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# *In vitro* Effects of *Persea americana* Aqueous Extracts Against Oxidants and $Fe^{2+}$-Induced Oxidative Stress in Rats' Pancreas

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ABSTRACT

Various mechanisms have been reported to orchestrate the antioxidant properties of bioactive compositions of medicinal plants in the management/treatment of many diseases. In this research, we focused on the antioxidant and anti-lipid peroxidation potential of aqueous extracts of *Persea americana*. Total phenol content, total flavonoid content, ferric reducing antioxidant property (FRAP), $OH^-$ radical scavenging ability, 1, 1-diphenylpicryhydrazyl (DPPH) radical scavenging ability and anti-lipid peroxidation strength of the extracts were estimated using 5 mg/mL. Leaf and seed extracts showed the highest total phenol and flavonoid contents respectively, while the FRAP of the seed was significantly ($P < 0.05$) higher than others. $IC_{50}$ values revealed that Peel ($IC_{50}$ = 0.335 ± 0.067) has the highest DPPH potential while $OH^-$ radicals and MDA were significantly ($P < 0.05$) reduced by the leaf extract in a dose dependent pattern. Characterization of avocado leaf and fruit parts revealed varied concentrations of phenolics (Syringic acid, eugenol, vnillic acid, isoeugenol, guaiacol, phenol, kaempherol, catechin, ρ-hydroxybenzoic acid, ferulic acid, apigenin, naringenin, epigallocatechin, lupeol and epigallocatechin-3-O-gallate). Our findings showed that the aqueous extract of the leaf and fruit parts of avocado pear (*P. americana*) protected the pancreas from $Fe^{2+}$ induced lipid peroxidation *in vitro* which could be attributed to their phenolic compounds and this may be due to their antioxidant activities, $Fe^{2+}$ chelating ability, radical scavenging strength and reducing power. This research therefore suggests the use of avocado pear as a functional food for the management of oxidative stress related diseases.

***Keywords:*** *Persea americana*, lipid peroxidation, malondialdehyde, phenolic compounds, antioxidant activities.

## Introduction

Oxidative stress stems from the decrease in natural cell antioxidant capacity or an increase amount of reactive oxygen species (ROS) in organisms.[1] Free radicals have been implicated in cell degeneration through oxidation of bio-molecules like DNA, membrane lipids and proteins. This oxidation can induce various pathologies which include atherogenesis, carcinogenesis, Parkinson's disease and ageing.[2] Health disorders like arthritis, cancer, diabetes mellitus, gastric ulcers, hypertension, inflammatory diseases and neurodegenerative diseases have been emphasized by scientists to emanate from unchecked activities of ROS.[3,4] However, functional foods and nutraceutical scientists emphasize the consumption of foods naturally blessed with antioxidant phytochemicals as a remedy, due to their ability to ameliorate degenerative diseases by improving the body's antioxidant status.

Natural foods have been reported to possess phytochemicals and health improving biochemicals. Antioxidants chelates heavy metal ions, reduces singlet oxygen, nitrates and other free radicals, and suppress the lipid peroxidation, thereby reducing the risk of degenerative diseases such as cancer, diabetes and alzheimer'sdisease.[5,6] They can also react with compounds like histamine and peroxides to reduce inflammatory symptoms.[7] Vitamin E, otherwise called α-tocopherol appears to be the first line of defense against peroxidation of Polyunsaturated Fatty Acids (PUFA) contained in cellular and sub-cellular membrane phospholipids. Their antioxidant mechanism involves the breaking of free radical chain reactions as a result of their ability to transfer phenolic hydrogen to a peroxyl free radical of a peroxidized PUFA.[7] Polyphenols found in diets are called phytochemicals and they have been used as drugs. Experiments from different laboratories in the world had shown that phytochemicals can reduce the risk of cancer, possibly due to dietary fibres, polyphenols antioxidant and anti-inflammatory effects.[8] Among edible plants with health promoting phytochemicals, dindolylmethane, from Brassica vegetables (Broccoli, cauliflower, cabbage, Kale, Brussels sprout) may be useful for recurring respiratory papillomatosis tumors (caused by the human papilloma virus). The compound is being studied for anti-viral, anti-bacterial and anti-cancer properties, making it a possible anti-cancer phytochemical as taxol resistance is a major problem for cancer patients.[7] Lycopene in tomatoes is a good example that has been tested in clinical trials for cardiovascular diseases and prostate cancer. Also, lutein and zeaxanthine from vegetables may affect visual performance and inhibit macular degeneration and cataracts.[7]Important flavonoid such as quercetin which is the aglycone form of a number of other flavonoid glycosides such as rutin and quercitrin are found in citrus fruit, buckwheat and spices such as onions. Quercetin forms the glycosides quercetin and rutin together with rhamnose and rutinose respectively. These compounds and their derivatives may help to prevent some types of cancer.[9]

