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# *In Vivo* Antioxidant Activity of Aqueous Stem Bark Extract of *Pycnanthus angolensis* Against Carbon Tetrachloride Induced Oxidative Stress in Wistar Rats

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**ABSTRACT**

Oxidative stress is involved in the pathogenesis of many chronic and degenerative diseases. This study aims to evaluate the effect of the aqueous stem bark extract of *Pycnanthus angolensis* on some oxidative stress biomarkers induced by carbon tetrachloride ($CCl_4$) in Wistar rats. Oxidative stress was induced in rats by intraperitoneal administration of 30% $CCl_4$ (in liquid paraffin) for 3 days. The animals were then treated for 11 days with either the aqueous stem bark extract of *P. angolensis* at doses of 100 or 200 mg/kg b.w., or silymarin (50 mg/kg b.w.). At the end of the treatment period, blood was collected from the rats and the serum was used to measure the levels of lipid peroxidation products, such as malondialdehyde (MDA), and of antioxidant enzymes, such as superoxide dismutase (SOD) and glutathione peroxidase (GPx). The results showed a significant increase in serum MDA levels and a significant decrease in serum antioxidant enzyme concentrations in $CCl_4$-injected rats and untreated. However, a decrease in MDA levels and an increase in SOD and GPx concentrations were observed in treated rats, with a significant effect in animals receiving the extract at 200 mg/kg b.w., and in those treated with silymarin. These results suggest that the aqueous stem bark extract of *Pycnanthus angolensis* exhibits antioxidant properties *in vivo*, enabling it to counteract oxidative stress.

**Keywords:** Oxidative stress, malondialdehyde, superoxide dismutase, glutathione peroxidase, *Pycnanthus angolensis.*

## INTRODUCTION

Oxidative stress is defined as the imbalance between oxidants and antioxidants[1]. It is a state in which the production of reactive oxygen species (ROS) exceeds the body's antioxidant capacity. This results in attacks on cellular components, such as plasma membranes, proteins and DNA, causing significant damage to cells[2,3]. Oxidative stress is involved in the pathogenesis of chronic diseases such as cardiovascular diseases, diabetes, neurodegenerative diseases, and cancer, which tend to become more prevalent with age[3-5].

The body's antioxidant system comprises a variety of endogenous molecules, including antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx)[6], as well as non-enzymatic antioxidants such as glutathione[7]. These substances neutralize free radicals, thereby protecting cells from the damaging effects of oxidative stress.

Regarding the resurgence of diseases due to oxidative stress, as well as the limitations of synthetic antioxidants, which are frequently expensive and carcinogenic[8], research into bioactive compounds of natural origin has become essential. In this context, we focused on *Picnanthus angolensis* (Myristicaceae), a plant whose bark is used to treat many diseases including dermatological infections, cough and chest pains, anemia, malaria, etc.[9] The bark is also used to treat various gynecological problems, ranging from sterility to gonorrhea[10]. Our previous study showed that the aqueous stem bark extract of this plant is able of scavenging DPPH radicals *in vitro*, with an $IC_{50}$ of 46.5 µg/mL, corresponding to an antiradical power of 2.15 µmol of DPPH scavenged per mg of extract. The total phenol content of this extract was also found to be 18.42 ± 0.36 milligrams of gallic acid equivalent per gram (mg GAE/g) of extract[11]. The aim of this study is to evaluate the effect of the aqueous stem bark extract of *Pycnanthus angolensis* on certain biomarkers of carbon tetrachloride ($CCl_4$)-induced oxidative stress in Wistar rats.

## Materials and methods

### Plant material

The plant material consists of stem barks from *Pycnanthus angolensis,* which were collected in the Agneby-Tiassa region (Southern Côte d'Ivoire). Samples of this plant were sent to the National Floristic Center at Felix Houphouët-Boigny University in Abidjan, where they were authenticated by comparison with specimens registered under the number UCJ012895.

