# Antioxidant and antimicrobial activities of ginseng extract, ferulic acid and noni juice, in the evaluation of their potential to be incorporated in food.

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## Abbreviations

A, asymptotic value; λ, lag time; μ, maximum growth; CFU, colony forming units; DPPH·, 2,2-diphenyl-1-picrylhydrazyl; FA, ferulic acid, FNJP, fermented noni juice poder; GE, ginseng extract; $IC_{50}$, half inhibitory concentration; MIC, minimal inhibitory concentration; PPO, polyphenol oxidase.

- Ginseng extract (GE), ferulic acid (FA) and fermented noni juice powder (FNJP) were studied.
- FA had the lowest IC50 and was able to inhibit polyphenoloxidase (PPO)(21.2-73.6 %).
- FNJP inhibited PPO (59.1-95.1 %) and also showed good antioxidant properties.
- MIC values of the three compounds against thirteen bacterial strains was evaluated
- Changes in lag phase, maximum growth rate and asymptotic value were elucidated.

**Abstract**

Ginseng extract (GE), ferulic acid (≥99%) (FA), and a fermented noni juice powder (FNJP), were investigated for their antioxidant and antimicrobial activities *in vitro*. Half inhibitory concentration ($IC_{50}$) was 29.87, 0.45 and 3.82 mg/mL, for GE, FA, and FNJP, respectively. The capacity of the three extracts to inhibit polyphenol oxidase from three vegetable matrices ranged between no inhibition and 95.1 % (depending on the extract and PPO source). In the study of peroxidation prevention of three types fats, only ferulic acid delayed lipid peroxidation of olive oil when applied at 10 mg/mL. The extracts' antimicrobial activity was studied on thirteen bacterial strains using the disk diffusion assay and the microdilution assay. Minimal inhibitory concentration (MIC) values were 5.5 mg/mL of GE for *Listeria monocytogenes*, 1.7 mg/mL of FA for *Staphylococcus aureus, L. monocytogenes* 1/2 and 4b, and 4.2 mg/mL of FNJP for *Bacillus cereus*. The increases in lag phase, and decreases in growth rate and in asymptotic value of the bacteria growing under different concentrations of the three compounds were described. The results obtained suggest the potential of GE, FA and FNJP for its further application in food industries.

**Keywords:**

Growth modelisation, microorganism, lipid peroxidation, natural source, bacteriostatic, bactericide

**Practical applications**

The exploration of new compounds for their antioxidant properties increases the range of ingredients to be used in food products with different purposes (e.g. browning inhibition, lipid peroxidation delay). Determining the antimicrobial properties and the minimum

54 inhibitory concentrations of these compounds for food-borne pathogens can help in
55 promote their use to enhance food safety.

## 56 1. Introduction

57 Food quality and safety maintenance during shelf-life is a major concern for both
58 producers and consumers. In order to extend shelf-life and facilitate access to low-cost
59 and palatable foods, the food industry has relied on the use of fats, sugars, and chemical
60 aids for decades. However, despite chemical additives being safe in commercial doses,
61 consumers tend to demand other alternatives. Some of them include novel technologies,
62 such as slightly acidified water (Hao, Wu, Li, & Liu, 2017), pulsed electric field (Martín-
63 Belloso & Sobrino-López, 2011) or innovations in packaging (Tsiraki & Savvaidis,
64 2013). Alternatively, plants are excellent sources of active molecules, possessing
65 antioxidant and/or antimicrobial properties (Negi, 2012). Plant-derived extracts,
66 including essential oils or volatile compounds(Kim, Kim, & Oh, 2021), are used in many
67 areas of the food industry to prevent microbial growth and undesirable quality changes
68 during storage (Nikmaram, Budaraju, Barba, & Lorenzo, 2018; Olatunde & Benjakul,
69 2018).

70 In a search for more sustainable and natural options, alternative plant-based extracts are
71 being explored to answer consumer requests (Abdul Qadir, Shahzadi, Bashir, Munir, &
72 Shahzad, 2017; Das, Singh, Dwivedy, Chaudhari, & Dubey, 2021; Ribeiro-Santos,
73 Andrade, Sanches-Silva, & de Melo, 2018; Teodoro, de Barros Fernandes, Botrel,
74 Borges, & de Souza, 2014). There are still plenty of plant-derived or plant by-products
75 that must be investigated in order to develop potential and functional ingredients, or
76 additives for food products. High-value plants such as ginseng (*Panax ginseng* L.) and its
77 derived compounds have been extensively described for their health-promoting properties
78 (Kim, Yi, Kim, & Cho, 2017). According to the European Food Safety Authority (EFSA),

ginseng is allowed to be used as a herbal medicinal product, to combat fatigue and asthenia and/or "strengthen the human body, supply of lacking energy, and positive life force, antioxidant" (Comittee on Herbal Medicinal Products, 2013; EFSA, 2008). Bacteriostatic and bactericidal effects have also been reported by some authors (Kachur & Suntres, 2016). The antimicrobial bioactivities were mainly attributed to ginsenosides, which are about thirty different saponin-type, triterpenoid glycosides (Santangelo, Silvestrini, & Mancuso, 2019). These compounds, which are part of the defence mechanism of the plant, also give the pharmacological properties attributed to ginseng: modulating blood pressure, metabolism, and inflammatory and immune functions (Leung & Wong, 2010). Some attempts have already been done in incorporating ginseng in food (Kim, Hwang, Eum, & Paik, 2019; Park, Lee, Kim, Park, & Paik, 2018), but deeper understanding of its features must be achieved in order to be able to exploit its use.

Other promising plant-based organic compounds include ferulic acid ([E]-3-[4-hydroxy-3-methoxy-phenyl] prop-2-enoic acid; FA) as an ubiquitous phenolic acid present in plant tissues (Mattila & Kumpulainen, 2002). FA is approved as a food additive in Japan, where it can be used as an antioxidant, while natural extracts with high contents of FA are permitted in the US and most European countries to prevent lipid peroxidation of foods (Quitmann, Fan, & Czermak, 2014). Its antimicrobial properties have also been explored against the pathogenic bacteria *Escherichia coli* and *Salmonella* Typhimurium *in vitro* (Pacheco-Ordaz, Wall-Medrano, Goñi, Ramos-Clamont-Montfort, Ayala-Zavala, et al., 2017) and in some ready-to-eat food (Takahashi et al. 2013). Although it has been relatively explored (Castagna, Dall'Asta, Chiavaro, Galaverna, & Ranieri, 2014; Guido & Moreira, 2017; Peanparkdee, Yamauchi, & Iwamoto, 2018) indepth evaluation on its applicability in food is still needed.

Finally, novel plants are under exploration as novel sources of bioactive compounds with potential food applications. Noni plant (*Morinda citrifolia* L.) is a 'superfruit', an exotic fruit that, according to studies, possesses strong antioxidant and functional activities (Fernandes, Rodrigues, Law, & Mujumdar, 2011; Kumoro, Retnowati, & Budiyati, 2011) and it has received the status of a novel food ingredient, which is defined as food that had not been consumed to a significant degree by humans in the EU before 15 May 1997. This is when the first regulation on novel food came into force, with the approval of the European Union Novel Foods Regulation (Regulation (EC) No 2015/2283). The use of noni plant purée and concentrates is allowed in a number of foods as an ingredient. Its functional action is attributed to flavonoids and polyphenols present in the fruit (Gironés-Vilaplana et al., 2014). Its antioxidant, antimicrobial, and immune-enhancing properties have been reviewed by several authors (Abou et al., 2017; Almeida, de Oliveira, & Hotza, 2019). Alternative food applications, including the use of noni fruit extract as sanitizer in washing steps for romaine lettuce, spinach, and kale, have demonstrated reductions of *L. monocytogenes* between 1.47-3.38 log CFU / g (Kang & Song, 2019).

The pure compound (FA) and the two extracts (GE, FNJP) are of actual interest in the food industry, for the increasing trend of using ginseng extract in food and beverages products (GVR, 2020), the incorporation of noni on the novel foods lists, and the potential extraction of ferulic acid from cereal by-products (Juhnevica-Radenkova et al., 2021), and for this reason, this paper aims to explore their potential application and functionality to be added in food products. Moreover, FA can be present in small concentrations in both GE (Yhung Jung, Sun Jeon, & Young Bock, 2002). and FNJP (Yan, et al., 2018), which could also contribute to the antioxidant and antimicrobial activities reported for those extracts.

Although some of the properties of the pure compound (FA) and the two extracts (GE, FNJP) have been described previously, this study aims to contribute to this growing area of research by exploring the antioxidant and antimicrobial activities of ginseng extract (GE), pure *trans*-ferulic acid (FA), and fermented juice extract powder (FNJP). This study makes an original contribution to the current knowledge, including the lipid peroxidation prevention of three different fats, and the polyphenol oxidase inhibition efficacy of the three compounds, tested *in vitro*. In most of the publications, the minimal inhibitory concentrations (MIC) values are only presented (Aziz & Almasi, 2018; Danh et al., 2013; Trojaike, Biondo, Padilha, Brandelli, & Sant'Anna, 2019). However, to deepen the understanding of the antimicrobial effects of the selected pure compound and extracts, the changes in the growth parameters of 13 bacterial strains were evaluated. A thorough understanding of the possibilities of a pure compound (FA) and plant-derived extracts reported to contain this compound (GE and FNJP) would be useful in their further application in food.

## 2. Experimental

### 2.1.Materials

Commercial ginseng extract containing 1 % of ginsenosides approved by the Comittee on Herbal Medicinal Products (2013) was purchased from EPSA (Torrent, España) Eand trans-ferulic acid (≥ 99% purity) was obtained from Sigma-Aldrich (ref. W518301, Steinheim, Germany). Noni juice extract was kindly provided by the University of Nayarit, Mexico, and was prepared as described by Ulloa, González-Tapia, Rosas-Ulloa, Ramírez-Ramírez, & Ulloa-Rangel (2015).

Scopoletin, ursolic acid, 2,2-diphenyl-1-picrylhydrazyl (DPPH), sodium carbonate, $K_2HPO_4$ and $KH_2PO_4$, 2-polyvinyl pirrolidone (PVPP), cistein, pyrocatechol, guayacol

and streptomycin were acquired from Sigma-Aldrich (Steinheim, Germany). Peroxide hydrogen, methanol, were procured from Panreac (Llinars del Vallès, Spain). Triptone soy broth (TSB) and Müeller-Hinton broth (MHB) were purchased from Biokar Diagnostics (Allonne, France).