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*Persea americana* Mill commonly called Avocado pear belongs to the Lauracea family. *Perseaamericana* mill (Lauraceae) is a tree plant also called avocado or alligator pear and its fruit is climacteric (i.e. matures on the tree but ripens off the tree). *Persea americana* consists of fixed oil (6-10%) and protein (1.3-6%). Leaves contain volatile oil (0.5%), with methyl-chavicol, d-d pinene and parafine. Also, the leave has been shown to contain 3d isorhamnetin, luteolin, rutin, quercetin and apigenin. Seed is rich in saponins, tannins, flavonoids and alkaloids.[10] The seed of *P. Americana* (avocado seed) has diverse application in ethno–medicine, ranging from treatment for diarrhea, dysentery, toothache, intestinal parasites, skin treatment and beautification. The avocado seed oil has several health benefit e.g. for controlling human weight (especially used for obese or weight loss).[11,12] The leaves have been reported to have or possess antiinflammatory and analgesic activities.[13] The edible part (fruit) is very popular in vegetarian cuisine, making a substitute for meat in sandwiches and salads, because of its high fat content and high in valuable, health-promoting fats.[14]

In an attempt to investigate the inhibitory potential of the aqueous extracts of *Persea americana* on carbohydrate hydrolyzing enzymes in order to suggest its possible antidiabetic mechanism in our laboratory, we chose to estimate its antioxidant potential and investigate the protective role of its leaves and fruit parts (peel, flesh and seed) aqueous extracts against free radical generation and $Fe^{2+}$ induced oxidative stress in rats' pancreas *in vitro*.

## Materials and Methods

*Sample Collection*

The leaves and fruit of avocado pear (*Persea americana*) were obtained from a farm land at Ijoka, Akure and authentication of the plants were carried out by Mr. Segun of the Department of Crop, Soil and Pest Management, The Federal University of Technology, Akure, Nigeria. A voucher specimen (OAU 2360) was deposited at the Herbarium of Faculty of Science, Obafemi Awolowo University, Ile-Ife.

*Animals*

Ten adult male Wistar rats were purchased from the Animal Production and Health Department, Federal University of Technology, Akure. The animals were acclimatized for 2 weeks, during which period they were maintained on standard rodent feeds and water *ad libitum*. The handling of animals was carried out in accordance with the recommended international standard (National Research Council 1988).

*Preparation of Extracts*

Contaminants of the leaves and fruits were washed with distilled water after which the fruits were separated into three parts (peel, flesh and seed), chopped into bits and air dried. Aqueous extracts of leaves and fruit parts were later prepared by soaking the grinded samples in distilled water for 24 h at 37ºC. The mixture was filtered after which the filtrates were stored in the refrigerator for subsequent assays.

*Determination of Total Phenol Content*

The total phenol content was determined according to the method of Singleton *et al*.[15] Briefly, appropriate dilutions of the extracts were oxidized with 500 µl 10% Folin-Ciocalteau's reagent (v/v) and neutralized by 2.0 mL of 7.5% sodium carbonate ($Na_2CO_3$). The reaction mixture was incubated for 40 min at 45ºC and the absorbance was measured at 765 nm in the spectrophotometer. The total phenol content was subsequently calculated as gallic acid equivalent (mg GAE/g).

*Determination of Total Flavonoid Content*

The total flavonoid content was determined using a slightly modified method reported by Meda *et al*.[16] Briefly 0.5 mL of appropriately diluted sample was mixed with 0.5 mL methanol, 50 µL 10% $AlCl_3$, 50 µL of 1 M Potassium acetate and 1.4 mL water and allowed to incubate at room temperature for 30 minutes. The absorbance of the reaction mixture was subsequently measured at 415 nm and the total flavonoid content was subsequently calculated using quercetin as standard (mg QUE/g).

