Acta Alimentaria 52 (2023) 1, 12–26
DOI: 10.1556/066.2022.00118

AKADÉMIAI KIADÓ

Biological functionality and characteristics of Jerusalem artichoke (*Helianthus tuberosus* L.) tuber extracts

N.A. Afoakwah[1**], Y. Zhao[2] and Y. Dong[2*]

[1] Department of Food Science and Technology, Faculty of Agriculture, Food and Consumer Sciences, University for Development Studies, P. O. Box 1882, Tamale, Ghana

[2] School of Food and Biological Engineering, Jiangsu University, 301 Xuefu Road, Zhenjiang, 212013, China

ORIGINAL RESEARCH PAPER

Received: June 20, 2022 • Accepted: November 29, 2022
Published online: February 22, 2023

ABSTRACT

Jerusalem artichoke tuber extracts (JAE) bioactivity including anticancer, antimicrobial, and digestion-inhibiting properties were investigated. The findings showed that the extracts were able to inhibit cancer growth in the HT-29 colon cancer cell line (HT-29 cc cell line) in a dose-dependent form. The suppression of cell proliferation rose to about 78.05 ± 3.9 percent at a dose of 250 μg mL^{-1}. The Annexin V assay showed dose-dependent DNA fragmentation and detected late apoptotic induction in the HT-29 cc cell line. Depending on the concentration, the extract was able to stop the cell cycle in the HT-29 cc cell line at the G1 phase. Also, JAE prevented the HT-29 cc cell line growth, which resulted in programmed cell death. Additionally, the extracts are potential antibacterial agents and may inhibit lipase and α-amylase.

KEYWORDS

Jerusalem artichoke tuber, bioactivity, functional activities, apoptotic-induction, cell-cycle arrest

* Corresponding author. E-mail: ydong@ujs.edu.cn
** Corresponding author. E-mail: nafoakwah@uds.edu.gh

1. INTRODUCTION

Helianthus tuberosus L., also known as Jerusalem artichoke (JA), is a plant that yields irregularly shaped, fibrous, and coloured tubers (Long et al., 2016; Afoakwah and Mahunu, 2022). It is eaten as a vegetable salad, used as animal feed, and added to sausages as well as pastries (Praznik et al., 2002; Panchev et al., 2011; Afoakwah et al., 2015; Afoakwah and Mahunu, 2022). Besides, it has been established that JA has a variety of nutrients, bioactive compounds, and inulin (Tchoné et al., 2006; Yuan et al., 2012; Afoakwah et al., 2015; Afoakwah and Mahunu 2022). Scientifically, inulin is a known prebiotic (Afoakwah and Mahunu, 2022), and the foremost dietary fibre in JA, which has been demonstrated to cause a drop in blood glucose concentration and ensures mineral bio-activity in humans (Afoakwah and Mahunu, 2022).

Recently, the research focus is on the intake of a healthy diet rich in polyphenols contents, because polyphenols are known to possess on coprotective (Nezbedova et al., 2021), antiinflammatory (Oliviero et al., 2018), antiallergic (Ding et al., 2018), chemopreventive (Nowacka-Jechalke et al., 2018), and hepatoprotective (Sobeh et al., 2018; Elansary et al., 2020) health benefits. Also, they have lipid peroxidation (Socrier et al., 2018), digestive enzymes (Prpa et al., 2020; Ayua et al., 2021), and platelet aggregation (Bojić et al., 2019; Prpa et al., 2020) inhibition strength. More so, due to consumers' preference for healthy products, several studies are being conducted on underutilised plants to elucidate both their polyphenolic profile and biological functions. For this reason, Gowadia and Vasudevan (2000); Kazeem et al. (2013), and Nyambe-Silavwe et al. (2015) conducted a study on the inhibition potential of dietary polyphenols extracts on α amylase and α glucosidase. Furthermore, Gonelimali et al. (2018); Atef et al. (2019), and Efenberger-Szmechtyk et al. (2021) studied the antibacterial effect of plant extracts on food pathogens and wound healing, while Rahim et al. (2021) and Magalhães et al. (2021) proved the antitumour capacity of polyphenol extracts on cancer cell lines.

However, literature on Jerusalem artichoke tuber extract (JAE) is limited on its biological functionality, particularly, on the inhibition action of gastric enzymes, cancer, and pathogenic bacteria. Thus, in this study, the biological properties of Jerusalem artichoke tuber extract (JAE) to suppress the action of gastric enzymes, cancer cell lines, and harmful microorganisms, were studied. Also, the main polyphenols in JAE in charge of its biological properties were found using LC-MS.

2. MATERIALS AND METHODS

2.1. Materials and reagents

The Cell Bank of the Chinese Academy of Sciences provided HT-29 colon cancer cells and DMSO for the antitumour assay. McCoy's 5A medium and foetal calf serum were bought from GIBCO (Grand Island, New York, USA). Trypsin-EDTA was obtained from Beyotime (Haimen, China). Penicillin, Hoechst dye 33342, propidium iodide (PI), annexin V-FITC, and RNaseA were all procured from Sigma (St. Louis, USA, and Minneapolis, USA). The MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay was bought from Beyotime, Shanghai, China. *Escherichia coli* and *Staphylococcus aureus* were isolated and stored for the antimicrobial test by Jiangsu University's School of Food and Biological Engineering. 10 g of peptone, 5 g of yeast extract, 10 g of sodium chloride, 6 g of agar powder, and 1,000 mL of

distilled water were used to make Luria-Bertani (LB) broth. The instruments utilised in this test were all sterile. In the test for the inhibition of enzymes and the extraction of polyphenols, the following reagents were used: ethanol, sodium carbonate, α-amylase, methanol, 3,5-dinitrosalicylic acid, sodium hydroxide, potassium sodium tartrate tetrahydrate, phenol, sodium sulphite, glucose, monopotassium phosphate, dipotassium phosphate, pyridine gallic acid, Folin–Ciocalteu's phenol reagent, etc. (Beyotime, Shanghai, China). The olive oil and soluble starch were bought from a hypermarket in Zhenjiang, China.