### Animals

Wistar albino rats aged 12 to 14 weeks and weighing between 160 and 185 g were used in this study. These animals were bred at the Normal Superior School of Abidjan's animal house, where they were fed pellets every other day and had free access to water. They were also kept at room temperature, with 12 hours of light during the day and 12 hours of darkness at night. The Ethical Committee of Health Sciences of Felix Houphouët-Boigny University of Abidjan has examined and approved all experimental procedures.

### Chemicals

Silymarin, trichloroacetic acid (TCA), butyl hydroxytoluene (BHT), hydrochloric acid (HCl) were purchased from Sigma Chemical Co. (St. Louis, USA). Carbon tetrachloride ($CCl_4$) and thiobarbituric acid (TBA) were from Merck Co. (Germany). ELISA kits for measuring superoxide dismutase (SOD) and glutathione peroxidase (GPx) concentrations were provided by Elabscience® (France). All other reagents were of analytical grade.

### Extract preparation

The collected stem barks of *Pycnanthus angolensis* were shade at room temperature for three weeks and were later pulverized using a grinder. One hundred (100) grams of plant powder were dissolved in one liter of distilled water. The mixture was homogenized for 5 min using an electronic mixer. The resulting homogenate was then filtered twice on cotton and once on Whatman filter paper (3 mm). The filtrate was concentrated to dryness under reduced pressure at 30°C using a Büchi rotary evaporator[12]. The resulting extract was the aqueous stem bark extract of *Pycnanthus angolensis*, which was stored at 4°C for later use.

### Acute toxicity test

The acute toxicity of the aqueous stem bark extract of *Pycnanthus angolensis* was assessed in accordance with Organization for Economic Cooperation and Development (OECD) guideline no. 423[13]. It is a method which uses predetermined doses of 5, 50, 300 and 2000 mg/kg of body weight (b.w.), and enables a range of values for the lethal dose 50 ($LD_{50}$) to be determined.

Thus, rats were divided into two groups of three, which were then fasted overnight. The first group received the extract solution orally at a dose of 2000 mg/kg b.w. in a single administration. The second group, which served as a control group, received distilled water. The rats were then deprived of food again for three hours, after which they were given access to it. They were observed regularly for 24 hours, and daily for 14 days in order for clinical signs of toxicity and death to be recorded.

### Antioxidant activity assessment

### Induction of oxidative stress and treatment of animals

Twenty-five (25) rats were divided into 5 groups of 5 rats each. Oxidative stress was induced by intraperitoneal administration of carbon tetrachloride (2 mL/kg b.w, 30% in liquid paraffin) over three consecutive days to four groups of rats (groups 2, 3, 4 and 5). Group 1 received liquid paraffin (2 mL/kg b.w.) for three consecutive days. The rats were then treated as follow:

- Group 1 (normal control) and group 2 ($CCl_4$ control) received distilled water orally for 11 days.

- Group 3 (reference control) was orally administered silymarin at 50 mg/kg b.w. for 11 days.

- Groups 4 and 5 were orally administered the aqueous stem bark extract of *Pycnanthus angolensis* at doses of 100 and 200 mg/kg b.w, respectively, for 11 days.

Twenty-four (24) hours after the last treatment, blood was collected from the rats via retro-orbital puncture and stored separately in sterile dry tubes. Blood samples were then subjected to centrifugation at 2500 rpm for 10 min, and the serum was used to measure certain markers of oxidative stress.