## 2.2.Methods

### 2.2.1. Determination of scopoletin, ursolic acid and rutin in fermented noni juice powder

Concentrations of scopoletin, ursolic acid and rutin in freeze-dried fermented noni juice FNJP were determined by UPLC-MS, using Acquity UPLC-Xevo TQS (Waters) by the Scientific and Technical Service of Chromatographic Techniques and Mass Spectrometry (TCEM) of the University of Lleida. Briefly, 50 mg of freeze-dried material was diluted and filtered with a PTFE hydrophylic 0.22µm filter. Internal standard method was used to identify and quantify the compounds. UPLC was performed using Acquity UPLC ® HSS T3 1.8 µm, 100 x 2.1 mm column, injecting 2.5 µL of sample at 10 ºC, in an isotherm column at 30 ºC, with two mobile phases: (A) water, methanol and formic acid (1.5:98:0.5 v:v:v), and (B) methanol and formic acid (99.5:0.5 v:v) at 0.3 mL/min. They were performed in a gradient as follows: from 0 to 0.51 min 5% B, from 0.51 to 3.50, up to 100% B, from 3.51 to 5.50, at 100% B, and finally, back at initial conditions for 8.00 min. Mass spectrometry was done with an ESI with negative ion mode, 2 kV capillarity, source and desolvation temperatures of 120 and 450 ºC, respectively. Cone and desolvation gas flow were 150 and 1000 L/h, respectively, and collision gas flow was 0.15 mL/min. Results were obtained by comparing the peaks on the chromatogram with the peaks produced by the internal standards, corresponding to mass ions 190.94 > 147.72 and 190.94 > 175.83 for scopoletin, 455.07 > 408.96 and 455.07 > 4550 for ursolic acid and 609.00 > 271.03 and 609.00 > 300.03 for rutin.

### 2.2.2 Antioxidant properties

#### 2.2.2.1 Half maximal inhibitory concentration ($IC_{50}$)

The compounds were serially diluted in distilled water in concentrations ranging from a stock solution (which, according to preliminary studies in which the compounds were diluted at increasing concentrations in water and precipitation of the compound after 2 h was not observed, corresponded to their maximum solubility in water) of 33.0 mg/mL GE, 10.0 mg/mL FA, and 100.0 mg/mL FNJP, to 0 mg/mL. Then, 0.1 mL of the diluted samples were added to 1.4 mL of 0.1 mM DPPH· solution. Methanol was used as a blank. After incubation at room temperature for 60 min in the dark, absorbance at 515 nm was read using a GENESYS$^{TM}$ 10S UV-Vis spectrophotometer (Thermo Fisher Scientific, MA, USA). For $IC_{50}$ calculation, the percentage of inhibition calculated using Eq. 1 was plotted against extract concentration. $IC_{50}$ corresponds to the necessary concentration of an extract to achieve 50% of inhibition (Eq. 2) (Kumawat, Gupta, & Singh, 2012).

$$\%I = [(A_b - A_s) / A_b] \cdot 100 \qquad \text{Eq. 1}$$

$$\%I = m \cdot C + n; \qquad IC_{50} = (50-n)/m \qquad \text{Eq 2.}$$

Where %I is the inhibition in percentage, $A_b$ and $A_s$ are the absorbance of the blank and the sample, respectively. m is the slope of the lineal adjustment when representing %I in front of C (mg/mL), concentration, and n is the intercept.

#### 2.2.2.2. Polyphenol oxidase (PPO) activity inhibition

To evaluate GE, FA and FNJP as natural inhibitors of enzymatic browning, determination of the enzyme inhibition was evaluated on different model systems containing PPO from different matrices, on apple, potato, and mushroom, following the method reported by (Bobo, 2014, Masuda 2015) with some modifications. PPO extraction was carried out by mixing 5.0 ± 0.5 g of frozen apple, potato, or mushroom with 0.5 g PVPP and 10 mL 0.1

M phosphate buffer solution pH 6 (PBS) with 0.05 mM cysteine in an Ultra-turrax® Tube drive P control (IKA, Staufen, Germany) for 1.5 min at 5,000 strokes/min. After filtration using a sterile cloth and centrifugation at 20,000 × *g* for 10 min at 4 ºC, the supernatant was kept in ice.

PPO inhibitory activity was assessed spectrophotometrically. For each compound, three concentrations were tested: 33.0, 25.0, and 16.5 mg/mL GE; 7.5, 5.0, and 2.5 mg/mL FA; and 100.0, 75.0, and 50.0 mg/mL FNJP. Briefly, 65 µL of PPO extract were incubated for 10 min with or without 65 µL of the solutions to be analysed. Then, 65 µL of 0.2 M pyrocatechol in PBS were added. After 10 min of incubation at 37 °C, absorbance was read at 400 nm. Inhibition was expressed as % of the PPO activity using Equation 3.

$$\% \text{ Inhibition} = [(A_0 - A_E) / A_0] \cdot 100 \qquad \text{Eq. 3}$$

Where $A_0$ is the absorbance at 400 nm after enzymatic reaction alone, and $A_E$ is the absorbance at 400 nm after the enzymatic reaction in presence of the extract studied.

#### 2.2.1.2. Prevention of lipid peroxidation

The effect of GE, FA and FNJP in the prevention of lipid peroxidation on olive oil, sunflower oil and butter was evaluated by the Oxidation stability of oils and fats – Rancimat method, following the recommendations of the manufacturer (Rancimat, Metrohm). Samples were prepared immediately before the test by homogenizing olive oil, sunflower oil, or butter with 10 mg/mL of each compound, using an Ultra-Turrax T-25 homogenizer (IKA Works GmbH & Co, Staufen, Germany) operating at 14,000 strokes/min. Samples were tested per quadruplicate (n=4) in a Rancimat 743 apparatus for oils and fats (Metrohm, Germany), at a temperature of 110 °C and using a gas flow of 10 L/h. Induction time was calculated using Rancimat 743 software 1.1.

### 2.2.3. Antimicrobial effects

#### 2.2.3.1. Strain and inoculum preparation for antimicrobial effect assays

The antimicrobial effect of each compound was tested against 13 strains (Table 1). Strains were grown for 22 ± 2 h in 50 mL of triptone soy broth (TSB), which was supplemented with 6 g/L of yeast extract, 2.5 g/L of glucose, and 2.5 g/L of $K_2HPO_4$ (TSBYE) for *Listeria monocytogenes* – at 37±1 °C in a rotatory shaker set at 150 rpm.

#### 2.2.3.2. Antimicrobial activity: Disk diffusion test

The disk diffusion test to investigate the susceptibility of bacteria to selected antimicrobials was performed to test serial 2-fold dilutions of the compounds, starting with stock solutions of 33.0, 20.0, and 100.0 mg/mL of GE, FA, and FNJP, respectively. Plates with a thin layer of TSB or TSBYE (for *L. monocytogenes* strains) were prepared in advance. Then, 5 mL of semi-solid TSB or TSBYE agar were prepared in glass tubes, where 50 µL of the inoculum, prepared as described in section 2.2.3.1 were added. After homogenization, the semi-solid agar was poured onto the plates to spread the microorganism over the entire surface. When it was solid, nine paper disks (6 mm diameter) per plate were placed separately on the agar. Then, 5 µL of the concentrations to study were discharged on each disk. Negative and positive controls were distilled water (no extract present) and streptomycin 1 mg/mL respectively. When more than 9 solutions were needed to be tested, two plates were used. Each extract and concentration was tested in triplicate (three plates). After 1 h at room temperature, plates were incubated at 37 ºC for 22 ± 2 h. The antimicrobial effect was stated when inhibition halos or zones with no microbial growth were observed.

#### 2.2.3.3. Effect of extracts on the kinetic parameters of studied strains and determination of the minimal inhibitory concentration (MIC)

The MIC of the different extracts for each strain was tested using the microdilution method (CLSI, 2012). The inoculum of the 13 tested microorganisms was prepared as described in section 2.2.3.1 and diluted to $7.5 \times 10^5$ CFU / mL in Mueller-Hinton Broth Cation Adjusted (MHB-CA) with 25 mg/L $Ca^{2+}$ and 12.5 mg/L $Mg^{2+}$. A stock solution that contained 33.0, 10.0, and 100.0 mg/mL, of GIN, FA, and FNJP, respectively, was prepared in sterile water under sterile conditions. From this, 5 more concentrations were prepared by making 2-fold serial dilutions. Then, 100 µL of the inoculum was poured into each microplate well, containing 50 µL of each compound at the prepared concentrations. Negative and positive controls were distilled water (no extract present) and streptomycin 1000 ppm respectively. A blank for each concentration, consisting of MHB-CA without inoculum, but with the corresponding concentration of the compound, was set in order to correct the compounds' color basis. The plate was incubated for 48 h at 37 ºC in a PowerWave HT (Biotek, Vermont, United States). Absorbance at 620 nm was read every 30 min (Andrews, 2001).

For the bacterial growth experiment, primary models were fitted using the DMFit 3.5 Excel add-in provided by ComBase predictive modelling tool (https://www.combase.cc) and growth parameters (lag time, growth rate, and maximum optical density) were determined using the re-parameterized Gompertz model described by Zwietering *et al*. (1990) based on Equation 4.

$$y = A \exp\left\{- exp\left(\frac{\mu \max e}{A}(\lambda - t) + 1\right)\right\} \quad \text{Eq. 4}$$

where y represents the absorbance at time t, $\mu_m$ is the maximum growth rate (OD × $10^3$ / min), t is the incubation time (min), λ corresponds to lag time (min), and A is the asymptotic value (or maximum growth, OD units).

To determine the minimum inhibitory concentration (MIC) of the three compounds, the value recorded was the lowest concentration of the agent that completely inhibited the growth of each bacterial strain studied (EUCAST, 2003).

### 2.3. Statistical analysis

Results were expressed as mean ± standard deviation (SD). All data was checked for normality and homoscedasticity, and significant differences by applying the analysis of variance test (ANOVA). The criterion for statistical significance was $p < 0.05$. When significant differences were observed, Tukey's Honest Significant Difference (HSD) of the means was applied. All statistical analysis was carried out using JMP 13 (SAS Institute Inc., Cary, USA).

