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Original Article

Neuroprotective Effect of Porelis, A Standardized Extract of Purple Tea (*Camellia sinensis*) Comprised of GHG in a 6-OHDA Induced Cellular Model of Parkinson's Disease

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ABSTRACT

Introduction: *Camellia sinensis* is the most frequent used plant source for making tea, which are categorized as green, white, yellow, oolong, black and dark, among them, purple tea is a new variant of green tea developed in Kenya, it is believed that purple tea has greater health advantages than green tea because of its unique composition, which includes significant concentrations of anthocyanins and anthocyanidins. **Objectives:** This study aimed to evaluate the protective effect of Porelis, a standardized purple tea extract against oxidative stress and apoptosis caused by 6-Hydroxydopamine (6-OHDA). **Materials and Methods:** To evaluate the vitality of the cells, the MTT test was employed, to create an *in vitro* Parkinson's disease model, SH-SY5Y cells were treated with 60 µM of 6-OHDA for 24 hr. The levels of antioxidant enzymes, anti-inflammatory mediators and apoptotic markers were evaluated using western blotting. **Results:** The Porelis pretreatment significantly increased the cell viability of damaged SHSY-5Y cells stimulated with 6-OHDA and restored the levels of antioxidant enzymes including Superoxide Dismutase (SOD) and Catalase (CAT). The Porelis effectively lowered 6-OHDA-induced neuroinflammation by subsiding the expression levels of COX-2 and iNOS. Additionally, via downregulating the expression of Bax and cleaved caspase 3, as well as upregulating the expression of B-cell lymphoma 2 (Bcl-2), the Porelis dramatically inhibited 6-OHDA-induced apoptosis. **Conclusion:** The Porelis could be utilised to treat or prevent neurodegenerative diseases. It subsided oxidative stress and apoptosis in 6-OHDA-induced SHSY-5Y cells.

Keywords: Antioxidative enzymes, Apoptosis, Neurodegenerative disease, Neuroinflammation, Standardized extract.

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INTRODUCTION

A new variety of *Camellia sinensis* with purple colour leaves, known as purple tea, has been developed by the Tea Research Foundation of Kenya (TRFK). The purple colour of the tea is due to the presence of anthocyanin pigments in the leaves, which are not found in traditional green tea. Additionally, purple tea contains several polyphenolic compounds such as Epigallocatechin Gallate (Egcg) and Epicatechin Gallate (ECG). It has been reported that purple tea also differs from other *Camellia sinensis* varieties in that it has a lower caffeine content than green and black tea. It is currently produced and processed in Kenya in the same way that green tea is.[1,2] It has been demonstrated that purple tea has antioxidant and anti-trypanosome effects on the brain.[3] According to epidemiological findings, eating foods high in anthocyanins and proanthocyanins may lower the risk of neurodegenerative disorders.[4] Neurodegenerative disorders include Alzheimer's and Parkinson's disease affects 1 to 4% of the senior population. Parkinson's disease is characterised by dopaminergic neuron loss, which can result in a variety of motor and non-motor symptoms. Although the exact mechanism of neuronal degeneration remains unknown, research suggests that oxidative stress and Reactive Oxygen Species (ROS) may have a role.[5-7] One of the key processes influencing the cause of neurodegenerative diseases is apoptosis. Studies in humans and experiments conducted in living animals and in a laboratory, setting have suggested that apoptosis induces cell death in dopaminergic neurons in PD.[8]

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A common neurotoxin, 6-Hydroxydopamine (6-OHDA), is widely used for cell damage induction. Studies of PD using this agent have been conducted both *in vivo* and *in vitro*. 6-OHDA toxicity is associated with oxidative stress and apoptosis.[9] Several studies have used 6-OHDA to screen medicinal plants for the treatment of Parkinson's disease.[10-13] The current investigation examines Porelis's neuroprotective properties and its underlying mechanism.

MATERIALS AND METHODS

Experimental procedures

Cell culture

The SHSY-5Y cell line of human neuroblastoma cells was obtained from NCCS Pune, India. and maintained on DMEM medium with 1% penicillin-streptomycin and 10% heat-inactivated foetal bovine serum added. The cells were maintained in a CO_2 incubator at 37°C and in a controlled environment of 95% CO_2 gas.

