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zucoppes@yahoo.com / zucoppes@fq.edu.uy

## Antioxidant Effects of Yerba Mate Tea

Hogans, V. J.<sup>1</sup>; Coppes Petricorena, Z. L.<sup>2</sup>; Harris, G. K.<sup>3</sup>; Leonard, S. S.<sup>4</sup>

<sup>1</sup>United States - <sup>1</sup>Health Effects laboratory Division, National Institute for Occupational Safety and Health Morgantown.; <sup>2</sup>Cátedra de Bioquímica, Faculty of Chemistry; <sup>3</sup>Department of Food Science, North Carolina State University; <sup>4</sup>Health Effects Laboratory Division, National Institute for Occupational Safety and Health

Yerba Mate (*Ilex paraguayensis*) tea is indigenous to Paraguay, Argentina, Brazil and Uruguay and is cultivated in other South American countries. The tea is made from the leaves of the Yerba Mate holly tree. The native Indians of Paraguay and Brazil have used Yerba Mate for health remedies since before the sixteenth century. The tea is traditionally believed to promote perspiration, immunity, digestion, memory, and weight loss. Our research explored the antioxidant effects of Yerba Mate (YM) tea using a chemical reaction, enzymatic reaction, and radicals produced by Cr(VI)-stimulated RAW 264.7 cells. We also investigated YM tea's effect on cellular O<sub>2</sub> consumption, DNA damage and lipid peroxidation. YM tea samples were prepared using an infusion method and several concentrations were used to measure antioxidant effects. An electron spin resonance trapping technique was used to analyze free radicals and the effect of YM tea. Results showed a significant decrease in trapped hydroxyl radicals produced from the Fenton reaction after addition of all concentrations of YM tea. A significant, yet less dramatic, reduction in superoxide radicals generated from an enzymatic xanthine/xanthine oxidase reaction was also seen with all concentrations of YM tea in a concentration dependent manner. The presence of YM tea significantly reduced Cr(VI)-induced radical formation in RAW 264.7 cells in a concentration dependent manner. Controls and measurements which confirm scavenging of the radicals, rather than the inhibition of enzymatic reactions were performed in all assays. We also performed the following cellular assays using RAW 264.7 cells: measurement of O<sub>2</sub> consumption, COMET assay for DNA damage, and the measurement of lipid peroxidation. Our results showed that YM tea reduced the amount of O<sub>2</sub> consumed in cells after stimulation by exposure to Cr(VI). YM tea also inhibited -OH radical generated DNA damage in a concentration dependent manner and inhibited lipid peroxidation in H<sub>2</sub>O<sub>2</sub>-exposed cells. These data indicate that YM tea possesses potent antioxidant and DNA-protective activity, properties that are common to natural anti-cancer agents. This study may help to explain the mechanisms behind the reported antioxidant and anti-cancer effects of YM tea. **Keywords:** Yerba Mate tea; ROS; ESR; DNA damage; Lipid peroxidation

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plaquetabr@yahoo.com.br

## Relation between antioxidants vitamins and cognitive impairments in institutionalized elderly women in Santa Maria - Brazil

Paniz, C. A.<sup>1</sup>; Boeira, S. P.<sup>1</sup>; Tessele, F.<sup>2</sup>; Bulcão, R. P.<sup>1</sup>; Charão, M. F.<sup>1</sup>; Tonello, R.<sup>1</sup>; Almeida, F. L.<sup>1</sup>; Roehrs, M.<sup>1</sup>; Garcia, S. C.<sup>1</sup>

<sup>1</sup>Brazil - <sup>1</sup>Federal University of Santa Maria; <sup>2</sup>Federal University of Rio Grande do Sul