2.2. Methods

2.2.1. Bioassay for the extracted Jerusalem artichoke tuber phenolics. Using a Discover® System S-Class CEM Cooperation microwave-assisted Soxhlet extraction (MASE), phenolics from homogenised Jerusalem artichoke tuber (JAT) were extracted (Afoakwah et al., 2015). In this paper, the extraction parameters employed were ethanol (60%), a microwave extraction duration of 12.31 min, a microwave degree of hotness (71.54 °C), and a microwave power of 11.18 W (Afoakwah et al., 2015). In a 50.0 mL rounded-bottomed bottle, 25 mL of 60% ethanol and 5 g of homogenised JAT were combined. After the MASE, Jerusalem artichoke tuber extract (JAE) was centrifuged at 3,500 r.p.m. for 30 min. To produce crude JAE, the top fluid was condensed using a rotating evaporator at 35.0 °C (Afoakwah et al., 2015). The crude JAE was freeze-dried and resolubilised in distilled water, and was applied to the Supelco LC-18 solid phase extraction columns (Bellefonte, PA). Methanol (20%) and methanol acidified with hydrochloric acid were used to elute the phenolic acids fraction and other bioactive components, respectively. The two fractions were combined, freeze-dried, and resolubilised in 0.25% DMSO for the antitumour, antibacterial, and digestive enzyme inhibition studies.

2.2.2. Cancer cell-line and culture. In McCoy's 5A medium containing 10 percent heat-inactivated foetal calf serum, 1 percent penicillin at 37 °C, and 5 percent CO_2 in a humidified atmosphere, the HT–29 human colon cancer cell line was grown, and measurements of cell proliferation, cell cycle, and apoptosis were made when the cells were up to 80 percent confluence density (Magalhães et al., 2021).

2.2.3. Determination of cytotoxicity of HT-29 colon cancer cells. As pronounced by Rahim et al. (2021) and Magalhães et al. (2021), the cytotoxicity was determined using MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay. Prior to treatment with various concentrations of JAE, HT-29 cc cells of 3×10^3 cells mL^{-1} were seeded in a 96-well micro-plate for 24 h. The cytotoxicity against the cancer cells was carried out by measuring the absorbance of the converted dye at 570 nm in an ELISA reader (Hercules, USA).

2.2.4. Cell cycle analysis. As proposed by Rahim et al. (2021), the HT-29 cc cell lines were seeded in 25 cm^2 Nunc flasks at a density of 0.4×10^6 cells mL^{-1} and were then treated with JAE at varying doses for 24 h at 37 °C. After treatment, cells were collected and suspended in 5 mL of cold PBS. The cell pellets were re-suspended in 3 mL of cold ethanol (70%) and were left overnight at 4 °C after being centrifuged at 500 *g* for 10 min. RNase A and propidium iodide were added after two cold PBS washes at a final concentration of 50 μg mL^{-1}. The sample was then allowed to incubate at 4 °C for 30 min before measurements were done. The cell cycle

distribution was computed using WinMDI 2.9 software (TSRI, La Jolla, CA, USA) after the cells were examined on a flow cytometer (Beckman Coulter, USA).

2.2.5. Quantification of the apoptotic ratio of HT-colon cancer cells. Using flow cytometry and the Annexin V-FITC labelling technique (Rahim et al., 2021), apoptosis was identified, after being exposed to JAE at various doses. HT-29 cc cell lines were treated with the extract, washed once in a buffer, and then re-suspended in the same buffer at a density of 10^6 cells mL^{-1} with annexin V-FITC (0.5 µL). Ten (10) µL of PI was added after 30 min of 4 °C incubation. The fluorescence of cells was analysed by flow cytometry for one hour and WinMDI 2.9 software (TSRI, La Jolla, CA, USA) was consequently used to calculate the results obtained.

2.2.6. Antimicrobial susceptibility test. *E. coli* or *S. aureus* was grown on a table shaker at 130 r.p.m. for 16–18 h at 37 °C after being injected into a liquid LB medium. *E. coli* or *S. aureus* concentrations were adjusted to 10^6 CFU mL^{-1}. After that, 1 mL of their suspension was added to a tube containing 9 mL of LB broth and was carefully mixed and incubated at 3 °C for 24 h. About 0.2 mL from the 24-h culture of *E. coli* or *S. aureus* was dispensed into 20 mL sterile LB-broth and incubated for 3–5 h to standardise the culture to 10^6 CFU mL^{-1}. Plates were inoculated within 15 min of standardising the inoculum, to avoid changes in inoculum density (Abalaka et al., 2012). The agar well diffusion method defined by Das et al. (2013) was used for the bacterial assay. Briefly, a sterile swab was used to spread 1 mL (10^6 CFU mL^{-1}) of *E. coli* or *S. aureus* culture onto an LB broth-agar plate. About 0.1 mL of JAE solution (100 mg mL^{-1}) was used to fill one 6 mm-diameter well that had been punched into the LB broth-agar aseptically, while 0.1 mL distilled water was used as a blank to fill another punched hole (6 mm). Prior to incubation, the plates were allowed to stand for an hour to allow pre-diffusion of the extract into the medium. After the incubation of the plates for 24–28 h at a temperature of 37 °C, the antibacterial potency of JAE was assessed using the zone of inhibition (mm) method. Also, the minimum inhibitory concentration (MIC) was determined as proposed by Greenwood (1989) and Usman et al. (2014).

2.2.7. α-Amylase activity inhibitory test. McCue and Shetty (2004) approach was modified for this experiment. A tube holding 1 mL of a gradient (1.0–12.0 mg mL^{-1}) concentration of JAE solutions received 1 mL of 0.02 M sodium phosphate buffer (pH 7) and 1 mL of α-amylase solution (0.5 mg mL^{-1}). The mixture was initially pre-incubated for 10 min at 37 °C and was incubated again at 37 °C for 1.5 h before 1 mL of 1% starch solution in 0.02 M sodium phosphate buffer (pH 7) was added at predetermined intervals. Afterward, 0.5 mL of dinitrosalicylic acid (DNS) reagent was added to stop the reaction. The tubes were incubated in boiling water for 5 min and then allowed to cool at room temperature. The reaction mixture was diluted with 1.5 mL of distilled water. Finally, a spectrophotometer was used to read the absorbance at 540 nm. A control sample was made using the same procedure but with distilled water instead of the extract, and the absorbance was assessed using a spectrophotometer at 540 nm. The percentage inhibition measurement of α-amylase was determined as:

$$\% \text{ Inhibition} = \left[\frac{\text{Abs}_{\text{control}} - \text{Abs}_{\text{extracts}}}{\text{Abs}_{\text{control}}}\right] \times 100. \quad (1)$$