**Estimation of lipid peroxidation products**

The thiobarbituric acid reactive substances (TBARS) method was used for the measurement of lipid peroxidation products. In an acidic and hot environment, lipid peroxidation products such as malondialdehyde (MDA) react with thiobarbituric acid (TBA) to form a pink-colored complex (TBA-MDA), which is measured at 532 nm. To do this, 400 µL of TBA reagent was added to 100 µL of serum. The TBA reagent is composed of trichloroacetic acid (20% w/v), thiobarbituric acid (0.375% w/v), butyl hydroxytoluene (0.01% w/v), and hydrochloric acid (1 N). The reaction mixture was then incubated in a water bath at 100°C for 15 min. After incubation, the mixture was cooled in a cold water bath, and centrifuged at 3,000 rpm for 5 min. The absorbance of the supernatant was measured at 532 nm using a spectrophotometer. The concentration of TBARS in the serum samples was determined using the molar extinction coefficient of MDA ($\varepsilon = 1.53 \times 10^5\ M^{-1}\ cm^{-1}$) according to the following formula:

$$A = \varepsilon \times C \times l$$

where: A = absorbance; C = molar concentration in mol/L; $\varepsilon$ = molar extinction coefficient in $M^{-1}\ cm^{-1}$; l = optical path length in cm.

The results were expressed in micromoles per liter (µmol/L)[14].

**Superoxide dismutase assay**

The serum concentration of superoxide dismutase (SOD) was determined by the competitive Enzyme-Linked Immunosorbent Assay (ELISA) method, using a commercial SOD kit, in accordance with the manufacturer's recommendations.

**Principle**

The assay is based on a competitive reaction between the free SOD present in the serum samples and the SOD immobilized on the microtiter plate for the binding of a specific anti-SOD antibody. The higher the concentration of SOD in the sample, the less the antibody binds to the antigen immobilized on the plate, resulting in a decrease in the colorimetric signal. The intensity of the colorimetric signal obtained after enzymatic development is therefore inversely proportional to the concentration of SOD in the sample.

**Experimental procedure**

A standard range of SOD was prepared by successive dilutions from the provided standard. The standards and serum samples were placed in the wells of the pre-coated plate at a rate of 50 µL per well. Immediately, an equivalent volume (50 µL) of biotinylated detection antibody solution (anti-SOD) was then added. The plate was then sealed, and the reaction mixture was incubated at 37°C for 45 min. After the incubation stage, a cycle of three successive washes was performed by adding 350 µL of wash buffer to each well. This was followed by aspiration and drying of the well to remove unbound fractions. Next, 100 µL of horseradish peroxidase (HRP) conjugate solution were added to each well of the plate which was left to incubate at 37 °C for 30 min. After a new cycle of five successive washes, the development stage was initiated by adding 90 µL of a tetramethylbenzidine (TMB) substrate solution to each well, and incubating at 37°C for 15 min. Finally, 50 µL of diluted sulfuric acid solution (stop solution) were added to each microplate well, and the absorbance of the resulting yellow colored solution was measured at 450 nm using a microplate reader. The standards were used to construct a calibration curve, which allowed the SOD concentrations of the samples to be determined. The results were expressed as picograms of SOD per milliliter (pg/mL).

**Glutathione peroxidase assay**

The serum concentration of glutathione peroxidase (GPx) was determined by the sandwich ELISA method, using a commercial GPx kit, in accordance with the manufacturer's recommendations.

**Principle**

This method uses two specific antibodies that target different epitopes of GPx. The GPx present in the serum samples first binds to the capture antibody immobilized on the microtiter plate. A second biotinylated detection antibody then binds to GPx, forming an

immunological complex "sandwiched" between the two antibodies. The next step is for an enzyme conjugate to attach to the biotin on the detection antibody. The intensity of the color produced when a chromogenic substrate is added is directly proportional to the concentration of GPx initially present in the sample.