*Lipid Peroxidation Assay*

*Preparation of Tissue Homogenates*

The rats were sacrificed under mild diethyl ether anaesthesia and the pancreas was rapidly isolated and placed on ice and weighed. This tissue was subsequently homogenized in cold saline (1:5 w/v) with about 10-up-and–down strokes at approximately 1200 rev/min in a Teflon glass homogenizer. The homogenate was centrifuged for 10 min at 3000 × *g* to yield a pellet that was discarded, and a low-speed supernatant (SI) was kept for lipid peroxidation assay.[17]

*Lipid Peroxidation and Thiobarbituric Acid Reactions (TBARS)*

The lipid peroxidation assay was carried out using the modified method of Ohkawa *et al*.[18] Briefly 100 µL of SI fraction was mixed with a reaction mixture containing 30 µL of 0.1 M Tris-HCl buffer (pH 7.4), extract (0 – 100 µL) and 30 µL of 250 µM freshly prepared $FeSO_4$. The volume was made up to 300 µL by water before incubation at 37ºC for 3 hrs. The colour reaction was developed by adding 300 µL 8.1% SDS (Sodium deodecyl sulphate) to the reaction mixture containing SI; this was subsequently followed by the addition of 600 µL of acetic acid/HCl (pH 3.4) mixture and 600 µL 0.8% TBA (Thiobarbituric acid). This mixture was incubated at 100ºC for 1 hr. TBARS (Thiobarbituric acid reactive species) produced were measured at 532 nm in the spectrophotometer and the absorbance was compared with that of standard curve using MDA (Malondialdehyde).

*$Fe^{2+}$Chelation Ability*

The $Fe^{2+}$ chelating ability of the aqueous extracts was determined using a modified method of Minotti and Aust[19] with a slight modification by Puntel *et al*.[20] Freshly prepared 500 µM $FeSO_4$ (150 µL) was added to a reaction mixture containing 168 µL 0.1M Tris-HCl (pH 7.4), 218 µL saline and the extracts (0 – 25 µL). The reaction mixture was incubated for 5 minutes, before the addition of 13 µL of 0.25% 1, 10-orthophenanthroline (w/v). The absorbance was subsequently measured at 510 nm in a spectrophotometer. The percentage (%) $Fe^{2+}$ chelating ability was subsequently calculated.

*Hydroxyl Radical Scavenging Activity*

The method of Halliwell and Gutteridge[21] was used to determine the ability of the extracts to prevent $Fe^{2+}$/ $H_2O_2$ induced decomposition of deoxyribose. The extracts (0 – 100 µL) was added to a reaction mixture containing 120 µL of 20 mMdeoxyribose, 400 µL of 0.1 M phosphate buffer, 40 µL of 500 µM of $FeSO_4$, and the volume were made up to 800 µL with distilled water. The reaction mixture was incubated at 37ºC for 30 minutes and the reaction was then stopped by the addition of 0.5 mL of 2.8% trichloroacetic acid. This was followed by addition of 0.4 mL of 0.6% thiobarbituric acid solution. The tubes were subsequently incubated in boiling water for 20 minutes. The absorbance was measured at 532 nm in a spectrophotometer.

*DPPH Free Radical Scavenging Ability*

The free radical scavenging ability of the aqueous extracts against DPPH (1, 1-diphenyl–2 picrylhydrazyl) free radical was evaluated as described by Gyamfi *et al*.[22] Briefly, appropriate dilution of the extracts (0 - 600 µL) was mixed with 600 mL, 0.4 mM methanol solution containing DPPH radicals, the mixture was left in the dark for 30 min and the absorbance was taken at 516 nm. The percentage (%) DPPH free radical scavenging ability was subsequently calculated.

*Determination of Reducing Property (FRAP)*

The reducing property of the aqueous extracts was determined by assessing the ability of the extract to reduce $FeCl_3$ solution as described by Oyaizu.[23] 2.5 mL aliquot was mixed with 2.5 mL 200 mM sodium phosphate buffer (pH 6.6) and 2.5 mL 1% potassium ferricyanide. The mixture was incubated at 50°C for 20 minutes and then 2.5 mL 10% trichloroacetic acid (TCA) was added. This mixture was centrifuged at 650 rpm for 10 min. 5 mL of the supernatant was then mixed with an equal volume of water and 1 mL 0.1% ferric chloride. The absorbance was measured at 700 nm. The ferric reducing antioxidant property was subsequently calculated as ascorbic acid equivalent (mg AAE/g).