Preparation of samples

Porelis, a standardized purple tea extract that contains 3-5% 1,2-di-O-galloyl-4,6-O-(S)-hexahydroxydiphenoyl-D-glucose, also known as GHG (Supplementary Figure S1) was obtained from Vidya Herbs Pvt. Ltd., in Bangalore, Karnataka, India. It was dissolved in 0.1% DMSO at the required concentrations.

Cell Cytotoxicity and Neuroprotection Assay

MTT assay was carried out to test the safety of Porelis using 96-well plates.[13] Each well was seeded with a density of 1×10^4 SHSY-5Y cells, which were then cultured for overnight at 37°C in a humidified incubator with 5% CO_2. Following incubation, cells were subjected to several concentrations (50-500 µg/mL) of Porelis for 24 hr. Subsequently, MTT reagent was added and the cells were incubated for an additional 4 hr. Moreover, the supernatants were extracted and substituted with 100 µL DMSO in order to dissolve the purple formazan crystals that resulted from cells that were metabolically alive. A multi-well microplate reader was used to record the absorbance of the resultant solution at 550 nm. The findings of the experiments were averaged after being run in triplicate.

Western blot

Porelis' possible defence mechanism against 6-OHDA-induced oxidative stress was analysed by western blot experiments[14] Briefly, SHSY5Y cells (4×10^5) were seeded in each well of a 6-well plate, after 12 hr of incubation the cells were exposed to two distinct Porelis doses (100 and 200 µg/mL) with or without 6-OHDA (60 µM) for 24 hr. After completion of treatment protein extraction was performed using RIPA buffer for subsequent Western blotting experiment, the sample containing 60 µg of protein from every treatment condition was separated by SDS/PAGE and subsequently transferred to PVDF membranes (Millipore Corporation). The membranes were then blocked for 2 hr at room temperature using 5% non-fat milk. Next, the samples were incubated with different primary antibodies, including CAT, SOD, COX-2, iNOS, Bax, Bcl-2, Caspase 3 and MAPK, shaking the mixture overnight at 4°C. Following three washes with TBST buffer, the membranes were treated with the anti-rabbit or antimouse IgG secondary antibody (1:8000) in TBST for 1 hr at room temperature, then washed three more times the bands were detected with an EZ-Western Detection kit. The findings were reported as a percentage in comparison to the control group, assumed to be 100%.

Statistical analysis

The three consecutive experiments were carried out and the results were represented as mean±SEM of three independent observations. The results were statistically analysed with one-way analysis of variance, followed by a Tukey test using GraphPad Prism. $p<0.01$ was statistically significant.

RESULTS

Effect of Porelis on cell cytotoxicity and Neuroprotection

The MTT assay was performed to evaluate the cell viability and neuroprotective effects of Porelis against 6-OHDA-mediated cytotoxicity in SH-SY5Y cells. Initially, the toxic dose and cell viability of SH-SY5Y cells were studied by treating them for 24 hr with various concentrations of Porelis (100-500 µg/mL). Lower concentration of Porelis slightly enhanced the cell viability, while higher concentration (500 µg/mL) exhibited significant difference in cell viability compared to control (untreated cells) (Figure 1A). Hence, further experiments utilized 100 and 200 µg/mL concentrations. In the 6-OHDA-mediated cytotoxicity 56.6% cell viability was observed following treatment of 60 µM 6-OHDA. However, 2 hr pre-treatment with Porelis (100 and 200 µg/mL) prevented 6-OHDA mediated SH-SY5Y cell death in a dose-dependent manner (Figure 1B).

Porelis, upregulated the expression levels of Catalase (CAT) and Superoxide Dismutase (SOD)

The antioxidant enzymes CAT and SOD were measured using western blot analysis in SH-SY5Y cells. 6-OHDA induced oxidative damage in SHSY-5Y cells (Figure 2C, D) resulting in decreased CAT and SOD protein expression levels. However, pre-treatment with Porelis increased the expression of CAT and SOD significantly ($p<0.001$) in SH-SY5Y cells. Thus, the findings indicated that Porelis, at the concentrations of 100 and 200 µg/mL had antioxidant effects in 6-OHDA-mediated oxidative stress in SH-SY5Y cells.