2.2.8. Lipase activity inhibitory test. A modified technique of Gowadia and Vasudevan (2000) was used for the lipase inhibition study. Briefly, 0.5 mL of JAE solutions were placed in a tube along with 1 mL of lipase solution (0.5 mg mL^{-1}) in 0.02 M sodium phosphate buffer (pH 7). At 37 °C, 1 mL of olive oil was added, agitated for 5 min, and then incubated for 2 min. After that, 1 mL of ethanol was added to stop the process. Ten millilitres of benzene was added to extract the free fatty acid. Additionally, 1 mL of copper acetate-pyridine reagent (pH 6.1) was added and the mixture was vortexed. Two layers were allowed to separate in the test tubes, and a spectrophotometer was used to measure the absorbance at 710 nm. The same process was performed to formulate the control sample, but distilled water was used in place of the extract. Finally, the inhibitory lipase activity was identified as:

$$\% \text{ Inhibition} = \left[\frac{\text{Abs}_{\text{control}} - \text{Abs}_{\text{extracts}}}{\text{Abs}_{\text{control}}}\right] \times 100. \quad (2)$$

2.2.9. LC-MS analysis of JAE. JAE (5.0 mg) was filtered through a 0.45 m micropore-membrane (PTFE, Waters, Milford, MA, USA) after being diluted in 1% formic acid in methanol (5.0 mL). A Surveyor LC Pump (Thermo Finnigan, San Jose, CA, USA), a twin-piston quaternary pump with an integrated vacuum degasser was used for the reversed-phase high-performance liquid chromatography. Using a Luna 5 C18 100 reverse phase column (150 × 2.00 mm, Phenomenex, USA) and a flow rate of 0.5 mL^{-1} JAE, phenolic compounds were separated at a column temperature of 25 °C. The mobile phase consisted of solvent A (methanol) and solvent B (10 mM ammonium acetate-water solution, pH = 3.5), while the elution conditions of 0–16 min of 95% B, 16–21 min of 25–75% B, 21–31 min of 5–95% B, 31–40 min of 5–95% B were applied. Five microlitres of injections were made using the Surveyor Autosampler (Thermo Finnigan). Electrospray ionisation (ESI) studies were conducted, employing Ion-Trap LXQ (Thermo Scientific) Mass Spectrometer. The following parameters were used to measure the negative ions: a capillary temperature of 275.0 °C, a sheath gas flow rate of 20 units, a spray voltage of 4.7 kV, and a capillary voltage of −23 V. The gas helium served as ion trap collision dampening gas with a 1 mTorr pressure setting. Xcalibur (Thermo Finnigan) was used for data processing (Fraternale et al., 2015).

3. RESULTS AND DISCUSSION

3.1. Anticancer evaluation of JAE

Following the application of various concentrations (50–250 μg mL^{-1}) of JAE for 24 h, the study assessed the impact of the JAE on cell proliferation using the MTT test. A dose-dependent decrease in cell growth that was significant ($P < 0.05$) occurred at a dosage of 250 μg mL^{-1}, with an inhibition of cell growth of about 78.05 ± 3.9 percent (Fig. 1). The finding suggests that JAE might be able to stop HT-29cc cell line proliferation, and can be linked to the JA tuber bio chemicals having cytotoxic potential (Pan et al., 2009; Baker et al., 2010; Yuan et al., 2013; Nizioł-Łukaszewska et al., 2018). Also, Zhang and Kim (2015) demonstrated that JAE showed a cell-specific toxic action in the murine macrophage (RAW 264.7) cell line and human-lung cancer epithelial (A549) cell line. They showed that an increase in JAE concentration prevented A549 cells to proliferate.

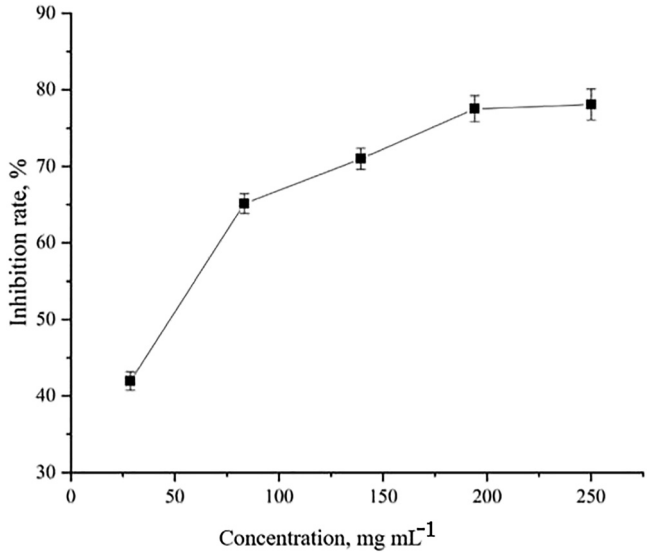

Fig. 1. JAE treatment to inhibit cell proliferation

Cell cycle research utilising PI staining was carried out to determine whether the growth-inhibitory impact of JAE on the HT-29 cc cell line was related to the cell-cycle arrest. For 24 h, JAE was applied to HT-29 cc cell line at concentrations of 0, 50 μg mL^{-1}, 150 μg mL^{-1}, and 250 μg mL^{-1}. As shown in Fig. 2, treatment with JAE led to a dose-dependent pattern of G1/S cell-cycle arrest in the HT-29 cc cell line. At the concentrations of 150 μg mL^{-1} and 250 μg mL^{-1} of JAE, respectively, and in comparison to the control, the percentage of cells in the G1-phase cells significantly ($P < 0.05$) increased by 28.2% and 31.4%. The percentage of G1-phase cells decreased, while that of the S-phase cells increased in the untreated HT-29 cc cell line. After being treated with JAE (250 μg mL^{-1}), a large percentage of HT-29 cc cell lines were still in the G1 phase, indicating that the extract may have stopped the cell-cycle progression at the G1 phase. These findings could mean that JAE caused HT-29 cc cell line to enter a G1 cell cycle arrest. Hence, apoptosis, cell proliferation, and the cell cycle activity in the HT-29 cc cell line may be controlled by JAE, which contains the three most significant bio-compounds such as chlorogenic acid, salicylic acid, and caffeic acid Fig. 5.

The impact of JAE on HT-29 colon cell death was examined using an annexin V-FITC-apoptosis detection kit. It was revealed that 68.8% of the cells were alive and non-apoptotic, and that the R4 population decreased when JAE was applied. Apoptosis-inducing cells were also seen. When JAE was administered to the HT-29 cc cell line, dead or necrotic cells were visible (Fig. 3A–D).