*Phenolics Characterization using Gas Chromatography*

The method reported by Kelly *et a*l. (1994)[24] was used to analyze the quality and quantity of phenolic compounds present in samples. After the phenolics were extracted from the samples using the method reported by Kelly *et al*. (1994),[24] the purified phenolic extracts

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(1µL:10:1 split) were analyzed for composition by comparison with phenolic standards (Aldrich Chemical Co., Milkwaukee, W1) and a chromatography with standards on a Hewlett-Packard 6890 gas chromatography (Hewlett-packard Corp., Palo Alto, CA) equipped with a derivatized, nonpacked injection liner, a Rtx-5MS (5% DIPHENTL-95% dimethyl polysiloxane) capillary column (30 m length, 0.25 mm column id., 0.25 µm flm thickness), and detected with a flame ionization detector (FID). The following conditions were employed PA separation; injection temperature, 230°C; temperature ramp, 80°C for 5 min then ramped to 250°C at 30°C/min; and a detector temperature of 320°C.

### Statistical Analysis

Three replicate experiments were pooled and expressed as mean ± standard deviation. A one-way analysis of variance (ANOVA) and the least significance difference (LSD) were carried out. Significance was accepted at $P \leq 0.05$.[25]

### Results and Discussion

The mechanisms behind antioxidants actions have been emphasized umpteen times to involve the scavenging of free radicals, chelation of metal catalysts, activation of antioxidant enzymes, reduction of tocopherol radicals and inhibition of oxidases.[26, 27] They have also been reported several times to prevent oxidative DNA strand breakage which could be caused by the interaction of some reactive oxygen and nitrogen species with DNA that ultimately leads to neurodegeneration and cardiovascular complications. Another interesting mechanism of these fascinating compounds is their potential to prevent protein modifications orchestrated by ROS nitration or chloration of protein building blocks.[28] Many plants are rich sources of phytochemicals, and intakes of these plant chemicals have protective role against degenerative diseases.[29] Phenolic compounds can protect human body from free radicals, whose formation is associated with the normal metabolism of aerobic cells. Their potent antioxidant activity had been attributed to the redox properties of their hydroxyl groups.[30,31] Flavonoids have antioxidant activity and could therefore lower cellular oxidative stress.[32]Polyphenols are considered to be strong antioxidants due to the redox properties of their hydroxyl groups which encode the power house of their reducing strength.[30] Although, it has been reported that natural antioxidants possess many drawbacks which include high usage level, undesirable flavor, low antioxidant efficiency, and possible loss during processing, nevertheless, their safety cannot be overemphasized because of their potential mutagenicity, carcinogenicity, teratogenicity, or other pathogenic roles.[33]

The total phenol content (mg GAE/g) and total flavonoid content (mg QUE/g) of *Persea americana* leaves and fruit parts were estimated and the results are presented in Table 1. The leaf showed highest significant ($P < 0.05$) total phenol content while the flesh was the least. Total flavonoid content of the seed was significantly ($P < 0.05$) high while the peel was the least. The antioxidant prowess of flavonoids gave them the strength to lower cellular oxidative stress while the redox potentials of the hydroxyl groups present in polyphenols contribute immensely to their antioxidant properties.[32,34] Red and white ginger have also been reported in our laboratory to possess phenolic contents with red ginger (*Zingiber officinale* roscoe) having the highest significant ($P < 0.05$) total phenol content and flavonoid content than white ginger (*Zingiber officinale* rubra).[35] These phenolic compounds are strong enough to inhibit free radical deleterious activities in human body and their formation has been associated with normal metabolisms that occur in aerobic cells. They are also capable of removing free radicals; they may chelate metalic catalysts, activate antioxidant enzymes, reduce α-tocopherol radicals and inhibit oxidases.[27]It is an established fact that phenolic compounds improve the quality of food in terms of colour modification, taste and flavor.[36]

Furthermore, antilipid peroxidation potential of *Persea americana* was investigated as shown in Figure 1 and Table 3 using a thiobarbituric Acid Reactive Substance (TBARS) called malondialdehyde (MDA) which has been established to be a prominent biomarker of oxidative stress because of its potential to cause oxidative damage.[37]*Persea americana* leaf and fruit parts aqueous extracts reduced MDA production in a dose dependent pattern with leaf showing the highest antilipid peroxidation strength.

