In vivo evidence of hepato- and reno-protective effect of garlic oil against sodium nitrite-induced oxidative stress

Hanaa A Hassan¹, Sherif M El-Agmy¹, Rajiv L Gaur², Augusta Fernando², Madhwa HG Raj³, Allal Ouhtit²

¹. Department of Zoology, Faculty of Science, Mansoura University, Mansoura, Egypt;
². Department of Pathology and Department of Genetics, Stanley S. Scott Cancer Center, Louisiana State University Health Science Center, New Orleans, Louisiana, USA;
³. Department of Ob Gyn and Biochemistry, Stanley S. Scott Cancer Center, Louisiana State University Health Science Center, New Orleans, Louisiana, USA.

Correspondence to: Allal Ouhtit, Ph D., M Ph., Stanley S. Scott Cancer Center, Louisiana State University Health Science Center, CSRB Building, Room# 748C, 533 Bolivar Street, New Orleans, LA 70112. Ph: 1-504-568-2896 (Office); Fax: 1-504-568-2932; aouhti@lsuhsc.edu

Received: 2008.12.21; Accepted: 2009.02.26; Published: 2009.03.10

Abstract

Sodium nitrite (NaNO₂), a food color fixative and preservative, contributes to carcinogenesis. We investigated the protective role of garlic oil against NaNO₂-induced abnormalities in metabolic biochemical parameters and oxidative status in male albino rats. NaNO₂ treatment for a period of three months induced a significant increase in serum levels of glucose, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), bilirubin, urea and creatinine as well as hepatic AST and ALT. However, significant decrease was recorded in liver ALP activity, glycogen content, and renal urea and creatinine levels. In parallel, a significant increase in lipid peroxidation, and a decrease in glutathione content and catalase activity were observed in the liver and the kidney. However, garlic oil supplementation showed a remarkable amelioration of these abnormalities. Our data indicate that garlic is a phytoantioxidant with powerful chemopreventive properties against chemically-induced oxidative stress.

Key words: Food additives; Sodium nitrite; Oxidative stress; Garlic oil; Liver; glycogen; Alanine aminotransferase; Aspartate aminotransferase; Alkaline phosphatase

Introduction

Natural and synthetic food additives approved by the U.S. Food and Drug Administration are commonly used to maintain or improve safety, the nutrient value, and the taste and texture of food [1]. Although many of the 3,000 these additives enhance our food supply, others are the subject of fierce controversy. The discovery that children at the age of nursery consume food containing great amounts of additives prompted the scientific community to oversee this issue. Sodium nitrite (NaNO₂) is present in vegetables and is routinely used as a color fixative and preservative for meats and fish [2]. The hazardous effect of NaNO₂ derives from the reaction of nitrites with amines to produce nitrosamines, and with amides to produce nitrosamides. The toxic effects of nitrates and nitrites are well documented in mammals, including impairment of reproductive function [3], hepatotoxicity and methaemoglobinemia [4], dysregulation of inflammatory responses and tissue injury [5], growth retardation [6], and endocrine dis-
turbance [7]. The wide use of nitrates as preservatives in food technology elevates the importance of studying their effects.

Although NaNO 2 is generally accepted as a weak carcinogen [8, 9], Wistar rats exposed to 0.3% NaNO 2 in their drinking water for at least one year developed squamous papillomas of the forestomach [10]. Using F344 rat in a multiorgan carcinogenesis model, 0.3% NaNO 2 given in the drinking water for 28 weeks increased the incidence of forestomach neoplasm in the post-initiation period [11]. Thus, it cannot be precluded that NaNO 2 has very weak carcinogenic potential, particularly in the squamous epithelium of the forestomach. Moreover, in combination with other chemicals, NaNO 2 has also been shown to form carcinogens or to enhance carcinogenesis. For instance, highly carcinogenic N-nitroso-compounds are produced when nitrite reacts with secondary amines and N-alkyl amides under acidic conditions in vitro [8, 12] and in vivo [13]. Other studies have demonstrated that treatment with NaNO 2 in combination with phenolic compounds [11, 14] or ascorbic acid [15] strongly enhanced forestomach carcinogenesis in a rat two-stage carcinogenesis model.

Recent trends in controlling and treating diseases tend to favor natural antioxidant compounds rather than synthetic ones [16]. The human diet, which contains large number of natural compounds, is essential in protecting the body against the development of diseases, and the garlic Allium sativum is one of the well known plant with remarkable anti-carcinogenic properties is [16, 17]. Garlic, is a commonly worldwide used food, and its medical properties have been well recognized since the ancient times. Garlic is known for its antibacterial [18], antitumorogenic [19], hypolipidemic [20], hypoglycemic [21], antifungal [22], and anti-atherosclerotic [23] properties, and an antioxidant against free radicals [24, 25].