## 3. Results and discussion

### 3.1. Antioxidant properties

#### 3.1.1. Half maximal inhibitory concentration ($IC_{50}$)

The half maximal inhibitory ($IC_{50}$) is a measure of the potential of a substance in inhibiting a specific biological or biochemical function, in this case, to inhibit the oxidation of DPPH· radical, in which there is an inverse correlation between IC50 value of a compound and its antioxidant activity. Ginseng extract ginsenosides of 1% showed an $IC_{50}$ value of 29.87 mg/mL. The results of the antioxidant capacity of GE, reported so far, are contradictory. Indeed, Kim, Guo, & Packer (2002) observed that 2.0 mg/mL of a red ginseng extract completely inhibited DPPH· radical. Results were comparable to those reported by Kitts, Wijewickreme, & Hu (2000), which showed a powerful antioxidant activity of a North American ginseng extract. In turn, Jung, Seog, Choi, Park, & Cho (2006) reported an $IC_{50}$ of a wild ginseng extract in water or methanol of around 30 mg/mL, which is in line with our results. Ginseng extracts have been tested on some food products for their antioxidant properties. For instance, 2% red ginseng extract was added to milk and yoghurt in order to increase their antioxidant capacities by a combined action of the ginsenosides and phenolic compounds that are present in the extract (Park, Lee, Kim, Park, & Paik, 2018b). Moreover, Kim, Hwang, Eum, & Paik (2019) added 0.5 and 1.0% of red ginseng extract to cheese. Although the authors reported color and texture changes, the antioxidant capacity of the cheese increased, and the acceptance of the product was not affected. The evidence of the studies on the effects of ginseng on the human body suggests that the ingestion of ginseng extracts with food, could not only be advantageous for the properties and shelf-life of the food itself, but also for the consumer's health. As an example, ginsenoside Rg3 acted as a mechanism for antiaging in human dermal fibroblasts (Lee et al., 2018), and ginsenoside Rg1 promoted the activity

of antioxidant proteins by reducing reactive oxygen species (ROS) and delaying apoptosis (Gao et al., 2019).

FA showed an $IC_{50}$ value of 0.45 mg/mL, which was the lowest of the three extracts studied. FA has repeatedly been reported as a powerful antioxidant. It is important to highlight that the FA studied in the current paper was chemically synthesized and its purity was of an analytical grade. Its action mode is primarily related to scavenging of free radicals by combining with reactive molecules. This makes the initiation of the complex cascade reaction that leads to the generation of further free radicals difficult (Rice-Evans, Miller, & Paganga, 1996). This compound may also act as a hydrogen donor, giving atoms directly to the radicals (Zduńska, Dana, Kolodziejczak, & Rotsztejn, 2018). Actually, that is the main reason why FA has been used for cholesterol control, prevention against thrombosis and atherosclerosis, anti-inflammatory effects, cancer, and as an anti-ageing agent (Kumar & Pruthi, 2014; Ou & Kwok, 2004). In fact, FA could have also contributed to the antioxidant and antimicrobial properties of GE and FNJP, as both extracts could have small concentrations of this compound. For instance, one of the major phenolics in ginseng root is ferulic acid (Kim, 2016) and also in noni fruit Sirithon, Chodsana & Pornpimol, 2014). However, in this study, only the main bioactive compounds of GE and FNJP were determined in this paper. A further quantification of FA present in FE and FNJP, as well as a more detailed characterization of the composition of each extract would be useful to explain in-depth the results presented in this manuscript.

The characterisation of FNJP showed an $IC_{50}$ value of 3.82 mg/mL. The antioxidant activity of this extract is mainly related to the active biomolecules of the fruit, such as phenolic acids, flavonoids, phytoestrogens, and vitamin C (Chang, Alasalvar, & Shahidi, 2018). Values of pH and total soluble solids of FNJP were 3.16 ± 0.05 and 5.5 g/L,

330 respectively. In addition, concentrations of 333.5 μg/g scopoletin and 346.5 μg/g of rutin
331 were also determined by UPLC-MS, which have been reported as the main bioactive
332 compounds present in noni fruit (Almeida et al., 2019). Gironés-Vilaplana et al. (2014)
333 reported higher $IC_{50}$ values (25 mg/mL) than those obtained in the present study using
334 DPPH· assay. This could be attributed to the differences in phenolic contents related to
335 maturity or origin of the fruit. This not only affects the juice yield but also the total
336 quantities of phenolic compounds, condensed tannins, flavonoids and scopoletin (Iloki
337 Assanga et al., 2014), or the fermentation conditions of the juice: longer times lead to
338 lower antioxidant content in the final product (Yang, Chen, Li, & Tsai, 2007). As assayed
339 *in vitro* in cells, noni juice has shown that its antioxidant capacity may prevent cancer
340 incidence, as it decreases intracellular ROS generation and mitochondrial membrane
341 potential in breast cancer cell lines (Sharma et al., 2015). The studies also report a
342 decrease in the level of lipid peroxidation and an increase in catalase activity in cervical
343 cancer cell lines (Gupta & Singh, 2013).

344 This section has reviewed the antioxidant capacities of the three studied compounds,
345 showing their potential for being used as valuable natural additives to prevent oxidation
346 and to increase the shelf-life of food.

347 **3.1.2. Polyphenol oxidase (PPO) activity inhibition**

348 PPO catalyzes two type of reactions, hydroxylation of monophenols to diphenols, which
349 results in colorless products, and oxidation of diphenols to quinines, which gives colored
350 products (Ioannou & Ghoul, 2013). Finding ways to inhibit such reactions is relevant to
351 food processors, because this enzyme has been directly related to browning reactions in
352 fruits and vegetables that decrease consumer acceptance (Sulaiman, Soo, Farid, & Silva,
353 2015). The percentage of inhibition of the PPO activity in potato, apple, or mushroom is

shown in Table 2. FA could only be studied at doses lower than 7.5 mg/mL, because it was not possible to read its absorbance at 400 nm at higher concentrations.

There is a lack of information about the effect that ginseng extract or ginsenosides can exert on PPO. In this study, GE did not show inhibition of potato and mushroom derived PPO. An increase in GE concentration may conceivably trigger the inhibition effect on the PPO obtained from these matrices. For this reason, and to increase solubility of GE, other suitable solvents should be tried. Contrarily, when testing 33 mg/mL of GE with apple-PPO, a 33.9 ± 2.5 % inhibition of the enzymatic activity was observed. The mechanisms of this inhibitory effect have not yet been described. To have a better understanding, it would be advisable to assess other matrices and the kinetics of this interaction.

Inhibition of PPO attributed to FA was matrix-dependent, and it has been stated that it can also be variety-dependent (Liao et al., 2020). Higher inhibitory activities were observed for the enzymes extracted from mushroom, obtaining a decrease in PPO activity of 73.6 ± 4.29 % at a concentration of 7.5 mg/mL. For potato and apple derived PPO, the same concentration showed a 37.8 ± 1.0 and 41.7 ± 1.4, respectively. In the case of FA, a higher inhibition was observed at higher doses. Shannon & Pratt (1967) suggested that FA acted as a competitive inhibitor of apple-derived PPO, preventing the substrate from binding to the enzyme by occupying its place in the active site. Nirmal & Benjakul (2009) added that hydroxyl group of FA also had a role in the decrease in the activity of PPO, by its electron donating to intermediate quinone. The results obtained show that FA has potential to be used in a food matrix to inhibit enzymatic browning. Nevertheless, interactions between FA, PPO, and other food components must be taken into account because they can determine the concentration and the effect of FA in food. As reported by Sukhonthara, Kaewka, & Theerakulkait (2016), concentrations of 390 mg/L only

reached a decrease of 15 % in PPO activities when added to potato and apple purees, indicating that higher amounts of FA were needed to prevent enzymatic browning. The increase in the concentration needed for a PPO inhibition in a food matrix could be attributed to the implication of other factors when compared to its effect on isolated PPO studied *in vitro*, e.g. other substrates or inhibitors present in the food matrix, suboptimal pH, other compounds that may interfere or potentiate the reaction (Cantos, Tudela, Gil, & Espín, 2002). FA was also used in Chinese water chest-nut to prevent yellowing, inhibiting not only PPO, but also preventing the increase of other compounds naturally present in chest-nut that contribute to its yellowing process, namely eriodyctiol and naringenin (Song et al., 2019). In addition, Liao et al. (2020) showed that there was no direct relationship between the inhibition of browning in pear puree and the inhibition of PPO.

FNJP reduced the PPO activity from the three matrices (Table 2). Maximum inhibition was observed in PPO from mushroom, being 89.5 ± 7.6 % when FNJP was used at 100 mg/mL. Percentage of inhibition of potato and apple PPO was not concentration-dependent. A saturation of the inhibitory effect of FNJP on PPO could explain this independence. The mechanisms underlying the decrease in PPO activity exerted by NJE are not yet described in the literature. A likely explanation is that noni fruit possesses plenty of antioxidant compounds that may promote this effect (Almeida et al., 2019). Nevertheless, there is a need for a deeper understanding on the role played by its characteristic active compounds, such as scopoletin or rutin.

In this study, FA and FNJP are revealed to be effective in inhibiting a remarkable percentage of PPO activity. However, in vitro studies give an idea of the potential of the compounds to be used in food matrices, and browning reactions are not only PPO

dependent. For this reason, for certain foods, specific experiments must be carried out prior to application.

### 3.1.3. Lipid peroxidation prevention

None of the extracts were able to delay the oxidation of sunflower oil or butter at the tested concentration (Table 3). When tested in olive oil, only FA increased the induction time to 37.57 ± 4.86 h, compared to the control (with no extract) which had an induction time of 15.13 ± 0.09 h. The effectiveness of FA might be explained by its chemical structure: its hydroxyl group can provide protons and inhibit the formation of free radicals, delaying the rate of oxidation. In fact, FA has been reported to have protective effects in linseed oil (Kyselka et al., 2017) and soybean oil (Luo, Zhang, Zheng, Wang, & Ji, 2012). However, in this study, it failed in preventing lipid peroxidation of sunflower oil and butter. This could be related to the composition of those lipid matrices, and differences in the main fatty acids (oleic acid in olive oil, linoleic acid in sunflower oil, and palmitic acid in butter), that as well as influencing their lipid peroxidation susceptibility when compared to olive oil (induction time was lower for sunflower oil and butter – 2.42 and 1.46 h, respectively – than it was for olive oil – 15.13 h –), it can influence the impact that one antioxidant compound may have on its prevention (Choe & Min, 2006). It is possible that a higher concentration of FA is necessary to perform a significant delay in lipid peroxidation of sunflower oil and butter fats. In this study, GE did not reveal any effect on the lipid peroxidation of the tested matrices. However, some authors reported a decrease in lipid peroxidation of up to 90% when adding 0.5 to 2% of red ginseng extract to milk (v:v) (J. E. Jung et al., 2020). Regarding FNJP, although no effect was observed in our study at the concentration used, its puree was used at concentrations ranging from 2 to 6% in beef patties, which showed a delay in their lipid

427 peroxidation in time, when compared to the control samples (Tapp, Yancey, Apple,
428 Dikeman, & Godbee, 2012).

