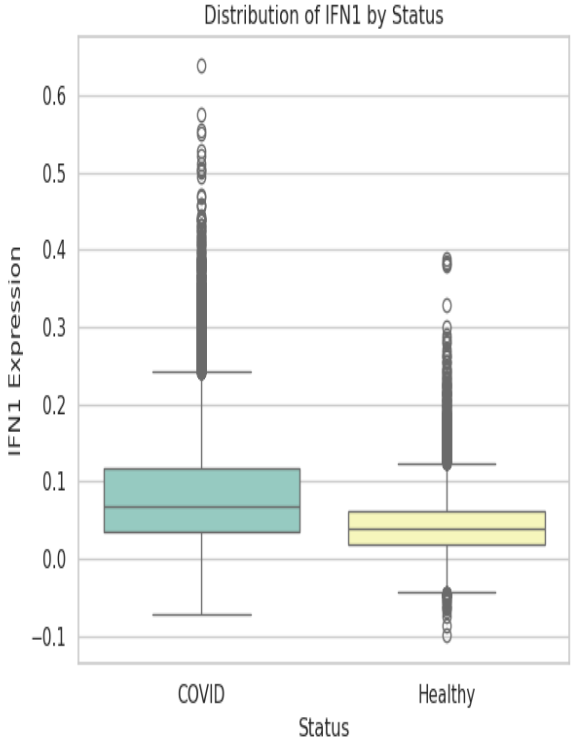
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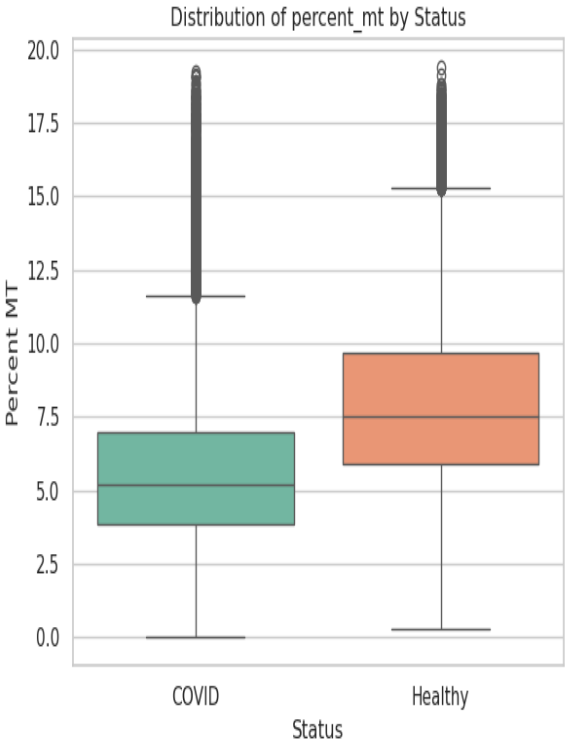
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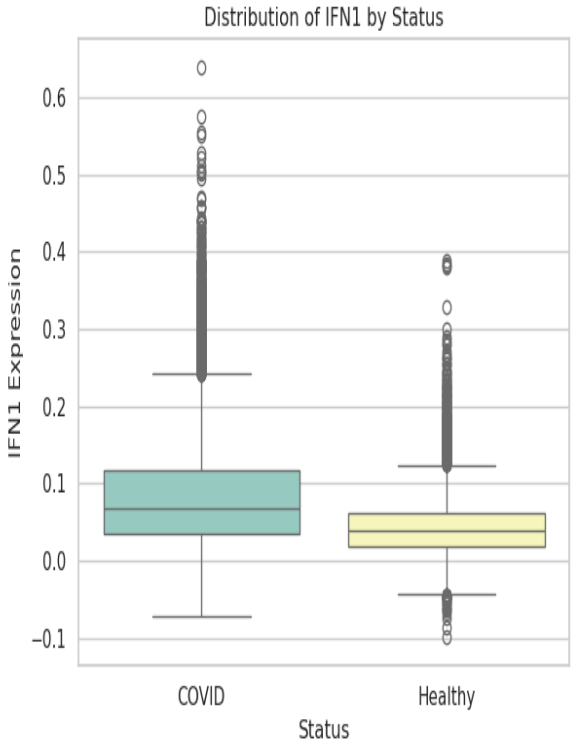
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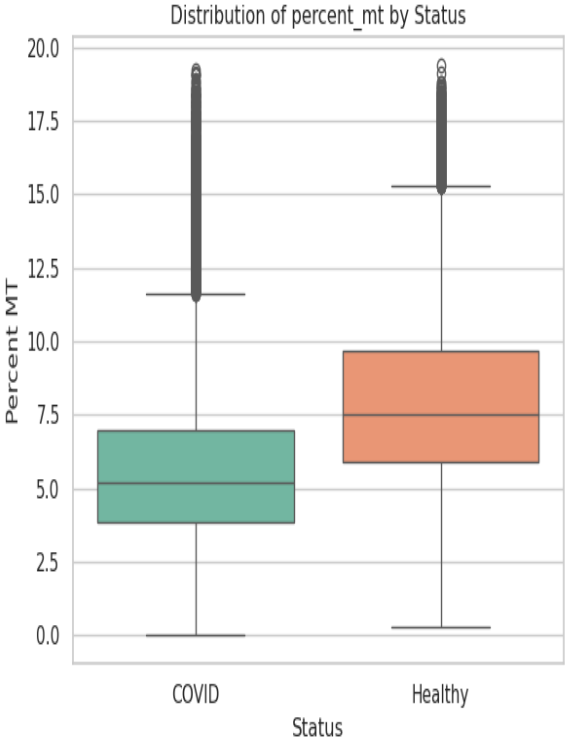
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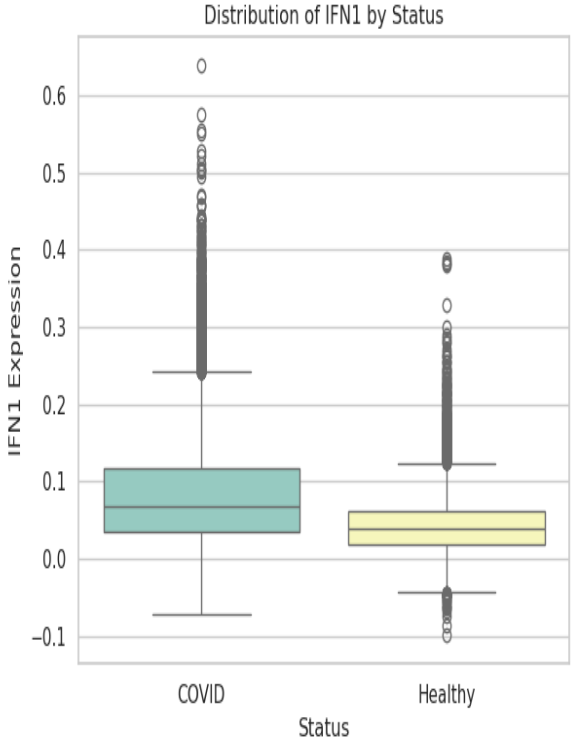