Effects of Porelis on iNOS and COX-2 protein expression

Two proteins, COX-2 and iNOS have been associated with inflammation We used western blot analysis to confirm that Porelis inhibits 6-OHDA-induced iNOS and COX-2 expression. 6-OHDA caused a significant increase in expression levels of COX-2 and iNOS whereas Porelis pre-treatment along with 6-OHDA significantly inhibited 6- iNOS and COX-2 expression in a dose dependent manner (Figure 3).

Anti-apoptotic effects of Porelis in 6-OHDA induced cellular PD model

The effect of Porelis on proapoptotic (Bax, Caspase3) and antiapoptotic (Bcl-2) factors was investigated in a cellular Parkinson's disease model. As shown in Figure 4 the findings demonstrated that treatment with 6-OHDA alone led to a significant upregulation in the proapoptotic proteins Bax and cleaved Caspase-3 levels as well as a considerable downregulation in the levels of the anti-apoptotic protein Bcl-2 than the control group. However, pre-treatment with Porelis markedly ($p<0.001$) improved the Bcl-2/Bax ratio and decreased the expression levels of cleaved caspase-3 in 6-OHDA induced SHSY-5Y cells.

DISCUSSION

Anti-trypanosome, cerebral antioxidant and pancreatic lipase inhibition are among the biological functions of purple tea extract that have been reported.[15-18] To the best of current understanding, this investigation is the first to assess the neuroprotective effect of Porelis, a standardized purple tea extract in 6-OHDA-stressed SH-SY5Y cells. *In vitro* models of human neuroblastoma SH-SY5Y cells are frequently employed in investigations of neurobiology and neuroprotective events in neuroscience. First, we demonstrated that 6-OHDA-induced cell viability damage could be prevented by Porelis. Our investigation revealed that, though 60 µM 6-OHDA reduced cell viability, Porelis pretreatment protected 6-OHDA-induced cell loss and morphological alterations. The protective effect of Porelis may be attributed to the presence of GHG and other active polyphenols and flavonoids. Our findings are comparable with the results of previously published reports.[19,20] The antioxidant property of Porelis was assessed through the evaluation of two common cellular biomarkers associated in this process CAT and SOD. In accordance with the previous reports, 6-OHDA exposure lowers the antioxidant protection of SH-SY5Y cells and the downregulation of SOD and CAT by 6-OHDA in the SH-SY5Y cells is comparable with that described by.[21] Several plant extracts and its bioactive constituents are acknowledged for hampering it; our findings showed that the antioxidative defence mechanism of SH-SY5Y cells was strongly protected by Porelis in a manner similar to previously published reports.[22,23] A key contributing component to the onset and progression of the PD-related neuronal degeneration is neuroinflammation. The occurrence and enhancement of inflammatory responses in PD depend on inflammatory mediators including iNOS and COX-2 as well as other proinflammatory cytokines like IL-6 and TNF-α. By decreasing the excessive secretion of these proinflammatory cytokines and mediators, PD-related neuronal damage can be mitigated.[24,25] Interesting to note that the levels of inflammatory