The aging process comes being explained for evidences that point the free radicals, and consequently oxidative stress, as the main factors that influence the cellular longevity. To protect itself of the oxidative damages, the organism possess endogenous antioxidative substances, however, also it needs exogenous molecules to balance the pro-oxidant activity. As an example of exogenous constituent, can be cited vitamins C and E. Vitamin C mainly acts in the oxide-reduction reactions, neutralizing the deleterious action of the free radicals, while vitamin E acts inhibiting the lipid peroxidation. Additionally, there are micronutrients such as folate e B12 that are indirectly related with the oxidative process. On the other hand, all of these vitamins can be directly or indirectly involved with the cognitive level in the aged ones. These vitamins may have a protective role against the possible brain dysfunction observed in some menopause women. Thus, the objective this work was to verify a possible correlation between the vitamins levels in blood from healthy women with cognitive deficit evaluated through the Mini Mental State Examination (MMSE). In this study, the vitamins E, C, B12 and folate were quantified in blood samples from 30 institutionalized healthy aged women with average of 70.5 ± 5.5 years old. The cognitive functions were evaluated through the MMSE. Results were expressed as means ± standard error (S.M.E). Pearson correlation was used to verify the correlations, in accordance with the distribution of the variable. The serum levels of vitamin C, E, folate and B12 were 44.09 ± 2.9 µmol/l, 29.6 ± 1.6 µmol/l, 11.47 ± 0.58 nmol/l e 498.1 ± 42.8 pmol/l, respectively. The results of MMSE score was showed that 90% of the subjects presented some cognitive impairment. A positive correlation was found between MMSE scores and vitamin C levels (r = 0.387; p < 0.04), however did not have correlation among MMSE and the other vitamins. Although the vitamin C levels were within the reference levels for adults, the positive association with cognitive losses demonstrates that, higher levels of this micronutrient, could be necessary to protect the aged ones against cognitives damages, providing a more healthful aging process.

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bia@ufpr.br

## Effect of galectin-3 on the cytotoxic activity of isatin-Schiff base copper (II) and zinc complexes in melanoma cells

Simeoni, R. B.<sup>1</sup>; Boroes, B. E.<sup>2</sup>; Francisco, J. C.<sup>1</sup>; Silveira, V. C.<sup>3</sup>; Abbott, M. P.<sup>3</sup>; Chammas, R.<sup>4</sup>; Zanata, S. M.<sup>2</sup>; Ferreira, A. M. D. C.<sup>2</sup>; Nakao, L. S.<sup>1</sup>

<sup>1</sup>Brazil - <sup>1</sup>Núcleo de Investigação Molecular Avançada, Pontifícia Universidade Católica do Paraná; <sup>2</sup>Departamento de patologia básica - Universidade Federal do Paraná; <sup>3</sup>Departamento de Química Fundamental, Instituto de Química, Universidade de São Paulo; <sup>4</sup>Faculdade de Medicina - Universidade de São Paulo

Anti-tumoral activities of metal complexes have been extensively investigated. Some copper(II) complexes with imine ligands derived from isatin have been described to induce apoptosis in promonocytic and neuroblastoma cells, probably due to the redox properties of Cu(II), which can be modulated by the ligands. We analyzed the cytotoxic effects of [Cu(isaepy)<sub>2</sub>](ClO<sub>4</sub>)<sub>2</sub>, [Zn(isaepy)Cl<sub>2</sub>] and [Cu(enim)H<sub>2</sub>O](ClO<sub>4</sub>)<sub>2</sub> at 75, 100 and 150 mM in B16F10 mouse melanoma cells. After 48h exposure, cellular viability and mortality were analyzed by Trypan Blue exclusion assay. Results showed that all three compounds were effective as cytotoxic agents in all tested concentrations compared to control cells. At 100 mM the mortality rate, determined by the number of dead cells related to total cell number, induced by [Cu(isaepy)<sub>2</sub>](ClO<sub>4</sub>)<sub>2</sub>, [Zn(isaepy)Cl<sub>2</sub>] and [Cu(enim)H<sub>2</sub>O](ClO<sub>4</sub>)<sub>2</sub> were 24, 100, and 27%, respectively. In addition, after 24h, 100 mM induced vacuoles formation, confirming their toxic effect. Next, since current research indicates that galectins, lectins with diverse biological activities, can contribute to neoplastic transformation, tumor cell survival, angiogenesis and tumour metastasis, we analyzed the effect of galectin-3 in the cytotoxic activities of such compounds. Galectin-3 has been demonstrated to be both pro- and anti-apoptotic. We employed melanoma cells stably transfected either with galectin-3 cDNA vector (Tm1-G3) or with the control vector (Tm1-vector). Similarly to the B16F10, all three compounds induced cellular mortality in a dose-dependent manner. The mortality rate induced by [Cu(isaepy)<sub>2</sub>](ClO<sub>4</sub>)<sub>2</sub> and [Cu(enim)H<sub>2</sub>O](ClO<sub>4</sub>)<sub>2</sub> (23 vs 49% for Tm1G3 and TM vector induced by [Cu(isaepy)<sub>2</sub>](ClO<sub>4</sub>)<sub>2</sub>, respectively; 33 vs 42% for Tm1G3 and TM vector induced by [Cu(enim)H<sub>2</sub>O](ClO<sub>4</sub>)<sub>2</sub>, respectively), indicating that galectin-3 has a role in tumor cell survival. Together, these results indicate that the isatin-Schiff base metal complexes [Cu(isaepy)<sub>2</sub>](ClO<sub>4</sub>)<sub>2</sub>, [Zn(isaepy)Cl<sub>2</sub>] and [Cu(enim)H<sub>2</sub>O](ClO<sub>4</sub>)<sub>2</sub> have anti-melanoma activity, and that galectin-3 increases melanoma resistance to such complexes-induced death.