429 **3.2.Antimicrobial effect of GE, AF and LFNJ**

430 **3.2.1. Disk diffusion test**

431 None of the extracts cause inhibition halos in any of the tested strains (Data not shown).
432 As will be described in section 3.2.2., the microdilution method showed growth
433 inhibition, so the methodology used for the determination of antimicrobial activity is
434 crucial. In other studies, the microdilution method was also more sensitive than the disk
435 diffusion was (Scorzoni et al., 2007). Disk diffusion methods cannot be used to determine
436 MICs, because the amount of the substance that is diffused in the agar is unknown, so it
437 is not possible to relate the inhibition halo with a determined inhibitory concentration
438 (Balouiri, Sadiki, & Ibnsouda, 2016). These methods may serve as a screening method to
439 ascertain whether the studied compounds have antimicrobial activity or not. The absence
440 of an inhibitory effect in our study could be explained by the low diffusion of the diluted
441 substances in the agar, or to the higher difficulty that the active compounds encounter in
442 order to be in contact with the microorganism when compared to a broth dilution method,
443 in which this contact is direct (Rios, Recio, & Villar, 1988).

444 **3.2.2. Effect of studied substances on the kinetic parameters of studied strains and**
445 **determination of the minimal inhibitory concentration (MIC)**

446 The growth curves of the microorganisms grown under the presence of different
447 concentrations of the compounds were adjusted to the 3-parametric Gompertz equation,
448 which has been proved to be a good mathematical model to describe biological parameters
449 of microorganism growth (Pla, Oltra, Esteban, Andreu, & Palop, 2015). In the present

study, the coefficient of determination ($R^2$) was always higher than 0.800 and averaged 0.988.

Kinetic growth parameters of studied microorganisms in relation to the presence of GE in the growth medium are shown in Table 4. GE had the greatest impact on *L. monocytogenes* serovar 1/2a, in which lag phase (λ) was 3-fold longer at 2.7 mg/mL, and MIC value (Table 7) was 5.5 mg/mL GE, when its growth was completely inhibited. In contrast, *L. monocytogenes* serovars 1/2 and 4b lag phase was affected only at the highest GE concentrations. These also slightly decreased the maximum growth rate (μ) and asymptotic value (A). Norajit & Ryu (2012) suggested that ginsenosides may induce the lysis of *L. monocytogenes*. The other Gram-positive bacteria studied, *S. aureus* or *E. faecalis,* were not significantly affected by GE. In most of the works published, GE was used as a compound that, when ingested or taken as a medical treatment, is able to diminish some of the virulent mode of action of the microorganisms. These include its ability to attach to the human gut cell, or its effect of inhibiting cytokines and chemoquines responsible for the inflammation when the body is infected by bacteria (Iqbal & Rhee, 2020; Szczuka et al., 2019). In this work, the assessment of the compounds to inhibit the growth of microorganisms in a possible direct food application is taken into account. Even though none of the tested strains of *S. enterica* were affected by GE, growth parameters of *B. cereus* were modified when GE was used at a concentration of 11.0 mg/mL. *E. aerogenes* and toxigenic *E. coli* respective lag phases (λ) were also longer when applying GE at 1.4 mg/mL, when compared to the control. Pina-Pérez, Rivas, Martínez, & Rodrigo (2018) investigated the effect that different heat treatments applied to GE had on the viability of bacteria. They reported that microwaved GE had a greater effect on microorganism growth than non-heated GE when using concentrations ranging from 10 to 100 mg/mL, especially on *E. coli* O157:H7, followed by *Cronobacter*

475 *sakazakii*. Other authors also reported that heating GE to 100 °C for 2 or 16 h was related
476 to an increase of its antimicrobial activity against *S. aureus* and *B. cereus* (Na, Young, &
477 Rhee, 2017). To date, the applications of ginseng extract or its derivatives as antimicrobial
478 agents for food preservation are scarce. Norajit & Ryu, (2012) developed an alginate
479 coating with ginseng extract, which was tested against *Staphylococcus epidermidis*,
480 *Bacillus subtilis* and *L. monocytogenes*. The results suggested that the incorporation of
481 ginseng extracts into edible films could be used to control food pathogens and improve
482 shelf life in food systems.

483 Regarding FA (Table 5), *L. monocytogenes* 4b and 1/2 growth were significantly affected,
484 as FA extended their lag time (λ), and decreased their maximum growth rate (μ) and
485 asymptotic value (A) at concentrations of 0.8 mg/mL. These strains were completely
486 inhibited with 1.7 mg/mL FA, (MIC value, Table 7) and *L. monocytogenes* 1/2a MIC was
487 2.5 mg/mL FA. For other Gram-positive bacteria studied, *S. aureus* and *E. faecalis* at 1.7
488 mg/mL, or *B. cereus*, MIC value was higher, 3.3 mg/mL FA. FA also had an antimicrobial
489 effect on Gram-negative bacteria, such as *E. aerogenes*, whose maximum growth rate (μ)
490 and asymptotic value (A) were reduced significantly at concentrations ≥ 0.8 mg/mL FA.
491 3.3 mg/mL were needed to completely inhibit its growth (MIC value). Although FA had
492 no effect on *S.* Typhiurium, MIC values for *S.* Montevideo and *S.* Gaminara were 2.5
493 mg/mL, and that of *S.* Agona was 3.3 mg/mL FA. Both strains of *E. coli* were also
494 completely inhibited by 3.3 mg/mL FA. The MICs found in literature for *Salmonella*
495 Typhi and *E. coli* were 20 mmol/L (3.9 mg/mL) (Pacheco-Ordaz et al., 2017). Pernin et
496 al. (2018) also tested the antimicrobial effect of FA against *L. monocytogenes* and
497 reported that the MIC was 13.6 mmol/L (2.6 mg/mL). These values are similar to the
498 MICs reported in the present study. Small differences could be explained by the existing
499 differences linked to strain resistance to a certain compound, as has already been stated

500 in the present study. When a number of phenolic compounds are studied, their mode of
501 action consists of a combination of two mechanisms: the acidic dissociation and the
502 intercalation in the phospholipid membrane of the bacteria. The effect of dissociation of
503 the acid, causing the acidification of the cell cytoplasm, the efflux of $K^+$ ions and the
504 eventual death of the microorganisms, is combined with the intercalation of the acid in
505 the phospholipid layers of the membrane. This disturbs the Van der Waals interactions
506 and inhibits the substrate transport of key enzymes (Pernin, Bosc, Maillard, & Dubois-
507 Brissonnet, 2019; Pernin, Guillier, & Dubois-brissonnet, 2019). Our study used a media,
508 MHB-CA, whose pH is 7, but according to Miyague, Macedo, Meca, Holley, & Luciano
509 (2015), MIC values can decrease at lower pH values. For instance, they found that at pH
510 5, MIC was 2.5 mmol/L (0.5 mg/mL), while in contrast, it was 10 mmol/L (1.9 mg/mL)
511 at pH 7. That could be explained by a combination of hurdle barriers against *L.*
512 *monocytogenes* growth. The antimicrobial effect of FA has been studied in some food
513 matrices by Takahashi et al. (2013, 2015) who evaluated the effect of FA in smoked
514 salmon, cheese and coleslaw at a concentration of 1.5 mg/mL in coleslaw and observed
515 reductions ≥1.5 log CFU/g in the counts of *L. monocytogenes* after 5 days.

516 Finally, FNJP also showed antimicrobial activity against most of the pathogenic bacteria
517 studied (Table 6). Concentrations of FNJP of 2.1 mg/mL led to a decrease in the
518 maximum growth rate (μ) of *L. monocytogenes* 4b, 1/2a and 1/2, *S.* Typhimurium, *S.*
519 Agona, and *B. cereus*. MIC value (Table 7) of all strains of *L. monocytogenes* and *S.*
520 *Typhimurium* was 16.6 mg/mL FNJP. For the other serovars of *S. enterica*, Agona,
521 Montevideo and Gaminara, 33.3 mg/mL were needed to completely inhibit their growth
522 (MICs). The same concentration was the MIC found for *E. coli* CECT-516 and O157:H7,
523 and *E. faecalis*. Lower concentrations of FNJP were needed to completely inhibit the
524 growth of *B. cereus*, (4.1 mg/mL) and *S. aureus* (16.6 mg/mL). However, this compound

was non-effective against *E. aerogenes* at the concentrations tested. Other authors have studied the effect of noni derivates on other species of *Staphylococcus*, but focusing only on the infective mechanisms of the bacteria, and not on their ability to grow (De La Cruz-Sánchez et al., 2019). In fact, noni is used in traditional medicine of some Asian countries for its antimicrobial properties, mostly attributed to its main coumarin, named scopoletin. As stated before, the FNJP used in this study had 333.5 µg/g scopoletin. It is suggested that this coumarin interacts with the membrane of microorganisms, destroying its integrity and increasing its permeability, leading to cell death (Yang et al., 2016). The antimicrobial effect could also be attributed to the low pH values of the extract, which are below the growth limits of most pathogens. Methanolic extracts of noni fruit have also shown *in vitro* antimicrobial activity against *Pseudomonas aeruginosa, Proteus morganii, S. aureus, B. subtilis, E. coli, Salmonella* spp., and *Shigella* spp. (Rosyida et al., 2019). Noni extract has already been proposed for washing fresh-cut kale, lettuce, and spinach, with reductions of *L. monocytogenes* ATCC 19111 and 19115 ranging from 1.47 to 3.38 log CFU/g, depending on the roughness of the surface of the product (Kang & Song, 2019).

It is important to note that studies *in vivo* should be carried out in order to test real conditions of these extracts, such as pH of the matrix, other nutrients or compounds that may interact with them, different surfaces, and water activities, amongst others. But as shown in this study, the three compounds analyzed have antimicrobial activities that may be used for increasing the safety of food products. Antimicrobial data of these compounds may provide more information concerning different ways to combat the emergent resistance of bacteria, by using sources from a natural origin.

## 4. Conclusions

In this article, the following *in vitro* properties of ginseng extract (GE), ferulic acid (FA), and a fermented noni juice powder (LFNJ) were studied: IC50, anti-lipid peroxidation, inhibition of PPO activity, and effect on lag time, maximum growth rate, and asymptotic value in the growth curves of 13 pathogenic strains.