3.2. Antimicrobial activity of JAE

In this study, the application of 100 mg mL^{-1} of JAE was able to give inhibition diameter values of 10.1 and 11.3 mm for *E. coli* and *S. aureus,* respectively, hence this concentration was manipulated for the MIC study (Mostafa et al., 2018) (Table 1). The MIC ranged between

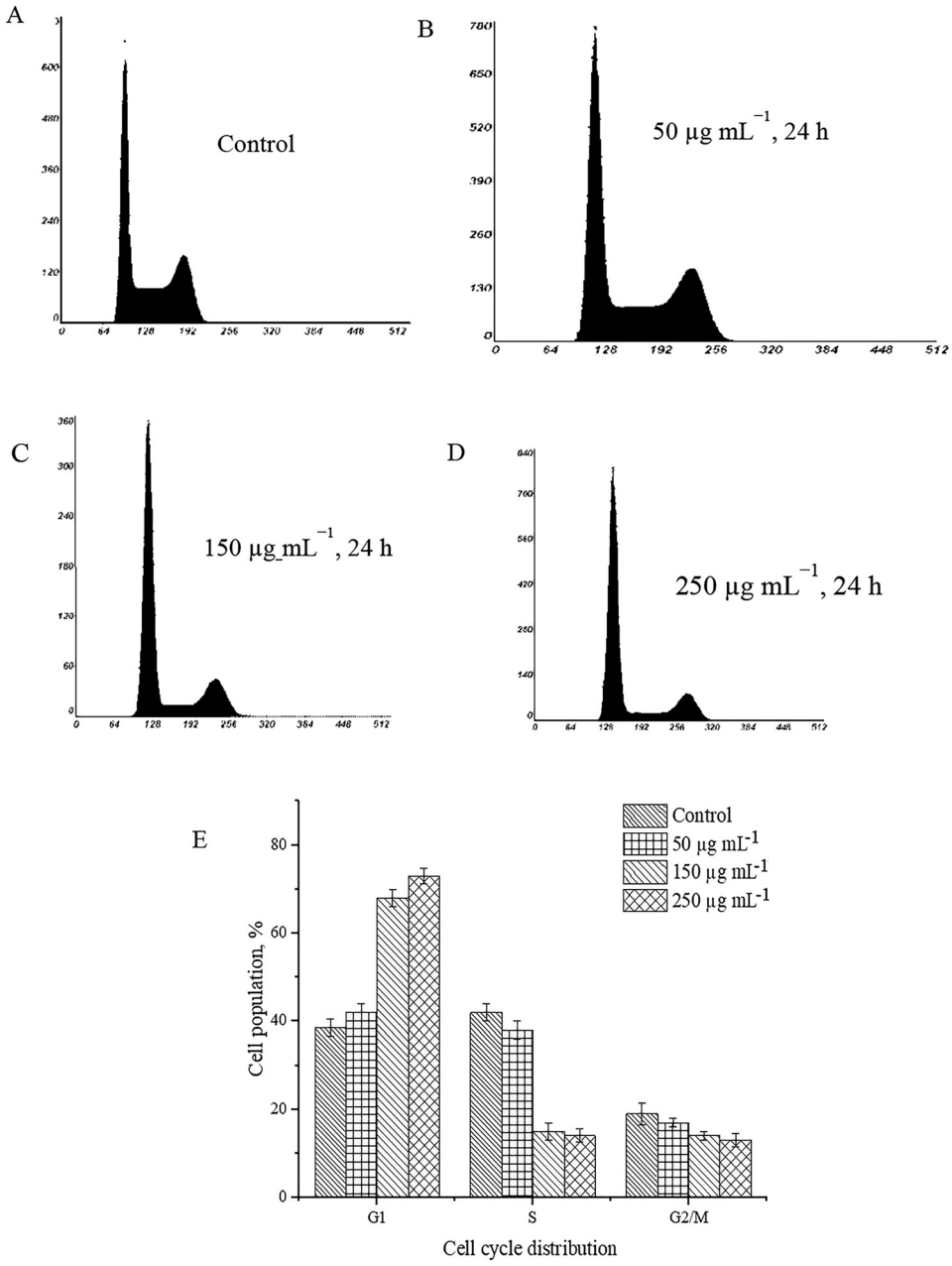

Fig. 2. JAE cell cycle arrest at G1/S phase (A): control cells for 24 h; (B): cell treated with 50 μg mL^{-1} of JAE for 24 h; (C): cell treated with 150 μg mL^{-1} for 24 h; (D): cell treated with 250 μg mL^{-1} of JAE for 24 h; (E): Cell population against cell cycle distribution. Data were expressed as mean ± standard error

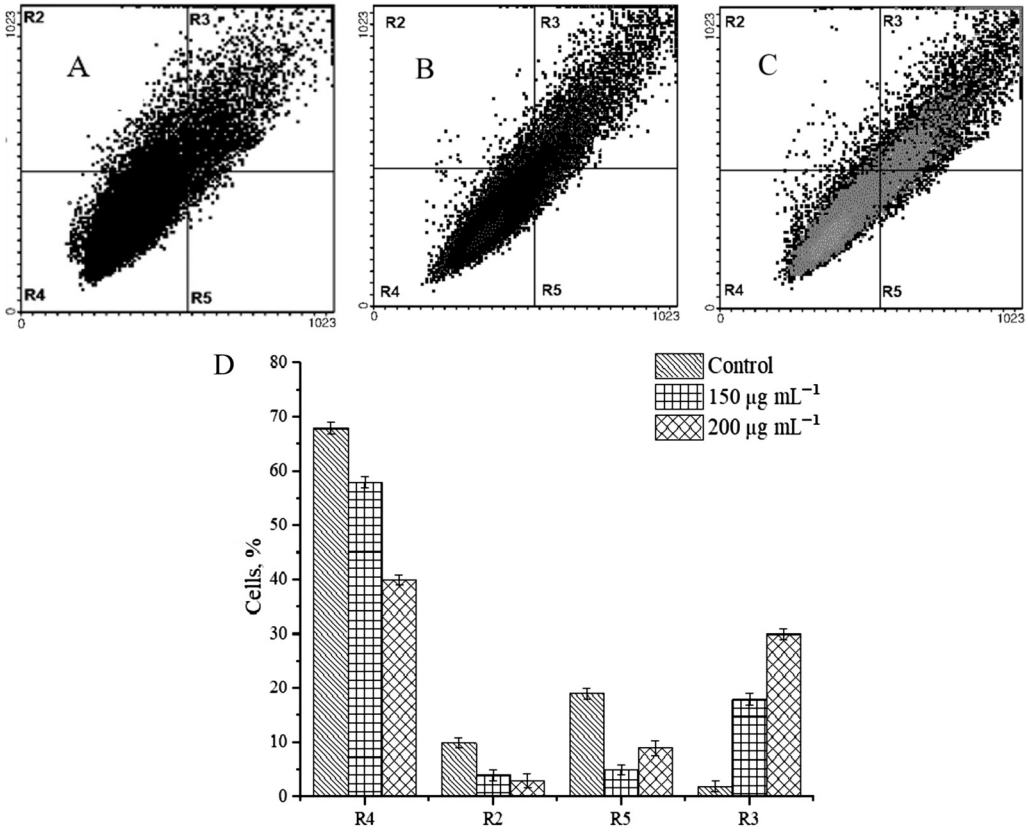

Fig. 3. The effect of JAE on apoptosis of HT-29 cells (A): HT-29 cells; (B): HT-29 cells were exposed to 150 μg mL^{-1} JAE and (C): HT-29 cells plus 200 μg mL^{-1} JAE for 24 h; Lower left (R4) quadrant: viable cells; lower right (R5) quadrant: early apoptotic cells; upper right (R3) quadrant: late apoptotic cells; (D): A graph showing cells (%) plotted against R4: viable cells; R5: early apoptotic cells; R2: debris/dead cells; and R3: ate apoptotic cells. Data were expressed as mean ± Standard error