Here, we investigated the role of garlic oil in preventing NaNO 2-induced abnormalities in the biochemical parameters associated with the oxidative stress in male albino rats.

Materials and methods

Chemicals

NaNO 2 (Sigma Aldrich, St Louis, MO) was applied as a freshly prepared solution and given by gavages at a dose of 80 mg/kg body weight as previously described [26]. Garlic oil was purchased from El-Captain Company (Cairo, Egypt). Garlic oil was given by gavages at a dose of 5ml/kg as described [27].

Animals

Male Albino Rats (Rattus rattus) weighing about 100-120 g were used in this study. The animals were kept under good ventilation and received a balanced diet and water ad libitum throughout the experimental period. Rats were divided into four main groups (n= 6) as follow: 1) Control group received, standard diet without any treatment; 2) Garlic oil-treated group, received standard diet supplemented orally with garlic oil at a dose of 5 ml/kg body weight for a period of 3 months; 3) NaNO 2-treated group, received standard diet supplemented orally with sodium nitrite at dose of 80 mg/kg body weight for a period of 3 months; and 4) NaNO 2+garlic oil-treated group, received standard diet and were supplemented orally with similar doses of NaNO 2 and garlic oil as group 3 for similar period of 3 months.

At the end of the experimental period, overnight fasted animals were sacrificed by cervical dislocation, and blood samples were collected in centrifuge tubes. Serum was separated from coagulant blood by centrifugation at 860g for 20 min, and then quickly frozen at -20°C for biochemical analysis. Small pieces of liver and kidney tissues were separated, weighed, homogenized in ice cold water and stored at -20°C for subsequent measurements.

Biochemical analysis

Serum glucose level was determined using the Biomerieux reagent kits [28]. Liver glycogen content was determined according to the method described by Van-Handle [29]. Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were determined according to the method described by Reitman and Frankel [30], whereas alkaline phosphatase (ALP) activity was estimated by Belfield method [31]. Total protein, bilirubin, urea and creatinine levels were determined using Diamond Diagnostic Kit as previously reported [32-33]. The product of lipid peroxidation, thiobarbituric acid reactive substances (TBARS), was determined as previously described [34]. Glutathione (GSH) content was estimated by the method of Prins and Loose [35], and the activity of catalase was determined by the method of Aebi [36].

Statistical analysis

The data was analyzed using the Statistical Package for Social Science program (S.P.S.S. 11). For comparison between different experimental rat
groups, one way analysis of variance (ANOVA) was used followed by Tukey’s test. The results were expressed as means ± SE and % of change, and P < 0.05 was considered to be statistically significant.

Results
A number of biochemical parameters were examined in the serum collected from each group and the results are summarized in Table 1. While rats fed on standard diet supplemented with garlic oil did not show any significant changes in the majority of the parameters examined, a significant increase in serum levels of glucose, bilirubin, urea and creatinine as well as the activity of the enzymes AST, ALT and ALP enzymes were observed in rats treated with NaNO2 for a period of three months. The total protein content significantly decreased in serum (Table 1). However, supplementation of NaNO2 intoxicated rats with garlic oil ameliorated the nitrate adverse effects as evidenced by a significant increase of serum total protein content, and a decrease of serum glucose, bilirubin, urea and creatinine levels, as well as the activity of AST, ALT and ALP enzymes (Table 1). The same parameters were examined in liver and kidney tissues, and the data are shown in Table 2. In the NaNO2-treated rats, a statistically significant inhibition of hepatic glycogen and total protein contents, and the activity of the enzymes ALT and AST as well as the levels of renal urea, creatinine and total protein content (Table 2). The activity of hepatic ALP was significantly increased in the NaNO2-treated rats (Table 2). However, administration of garlic oil to the NaNO2-intoxicated rats significantly restored these parameters in the liver and kidney organs (Table 2).

Further, we assessed oxidative stress parameters and antioxidant activity in the liver and the kidney and the results are summarized in Table 3. The data indicate that TBARS concentration increased significantly, while GSH content, as well as catalase activity were decreased in both organs of NaNO2-intoxicated rats (Table 3). However, combination of garlic oil with NaN02 reduced TBARS concentration and restored the levels of GSH as well as the activity of catalase (Table 3).