GE decreased the activity of mushroom-derived PPO and caused the complete inhibition of *Listeria monocytogenes* 1/2a when used at 16.5 mg/mL. It also extended the lag phase of *E. aerogenes* and *E. coli* O157:H7 at 8.2 mg/mL. FA, in turn, showed potential to be used as an antioxidant, as its IC50 was 0.45 mg/mL and it showed a delay of lipid peroxidation in olive oil. FA also showed antimicrobial effects: the MIC value for *S. aureus,* and *L. monocytogenes* 4b and 1/2 was 5.0 mg/mL, and for *S.* Montevideo *and S.* Gaminara was 7.5 mg/mL. FNJP proved to be antioxidant and a natural inhibitor of PPO in apple, mushroom and potato. It also acted as an antimicrobial agent, including the complete inhibition of *B. cereus* at concentrations of 12.5 mg/mL.

As shown in this article, GE, FA and FNJP might constitute control strategies for food preservation. These products, which can be obtained from natural sources, may constitute an added value for the food products. However, data shown hereby has been obtained *in vitro* and within a limited range of concentrations. As discussed above, further *in vivo* studies in food matrices should be carried out due to their complex composition and the interaction with the different intrinsic and extrinsic parameters, such as storage conditions, which may exert an effect on the activity of the compounds.

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## Conflict of interests

The authors declare no conflict of interests.

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**Table 1.** Bacterial strains used for the antimicrobial analyses

| **Specie** | | **Collection number** |
|---|---|---|
| *Listeria monocytogenes* | 4b | CECT[1]-935 |
| *Listeria monocytogenes* | 1/2 a | Isoleated in Lab |
| *Listeria monocytogenes* | 1/2 | CECT-4031 |
| *Salmonella enterica* subsp. Enterica | Typhimurium | CECT-4594 |
| *Salmonella enterica* subsp. Enterica | Agona | ATCC[2] BAA-707 |
| *Salmonella entérica* subsp. Enterica | Montevideo | ATCC BAA-710 |
| *Salmonella enterica* subsp. Enterica | Gaminara | ATCC BAA-711 |
| *Escherichia. coli* (virulent factor deleted) | O157:H7 | NCTC[3]-12900 |
| *Escherichia. coli* | | CECT-516 |
| *Staphylococcus. aureus* | | CECT-435 |
| *Bacillus cereus* | | CECT-131 |
| *Enterococcus faecalis* | | CECT-795 |
| *Enterobacter aerogenes* | | CECT-684 |

[1] Colección Española de Cultivos Tipo

[2] American Type Culture Collection

[3] National Collection of Type Cultures

**Table 2.** Inhibition of potato-, apple- or mushroom-derived polyphenol oxidase (PPO) activity, caused by ginseng extract (GE), ferulic acid (FA), and lyophilized of a spontaneously fermented noni juice (FNJP). Different letters indicate significant differences ($p < 0.05$) among extract tested concentration according to a Tukkey's Honest Significant Difference test

| **Extract** | **Concentration (mg/mL)** | **Inhibition of potato PPO (%)** | **Inhibition of apple PPO (%)** | **Inhibition of mushroom PPO (%)** |
|---|---|---|---|---|
| GE | 33.0 | No inhibition | 33.9 ± 2.5 [a] | No inhibition |
| | 25.0 | No inhibition | 24.2 ± 2.6 [b] | No inhibition |
| | 16.5 | No inhibition | 16.3 ± 1.9 [c] | No inhibition |
| FA | 7.5 | 37.8 ± 1.0 [a] | 41.7 ± 1.4 [a] | 73.6 ± 4.29 [a] |
| | 5.0 | 35.8 ± 1.4 [a] | 35.3 ± 9.9 [b] | 42.9 ± 0.05 [b] |
| | 2.5 | 36.3 ± 7.4 [a] | 21.2 ± 1.9 [b] | 39.4 ± 1.07 [b] |
| FNJP | 100.0 | 86.3 ± 7.7 [a] | 71.7 ± 6.3 [a] | 89.5 ± 7.6 [a] |
| | 75.0 | 78.5 ± 1.0 [a] | 86.3 ± 5.8 [ab] | 74.0 ± 0.4 [a] |
| | 50.0 | 86.7 ± 5.9 [a] | 95.1 ± 1.3 [b] | 59.1 ± 2.4 [a] |

**Table 3**. Effect of **10 mg/mL** of ginseng extract (GE), ferulic acid (FA) and fermented noni juice powder (FNJP) on the peroxidation of three different fats, expressed as induction time (h). Values are the mean ± standard deviation of 4 reps. Different letters indicate significant differences ($p < 0.05$) among extract tested according to a Tukkey's Honest Significant Difference test

| **Extract** | **Sunflower oil** | **Olive oil** | **Butter** |
|---|---|---|---|
| - | 2.42 ± 0.18 [a] | 15.13 ± 0.09 [b] | 1.46 ± 0.31 [ab] |
| GE | 2.43 ± 0.08 [a] | 14.76 ± 0.13 [b] | 1.76 ± 0.10 [a] |
| FA | 2.10 ± 0.91[a] | 37.57 ± 4.86 [a] | 1.50 ± 0.31 [ab] |
| FNJP | 1.81 ± 0.19 [a] | 14.31 ± 0.18 [b] | 1.11 ± 0.83 [b] |

**Table 4.** Effect of ginseng extract (GE) on lag time (λ, min), maximum growth rate (μ, ΔD· $10^3$/s), and asymptotic value (A, optical density) of the modelized growth of foodborne bacterial strains. For each strain and kinetic parameter, different letters indicate significant differences ($p < 0.05$) among extract tested concentration according to a Tukkey's Honest Significant Difference test.

| Microorganism | Strain | P | Control | 0.7 mg/mL | 1.4 mg/mL | 2.7 mg/mL | 5.5 mg/mL | 11.0 mg/mL |
|---|---|---|---|---|---|---|---|---|
| ***Listeria monocytogenes* 4b** | CECT -935 | λ | 283.6 ± 3.7 $^{a}$ | 248.6 ± 12.3 $^{ab}$ | 241.4 ± 8.9 $^{b}$ | 253.0 ± 23.1 $^{b}$ | 269.8 ± 3.7 $^{ab}$ | 236.4 ± 6.2 $^{b}$ |
| | | μ | 0.66 ± 0.02 $^{a}$ | 0.60 ± 0.05 $^{ab}$ | 0.58 ± 0.03 $^{abc}$ | 0.51 ± 0.04 $^{bc}$ | 0.48 ± 0.02 $^{cd}$ | 0.41 ± 0.01 $^{d}$ |
| | | A | 0.18 ± 0.00 $^{ab}$ | 0.20 ± 0.00 $^{a}$ | 0.19 ± 0.01 $^{a}$ | 0.19 ± 0.02 $^{a}$ | 0.15 ± 0.02 $^{bc}$ | 0.14 ± 0.00 $^{c}$ |
| ***L. monocytogenes 1/2 a*** | Lab | λ | 422.4 ± 33.8 $^{a}$ | 530.7 ± 187.7 $^{a}$ | 633.4 ± 167.5 $^{a}$ | 1261.5 ± 510.5 $^{b}$ | c.i. | c.i. |
| | | μ | 0.46 ± 0.08 $^{a}$ | 0.28 ± 0.14 $^{a}$ | 0.28 ± 0.25 $^{a}$ | 0.46 ± 0.01 $^{a}$ | c.i. | c.i. |
| | | A | 0.17 ± 0.02 $^{a}$ | 0.16 ± 0.05 $^{a}$ | 0.14 ± 0.01 $^{a}$ | 0.08 ± 0.09 $^{a}$ | c.i. | c.i. |
| ***Listeria monocytogenes 1/2*** | CECT-4031 | λ | 337.1 ± 10.1 $^{ab}$ | 336.3 ± 8.8 $^{ab}$ | 315.0 ± 17.2 $^{abc}$ | 291.1 ± 4.6 $^{bc}$ | 285.9 ± 8.3 $^{c}$ | 253.7 ± 25.2 $^{c}$ |
| | | μ | 0.99 ± 0.06 $^{a}$ | 0.97 ± 0.15 $^{a}$ | 0.83 ± 0.07 $^{ab}$ | 0.75 ± 0.06 $^{ab}$ | 0.65 ± 0.09 $^{b}$ | 0.42 ± 0.12 $^{c}$ |
| | | A | 0.22 ± 0.02 $^{a}$ | 0.25 ± 0.03 $^{a}$ | 0.23 ± 0.03 $^{a}$ | 0.22 ± 0.01 $^{a}$ | 0.20 ± 0.02 $^{a}$ | 0.20 ± 0.03 $^{a}$ |
| ***Salmonella enterica* subsp. Enterica Typhimurium** | CECT -4594 | λ | 30.0 ± 0.1 $^{a}$ | 29.7 ± 2.1 $^{a}$ | 32.7 ± 8.0 $^{a}$ | 37.2 ± 7.0 | 35.0 ± 2.1 $^{a}$ | 34.7 ± 4.6 $^{a}$ |
| | | μ | 0.96 ± 0.05 $^{a}$ | 0.76 ± 0.5 $^{a}$ | 1.10 ± 0.7 $^{a}$ | 1.37 ± 0.3 $^{a}$ | 1.32 ± 0.05 $^{a}$ | 1.37 ± 0.13 $^{a}$ |
| | | A | 0.29 ± 0.09 $^{a}$ | 0.21 ± 0.02 $^{a}$ | 0.25 ± 0.06 $^{a}$ | 0.23 ± 0.04 $^{a/}$ | 0.20 ± 0.02 $^{a}$ | 0.23 ± 0.01 $^{a}$ |
| ***Salmonella enterica* subsp. Enterica Agona** | ATCC BAA-707 | λ | 171.2 ± 5.2 $^{a}$ | 161.0 ± 4.3 $^{a}$ | 159.6 ± 8.3 $^{a}$ | 164.7 ± 2.9 $^{a}$ | 164.6 ± 2.3 $^{a}$ | 162.8 ± 3.13 $^{a}$ |
| | | μ | 1.13 ± 0.08 $^{ab}$ | 1.28 ± 0.12 $^{a}$ | 1.09 ± 0.12 $^{ab}$ | 1.06 ± 0.08 $^{ab}$ | 1.00 ± 0.03 $^{b}$ | 1.01 ± 0.03 $^{b}$ |
| | | A | 0.19 ± 0.00 $^{a}$ | 0.19 ± 0.01$^{a}$ | 0.19 ± 0.01 $^{a}$ | 0.17 ± 0.01 $^{b}$ | 0.17 ± 0.01 $^{b}$ | 0.18 ± 0.01 $^{ab}$ |

