

# The Impact of E-Cigarette Smoke on Endogenous Antioxidant Levels in Rats

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## ABSTRACT

This research work is concerned with the effect of e-cigarette smoke on the antioxidant defense system in rats, using GPx and CAT as the marker enzymes. The quantitative study investigated the effects of different nicotine concentrations (3, 6, and 9 mg) over a period of 30 days. Five hypotheses were developed to study the relationship between nicotine exposure, GPx and CAT levels, total oxidative stress, and metabolic rates. The results of these findings confirmed that high concentrations of nicotine significantly decrease GPx and CAT activity, enhance oxidative stress, and modify metabolic stability. Such an effect demonstrates the dangers from the perspective of e-cigarettes smoking and underlines a significant necessity for further scientific investigations on long-term implications from its influence on antioxidant systems and metabolic functions. Thus, the paper discusses essential biochemical consequences of cigarette use and emphasizes the role of antioxidant defenses in compensating for oxidative stress.

## 1. Introduction

This chapter discussed the influence of smoke from e-cigarettes on endogenous levels of antioxidants, such as glutathione peroxidase and catalase in rats. The work discusses the implications of this theory for understanding oxidative stress, as well as the practical implication for public health regarding e-cigarettes. The core research question investigates how e-cigarette smoke influences GPx and CAT levels, with five sub-research questions: the impact of varying nicotine levels in e-cigarette smoke on GPx levels, the effect on CAT levels, the relationship between GPx and CAT levels under oxidative stress conditions, the influence on overall oxidative stress in the body, and the implications of antioxidant level changes on metabolic rates. This is a quantitative study with the independent variables being the levels of nicotine and dependent variables being GPx and CAT levels. The paper is set out to contain a literature review, methodology, results, and conclusion. It discusses the association of antioxidant defenses and e-cigarette smoke.

## 2. Literature Review

This section discusses previously published literature that reviews how e-cigarette smoke impacts endogenous antioxidants. The five sub-research questions are categorized into this. It details the findings of how e-cigarette smoke impacts GPx and CAT levels, oxidative stress, and metabolic impact. Even with the advanced knowledge, areas still open to discussion are the little-known effects on the long run and its interactions with other factors of oxidative stress. The paper looks to fill the gaps as it formulates hypotheses from the identified relations.

### 2.1 Impact of Nicotine Levels on CAT

Initial studies focused on the short-term effects of nicotine on GPx levels, which indicated immediate responses in terms of oxidative stress but did not provide long-term data. Further studies were more comprehensive but still did not capture sustained impacts. The recent studies aim to

address this by including broader timeframes but are limited in scope. Hypothesis 1: Higher nicotine levels in e-cigarette smoke are associated with a significant reduction in GPx levels is proposed.

## **2.2 Relationship between GPx and CAT under Oxidative Stress**

Initial studies associated oxidative stress with alterations in GPx and CAT but did not dig deep into the interaction of their combination. Later studies enhanced this knowledge but did not dig out the details. Current studies shed more light but still do not provide sufficient details about these interactions. Hypothesis 3: There is an important interaction between GPx and CAT levels about the response they show due to oxidative stress caused by e-cigarette smoke.

## **2.3 Effect on Overall Oxidative Stress**

The early studies highlighted oxidative stress from e-cigarettes, focusing on short-term markers. Later research expanded the indicators but lacked the long-term assessment. Recent studies seek to close this gap but are limited by a lack of comprehensive appraisal. Hypothesis 4: E-cigarette smoke significantly increases overall oxidative stress in the body is proposed.

## **2.4 Changes in antioxidant levels and metabolic rates**

The preliminary research showed that alterations in antioxidant levels affect metabolism but did not provide detailed results. The subsequent studies extended this, but still, the metabolic effects were to be investigated in detail. Recent attempts have been made to bridge this gap, but further detailed analysis is still needed. Hypothesis 5: Alteration in GPx and CAT levels due to e-cigarette smoke has a significant effect on metabolic rates.

## **3. Method**

This part gives an account of the quantitative method undertaken to assess the impact of e-cigarette smoke exposure on GPx and CAT levels in rats. Information about data collection processes together with the variables applied ensure an effective and reliable output.

### **3.1 Data**

Data involved collecting during an experiment on 30 rats exposed to e-cigarette smoke with levels of nicotine at 3, 6, and 9 mg over 30 days. The data collected entailed GPx and CAT levels, body weight changes, and metabolic rate. The rats were set apart into two groups: the control and treatment group, and data evaluation was done applying one-way ANOVA to ensure more robust statistical assessment of the impact of different levels of nicotine.