Table 1. Serum biochemical parameters in different rat groups. Results are presented as means ± SE (n=5) and % of change. AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase

| Parameters                  | Control | Garlic Oil (GO) | NaN02 | NaN02 + GO | ANOVA |
|----------------------------|---------|----------------|-------|------------|-------|
| Glucose (mg/dl)            | 102.4±1.5 | 93.5±2.5       | 207.3±1.5 | 105.9±1.4 |       |
| AST (U/ml)                 | 88.8±1.6 | 88.6±1.5       | 138.2±2.1 | 115.1±0.8  |       |
| ALT (U/ml)                 | 22.8±0.8 | 20.8±0.6       | 32.2±1.1  | 26.0±0.6   |       |
| ALP (K.Arm.U/100ml)        | 21.5±0.8 | 19.9±0.8       | 27.8±0.7  | 22.5±0.2   |       |
| Bilirubin (mg/dl)          | 0.29±0.006 | 0.28±0.002     | 0.47±0.005 | 0.34±0.005 |       |
| Total protein (g/dl)       | 6.8±0.17  | 7.1±0.45       | 4.9±0.16  | 6.7±0.19   |       |
| Urea (mg/dl)               | 37.9±0.45 | 36.5±0.52      | 49.8±0.93 | 38.1±0.47  |       |
| Creatinine (mg/dl)         | 1.52±0.11 | 1.48±0.08      | 1.98±0.14 | 1.22±0.04  |       |

a: Compared to control group; b: Compared to NaN02 treated rats group
* % of change compared with control group.
** % of change compared with NaN02 treated rats group.

http://www.biolsci.org
**Table 2. Hepatic and renal biochemical parameters in different rat groups.** Results are presented as means ± SE (n=5) and % of change. AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase.

| Parameters          | Control   | Garlic Oil (GO) | NaNO₂ | NaNO₂ +GO | ANOVA |
|---------------------|-----------|-----------------|-------|-----------|-------|
| Glycogen (mg/100g)  | 36.7±1.6  | 42.4±1.5a       | 12±0.5 | 19±1.5b   | 115.4 | P<0.05 |
|                     |           | +13.6*          | -67.9* | -49.2* & +58.3** |
| AST (U/mg)          | 11.4±0.20 | 11.7±0.10       | 10.1±0.21a | 11.6±0.24b | 98.2  | P<0.05 |
|                     |           | +2.6*           | -11.4* | +1.8* & +14.9** |
| ALT (U/mg)          | 2.7±0.09  | 2.9±0.13        | 2.1±0.05a | 2.6±0.05b | 110.9 | P<0.05 |
|                     |           | +7.4*           | -22.2* | -3.7* & +23.8** |
| ALP (K.Arm.U/g)     | 170.4±2.1 | 168.2±1.0       | 271.2±2.1a | 233.4±1.7b | 1633.1 | P<0.05 |
| Total protein (g/100g) | 34.5±0.8 | 33.4±0.6        | 31.2±0.5a | 29.6±0.6a | 13.1  | P<0.05 |
|                     |           | -3.2*           | -9.6*  | -14.2* & -5.1** |
| Urea (mg/100g)      | 34.3±14.4 | 35.5±12.6       | 24.5±13.9a | 27.1±13.3b | 1770.5 | P<0.05 |
|                     |           | +3.5*           | -28.6* | -21* & +10.7** |
| Creatinine (mg/g)   | 1.02±0.01 | 1.04±0.02       | 0.95±0.02a | 1.13±0.01b | 25.1  | P<0.05 |
|                     |           | +2.0*           | -6.9*  | +10.8* & 18.9** |
| Total protein (g/100g) | 42.4±1.5 | 42.6±2.2        | 21.1±1.4a | 39.1±2b | 40.8  | P<0.05 |

a: Compared to control group; b: Compared to NaNO₂ treated rats group
* % of change compared with control group.
** % of change compared with NaNO₂ treated rats group.