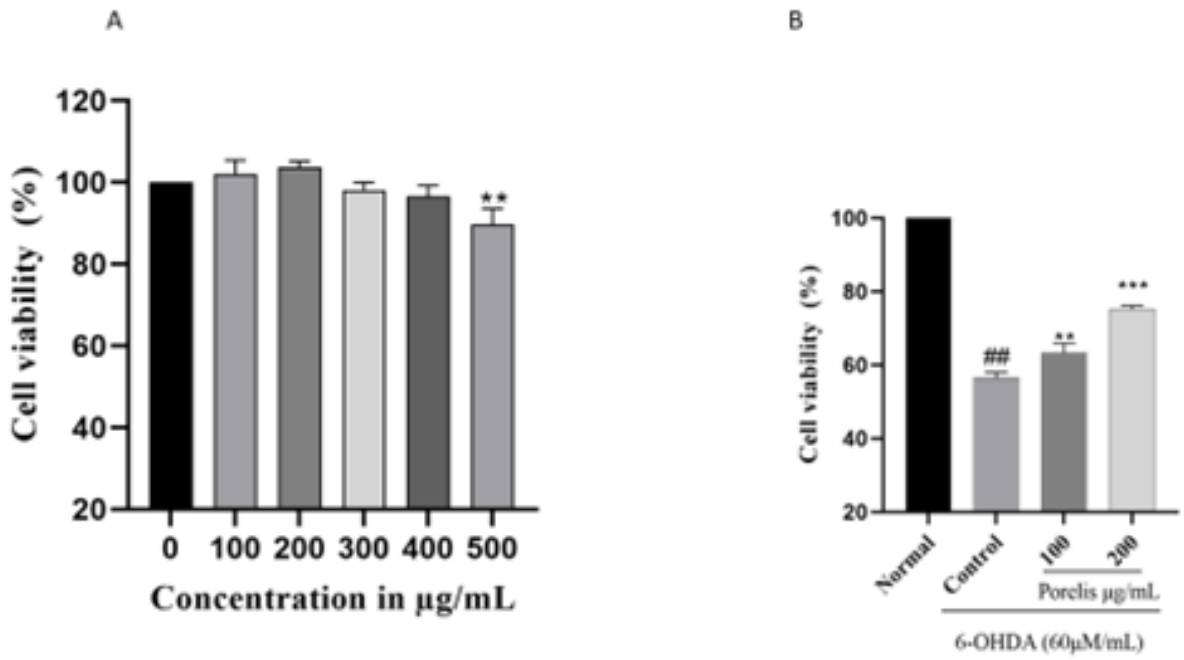

Figure 1: Effects of Porelis on cell viability of SHSY-5Ycells (A) and Neuroprotective activity against 6-OHDA neurotoxicity. (B) Data are presented as mean±SEM (triplicate experiments), one-way ANOVA followed by Tukey tests. (**$p<0.001$) compared with untreated cells and (##$p<0.001$) compared with normal (untreated cells). statistically significant (**$p<0.001$, ***$p<0.0001$) vs. 6-OHDA treated cells.

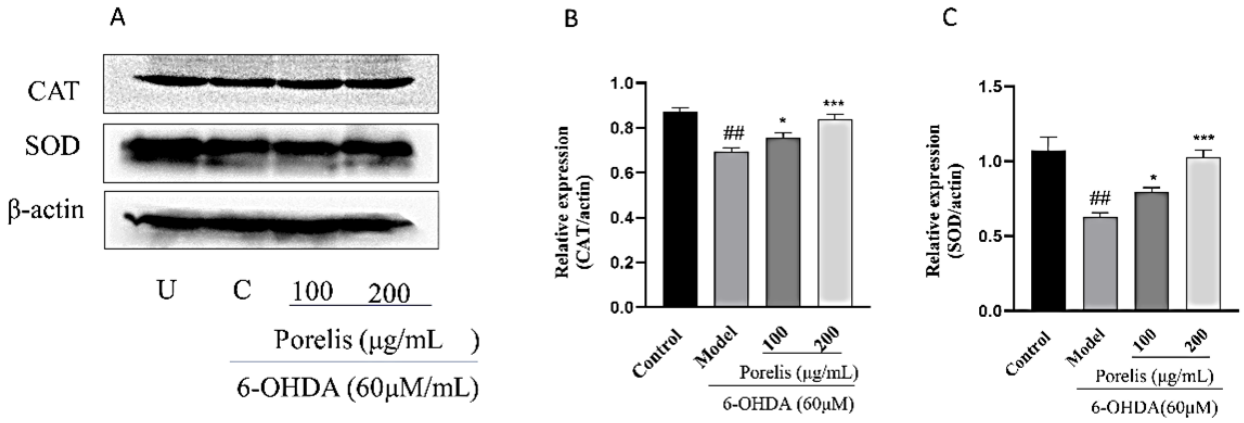

Figure 2: Effect of Porelis pretreatment on antioxidant enzymes in 6-OHDA induced oxidative stress in SH-SY5Y cells. (A) Western blot analysis of catalase and SOD-1 protein expression levels. (B, C) Densitometric quantitation. Data are expressed as mean±SE, Significance was analyzed by a one-way ANOVA followed by the followed by a Tukey test. ##*P*<0.001 versus control(untreated) cells. ∗∗*p*<0 01 and ∗∗∗*p*<0 001 versus model group (6-OHDA) alone-treated cells.