**Experimental procedure**

A GPx standard range was prepared by successive dilutions from the standard provided. The standards and serum samples were placed in the wells of the pre-coated plate at a rate of 100 µL per well. The plate was then sealed, and the reaction mixture was incubated at 37°C for 45 min. The plate was covered with a sealant and then incubated at 37 °C for 90 min. At the end of this step, 100 µL of biotinylated detection antibody solution were added to each well of the plate, which was sealed and incubated again at 37 °C for 1 hour. After the incubation, a cycle of three successive washes was performed by adding 350 µL of wash buffer to each well in order to remove unbound fractions. Then, 100 µL of horseradish peroxidase (HRP) conjugate solution were added to each well of the plate, and the reaction mixture was left to incubate at 37 °C for 30 min. A second washing phase was then carried out by repeating the cycle five times. This was followed by the addition of 90 µL of TMB substrate solution to each well and an incubation period of 15 min at 37°C. Finally, 50 µL of diluted sulfuric acid solution were added to each microplate well, and the absorbance of the resulting yellow colored solution was measured at 450 nm using a microplate reader. The GPx concentrations present in the samples were determined using a calibration curve constructed from the standards. The results were expressed as picograms of GPx per milliliter (pg/mL).

**Statistical analysis**

Statistical analysis of the data was performed using Graph Pad Prism 10.0 software (Microsoft, USA). Results were expressed as means ± SEM (standard error of the mean) to describe the distribution of data. Differences between means were determined using one-way analysis of variance (ANOVA), followed by Tukey's multiple comparison test. Significant differences between means were identified at the theoretical threshold of $\alpha = 5\%$.

**Results**

**Acute toxicity**

The general physical appearance and somatomotor activity of the rats remained unaffected during the observation period by oral administration of a maximum dose of 2000 mg/kg bw of the aqueous stem bark extract of *Pycnanthus angolensis*. No signs of salivation, diarrhea, agitation, torsion, convulsion or coma were observed. In addition, no deaths were recorded among these animals during the two-week observation period. Therefore, the lethal dose 50 ($LD_{50}$) of this plant extract can be estimated to be greater than 2000 mg/kg b.w.

**Effect of *Pycnanthus angolensis* extract on lipid peroxidation**

The effect of the aqueous stem bark extract of *Pycnanthus angolensis* on malondialdehyde (MDA) concentration in rats is shown in Figure 1. The results show that the administration of carbon tetrachloride ($CCl_4$) led to a significant increase in MDA concentration in untreated rats ($CCl_4$ control) (4.75 ± 0.44 µmol/L), compared to normal control rats (2.59 ± 0.19 µmol/L). Treatment of rats with the plant extract and silymarin resulted in a decrease of the MDA level in the corresponding groups compared to that of the intoxicated and untreated group ($CCl_4$ control). This decrease was significant ($p < 0.05$) in groups of rats treated with silymarin (2.73 ± 0.32 µmol/L), and with *P. angolensis* extract at 200 mg/kg b.w. (3.14 ± 0.33 µmol/L). The MDA concentration in these groups was comparable to that in the normal control group (2.59 ± 0.19 µmol/L). In rats that received the plant extract at a dose of 100 mg/kg b.w. (Group 4), the decrease in MDA concentration was not significant. However, the value obtained (3.29 ± 0.27 µmol/L) was within the range of that observed in normal control rats (Group 1).

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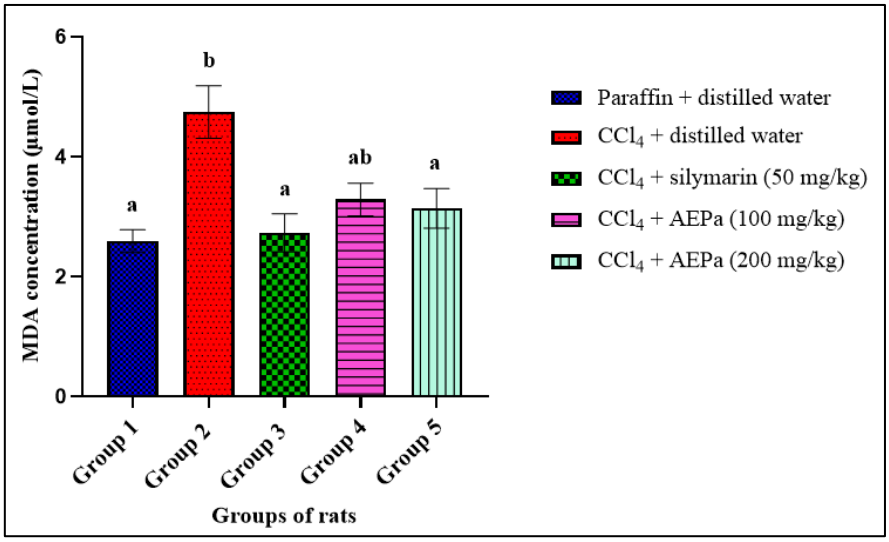