---

**Table 3. Hepatic and renal oxidative stress and antioxidant parameters in different rat groups.** Results are presented as means ± SE (n=5) and % of change. TBARS: Thiobarbituric acid reactive substance; GSH: Glutathione; CAT: Catalase; MDA: malondialdehyde.

| Parameters          | Control   | Garlic Oil (GO) | NaNO₂ | NaNO₂ +GO | ANOVA |
|---------------------|-----------|-----------------|-------|-----------|-------|
| TBARS (nmol/g)      | 102.5±1.3 | 70.4±2.2a       | 253.6±2.9a | 148.4±1.3a | 1580.5 | P<0.05 |
|                     |           | -31.3*          | +147.4* | 44.8* & -41.5** |
| GSH (mg/g)          | 0.65±0.02 | 0.69±0.01       | 0.52±0.01a | 0.66±0.01b | 35.8  | P<0.05 |
|                     |           | +6.2*           | -20*   | +1.5* & +27** |
| CAT (KU/mg)         | 0.17±0.01 | 0.19±0.01       | 0.05±0.01a | 0.08±0.01b | 66.3  | P<0.05 |
|                     |           | +11.8*          | -70.6* | -52.9* & +60** |
| MDA (nmol/g)        | 176.6±1.4 | 139.7±2a        | 234±7.6a | 203.1±0.6a | 99.8  | P<0.05 |
|                     |           | -20.9*          | +32.5* | +15* & -13.2** |
| GSH (mg/g)          | 0.76±0.01 | 0.77±0.02       | 0.63±0.02a | 0.75±0.01b | 20.5  | P<0.05 |
|                     |           | +1.3*           | -17.1* | -1.3* & +19** |
| CAT (KU/mg)         | 0.14±0.01 | 0.15±0.01       | 0.06±0.01a | 0.10±0.01ab | 23.7  | P<0.05 |

a: Compared to control group; b: Compared to NaNO₂ treated rats group
* % of change compared with control group.
** % of change compared with NaNO₂ treated rats group.
Discussion

The NaNO₂ and other additives may react with amines of the foods in the stomach and produce nitrosamines and free radicals. Such products may increase lipid peroxidation, which can be harmful to different organs including liver and kidney [37]. On the other hand, these free radicals, known to cause oxidative stress, can be prevented or reduced by dietary natural antioxidants through their capacity to scavenge these products [38]. The present study was undertaken to determine whether garlic oil can prevent and/or reduce NaNO₂-induced oxidative stress by examining different biochemical parameters of oxidative damage in the serum, the liver and the kidney of male rats.

Our results clearly showed that there was a significant increase in serum glucose concentration and a decrease in liver glycogen content of NaNO₂-treated rats. The findings suggest nitrate-stimulation of gluconeogenesis [39], and glucose shift from tissue to blood or an impairment of glucose mobilization. Furthermore, nitroso-compounds can alter the antioxidant system causing disturbance in the metabolic processes leading to hyperglycemia [40]. However, serum glucose and liver glycogen levels were ameliorated upon garlic oil supplementation. The hypoglycemic effect of garlic oil and its organo-sulfur compounds might be due to their ability to enhance insulin secretion [41, 42].

Our results also indicate an inhibitory effect NaNO₂ on the biosynthesis of protein, which was restored by garlic oil supplementation. These data suggest a stimulation of the thyroid and the adrenal glands by NaNO₂ which can lead to a blockade in protein synthesis, fast breakdown, increased rate of free amino acids, and decreased protein turnover [43]. In addition, nitrite interactions results into nitric oxide release, which can inhibit total protein synthesis [44]. However, the increase in bilirubin concentration as well as the activity of AST, ALT and ALP enzymes in the serum of NaNO₂-treated rats could be attributed to the toxic effect of nitroso-compounds, formed in the acidic environment of the stomach, in causing severe hepatic necrosis [45]. These abnormalities were prevented by supplementation of garlic oil, perhaps due to its role in stabilizing the cell membrane and protect the liver from free radical-mediated liver cell toxicity [46].

In response to NaNO₂ treatment, urea and creatinine were increased in the serum but decreased in the kidney, suggesting an impairment of kidney functions. These effects could also be attributed to the changes in the threshold of tubular re-absorption, renal blood flow and glomerular filtration rate [47]. Garlic oil showed a clear improvement in kidney functions, perhaps due to the antioxidant properties of garlic in scavenging free radicals leading to reduced levels of nitric oxide and lipid peroxidation. Moreover, NaNO₂-inhibited glutathione content and catalase enzyme activity in the liver and the kidney may be attributed to the observed induction of lipid peroxidation [48]. However, garlic improved the antioxidant mechanism due to the ability of Diallyl disulfide and Diallyl trisulfide present in garlic oil in modulating the oxidative stress and detoxifying enzyme system [49, 50, 51].

In conclusion, from the results achieved it can be concluded that the administration of garlic has an extremely beneficial role in overcoming the occurred adverse effects of chronic ingestion of sodium nitrite, which is probably through its excellent antioxidant properties and highly nutritional values.