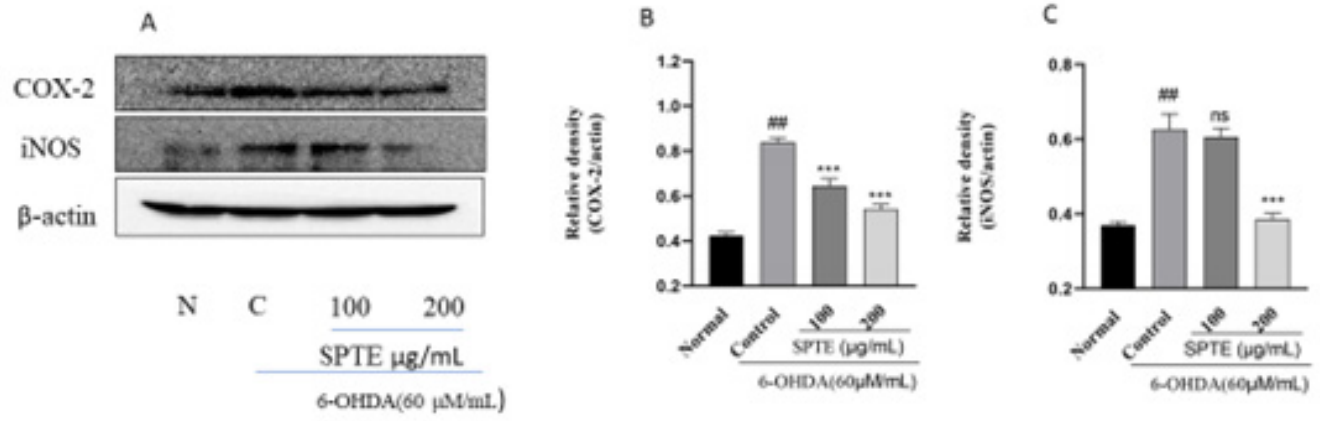

Figure 3: Effect of Porelis pretreatment on the expression of iNOS and COX-2 in 6-OHDA induced inflammation in SH-SY5Y cells. (A) Western blot analysis of iNOS and COX-2 protein expression levels. (B, C) Densitometric quantitation. Data are expressed as mean±SE, ##*p*<0.001 versus normal (untreated) cells. ∗∗*p*<0 01 and ∗∗∗*p*<0 001 versus model group (6-OHDA) alone-treated cells.

cytokines including iNOS and COX-2 were subsided in the Porelis treatment group. These findings indicate that Porelis may reduce inflammation in PD to have its neuroprotective benefits.

According to earlier studies, activation of pro-apoptotic and antiapoptotic proteins (Bax, caspases and PARP) caused by 6-OHDA to have lethal effects in several *in vitro* models. The Bcl-2 family of proteins, of which Bcl-2 and Bax are prominent members, are signalling molecules that are essential in controlling cell apoptosis. The pro-apoptotic protein Bax, on the other hand, may accelerate apoptosis by penetrating the mitochondrial membrane and subsequently changing the precise transport of ions through the membrane. The anti-apoptotic protein Bcl-2 prevents apoptosis by reducing ROS. Because of this, the Bcl-2/BAX ratio is commonly used as a prognostic indicator to assess a cell's capacity to defend against apoptosis.[26,27]

In this study, we examined whether pretreatment with or without Porelis affected the apoptosis of 6-OHDA-stimulated SH-SY5Y cells. The results revealed that pre-incubation with Porelis enhanced the ratio of Bcl-2/BAX and that exposure to 6-OHDA dramatically decreased the ratio of Bcl-2/BAX in a dose-dependent manner, which is consistent with the findings of earlier research.[28,29]