In order to elucidate the reason behind the remarkable antilipid peroxidation potential of these aqueous extracts, we further investigated their $Fe^{2+}$chelation and Ferric Reducing Antioxidant Potential and presented as Figure 2. Our results spoke that all the extracts chelate $Fe^{2+}$ in a dose dependent pattern. However, the flesh aqueous extract exhibited the highest $Fe^{2+}$ chelation ability while the leaf had the least. It is therefore reasonable to infer that the ability of the extracts to chelate metals may delay the initiation of lipid peroxidation because these metals participate in the initiation of lipid peroxidation.[32]These extracts may contain one or two functional groups such as –OH, -SH, -COOH, $PO_3H_2$, C=O, $-NR_2$ and –S which may be responsible for their $Fe^{2+}$ chelation ability.[38, 39] It has been reported that $Fe^{2+}$ chelation potential of plant extracts is more available to put oxidative stress at bay due to the extreme reactivity of $OH^-$ radical which requires extraordinary high concentration of antioxidants to nullify their deleterious effects.[40] The ability of this aqueous extract to degrade deoxyribose was also carried out and the result is presented in Table 2 and Figure 3. Our results showed the leaf to be the most potent with the peel exhibiting the lowest deoxyribose degradation potential.

The ability of the extract to reduce DPPH was also investigated and the result is presented in Figure 4 and Table 2. The Leaf aqueous extract had the highest significant ($P < 0.05$) reducing strength while the flesh was the least. The hydroxyl groups present in the phenolics of these extracts might be responsible for this obvious reducing power.[29]

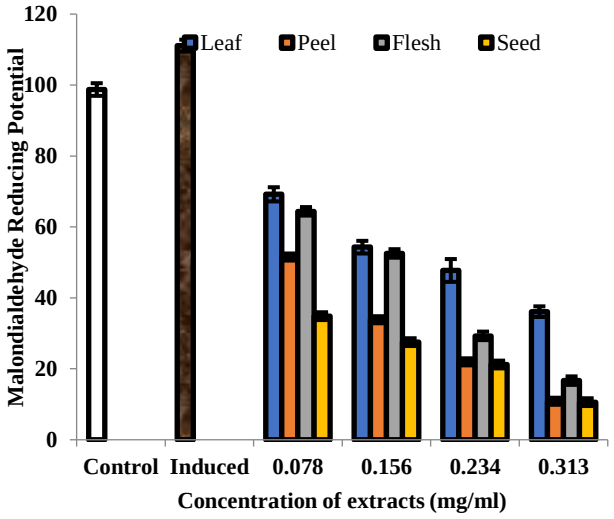


**Figure 1:** Inhibition of $Fe^{2+}$- Induced Lipid Peroxidation in rat's pancreas by aqueous extract of leaves and fruit parts of avocado pear (*Persea americana*)

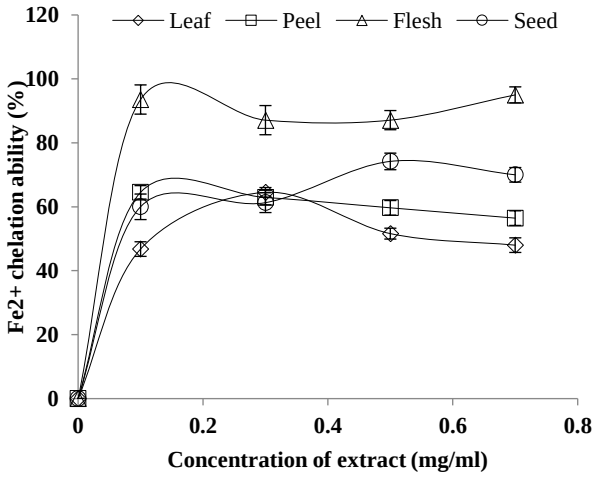


**Figure 2:** $Fe^{2+}$Chelating ability of aqueous extract of leaves and fruit parts of avocado pear (*Persea americana*).

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It has also been reported that the higher the Ferric Reducing Antioxidant Potential (FRAP) value, the higher the electron donating potential of the antioxidants in the extracts.[40] Our results also revealed the peel as the significant ($P < 0.05$) ferric reducer while the flesh aqueous extractis the least.