| Microorganism | Strain | P | Control | 0.7 mg/mL | 1.4 mg/mL | 2.7 mg/mL | 5.5 mg/mL | 11.0 mg/mL |
|---|---|---|---|---|---|---|---|---|
| ***Salmonella entérica*** **subsp. Enterica Montevideo** | ATCC BAA-710 | λ | 120.7 ± 17.8 [a] | 137.2 ± 3.5 [a] | 141.8 ± 6.4 [a] | 130.3 ± 1.2 [a] | 131.0 ± 23.5 [a] | 148.3 ± 2.0 [a] |
| | | μ | 0.90 ± 0.03 [a] | 0.94 ± 0.06 [a] | 0.99 ± 0.05 [a] | 0.94 ± 0.02 [a] | 0.96 ± 0.04 [a] | 0.96 ± 0.04 [a] |
| | | A | 0.22 ± 0.01 [a] | 0.20 ± 0.01 [a] | 0.19 ± 0.01 [a] | 0.18 ± 0.01 [a] | 0.19 ± 0.04 [a] | 0.18 ± 0.01 [a] |
| ***Salmonella enterica*** **subsp. Enterica Gaminara** | ATCC BAA-711 | λ | 169.0 ± 9.2 [a] | 165.5 ± 2.6 [a] | 162.5 ± 0.3 [a] | 172.1 ± 10.8 [a] | 177.5 ± 8.1 [a] | 177.3 ± 7.4 [a] |
| | | μ | 1.75 ± 0.53 [a] | 1.48 ± 0.38 [a] | 1.32 ± 0.14 [a] | 1.55 ± 0.15 [a] | 1.68 ± 0.13 [a] | 1.49 ± 0.15 [a] |
| | | A | 0.27 ± 0.08 [a] | 0.26 ± 0.03 [a] | 0.25 ± 0.02 [a] | 0.27 ± 0.03 [a] | 0.24 ± 0.01 [a] | 0.24 ± 0.02 [a] |
| ***Escherichia coli*** **(virulent factor deleted)** | NCTC -12900 | λ | 131.8 ± 7.2 [a] | 130.2 ± 7.1 [a] | 112.8 ± 1.9 [bc] | 115.9 ± 6.1 [c] | 122.1 ± 2.5 [ab] | 130. ± 0.0 [ab] |
| | | μ | 2.46 ± 0.83 [a] | 1.39 ± 0.05 [b] | 1.41 ± 0.12 [b] | 1.46 ± 0.14 [b] | 1.36 ± 0.10 [b] | 1.34 ± 0.02 [b] |
| | | A | 0.42 ± 0.02 [a] | 0.49 ± 0.04 [a] | 0.42 ± 0.03 [a] | 0.42 ± 0.06 [a] | 0.43 ± 0.04 [a] | 0.31 ± 0.00 [a] |
| ***Escherichia coli*** | CECT -516 | λ | 61.1 ± 9.7 [a] | 106.6 ± 4.5 [ab] | 112.0 ± 5.9 [b] | 110.5 ± 1.4 [b] | 113.1 ± 1.3 [b] | 111.9 ± 4.0 [b] |
| | | μ | 1.42 ± 0.14 [a] | 0.71 ± 0.01 [d] | 1.02 ± 0.02 [bc] | 1.23 ± 0.12 [ab] | 0.95 ± 0.10 [cd] | 0.90 ± 0.06 [cd] |
| | | A | 0.31 ± 0.02 [a] | 0.23 ± 0.01 [b] | 0.25 ± 0.01 [b] | 0.27 ± 0.04 [ab] | 0.23 ± 0.03 [b] | 0.20 ± 0.01 [b] |
| ***Staphylococcus aureus*** | CECT-435 | λ | 413.9 ± 27.3 [a] | 404.3 ± 7.0 [ab] | 377.2 ± 3.0 [bc] | 426.9 ± 3.0 [a] | 423.1 ± 8.1 [a] | 359.0 ± 3.5 [c] |
| | | μ | 0.55 ± 0.1 [a] | 0.59 ± 0.03 [a] | 0.53 ± 0.04 [a] | 0.59 ± 0.07 a | 0.55 ± 0.05 [a] | 0.44 ± 0.01 [a] |
| | | A | 0.18 ± 0.02 [a] | 0.17 ± 0.01 [ab] | 0.15 ± 0.01 [ab] | 0.15 ± 0.01 [ab] | 0.14 ± 0.01 [b] | 0.15 ± 0.01 [ab] |
| ***Bacillus cereus*** | CECT -131 | λ | 64.6 ± 1.8 [b] | 59.5 ± 8.7 [b] | 69.1 ± 5.6 [b] | 80.1 ± 2.2 [b] | 83.6 ± 15.5 [b] | 155.0 ± 26.0 [a] |
| | | μ | 1.03 ± 0.30 [ab] | 0.89 ± 0.24 [ab] | 1.12 ± 0.04 [a] | 1.14 ± 0.04 [a] | 1.03 ± 0.12 [ab] | 0.59 ± 0.10 [b] |
| | | A | 0.22 ± 0.01 [a] | 0.23 ± 0.02 [a] | 0.21 ± 0.01 [a] | 0.21 ± 0.01 [a] | 0.21 ± 0.01 [a] | 0.15 ± 0.04 [b] |
| ***Enterococcus faecalis*** | CECT -795 | λ | n.d. | n.d. | n.d. | n.d. | n.d. | n.d. |
| | | μ | 0.74 ± 0.06 [ab] | 0.78 ± 0.09 [a] | 0.80 ± 0.02 [a] | 0.75 ± 0.01 [ab] | 0.64 ± 0.01 [b] | 0.64 ± 0.02 [b] |

| Microorganism | Strain | P | Control | 0.7 mg/mL | 1.4 mg/mL | 2.7 mg/mL | 5.5 mg/mL | 11.0 mg/mL |
|---|---|---|---|---|---|---|---|---|
| | | A | 0.22 ± 0.01 [a] | 0.23 ± 0.01 [a] | 0.22 ± 0.01 [a] | 0.23 ± 0.01 [a] | 0.23 ± 0.01 [a] | 0.24 ± 0.03 [a] |
| ***Enterobacter aerogenes*** | CECT -684 | λ | 26.1 ± 15.4 [a] | 25.0 ± 23.2 [a] | 48.7 ± 1.5 [b] | 49.0 ± 19.8 [b] | 50.0 ± 16.4 [b] | 60.0 ± 0.0 [b] |
| | | μ | 2.09 ± 0.23 [a] | 2.11 ± 0.08 [a] | 2.07 ± 0.08 [a] | 2.05 ± 0.07 [a] | 1.67 ± 0.09 [b] | 1.40 ± 0.04 [b] |
| | | A | 0.97 ± 0.01 [a] | 0.98 ± 0.06 [a] | 1.02 ± 0.00 [a] | 0.99 ± 0.00 [a] | 0.88 ± 0.01 [b] | 0.87 ± 0.03 [b] |

c.i.: complete inhibition

**Table 5.** Effect of ferulic acid (FA) on lag time (λ, min), maximum growth rate (μ, ΔD· $10^3$/s), and asymptotic value (A, optical density) of modelized growth of foodborne bacterial strains. For each strain and kinetic parameter, different letters indicate significant differences ($p < 0.05$) among extract tested concentration according to a Tukkey's Honest Significant Difference test

| Microorganism | Strain | P | Control | 0.2 mg/mL | 0.4 mg/mL | 0.8 mg/mL | 1.7 mg/mL | 2.5 mg/mL | 3.3 mg/mL |
|---|---|---|---|---|---|---|---|---|---|
| ***Listeria monocytogenes* 4b** | CECT - 935 | λ | 398.5 ± 2.8 [a] | 335.9 ± 37.9 [a] | 343.1 ± 37.8 [a] | 473.9 ± 15.8 [b] | c.i. | c.i. | c.i. |
| | | μ | 1.03 ± 0.12 [a] | 0.27 ± 0.06 [b] | 0.20 ± 0.01 [bc] | 0.08 ± 0.03 [c] | c.i. | c.i. | c.i. |
| | | A | 0.25 ± 0.01 [a] | 0.13 ± 0.02 [b] | 0.11 ± 0.01 [b] | 0.06 ± 0.01 [c] | c.i. | c.i. | c.i. |
| ***Listeria monocytogenes 1/2 a*** | Lab | λ | 356.4 ± 11.6 [a] | 371.4 ± 48.5 [a] | 372.1 ± 48.7 [a] | 466.1 ± 87.6 [a] | 492.1 ± 43.3 [a] | c.i. | c.i. |
| | | μ | 1.19 ± 0.19 [a] | 0.53 ± 0.07 [b] | 0.53 ± 0.07 [b] | 0.61 ± 0.11 [b] | 0.55 ± 0.20 [b] | c.i. | c.i. |
| | | A | 0.33 ± 0.02 [ab] | 0.36 ± 0.04 [a] | 0.36 ± 0.04 [a] | 0.22 ± 0.08 [bc] | 0.17 ± 0.01 [c] | c.i. | c.i. |
| ***Listeria monocytogenes 1/2*** | CECT-4031 | λ | 432.5 ± 4.3 [a] | 446.6 ± 9.1 [a] | 530.9 ± 11.5 [a] | 1240.0 ± 143.0 [b] | c.i. | c.i. | c.i. |
| | | μ | 0.63 ± 0.06 [a] | 0.30 ± 0.01 [b] | 0.20 ± 0.01 [c] | 0.03 ± 0.01 [d] | c.i. | c.i. | c.i. |
| | | A | 0.17 ± 0.01 [a] | 0.11 ± 0.01 [b] | 0.08 ± 0.01 [c] | 0.03 ± 0.01 [d] | c.i. | c.i. | c.i. |
| ***Salmonella enterica* subsp. Enterica Typhimurium** | CECT - 4594 | λ | 127.5 ± 8.3 [a] | 113.4 ± 24.2 [a] | 112.6 ± 29.1 [a] | 127.9 ± 17.7 [a] | 144.1 ± 3.9 [ab] | 172.3 ± 29.8 [ab] | 1932. ± 31.3 [b] |
| | | μ | 1.76 ± 0.50 [a] | 1.32 ± 0.41 [a] | 1.40 ± 0.90 [a] | 1.46 ± 0.59 [a] | 1.45 ± 0.62 [a] | 1.17 ± 0.45 [a] | 0.87 ± 0.23 [a] |
| | | A | 0.29 ± 0.05 [a] | 0.29 ± 0.07 [a] | 0.23 ± 0.07 [a] | 0.21 ± 0.04 [a] | 0.20 ± 0.04 [a] | 0.17 ± 0.05 [a] | 0.15 ± 0.05 [a] |
| ***Salmonella enterica*** | | λ | 181.8 ± 4.7 [a] | 179.9 ± 5.6 [a] | 155.2 ± 29.2 [a] | 168.8 ± 5.5 [a] | 197.3 ± 20.4 [a] | 1129.88 ± 476.2 [b] | c.i. |