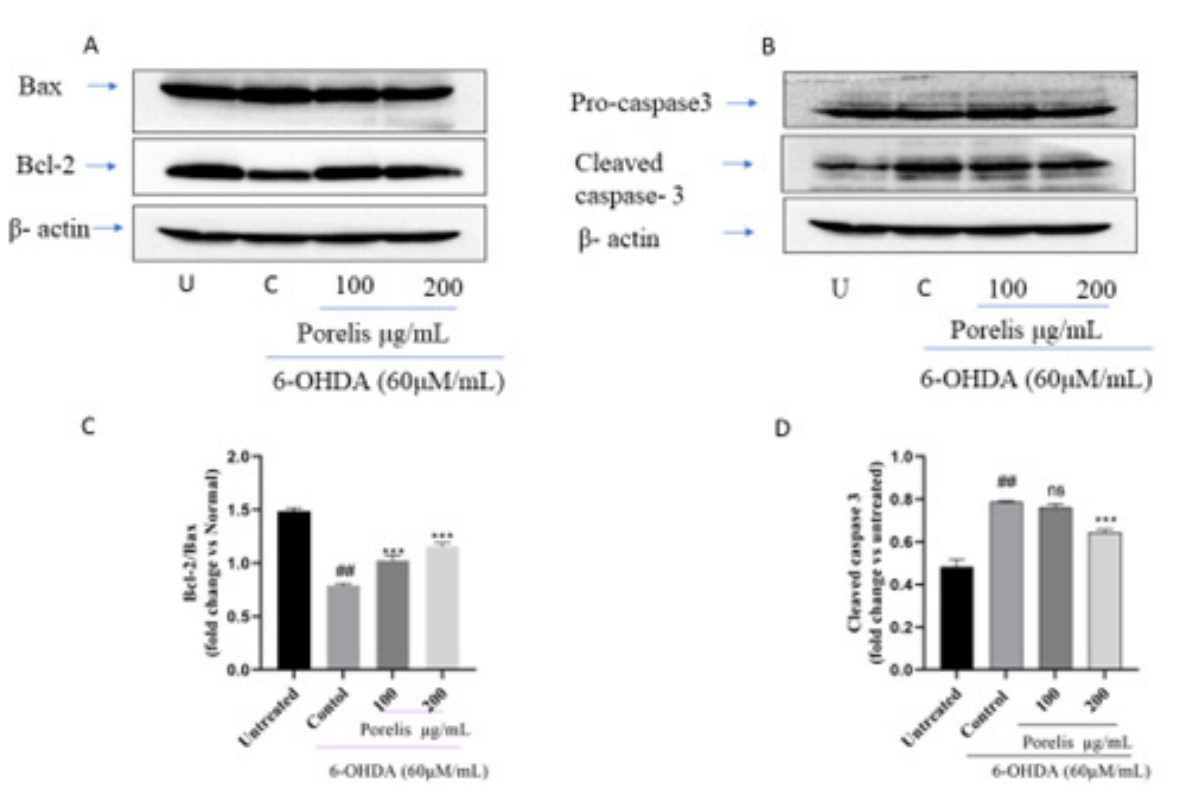

Figure 4: Effect of Porelis on the expression of apoptosis-related proteins in SH-SY5Y neuroblastoma cells. (A) Expression levels of Bcl-2, Bax, (B) caspase-3, (C, D) Densitometric analysis of three independent blots. Data are expressed as mean±SEM. ##p<0.001 versus control(untreated) cells. ∗∗p<0 01 and ∗∗∗p<0 001 versus model group (6-OHDA) alone-treated cells.

CONCLUSION

In conclusion, this study demonstrated the protective effects of standardized purple tea extract against 6-OHDA-induced Neurotoxicity through its antioxidant, anti-inflammatory and anti-apoptotic properties. These findings have important significance in the development of antioxidant, anti-inflammatory and antiapoptotic therapies for neurological disorders. Porelis, a standardized purple tea extract might be effective treatment option for neurological disorders.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

MTT: 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-2H-tetrazolium bromide; **NO:** Nitric Oxide; **TNF-α:** Tumor Necrosis Factor, **IL-6:** Interleukin 6; **COX-2: Cox-2:** Cyclooxygenase-2, **iNOS:** Inducible Nitric Oxide Synthase 6-OHDA; **CAT:** Catalase; **SOD:** Superoxide Dismutase; **ROS:** Reactive Oxygen Species; **DMSO:** Dimethyl Sulfoxide; **PVDF:** Polyvinylidene Fluoride.