Table 4 showed the various concentrations of phenolics in the leaves and fruit parts of *Persea americana* when GC/MS was carried out. Medicinal plants have continued to attract attention in the global search for effective methods of using plants' parts (e.g. seeds, stems, leaves, roots and bark etc) for the management/treatment of many diseases.[41] The antioxidant prowess of *Persea americana* aqueous extract of both leaves and fruit parts may be attributed to the presence of high concentration of some phenolics as revealed by the GC/MS analysis which include syringic acid, eugenol, vnillic acid, isoeugenol, guaiacol, phenol, kaemferol, catechin, para-hydroxybenzoic acid, ferulic acid, apigenin, naringenin, epigallocatechin, epicatechin, lupeol and epi-gallocatechin-3-O-gallate.

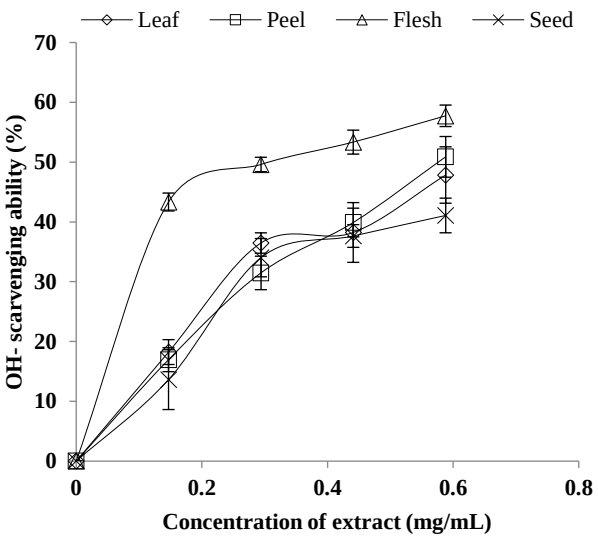


**Figure 3:** Hydroxyl radical scavenging ability of aqueous extract of leaves and fruit parts of avocado pear (*Persea americana*).

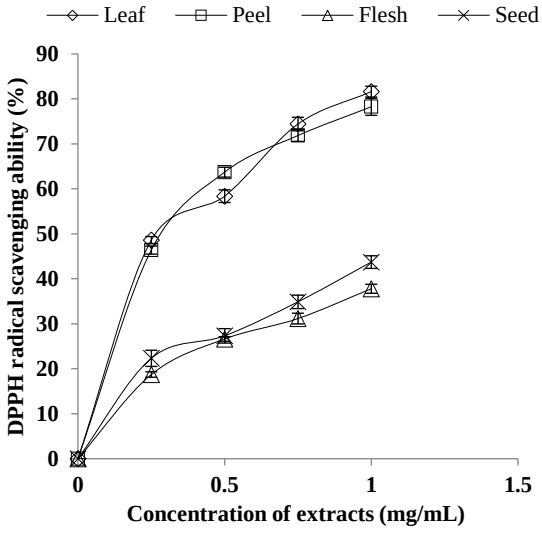


**Figure 4:** DPPH Free radical scavenging ability of aqueous extract of leaves and fruit parts of avocado pear (*Persea americana*).

**Table 1:** Total Phenol content, Total Flavonoid content and Ferric Reducing Antioxidant Property (FRAP) of leaves and fruit parts of avocado pear (*Persea americana*).

| Samples | Total Phenol (mg GAE/g) | Total Flavonoid (mg QE/g) | FRAP (mg/g) |
|---|---|---|---|
| Leaf | $21.06^{a} \pm 0.019$ | $2.04^{a} \pm 0.001$ | $182.72^{a} \pm 2.180$ |
| Peel | $19.45^{a} \pm 0.095$ | $1.99^{b} \pm 0.022$ | $239.56^{b} \pm 3.431$ |
| Flesh | $8.71^{b} \pm 0.227$ | $3.17^{c} \pm 0.002$ | $139.21^{c} \pm 4.112$ |
| Seed | $17.35^{c} \pm 0.038$ | $3.38^{c} \pm 0.001$ | $148.78^{d} \pm 2.111$ |

Values represent Mean ± Standard Deviation of triplicate readings. Values with the same superscript along the column are not significantly different ($P < 0.05$). GAE - Gallic Acid Equivalent QE - Quercetin Equivalent.