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aldafre@ccb.ufsc.br

## Antioxidant status in a comparative physiology approach on the positive and negative effects of zinc

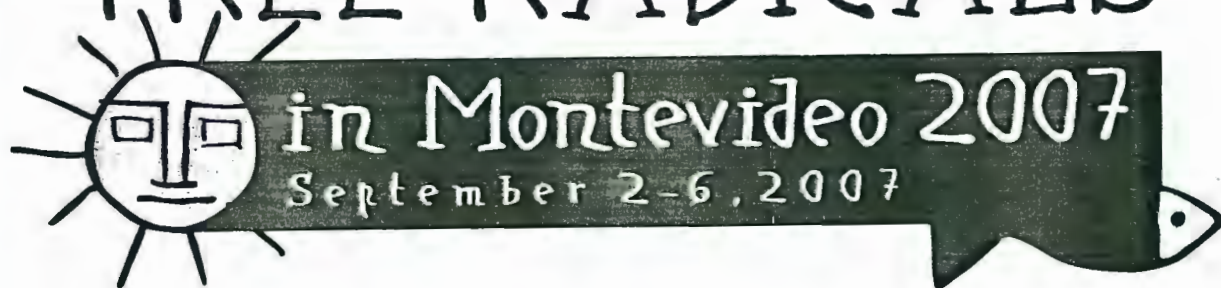
Dafre, A. L.<sup>1</sup>; Franco, J. L.<sup>1</sup>; Trevisan, R.<sup>1</sup>; Uliano-Silva, M.<sup>1</sup>

<sup>1</sup>Brazil - <sup>1</sup>Laboratório de Defesas Celulares, Depto. de Ciências Fisiológicas, Centro de Ciências Biológicas, Universidade Federal de Santa Catarina, 88040-900 Florianópolis, SC, Brazil

Zinc, at low levels, has several basic housekeeping functions in metalloenzymes, transcription factors, immunoregulation, growth, and cytoprotection, displaying anti-oxidant, anti-apoptotic, and anti-inflammatory roles. However, at high levels, this metal can be highly toxic to the cell. The aim of this work was to investigate some oxidative stress-related parameters in the mussel *Perna perna*, in the fish juvenile carp *Cyprinus carpio* and in the rat (*in vitro* and *in vivo*), in a comparative approach. Short term exposure to zinc chloride *in vivo* to mussel, fish (Zn, 10, 30 and 100 µM) and rat (1-30 mg/kg, i.p.), produced a dose-dependent inhibition in the glutathione reductase (GR) activity. This effect was also observed in an *in vitro* model using rat hippocampal slices. In mussel, fish and rat some indices of oxidative damage were increased, indicating the toxic potential of zinc. In the rat we studied the protective face of Zn, which, if treated orally for 30 days (300 mg/L in drinking water), produced a neuroprotective effect against acute malathion exposure. In this oral model, the decrease in GR, glutathione peroxidase, glutathione S-transferase, superoxide dismutase, catalase was prevented by Zn. In a published work (Toxicol Sci, 97:140) zinc was also neuroprotective against malathion in a short term treatment. This protective effect was parallel to an antidepressant effect, which may indicate that this effect is related to an improvement in the antioxidant power. Corroborating this idea, oral Zn treatment was able to induce neuroprotective effectors such as glutathione, BDNF expression and ERK1/2 phosphorylation. In conclusion, acute zinc exposure is pro-oxidative in several animal models, impairing antioxidant defenses, especially GR activity. The high effectiveness in reducing GR activity deserve further investigation to test if this lower activity impacts on the antioxidant capacity of exposed animals. On the positive side, chronic oral treatment to rats is neuroprotective, indicating the potentially beneficial effect of this essential metal. Support: International Foundation for Science grant W/3636 and Fapescc.



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