SUMMARY

Purple tea extract derived from a variety of *Camellia sinensis,* is notable for its distant colour and unique phytochemical composition. This variety primarily cultivated in Kenya, contains high levels of anthocyanins, which are responsible for its purple colour.

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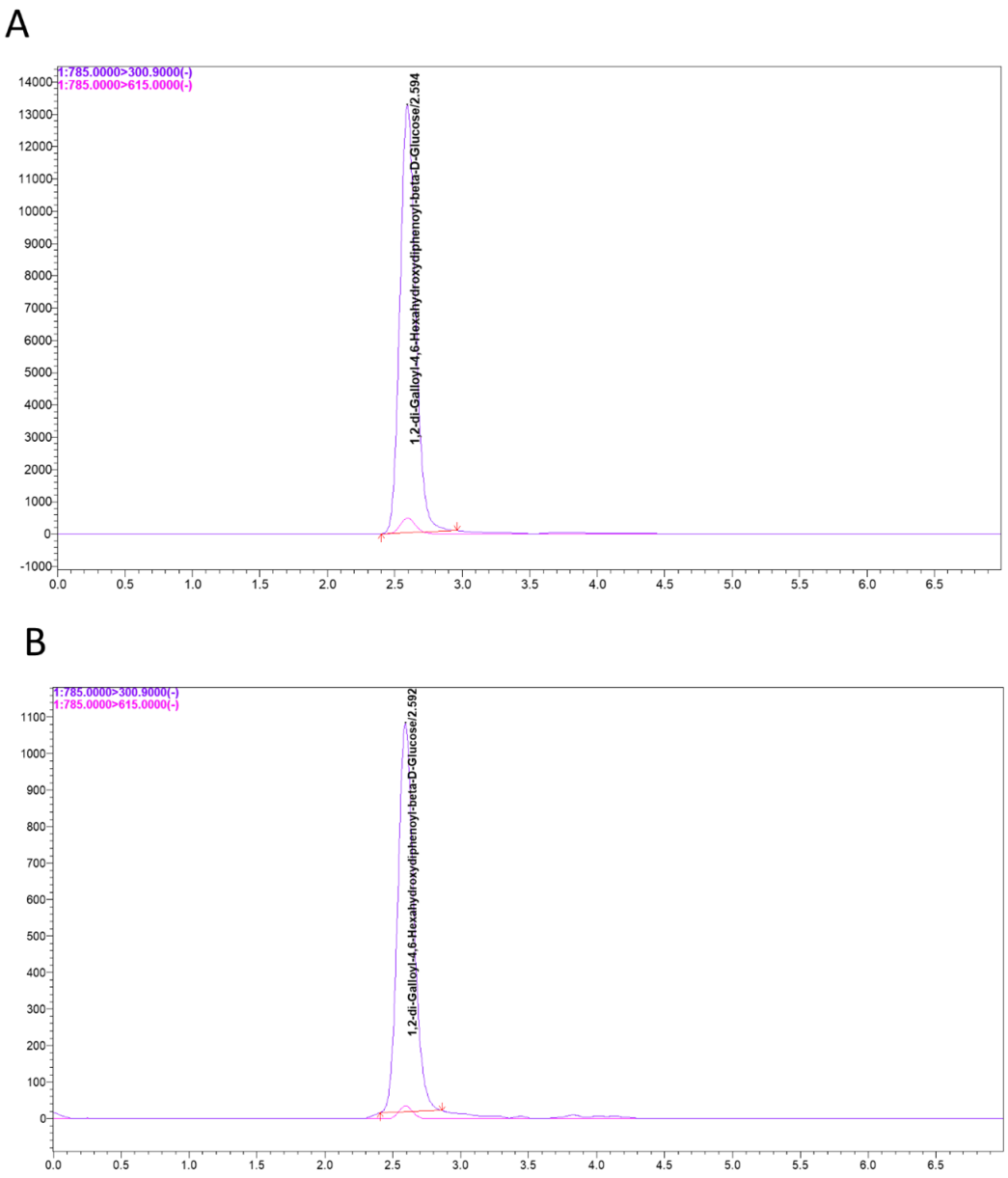

Supplementary Figure S1: HPLC chromatogram of standard GHG (A) GHG in Purple tea extract (B).