Table 1. Diameter of bacterial inhibition ring (mm) by Jerusalem artichoke tuber extract

Sample	*E. coli*	*S. aureus*
Jerusalem artichoke tuber extract	10.1 ± 0.1	11.3 ± 0.1
Sterile water	–	–

6.30 mg mL^{-1} and 13.0 mg mL^{-1}, while JAE (100 mg mL^{-1}) was able to stop the growth of *S. aureus* and *E. coli* (Table 2). This study agrees with the conclusions that antimicrobial actions have a direct association with increasing the concentration of extracts (Gonelimali et al., 2018; Mostafa et al., 2018). The potency of the JAE to inhibit bacteria growth may be associated with the presence of bioactive phenolic composites in the extracts (Zhang and Kim, 2015;

Table 2. The result of the minimum inhibitory concentration (MIC) test

Concentration of JAE (mg mL^{-1})	*E. coli*	*S. aureus*
100	–	–
50	–	–
25	+	+
13.0	+	+
6.3	++	++

–: no growth or weak growth, +: growth; ++: enhanced growth; JAE: Jerusalem artichoke tuber extract.

Gonelimali et al., 2018; Michalska-Ciechanowska et al., 2019; Simonetti et al., 2020). Gonelimali et al. (2018) and Simonetti et al. (2020) reported that crude extracts of some herbs and plants displayed antibacterial properties against a wide range of Gram-positive and Gram-negative bacteria due to the presence of phenolic compounds.

3.3. Inhibition of α-amylase activity by JAE

One of the human enzymes responsible for converting starch to simple sugars is α-amylase. As a result, the inhibitors of α-amylase can slow down the absorption of glucose and delay the digestion of carbohydrates, hence, the postprandial rise in plasma glucose may be reduced, thereby improving glucose tolerance in people with diabetes. In a concentration-dependent way, JAE was able to cause a strong inhibition against α-amylase (Fig. 4A and B). According to the results of this study, JAE may help regulate postprandial hyperglycaemia by preventing the release of α-amylase. Hence, JAE can help in the management of diabetes mellitus. In a study, Aslan et al. (2010) used JAE and found that diabetic rats given streptozotocin showed a higher response to JAE than normal rats. Additionally, other plant extracts and JAE have been shown to suppress the activities of α-glucosidase and α-amylase (Gowadia and Vasudevan, 2000; Kazeem et al., 2013; Nyambe-Silavwe et al., 2015; Orhan and Orhan, 2016).

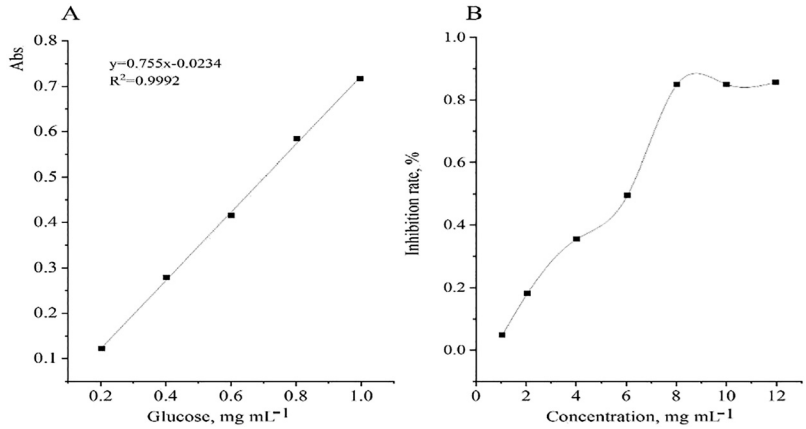

Fig. 4. Standard curve of glucose (A) and α-amylase inhibitory activity by different concentrations of JAE (B)

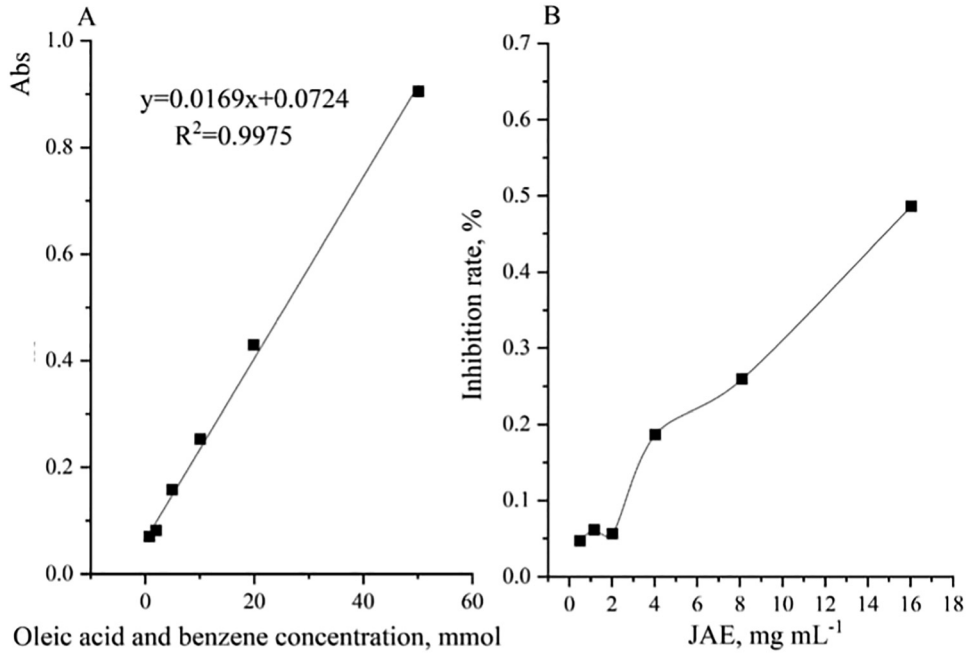

Fig. 5. Effects of oleic acid-benzene concentration (A) and gradient concentrations of JAE (B) on lipase inhibition rate %