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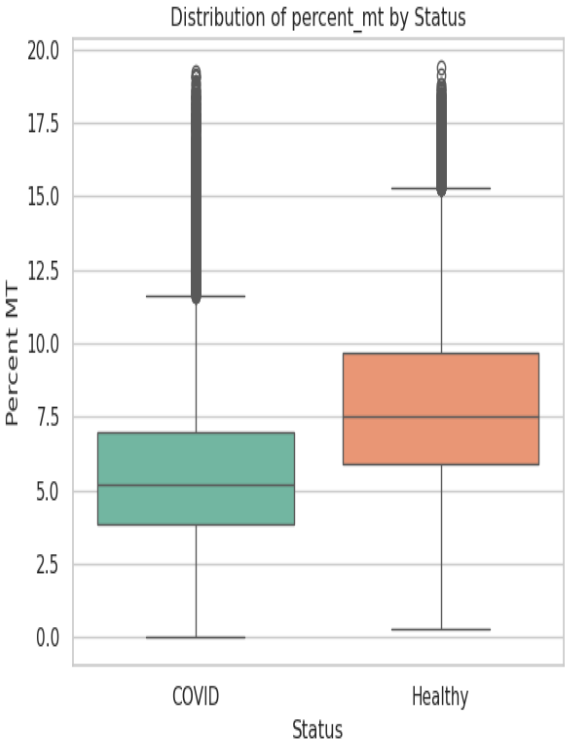
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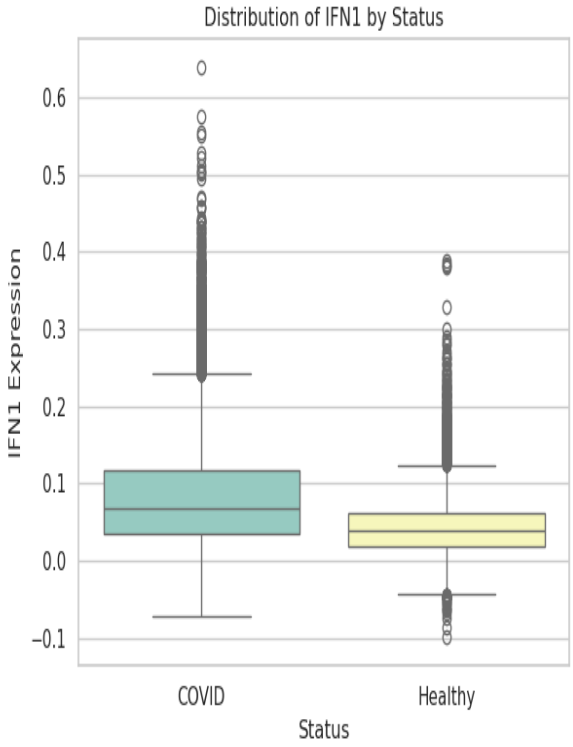
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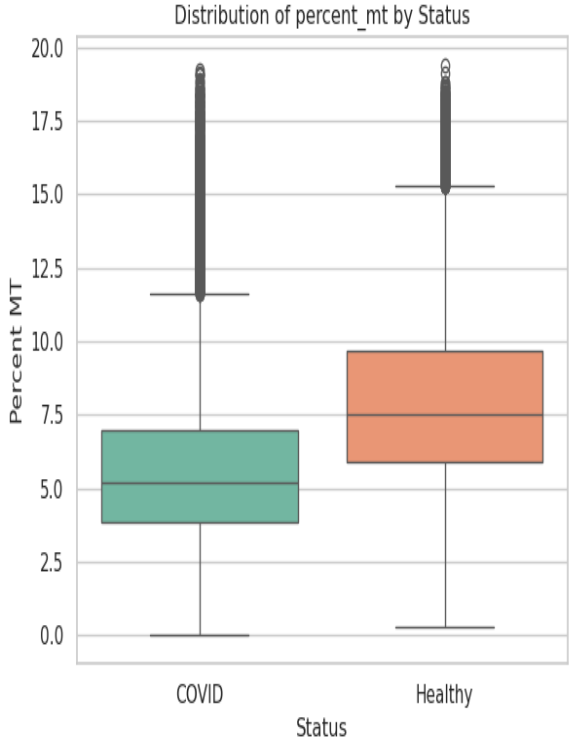
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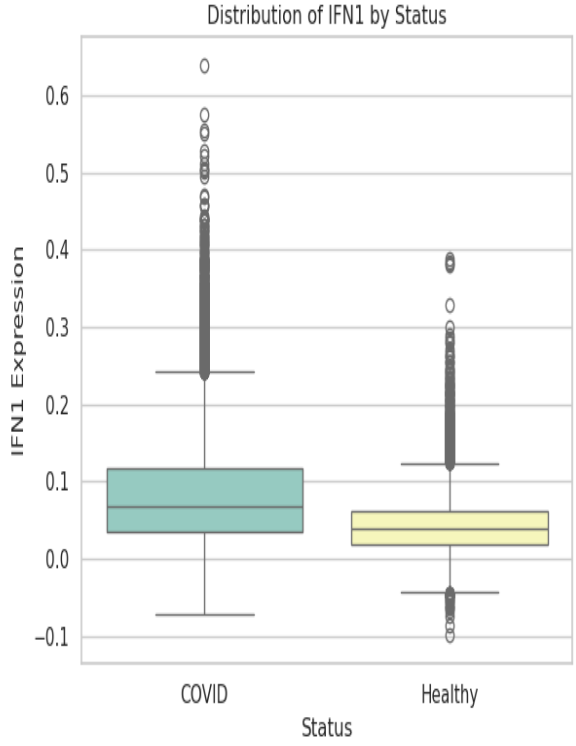
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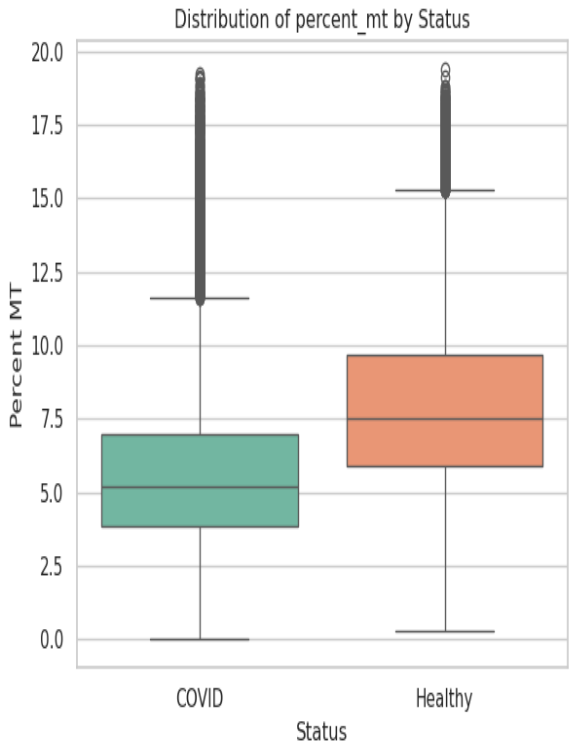