| Microorganism | Strain | P | Control | 0.2 mg/mL | 0.4 mg/mL | 0.8 mg/mL | 1.7 mg/mL | 2.5 mg/mL | 3.3 mg/mL |
|---|---|---|---|---|---|---|---|---|---|
| **subsp. Enterica Agona** | ATCC BAA-707 | μ | 1.16 ± 0.02 [a] | 0.89 ± 0.19 [a] | 0.84 ± 0.17 [ab] | 0.69 ± 0.10 [ab] | 0.57 ± 0.53 [ab] | 0.20 ± 0.11 [b] | c.i. |
| | | A | 0.15 ± 0.01 [a] | 0.12 ± 0.01 [a] | 0.11 ± 0.01 [a] | 0.10 ± 0.01 [a] | 0.10 ± 0.05 [a] | 0.14 ± 0.04 [a] | c.i. |
| ***Salmonella entérica* subsp. Enterica Montevideo** | ATCC BAA-710 | λ | 105.9 ± 5.1 [a] | 141.8 ± 3.2 [a] | 127.0 ± 6.7 [a] | 149.9 ± 25.6 [a] | 137.0 ± 40.3 [a] | c.i. | c.i. |
| | | μ | 0.80 ± 0.04 [a] | 0.89 ± 0.03 [a] | 0.76 ± 0.06 [a] | 0.74 ± 0.03 [ab] | 0.36 ± 0.03 [b] | c.i. | c.i. |
| | | A | 0.23 ± 0.01 [a] | 0.14 ± 0.01 [b] | 0.14 ± 0.01 [b] | 0.13 ± 0.01 [bc] | 0.12 ± 0.01 [c] | c.i. | c.i. |
| ***Salmonella enterica* subsp. Enterica Gaminara** | ATCC BAA-711 | λ | 165.5 ± 13.6 [a] | 181.7 ± 6.3 [ab] | 180.7 ± 4.1 [ab] | 194.4 ± 1.6 [b] | 182.5 ± 9.9 [ab] | c.i. | c.i. |
| | | μ | 1.38 ± 0.54 [a] | 0.87 ± 0.10 [ab] | 0.95 ± 0.04 [ab] | 0.86 ± 0.24 [ab] | 0.60 ± 0.02 [b] | c.i. | c.i. |
| | | A | 0.41 ± 0.13 [a] | 0.20 ± 0.01 [b] | 0.20 ± 0.01 [b] | 0.17 ± 0.01 [b] | 0.15 ± 0.01 [b] | c.i. | c.i. |
| ***Escherichia coli* (virulent factor deleted)** | NCTC - 12900 | λ | 141.5 ± 24.2 [a] | 143.8 ± 0.8 [a] | 131.7 ± 39.5 [a] | 203.4 ± 3.0 [ab] | 247.0 ± 21.1 [b] | 254.3 ± 34.1 [b] | c.i. |
| | | μ | 2.19 ± 0.87 [a] | 1.20 ± 0.02 [abc] | 1.03 ± 0.29 [bc] | 1.40 ± 0.05 [ab] | 0.71 ± 0.07 [bc] | 0.26 ± 0.04 [c] | c.i. |
| | | A | 0.43 ± 0.05 [b] | 0.57 ± 0.05 [a] | 0.34 ± 0.09 [bc] | 0.39 ± 0.01 [b] | 0.24 ± 0.03 [cd] | 0.11 ± 0.03 [d] | c.i. |
| ***Escherichia coli*** | CECT - 516 | λ | 126.2 ± 0.3 [a] | 112.3 ± 32.4 [a] | 71.3 ± 13.7 [a] | 95.5 ± 59.6 [a] | 136.3 ± 10.0 [a] | 143.4 ± 6.2 [a] | c.i. |
| | | μ | 2.26 ± 0.01 [a] | 1.44 ± 0.14 [ab] | 1.24 ± 0.11 [b] | 1.60 ± 0.65 [ab] | 1.97 ± 0.32 [ab] | 1.44 ± 0.37 [ab] | c.i. |
| | | A | 0.32 ± 0.01 [a] | 0.36 ± 0.01 [a] | 0.34 ± 0.01 [a] | 0.29 ± 0.04 [a] | 0.21 ± 0.03 [a] | 0.18 ± 0.04 [a] | c.i. |
| ***Staphylococcus aureus*** | CECT-435 | λ | 236.5 2.2 [a] | 236.6 17.8 [a] | 237.0 18.2 [a] | 328.9 70.0 [b] | c.i. | c.i. | c.i. |
| | | μ | 1.50 0.02 [a] | 1.17 0.15 [b] | 0.91 0.10 [b] | 0.37 0.01 [c] | c.i. | c.i. | c.i. |
| | | A | 0.43 0.01 [a] | 0.35 0.01 [b] | 0.29 0.02 [c] | 0.13 0.05 [d] | c.i. | c.i. | c.i. |
| | | λ | 263.8 ± 1.0 [a] | 865.3 ± 100.6 [a] | 1112.9 ± 183.8 [ab] | 1178.8 ± 42.8 [ab] | 1425.7 ± 289.9 [ab] | 2183.8 ± 187.9 [b] | c.i. |

| Microorganism | Strain | P | Control | 0.2 mg/mL | 0.4 mg/mL | 0.8 mg/mL | 1.7 mg/mL | 2.5 mg/mL | 3.3 mg/mL |
|---|---|---|---|---|---|---|---|---|---|
| ***Bacillus cereus*** | CECT - 131 | μ | 0.48 ± 0.12 [a] | 0.63 ± 0.35 [a] | 0.77 ± 0.32 [a] | 0.84 ± 0.39 [a] | 0.45 ± 0.21 [a] | 0.57 ± 0.13 [a] | c.i. |
| | | A | 0.74 ± 0.22 [a] | 0.79 ± 0.20 [a] | 0.81 ± 0.19 [a] | 0.79 ± 0.27 [a] | 0.63 ± 0.37 [a] | 0.38 ± 0.13 [b] | c.i. |
| ***Enterococcus faecalis*** | CECT - 795 | λ | 761.4 ± 32.3 [a] | 689.4 ± 102.7 [a] | 502.3 ± 44.8 [a] | 492.5 ± 177.2 [a] | c.i. | c.i. | c.i. |
| | | μ | 0.80 ± 0.10 [a] | 0.43 ± 0.29 [a] | 0.37 ± 0.10 [a] | 0.44 ± 0.15 [a] | c.i. | c.i. | c.i. |
| | | A | 0.18 ± 0.03 [a] | 0.19 ± 0.06 [a] | 0.16 ± 0.01 [a] | 0.12 ± 0.04 [a] | c.i. | c.i. | c.i. |
| ***Enterobacter aerogenes*** | CECT - 684 | λ | 185.0 ± 24.7 [a] | 204.2 ± 19.1 [a] | 182.9 ± 39.3 [a] | 174.1 ± 14.8 [a] | 154.3 ± 27.2 [a] | 152.9 ± 46.5 [a] | c.i. |
| | | μ | 2.25 ± 0.59 [a] | 2.08 ± 0.24 [ab] | 1.66 ± 0.22 [ab] | 1.37 ± 0.11 [bc] | 0.69 ± 0.06 [c] | 0.60 ± 0.13 [c] | c.i. |
| | | A | 0.64 ± 0.14 [a] | 0.74 ± 0.07 [a] | 0.65 ± 0.05 [a] | 0.59 ± 0.06 [a] | 0.18 ± 0.05 [b] | 0.15 ± 0.05 [b] | c.i. |

c.i.: complete inhibition

**Table 6.** Effect of the fermented noni juice powder (FNJP) on lag time (λ, min). Maximum growth rate (μ, ΔD· $10^3$/s). and asymptotic value (A, optical density) of the modelized growth of foodborne bacterial strains. For each strain and kinetic parameter, different letters indicate significant differences ($p < 0.05$) among extract tested concentration according to a Tukkey's Honest Significant Difference test.

| Microorganism | Strain | P | Control | 2.1 mg/mL | 4.2mg/mL | 8.3 mg/mL | 16.7 mg/mL | 33.3 mg/mL |
|---|---|---|---|---|---|---|---|---|
| ***Listeria monocytogenes* 4b** | CECT -935 | λ | 283.6 ± 3.7 $^{b}$ | 216.7 ± 8.0 $^{a}$ | 213.4 ± 18.3 $^{a}$ | 308.2 ± 23.6 $^{b}$ | c.i. | c.i. |
| | | μ | 0.66 ± 0.02 $^{a}$ | 0.49 ± 0.06 $^{b}$ | 0.46 ± 0.07 $^{b}$ | 0.26 ± 0.04 $^{c}$ | c.i. | c.i. |
| | | A | 0.18 ± 0.00 $^{a}$ | 0.17 ± 0.00 $^{a}$ | 0.18 ± 0.02 $^{a}$ | 0.09 ± 0.03 $^{b}$ | c.i. | c.i. |
| ***Listeria monocytogenes 1/2 a*** | Lab | λ | 422.4 ± 33.8 $^{a}$ | 487.0 ± 14.4 $^{a}$ | 495.7 ± 15.2 $^{a}$ | 737.4 ± 11.6 $^{b}$ | c.i. | c.i. |
| | | μ | 0.46 ± 0.08 $^{a}$ | 0.29 ± 0.07 $^{b}$ | 0.32 ± 0.03 $^{ab}$ | 0.17 ± 0.01 $^{c}$ | c.i. | c.i. |
| | | A | 0.17 ± 0.02 $^{ab}$ | 0.18 ± 0.03 $^{ab}$ | 0.19 ± 0.01 $^{a}$ | 0.13 ± 0.02 $^{b}$ | c.i. | c.i. |
| ***Listeria monocytogenes 1/2*** | CECT -4031 | λ | 337.1 ± 10.1 $^{a}$ | 257.4 ± 25.4 $^{b}$ | 281.9 ± 6.1 $^{b}$ | 418.7 ± 14.8 $^{c}$ | c.i. | c.i. |
| | | μ | 0.99 ± 0.06 $^{a}$ | 0.69 ± 0.14 $^{b}$ | 0.61 ± 0.24 $^{b}$ | 0.51 ± 0.13 $^{b}$ | c.i. | c.i. |
| | | A | 0.22 ± 0.02 $^{a}$ | 0.27 ± 0.05 $^{b}$ | 0.26 ± 0.05 $^{b}$ | 0.18 ± 0.04 $^{b}$ | c.i. | c.i. |
| ***Salmonella enterica* subsp. Enterica Typhimurium** | CECT -4594 | λ | 30.0 ± 0.1 $^{a}$ | 30.0 ± 0.1 $^{a}$ | 30.0 ± 0.1 $^{a}$ | 38.5 ± 2.7 $^{b}$ | c.i. | c.i. |
| | | μ | 0.95 ± 0.05 $^{a}$ | 0.6 ± 0.05 $^{b}$ | 0.44 ± 0.01 $^{c}$ | 0.20 ± 0.03 $^{d}$ | c.i. | c.i. |
| | | A | 0.29 ± 0.09 $^{a}$ | 0.19 ± 0.09 $^{ab}$ | 0.15 ± 0.01 $^{b}$ | 0.09 ± 0.00 $^{b}$ | c.i. | c.i. |
| ***Salmonella enterica* subsp. Enterica Agona** | ATCC BAA-707 | λ | 171.2 ± 5.2 $^{a}$ | 156.2 ± 1.4 $^{a}$ | 156.6 ± 4.1 $^{a}$ | 233.4 ± 2.1 $^{a}$ | 268.1 ± 14.4 $^{b}$ | c.i. |
| | | μ | 1.13 ± 0.08 $^{a}$ | 0.88 ± 0.13 $^{b}$ | 0.85 ± 0.01 $^{b}$ | 0.69 ± 0.06 $^{b}$ | 0.37 ± 0.03 $^{c}$ | c.i. |
| | | A | 0.19 ± 0.00 $^{a}$ | 0.17 ± 0.01 $^{b}$ | 0.15 ± 0.00 $^{c}$ | 0.13 ± 0.00 $^{d}$ | 0.06 ± 0.01 $^{e}$ | c.i. |