**Figure 1:** Effect of aqueous stem bark extract of *Pycnanthus angolensis* and silymarin on MDA concentration in rats subjected to oxidative stress induced by $CCl_4$.

AEPa: aqueous stem bark extract of *P. angolensis*. Values are expressed as means ± SEM (n = 5). Letters represent statistical significance. MDA concentrations with different letters are significantly different ($p < 0.05$).

**Effect of *Pycnanthus angolensis* extract on serum superoxide dismutase concentration**

Figure 2 shows the effect of the aqueous stem bark extract of *P. angolensis* on serum superoxide dismutase (SOD) concentration in rats. In $CCl_4$-injected rats and untreated, the serum SOD concentration (20.72 ± 0.986 pg/mL) decreased significantly ($p < 0.05$) compared to normal control rats (28.80 ± 0.70 pg/mL). In contrast, the concentration of this enzyme increased in the various treated groups. This increase was significant ($p < 0.05$) in rats treated with a 200 mg/kg b.w. dose of *P. angolensis* extract, where the SOD concentration (28.69 ± 0.57 pg/mL) was similar to that of the normal control rats (28.80 ± 0.70 pg/mL). Groups of rats that were injected with $CCl_4$ and then treated with either 50 mg/kg b.w. of silymarin or 100 mg/kg bw of *P. angolensis* extract did not exhibit a significant increase in SOD concentration. However, the values obtained (25.14 ± 2.08 pg/mL and 24.99 ± 2.65 pg/mL, respectively) were within the range of normal control rats (28.80 ± 0.70 pg/mL).

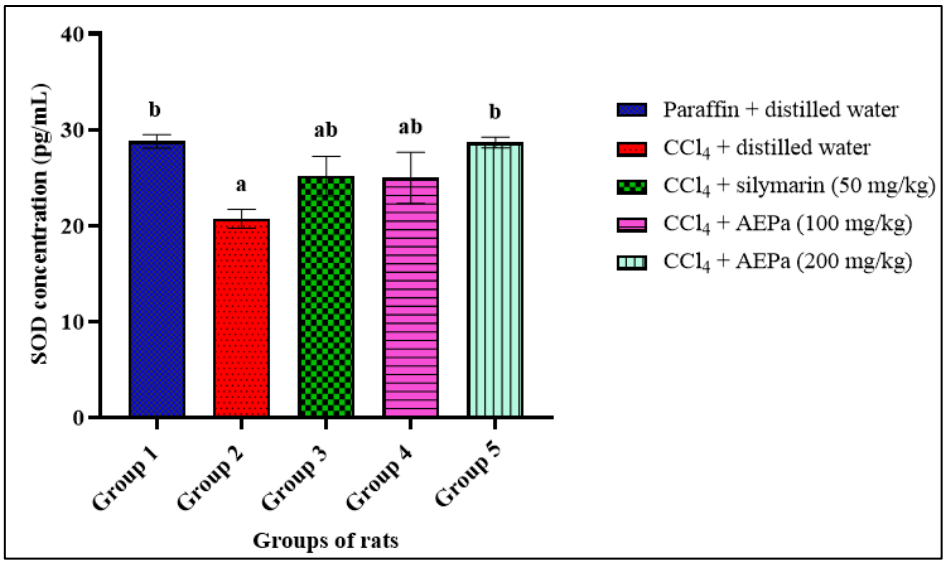


**Figure 2:** Effect of aqueous stem bark extract of *Pycnanthus angolensis* and silymarin on SOD concentration in rats subjected to oxidative stress induced by $CCl_4$.