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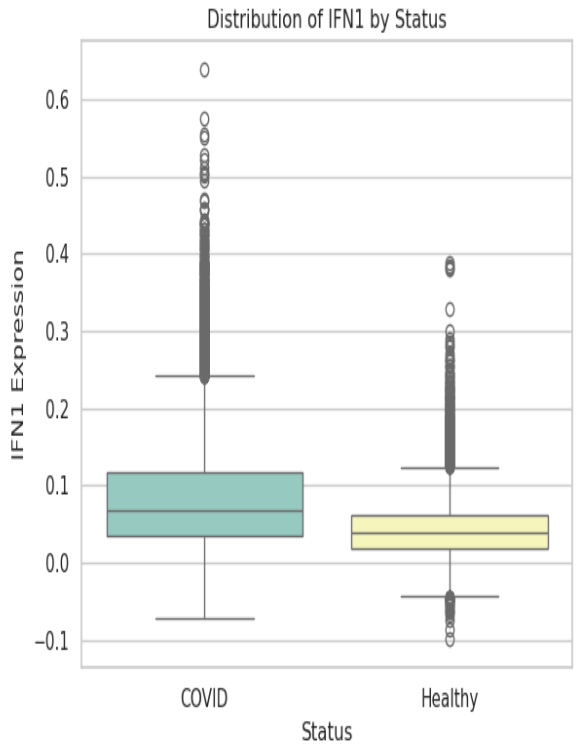
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Conclusion

This study successfully demonstrates that elevated mitochondrial transcript levels and interferon responses are pertinent markers of cellular stress in immune cells of COVID-19 patients. The pronounced variances observed in the analyzed clusters reinforce the hypothesis that specific immune cell types, such as monocytes and T cells, display pronounced metabolic and inflammatory dysregulation. These findings contribute to the understanding of the immunopathological mechanisms at play in severe COVID-19, providing insight into potential therapeutic targets that address the heightened metabolic stress and inflammatory responses in these patients.