| Microorganism | Strain | P | Control | 2.1 mg/mL | 4.2mg/mL | 8.3 mg/mL | 16.7 mg/mL | 33.3 mg/mL |
|---|---|---|---|---|---|---|---|---|
| ***Salmonella entérica* subsp. Enterica Montevideo** | ATCC BAA-710 | λ | 120.7 ± 17.8 $^{a}$ | 136.6 ± 15.0 $^{a}$ | 125.6 ± 3.8 $^{a}$ | 209.3 ± 5.5 $^{a}$ | 243.2 ± 13.1 $^{b}$ | c.i. |
| | | µ | 0.90 ± 0.03 $^{a}$ | 0.83 ± 0.06 $^{a}$ | 0.77 ± 0.03 $^{a}$ | 0.90 ± 0.05 $^{a}$ | 0.50 ± 1.13 $^{b}$ | c.i. |
| | | A | 0.22 ± 0.01 $^{a}$ | 0.16 ± 0.01 $^{b}$ | 0.15 ± 0.01 $^{b}$ | 0.15 ± 0.01 $^{b}$ | 0.1 ± 0.02 $^{c}$ | c.i. |
| ***Salmonella enterica* subsp. Enterica Gaminara** | ATCC BAA-711 | λ | 169.0 ± 9.2 $^{a}$ | 149.9 ± 15.5 $^{a}$ | 144.2 ± 14.6 $^{a}$ | 259.1 ± 2.0 $^{a}$ | 309.1 ± 14.8 $^{b}$ | c.i. |
| | | µ | 1.75 ± 0.53 $^{a}$ | 1.19 ± 0.13 $^{ab}$ | 0.93 ± 0.10 $^{ab}$ | 0.96 ± 0.12b $^{c}$ | 0.41 ± 0.09 $^{c}$ | c.i. |
| | | A | 0.27 ± 0.08 $^{a}$ | 0.24 ± 0.01 $^{a}$ | 0.20 ± 0.02 $^{a}$ | 0.23 ± 0.02 $^{a}$ | 0.06 ± 0.01 $^{b}$ | c.i. |
| ***Escherichia coli* (virulent factor deleted)** | NCTC - 12900 | λ | 131.8 ± 7.2 $^{ab}$ | 116.7 ± 26.4 $^{b}$ | 157.8 ± 1.9 $^{a}$ | 194.2 ± 7.3 $^{c}$ | c.i. | c.i. |
| | | µ | 2.46 ± 0.83 $^{a}$ | 1.35 ± 0.09 $^{a}$ | 1.68 ± 0.07 $^{a}$ | 1.57 ± 0.04 $^{a}$ | c.i. | c.i. |
| | | A | 0.42 ± 0.02 $^{ab}$ | 0.50 ± 0.02 $^{a}$ | 0.47 ± 0.02 $^{bc}$ | 0.38 ± 0.01 $^{c}$ | c.i. | c.i. |
| ***Escherichia coli*** | CECT -516 | λ | 61.1 ± 9.7 $^{a}$ | 76.0 ± 32.7 $^{a}$ | 142.0 ± 19.9 $^{b}$ | 189.4 ± 6.8 $^{b}$ | 205.2 ± 7.8 $^{c}$ | c.i. |
| | | µ | 1.42 ± 0.14 $^{abc}$ | 1.07 ± 0.14 $^{bc}$ | 2.02 ± 0.10 $^{ab}$ | 2.07 ± 1.01 $^{b}$ | 0.19 ± 0.02 $^{c}$ | c.i. |
| | | A | 0.31 ± 0.02 $^{a}$ | 0.39 ± 0.03 $^{a}$ | 0.44 ± 0.03 $^{a}$ | 0.41 ± 0.17 $^{a}$ | 0.04 ± 0.00 $^{b}$ | c.i. |
| ***Staphylococcus aureus*** | CECT - 435 | λ | 413.9 ± 27.3 $^{bc}$ | 375.6 ± 4.1 $^{ab}$ | 361.4 ± 20.2 $^{a}$ | 463.2 ± 18.6 $^{c}$ | c.i. | c.i. |
| | | µ | 0.55 ± 0.11 $^{a}$ | 0.46 ± 0.01 $^{ab}$ | 0.42 ± 0.01 $^{ab}$ | 0.28 ± 0.12b $^{c}$ | c.i. | c.i. |
| | | A | 0.18 ± 0.02 $^{a}$ | 0.15 ± 0.01 $^{a}$ | 0.13 ± 0.01 $^{a}$ | 0.07 ± 0.03 $^{b}$ | c.i. | c.i. |
| ***Bacillus cereus*** | CECT -131 | λ | 64.4 ± 1.3 $^{a}$ | 64.4 ± 1.3 $^{a}$ | c.i. | c.i. | c.i. | c.i. |
| | | µ | 1.03 ± 0.30 $^{a}$ | 0.52 ± 0.30 $^{b}$ | c.i. | c.i. | c.i. | c.i. |
| | | A | 0.22 ± 0.01 $^{a}$ | 0.15 ± 0.01 $^{b}$ | c.i. | c.i. | c.i. | c.i. |
| ***Enterococcus faecalis*** | CECT -795 | λ | n.d. | n.d. | n.d. | n.d. | n.d. | c.i. |
| | | µ | 0.74 ± 0.06 $^{a}$ | 0.63 ± 0.02 $^{ab}$ | 0.47 ± 0.16 $^{ab}$ | 0.39 ± 0.05 $^{b}$ | 0.42 ± 0.01 $^{ab}$ | c.i. |

| Microorganism | Strain | P | Control | 2.1 mg/mL | 4.2mg/mL | 8.3 mg/mL | 16.7 mg/mL | 33.3 mg/mL |
|---|---|---|---|---|---|---|---|---|
| | | A | 0.26 ± 0.01 [a] | 0.26 ± 0.0 [a] | 0.24 ± 0.01 [b] | 0.24 ± 0.01 [b] | 0.24 ± 0.01 [b] | c.i. |
| ***Enterobacter aerogenes*** | CECT -684 | λ | 51.2 ± 20.1 [a] | 61.2 ± 10.8 [ab] | 74.8 ± 36.7 [ab] | 119 ± 27.8 [ab] | 153.7 ± 87.1 [ab] | 772.7 ± 36.8 [c] |
| | | μ | 2.09 ± 0.23 [ab] | 1.98 ± 0.28 [ab] | 2.57 ± 0.15 [a] | 2.21 ± 0.26 [ab] | 1.66 ± 0.21 [b] | 0.69 ± 0.45 [c] |
| | | A | 0.97 ± 0.01 [a] | 0.97 ± 0.11 [a] | 0.96 ± 0.01 [a] | 0.97 ± 0.05 [a] | 0.81 ± 0.15 [ab] | 0.46 ± 0.28 [b] |

c.i.: complete inhibition

**Table 7.** MIC values (mg/mL) of ginseng extract (GE), ferulic acid (FA) and fermented noni juice powder (FNJP) for the bacterial strains studied.

| Specie | Strain | GE | FA | FNJP |
|---|---|---|---|---|
| ***Listeria monocytogenes* 4b** | CECT -935 | > 11.0 | 1.7 | 16.7 |
| ***L. monocytogenes 1/2 a*** | Lab | 16.5 | 2.5 | 16.7 |
| ***Listeria monocytogenes 1/2*** | CECT-4031 | > 11.0 | 1.7 | 16.7 |
| ***Salmonella enterica* subsp. Enterica Typhimurium** | CECT -4594 | > 11.0 | > 3.3 | 16.7 |
| ***Salmonella enterica* subsp. Enterica Agona** | ATCC BAA-707 | > 11.0 | 3.3 | 33.3 |
| ***Salmonella entérica* subsp. Enterica Montevideo** | ATCC BAA-710 | > 11.0 | 2.5 | 33.3 |
| ***Salmonella enterica* subsp. Enterica Gaminara** | ATCC BAA-711 | > 11.0 | 2.5 | 33.3 |
| ***Escherichia coli* (virulent factor deleted)** | NCTC - 12900 | > 11.0 | 3.3 | 16.7 |
| ***Escherichia coli*** | CECT -516 | > 11.0 | 3.3 | 33.3 |
| ***Staphylococcus aureus*** | CECT- 435 | > 11.0 | 1.7 | 16.7 |
| ***Bacillus cereus*** | CECT -131 | > 11.0 | 3.3 | 4.1 |
| ***Enterococcus faecalis*** | CECT -795 | > 11.0 | 3.3 | 33.3 |
| ***Enterobacter aerogenes*** | CECT -684 | > 11.0 | 3.3 | > 33.3 |