AEPa: aqueous stem bark extract of *P. angolensis*. Values are expressed as means ± SEM (n = 5). Letters represent statistical significance. SOD concentrations with different letters are significantly different ($p < 0.05$).

**Effect of *Pycnanthus angolensis* extract on serum glutathione peroxidase concentration**

Figure 3 shows the effect of the aqueous stem bark extract of *P. angolensis* on serum glutathione peroxidase (GPx) concentration in rats. Analysis of the results revealed a significant decrease in serum GPx concentration ($p < 0.05$) in $CCl_4$-injected rats and untreated (8.71 ± 2.51 pg/mL) compared to normal control rats (16.59 ± 2.04 pg/mL). The serum concentration of this enzyme increased significantly ($p < 0.05$) in all treated groups, reaching levels comparable to those in the normal control group (Group 1). The values obtained were 16.99 ± 0.94 pg/mL, 17.59 ± 1.165 pg/mL and 18.92 ± 1.113 pg/mL, respectively, in rats treated with silymarin (Group 3), and with *P. angolensis* extract at doses of 100 mg/kg b.w. (Group 4) and 200 mg/kg b.w. (Group 5).

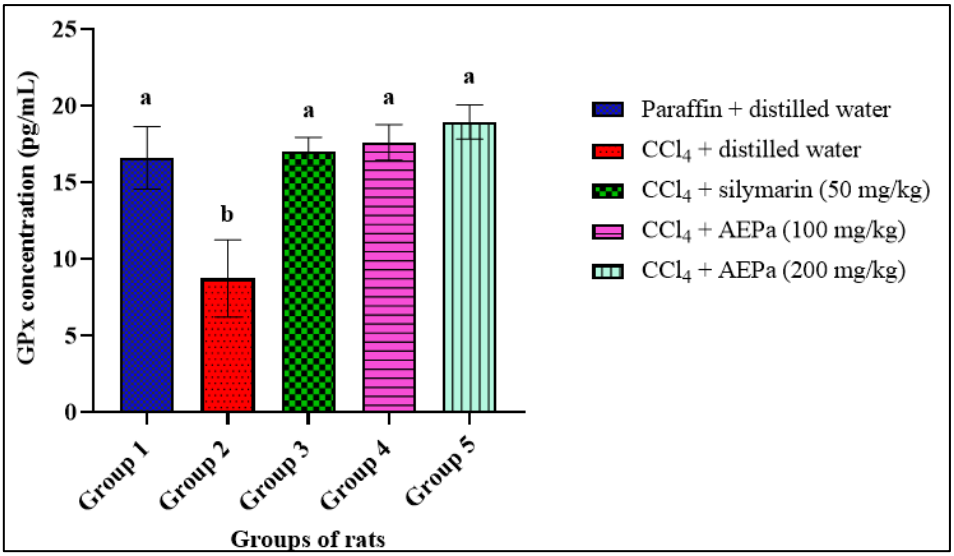


**Figure 3:** Effect of aqueous stem bark extract of *Pycnanthus angolensis* and silymarin on GPx concentration in rats subjected to oxidative stress induced by $CCl_4$.

AEPa: aqueous stem bark extract of *P. angolensis*. Values are expressed as means ± SEM (n = 5). Letters represent statistical significance. GPx concentrations with different letters are significantly different ($p < 0.05$).

**Discussion**

Various toxic substances to which the body is exposed can result in the generation of reactive oxygen species (ROS) such as superoxide anion and peroxides[15]. Increased ROS levels can cause antioxidant depletion and induce oxidative stress, a deleterious process that can damage biomolecules[15]. This process is involved in the development of many chronic diseases, including diabetes, cancer, cardiovascular diseases and neurodegenerative diseases[3-5].