3.4. Inhibition of lipase activity by JAE

The investigation into the potential of JAE's to inhibit lipase *in vitro* is presented in Fig. 5A and B. By using increasing quantities of JAE, the results demonstrated an inhibitory potential of JAE on lipase. Even though the study was conducted *in vitro*, the information allows us to surmise that the inhibitory effects of JAE may also be observed *in vivo*. Dietary fats have a high caloric value and are very important in the progression of obesity. Therefore, a decrease in fat digestion is viewed as a unique strategy for managing obesity. A gastrointestinal-lipase inhibitor called xenical was recently predicted to encourage weight loss in obese people. Given that JAE's *in vitro* inhibitory impact on lipase is demonstrated, it may be useful for limiting fat digestion, lowering body weight in obese people, and improving the metabolic state of humans. Numerous plant extracts have been reported to exhibit lipase inhibitory activity (Gowadia and Vasudevan, 2000; Mhatre et al., 2016), and the outcome suggests that the plant having abundant polyphenolic contents does have gastral inhibitory activity, thus supporting the findings of this study.

3.5. Identification of JAE phytochemicals

LC-MS was used to track the phenolic components in JAE. It was insufficient to distinguish the phenolic compounds precisely using retention time alone, but high confidence in identification was, however, achieved with online mass spectrum analysis. The JAE was examined based on the exact molecular weight and retention time of the standards for the effect-quality comparability.

The compounds identified are presented in Fig. 6 and Table 3. According to earlier research (Tchoné et al., 2006; Yuan et al., 2012; Zhang and Kim, 2015; Michalska-Ciechanowska et al., 2019), the Jerusalem artichoke has a high concentration of phenolic compounds in both its leaves and tubers. The tuber is found to be a good source of bio-chemicals such as chlorogenic acid, caffeic acid, etc. The bio-components identified in this work are consistent with previous

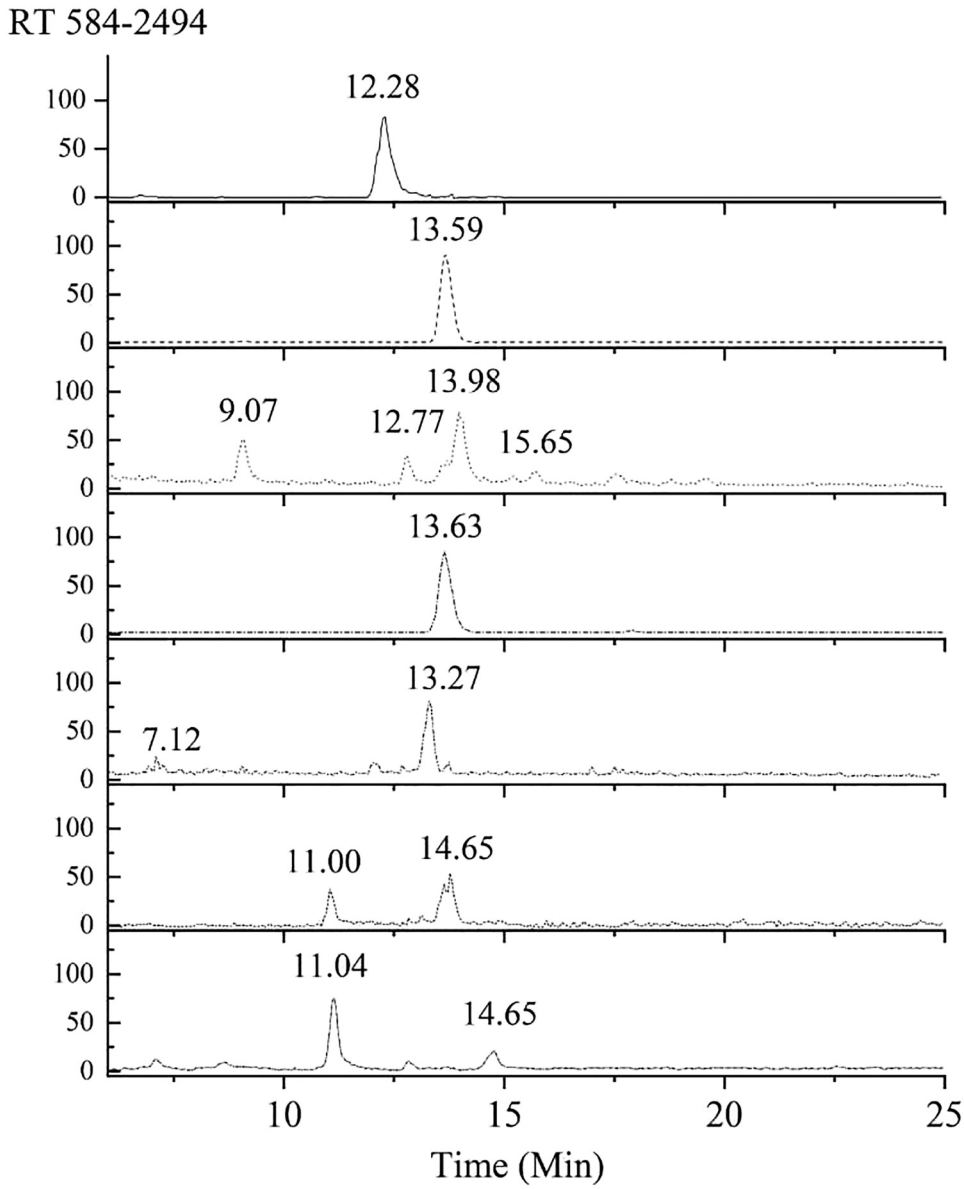

Fig. 6. Chromatograms of the identified phenolic compounds from JAE

Table 3. Phenolic compounds identified by LC-MS

No.	Retention time (min)	Name	Molecular formula	Molecular weight	CAS NO.
1	12.28	4-hydroxybenzoic acid	$C_7H_6O_3$	138.12	99-96-7
2	13.59	chlorogenic acid	$C_{16}H_{18}O_9$	354.31	327-97-9
3	13.98	caffeic acid	$C_9H_8O_4$	180.16	331-39-5
4	13.63	7-hydroxy-6-methoxycumarin	$C_{10}H_8O_4$	192.17	92-61-5
5	13.27	*p*-cumaric acid	$C_9H_8O_3$	164.16	7400-08-0
6	13.72	ferulic acid	$C_{10}H_{10}O_4$	194.18	1,135-24-6
7	14.65	sinapic acid	$C_{11}H_{12}O_5$	224.21	530-59-6

studies, which showed the existence of phenolic compounds in tubers of Jerusalem artichoke (Zhang and Kim, 2015; Michalska-Ciechanowska et al., 2019).