**Table 2:** $IC_{50}$ values of NO radical scavenging ability and MDA inhibitory activity by aqueous extract of avocado pear (*Persea Americana*) leaves and fruit parts.

| Samples | $IC_{50}$ (mg/mL) DPPH Scavenging activity | $IC_{50}$ (mg/mL) $OH^{-}$ Scavenging activity |
|---|---|---|
| Leaf | $0.348^{a} \pm 0.080$ | $0.595^{a} \pm 0.091$ |
| Peel | $0.335^{b} \pm 0.067$ | $1.427^{b} \pm 0.206$ |
| Flesh | $1.710^{c} \pm 0.125$ | $0.640^{c} \pm 0.096$ |
| Seed | $1.700^{c} \pm 0.192$ | $0.856^{d} \pm 0.097$ |

Values represent Mean ± Standard Deviation of triplicate readings. Values with the same superscript along the column are not significantly different ($P < 0.05$).

**Table 3:** $IC_{50}$ values of $Fe^{2+}$ chelating ability and MDA inhibitory potential of aqueous extract of avocado pear (*Persea americana*) leaves and fruit parts.

| Samples | $IC_{50}$ (mg/mL) $Fe^{2+}$ Chelating ability | $IC_{50}$ (mg/mL) MDA inhibitory potential |
|---|---|---|
| Leaf | $0.20^{a} \pm 0.072$ | $0.595^{a} \pm 0.091$ |
| Peel | $0.16^{b} \pm 0.036$ | $1.427^{b} \pm 0.206$ |
| Flesh | $0.08^{c} \pm 0.010$ | $0.640^{c} \pm 0.096$ |
| Seed | $0.16^{c} \pm 0.012$ | $0.856^{d} \pm 0.097$ |

Values represent Mean ± Standard Deviation of triplicate readings. Values with the same superscript along the column are not significantly different ($P < 0.05$).

**Table 4:** Key phenolics and concentration (mg/100 g) of *Persea americana* leaves and fruit parts.

| Phenolics | Leaves | Peel | Flesh | Seed |
|---|---|---|---|---|
| **Apigenin** | $6.06 \times 10^{-5}$ | 2.13 | 2.84 | $2.78 \times 10^{-4}$ |
| **Catechin** | - | 4.39 | - | - |
| **Epicatechin** | $2.84 \times 10^{-4}$ | 37.12 | 46.82 | $5.53 \times 10^{-4}$ |
| **Epigallocatechin** | $2.31 \times 10^{-4}$ | 19.29 | 34.84 | $3.06 \times 10^{-4}$ |
| **Epi-gallocatechin-3-O-gallate** | - | 13.48 | 6.03 | - |
| **Eugenol** | 21.52 | $7.22 \times 10^{-4}$ | $6.48 \times 10^{-4}$ | 14.53 |
| **Ferulic Acid** | $7.09 \times 10^{-5}$ | 8.92 | 11.07 | $1.54 \times 10^{-4}$ |
| **Guaiacol** | 12.79 | - | - | 10.13 |
| **Isoeugenol** | 12.92 | $2.07 \times 10^{-4}$ | $1.89 \times 10^{-4}$ | 10.73 |
| **Kaempferol** | 8.39 | $4.48 \times 10^{-1}$ | $8.68 \times 10^{-1}$ | 9.07 |
| **Lupeol** | $2.61 \times 10^{-6}$ | 10.89 | 19.73 | $2.64 \times 10^{-6}$ |
| **Naringenin** | $7.29 \times 10^{-4}$ | 3.20 | 1.86 | $1.25 \times 10^{-3}$ |
| **Phenol** | 8.47 | - | - | 9.20 |
| **P-hydroxybenzoic acid** | $2.51 \times 10^{-4}$ | 1.37 | 1.24 | $7.12 \times 10^{-4}$ |
| **Syringic acid** | 31.65 | $5.86 \times 10^{-4}$ | $5.61 \times 10^{-4}$ | 27.38 |
| **Vnillic acid** | 13.33 | $1.64 \times 10^{-3}$ | 14.54 | 10.46 |

## Conclusion

Our data unraveled the brain behind the traditional usage of *Persea americana* leaf extracts for the management of various kinds of diseases as it possesses high phenolic content, $OH^-$ radical scavenging prowess and malondialdehyde reducing capacity. The edible part (flesh) can serve as functional food while the peel, seed and leaves could be used as nutraceuticals for the management of oxidative stress-related diseases. Therefore, the phenolics revealed by GC/MS analysis might be contributory to the formidable antioxidant potentials exhibited by these extract in inhibiting oxidative stress and its downstream degenerative diseases.

## Conflict of interest

The authors declare no conflict of interest.

## Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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