### **3.2 Variables**

The independent variables are the nicotine levels in e-cigarette smoke: 3, 6, and 9 mg. The dependent variables include GPx levels measured in U mg<sup>-1</sup> and CAT levels measured in nmol mL<sup>-1</sup>. The control variables will be the age and weight of rats and the baseline antioxidant levels to ensure that the assessment of the effect of different levels of nicotine on antioxidant defenses is precise. Such measurements are backed by literature, allowing for credible analysis of the relationship between e-cigarette smoke exposure and antioxidant levels.

## **3 Results**

The results give an elaborate insight into the impact of e-cigarette smoke on endogenous antioxidants in rats. Descriptive statistics and regression analyses support the hypotheses developed, which depicts the effects of different levels of nicotine on GPx and CAT levels, oxidative stress, and metabolic rates.

### **4.1 Effect of Levels of Nicotine on GPx**

This result confirms Hypothesis 1, indicating that higher levels of nicotine in e-cigarette smoke significantly decrease GPx levels in rats. Data analysis from the experiment indicates that rats

exposed to 9 mg nicotine have the lowest GPx levels, at  $40.25 \pm 2.03$  U mg<sup>-1</sup>, showing a clear negative correlation between the level of nicotine exposure and antioxidant capacity. Empirical significance suggests a direct impact of nicotine on GPx activity, which is in line with theories on oxidative stress and antioxidant depletion. This result addresses previous gaps in understanding the sustained impact of nicotine on GPx, underscoring the role of e-cigarettes in oxidative stress.

#### **4.2 Impact of Nicotine Levels on CAT**

This finding supports Hypothesis 2, demonstrating that increased nicotine levels in e-cigarette smoke lead to significant reductions in CAT levels in rats. Data analysis indicates that 9 mg nicotine exposure results in the lowest CAT levels ( $2.46 \pm 0.50$  nmol mL<sup>-1</sup>), confirming the negative effect of nicotine on this antioxidant enzyme. The empirical significance reinforces theories on oxidative stress mechanisms, emphasizing the vulnerability of CAT to nicotine-induced oxidative challenges. By filling gaps in long-term exposure effects, this finding highlights the detrimental impact of e-cigarette smoke on antioxidant defenses.

#### **4.3 Relationship between GPx and CAT under Oxidative Stress**

This finding confirms Hypothesis 3, indicating a significant interaction between GPx and CAT levels in response to oxidative stress from e-cigarette smoke. The analysis shows that reductions in GPx are accompanied by decreases in CAT, indicating a coordinated antioxidant response to counteract oxidative damage. The empirical significance indicates that both enzymes function synergistically to counteract oxidative stress, in agreement with theories on antioxidant defense mechanisms. This result fills the gaps in understanding how GPx and CAT act together to protect against e-cigarette-induced oxidative stress.

#### **4.4 Impact on Overall Oxidative Stress**

This result confirms Hypothesis 4, which states that e-cigarette smoke significantly increases overall oxidative stress in rats. Analysis of oxidative markers shows that treatment groups have higher levels than controls, suggesting an increased oxidative challenge. The empirical significance emphasizes the role of e-cigarette smoke in enhancing oxidative stress, consistent with theories of free radical generation and antioxidant depletion. This finding underlines the gaps in long-term oxidative effects and thus highlights the health risks of using e-cigarettes and the importance of antioxidant defenses.

#### **4.5 Changes in Antioxidant Levels and Metabolic Rates**

This finding supports Hypothesis 5, indicating that changes in GPx and CAT levels due to e-cigarette smoke significantly affect metabolic rates in rats. Data analysis reveals fluctuations in body weight and metabolic activity, suggesting a link between antioxidant depletion and metabolic instability. The empirical significance highlights the broader implications of antioxidant changes on physiological functions, aligning with theories on oxidative stress and metabolism. By filling gaps in metabolic impacts, this finding underlines the potential health consequences of e-cigarette smoke on metabolic regulation.

### **5. Conclusion**

This study synthesized findings on the impact of e-cigarette smoke on endogenous antioxidants in rats, pointing out the negative effects on GPx and CAT levels, oxidative stress, and metabolic rates. These insights underlined the health risks associated with e-cigarette use and the importance of antioxidant defenses in mitigating oxidative challenges. However, the study has several weaknesses since it focuses only on short-term exposure to limited specific levels of nicotine; this may not

present all of the effects caused by e-cigarettes. More long-term exposure durations, variable nicotine concentrations, and other oxidative stress markers in further studies would elucidate e-cigarette impacts much more profoundly. By addressing these areas, future studies can provide comprehensive insights into the health implications of e-cigarette use and inform public health strategies to mitigate associated risks.

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