4. CONCLUSIONS

This study showed that JAE prevented the multiplication of H-29 colon cancer cells. A cell cycle arrest at the G1/S phase and induction of apoptosis was caused by the treatment of HT-29 cells with JAE. *E. coli* and *S. aureus* stopped growing when JAE was used. Also, JAE reduced the activity of lipase and α-amylase. JAE's primary effective bioactive components included 4-hydroxybenzoic acid, chlorogenic acid, caffeic acid, 7-hydroxy-6-methoxycumarin, *p*-coumaric acid, ferulic acid, and sinapic acid. The conclusion suggests that including Jerusalem artichoke tuber in the diet may have a positive impact on health status.

ACKNOWLEDGEMENT

This work was sponsored by the 2012 Agricultural Sci-Tech project of Zhenjiang (No.: NY2012015) and the Senior Talent Special Fund of Jiangsu University (No.:1 28136002).

REFERENCES

Abalaka, M.E., Daniyan, S.Y., Oyeleke, S.B., and Adeyemo, S.O. (2012). The antibacterial evaluation of *Moringa oleifera* leaf extracts on selected bacterial pathogens. *Journal of Microbiology Research*, 2(2): 1–4.

Afoakwah, N.A. and Mahunu, G.K. (2022). Utilization of Jerusalem artichoke (*Helianthus tuberosus* L.) tuber as a prebiotic and a synbiotic. In: Sulieman, A.M.E. and Mariod, A.A. (Eds.), *African fermented food products - New trends*, Springer, pp. 525–536.

Afoakwah, N.A., Dong, Y., Zhao, Y., Xiong, Z., Owusu, J., Wang, Y., and Zhang, J. (2015). Characterization of Jerusalem artichoke (*Helianthus tuberosus* L.) powder and its application in emulsion-type sausage. *LWT – Food Science and Technology*, 64(1): 74–81.

Aslan, M, Orhan, N., Orhan, D.D., and Ergun, F. (2010). Hypoglycemic activity and antioxidant potential of some medicinal plants traditionally used in Turkey for diabetes. *Journal of Ethnopharmacolology*, 128(2): 384–389.

Atef, N.M., Shanab, S.M., Negm, S.I., and Abbas, Y.A. (2019). Evaluation of antimicrobial activity of some plant extracts against antibiotic susceptible and resistant bacterial strains causing wound infection. *Bulletin of the National Research Centre*, 43(1): 144.

Ayua, E.O., Nkhata, S.G., Namaumbo, S.J., Kamau, E.H., Ngoma, T.N., and Aduol, K.O (2021). Polyphenolic inhibition of enterocytic starch digestion enzymes and glucose transporters for managing type 2 diabetes may be reduced in food systems. *Heliyon*, 7(2): e06245.

Baker, D.A.H., El-Gengaihi, S.E., Enein, A.A.H., and El-Ella., F.M.A. (2010). Biochemical study of some active ingredients in *Helianthus tuberosus* L. *Medicinal and Aromatic Plant Science and Biotechnology*, 4(1): 66–68.

Bojić, M., Maleš, Ž., Antolić, A., Babić, I., and Tomičić, M. (2019). Antithrombotic activity of flavonoids and polyphenols rich plant species. *Acta Pharmaceutica*, 69(4): 483–495.

Das, D.C., De, S., Battacharya, S., and Das, M. (2013). Antibacterial activity and photochemical analysis of *Cardanthera difformis* druce leaf extracts from West Bengal, India. *International Journal of Phytomedicine*, 5(4): 446–451.

Ding, S., Jiang, H., and Fang, J. (2018). Regulation of immune function by polyphenols. *Journal of Immunology Research*, 2018: 1264074.

Efenberger-Szmechtyk, M., Nowak, A., and Czyzowska, A. (2021). Plant extracts rich in polyphenols: antibacterial agents and natural preservatives for meat and meat products. *Critical Reviews in Food Science and Nutrition*, 61(1): 149–178.

Elansary, H.O., Szopa, A., Klimek-Szczykutowicz, M., Ekiert, H., Barakat, A.A., and Al-Mana, F.A. (2020). Activities of polyphenol extracts from *Ferocactus* species. *Processes*, 8(2): 138.

Fraternale, D., Ricci, D., Verardo, G., Gorassini, A., Stocchi, V., and Sestili, P. (2015). Activity of *Vitis vinifera* tendrils extract against phytopathogenic fungi. *Natural Product Communications*, 10(6): 1037–1042.

Gonelimali, F.D., Lin, J., Miao, W., Xuan, J., Charles, F., Chen, M., and Hatab, S.N. (2018). Antimicrobial properties and mechanism of action of some plant extracts against food pathogens and spoilage microorganisms. *Frontiers in Microbiology*, 9: 1639.

Gowadia, N. and Vasudevan, T.N. (2000). Studies on effect of some medicinal plants on pancreatic lipase activity using spectrophotometric method. *Asian Journal of Chemistry*, 12(3): 847–852.

Greenwood, D. (1989). Antibiotic sensitivity testing. In: Greenwood, D. (Ed.), *Antimicrobial chemotherapy*, Oxford University Press, pp. 91–100.

Kazeem, M.I., Adamson, J.O., and Ogunwande, I.A. (2013). Modes of inhibition of α-amylase and α-glucosidase by aqueous extract of *Morinda lucida* Benth leaf. *BioMed Research International*, 2013: 527570.

Long, X.H., Shao, H.B., Liu, L.P., Liu, L., and Liu, Z.P. (2016). Jerusalem artichoke: a sustainable biomass feedstock for biorefinery. *Renewable and Sustainable Energy Reviews*, 54: 1382–1388.

Magalhães, C.M., González-Berdullas, P., Duarte, D., Correia, A.S., Rodríguez-Borges, J.E., Vale, N., Esteves da Silva, J.C.G., and Pinto da Silva, L. (2021). Target-oriented synthesis of marine coelenterazine derivatives with anticancer activity by applying the heavy-atom effect. *Biomedicines*, 9(9): 1199. https://doi.org/10.3390/biomedicines9091199.

McCue, P.P. and Shetty, K. (2004). Inhibitory effects of rosmarinic acid extracts on porcine pancreatic amylase *in vitro*. *Asia Pacific Journal of Clinical Nutrition*, 13(1): 101–106.

Mhatre, V.S., Bhagit, A.A., and Yadav, R.P. (2016). Pancreatic lipase inhibitor from food plant: potential molecule for development of safe anti-obesity drug. *MGM Journal of Medical Sciences*, 3(1): 34–41.