In this study, the aqueous stem bark extract of *Pycnanthus angolensis* was tested for its ability to combat carbon tetrachloride-induced oxidative stress in rats. The acute toxicity study showed that the oral administration of 2000 mg/kg b.w. of this extract did not cause any clinical signs of toxicity or mortality during the 14-day observation period. The lethal dose 50 ($LD_{50}$), estimated to be greater than 2,000 mg/kg b.w., indicates that this plant extract is classified as a substance of low toxicity (category 5) according to the Globally Harmonized System of Classification of Chemicals[13].

The antioxidant activity of the aqueous stem bark extract of *P. angolensis* was evaluated using three key biomarkers of oxidative stress: malondialdehyde (MDA), a product of lipid peroxidation, and superoxide dismutase (SOD) and glutathione peroxidase (GPx), which are enzymes involved in the antioxidant defense system. SOD catalyzes the dismutation reaction of superoxide radical anion ($O_2^{\bullet -}$) to hydrogen peroxide ($H_2O_2$), which is then catalyzed into harmless oxygen ($O_2$) and water ($H_2O$) by glutathione peroxidase and catalase[6]. These parameters can be used to assess both the intensity of lipid peroxidation and the capacity of enzymatic defenses simultaneously. In fact, a high concentration of lipid peroxidation products in an organism, coupled with impaired enzymatic antioxidant capacity, indicates significant oxidative stress. Oxidative stress was induced in rats by intraperitoneal administration of carbon tetrachloride ($CCl_4$) prior to treatment. The results revealed that animals injected with $CCl_4$

but not treated showed a significant increase in MDA levels and a decrease in SOD and GPx concentrations. These observations confirm the role of $CCl_4$ as an inducer of oxidative stress via the production of hepatotoxic free radicals[16,17]. The hepatotoxicity of $CCl_4$ is due to its metabolism by cytochrome P450 (specifically CYP2E1) into the highly reactive trichloromethyl ($CCl_3$•) and trichloromethyl peroxyl ($CCl_3OO$•) free radicals, which are the primary drivers of hepatocyte injury and lipid peroxidation[16,18]. These active metabolites covalently bind to macromolecules, triggering the peroxydative degradation of polyunsaturated fatty acids in membrane lipids. This reaction leads to the formation of lipid peroxides, which affect both the permeability of hepatocyte membranes and the cellular antioxidant capacity.

Silymarin was used in this study as a reference product due to its well-established role as a natural hepatoprotective and standard antioxidant agent in pharmacological studies[19]. Its repeated administration to rats over a period of 11 days was found to significantly reduce MDA levels and restore SOD and GPx enzyme concentration. These results corroborate those of previous studies which have shown that silymarin acts by directly trapping free radicals and stimulating the production of endogenous antioxidant enzymes[20]. A significant decrease in MDA levels and a significant increase in SOD and GPx concentrations were also observed in $CCl_4$-injected rats treated with the aqueous stem bark extract of *P. angolensis* at a dose of 200 mg/kg b.w. These results suggest that *P. angolensis* extract exerts an antioxidant effect by trapping the free radicals produced by $CCl_4$ and limiting their impact. The effect of *P. angolensis* extract at a dose of 200 mg/kg bw, which is comparable to that of silymarin, could also result in stimulation of the synthesis of these antioxidant proteins. The antioxidant potential of this plant extract could be attributed to the presence of polyphenols, whose content was estimated to 18.42 ± 0.36 mg GAE/g of extract[11], and which are well-known for their antioxidant properties[21,22].

**Conclusion**

This study demonstrated that the aqueous stem bark extract of *Pycnanthus angolensis* exhibits antioxidant activity *in vivo*, reflected in its capacity to reduce lipid peroxidation and improve the antioxidant enzyme defense system in rats. This effect is dose-dependent, with notable efficacy observed in animals that received the extract at a dose of 200 mg/kg b.w.

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**Conflicts of Interests**

The authors state that they have no competing interests.

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