Michalska-Ciechanowska, A., Wojdyło, A., Bogucka, B., and Dubis, B. (2019). Moderation of inulin and polyphenolics contents in three cultivars of *Helianthus tuberosus* L. by potassium fertilization. *Agronomy*, 9(12): 884.

Mostafa, A.A., Al-Askar, A.A., Almaary, K.S., Dawoud, T.M., Sholkamy, E.N., and Bakri, M.M. (2018). Antimicrobial activity of some plant extracts against bacterial strains causing food poisoning diseases. *Saudi Journal of Biological Sciences*, 25(2): 361–366. https://doi.org/10.1016/j.sjbs.2017.02.004.

Nezbedova, L., McGhie, T., Christensen, M., Heyes, J., Nasef, N.A., and Mehta, S. (2021). Onco-preventive and chemo-protective effects of apple bioactive compounds. *Nutrients*, 13(11): 4025.

Nizioł-Łukaszewska, Z., Furman-Toczek, D., and Zagórska-Dziok, M. (2018). Antioxidant activity and cytotoxicity of Jerusalem artichoke tubers and leaves extract on HaCaT and BJ fibroblast cells. *Lipids in Health and Disease*, 17(1): 280.

Nowacka-Jechalke, N., Olech, M., and Nowak, R. (2018). Mushroom polyphenols as chemopreventive agents. In: Watson, R., Preedy, V., and Zibadi, S. (Eds.), *Polyphenols: prevention and treatment of human disease*, 2nd ed., Elsevier Inc., pp. 137–150.

Nyambe-Silavwe, H., Villa-Rodriguez, J.A., Ifie, I., Holmes, M., Aydin, E., Jensen, J.M., and Williamson, G. (2015). Inhibition of human α-amylase by dietary polyphenols. *Journal of Functional Foods, Part A*, 19: 723–732. https://doi.org/10.1016/j.jff.2015.10.003.

Oliviero, F., Scanu, A., Zamudio-Cuevas, Y., Punzi, L., and Spinella, P. (2018). Anti-inflammatory effects of polyphenols in arthritis. *Journal of the Science of Food and Agriculture*, 98(5): 1653–1659.

Orhan, D.D. and Orhan, N. (2016). Assessment of *in vitro* antidiabetic and antioxidant effects of *Helianthus tuberosus, Cydonia oblonga* and *Allium porrum*. *Turkish Journal of Pharmaceutical Science*, 13(2): 181–188.

Pan, L., Sinden, M.R., Kennedy, A.H., Chai, H., Watson L.E., and Graham, T.L. (2009). Bioactive constituents of *Helianthus tuberosus* L. (Jerusalem artichoke). *Phytochemistry Letters*, 2(1): 15–18.

Panchev, I., Delchev, N., Kovacheva, D., and Slavov, A. (2011). Physicochemical characteristics of inulins obtained from Jerusalem artichoke (*Helianthus tuberosus* L.). *European Food Research and Technology*, 233(5): 889.

Praznik, W., Cieślik, E., and Filipiak-Florkiewicz, A. (2002). Soluble dietary fibres in Jerusalem artichoke powders: composition and application in bread. *Food/Nahrung*, 46(3): 151–157.

Prpa, E.J., Bajka, B.H., Ellis, P.R., Butterworth, P.J., Corpe, C.P., and Hall, W.L. (2020). A systematic review of *in vitro* studies evaluating the inhibitory effects of polyphenol-rich fruit extracts on carbohydrate digestive enzymes activity: a focus on culinary fruits consumed in Europe. *Critical Reviews in Food Science and Nutrition*, 61(22): 3703–3808.

Rahim, N.F.C., Hussin, Y., Aziz, M.N.M., Mohamad, N.E., Yeap, S.K., Masarudin, M.J., Abdullah, R., Akhtar, M.N., and Alitheen, N.B. (2021). Cytotoxicity and apoptosis effects of curcumin analogue (2E, 6E)-2, 6-Bis (2,3-Dimethoxybenzylidine) Cyclohexanone (DMCH) on human colon cancer cells HT29 and SW620 *in vitro*. *Molecules*, 26(5): 1261.

Simonetti, G., Brasili, E., and Pasqua, A. (2020). Antifungal activity of phenolic and polyphenolic compounds from different matrices of *Vitis vinifera* L. against human pathogens. *Molecules*, 25(16): 3748.

Sobeh, M., Mahmoud, M.F., Petruk, G., Rezq, S., Ashour, M.L., Youssef, F.S., El-Shazly, A.M., Monti, D.M., Abdel-Naim, A.B., and Wink, M. (2018). *Syzygium aquarium*: a polyphenol-rich leaf extract exhibits antioxidant, hepatoprotective, pain-killing and anti-inflammatory activities in animal models. *Frontiers in Pharmacology*, 9: 566.

Socrier, L., Quéro, A., Verdu, M., Song, Y., Molinié, R., Mathiron, D., Pilard, S., Mesnard, F., and Morandat, S. (2018). Flax phenolic compounds as inhibitors of lipid oxidation: elucidation of their mechanisms of action. *Food Chemistry*, 274: 651–658. https://doi.org/10.1016/j.foodchem.2018.08.126.

Tchoné, M., Barwald, G., Annemuller, G., and Fleischer, L. (2006). Separation and identification of phenolic compounds in Jerusalem artichoke (*Helianthus tuberosus* L.). *Sciences des Aliments*, 26(5): 394–408.

Usman, J.G., Sodipo, O.A., and Sandabe, U.K. (2014). In vitro antimicrobial activity of *Cucumis metuliferus* E. Mey. Ex. Naudin fruit extracts against *Salmonella gallinarum*. *International Journal of Phytomedicine*, 6(2): 268–274.

Yuan, X., Gao, M., Xiao, H., Tan, C., and Du, Y. (2012). Free radical scavenging activities and bioactive substances of Jerusalem artichoke (*Helianthus tuberosus* L.) leaves. *Food Chemistry*, 133(1): 10–14.

Yuan, X., Cheng, M., Gao, M., Zhuo, R., Zhang, L. and Xiao, H. (2013). Cytotoxic constituents from the leaves of Jerusalem artichoke (*Helianthus tuberosus* L.) and their structure–activity relationships. *Phytochemistry Letters*, 6(1): 21–25.

Zhang, Q. and Kim, H.Y. (2015). Antioxidant, anti-inflammatory and cytotoxicity on human lung epithelial A549 cells of Jerusalem artichoke (*Helianthus tuberosus* L.) tuber. *Korean Journal of Plant Resources*, 28(3): 305–311.