Acknowledgments

Dr. Ouhtit is supported by the Louisiana Cancer Research Consortium in New Orleans.

Conflict of Interest

The authors have declared that no conflict of interest exists.

References

1. National Toxicology Program. NTP toxicology and carcinogenesis studies of sodium nitrite (CAS No. 7632-00-0) drinking water studies in F344/N rats and B6C3F1 mice. Natl Toxicol Program Tech Rep Ser 2001; 495: 1-274.
2. Kilgore WW and Li MY. Food Additives and Contaminations. In: Doull J, Khassen CD, Amdur MD, eds. Cassarett and Doulls, Toxicology: The Basic Science of Poisons, 2nd ed. New York: Macmillian, 1980:593-607.
3. Sleight SD, Sinha DP and Uzoukwa M. Effect of sodium nitrate on reproductive performance of pregnant sows. J Am Vet Med Assoc 1972; 161:819-823.
4. Swann PF. The toxicology of nitrate, nitrite and N-nitroso compounds. J Sci Food Agrec 1975; 26:1761-1770.
5. Blanquat DG, Fritsch F and Cazottes C. Effect of dietary nitrate and nitrite on experimentally induced inflammation in the rat. Intern. J Tiss React 1983; 27:173-180.
6. Prasad J. Effect of high nitrate diet on thyroid glands in goats. Ind J Animal Sci 1983; 53:791-794.
7. Jahries G, Hesse VI, Schone LH and Mehnert E. Influence of nitrates and plant goitrogens on thyroid hormone, somatized in status and growth of swine. Mj Vet Med 1986; 41:528-530.
8. Maekawa A, Ogiu T, Onodera H, Furuta K, Matsuoka C, Ohno Y, Odashima S. Carcinogenicity studies of sodium nitrite and sodium nitrate in F-344 rats. Food Chem Toxicol 1982; 20: 25-33

http://www.biolsci.org
9. Inai K, Aoki Y, Tokuoka S. Chronic toxicity of sodium nitrite in mice, with reference to its tumorigenicity. Gann 1979; 70: 203-8.
10. Mirvish SS, Bulay O, Runge RG, Patil K. Study of the carcinogenicity of large doses of dimethylnitramine. N-nitroso-L-proline, and sodium nitrite administered in drinking water to rats. J Natl Cancer Inst 1980; 64: 1435-42.
11. Hirose M, Tanaka H, Takahashi S, Futakuchi M, Fukushima S, Ito N. Effects of sodium nitrite and catechol, 3-methoxycatechol, or butylated hydroxyanisole in combination in a rat multiorgan carcinogenesis model. Cancer Res 1983; 53: 32-7.
12. Aoyagi M, Matsukura N, Uchida E, Kawachi T, Sugimura T, Takayama S, Matsui M. Induction of liver tumors in Wistar rats by sodium nitrite given in pellet diets. J Natl Cancer Inst 1980; 65: 411-4.
13. Hecht SS. Approaches to cancer prevention based on an understanding of N-nitrosamine carcinogenesis. Proc Soc Exp Biol Med 1997; 216: 181-91.
14. Kawabe M, Takaba K, Yoshiya Y, Hirose M. Effects of combined treatment with phenolic compounds and sodium nitrite on two-stage carcinogenesis and cell proliferation in the rat stomach. Jpn J Cancer Res 1994; 85: 17-25.
15. Yoshiya Y, Hirose M, Takaba K, Kimura J, Ito N. Induction and promotion of forestomach tumors by sodium nitrite in combination with ascorbic acid or sodium ascorbate in rats with or without N-methyl-N-nitro-N-nitrosoguanidine pre-treatment. Int J Cancer 1994; 56: 124-8.
16. Craig W, Beck L. Phytochemicals: Health Protective Effects. Can J Diet Pract Res. 1999; 60:78-84.
17. Agarwal MK, Iqbal M, Athar M. Garlic oil ameliorates ferric nitroltriacetate (Fe-NTA)-induced damage and tumor promotion: implications for cancer prevention. Food Chem Toxicol. 2007; 45:1634-40.
18. Johnson MG and Vaughn RH. Death of Salmonella typhimurium and Escherichia coli in the presence of freshly reconstituted dehydrated garlic and onion. Appl Microbiol 1969; 17:903-905.
19. Hussain SP, Jannu LN and Rea AR. Chemopreventive action of garlic on methylcholanthrene induced carcinogenesis in the uterine cervix of mice. Cancer Lett 1990; 49:175-180.
20. Bordia A, Bansal HC, Arora SK and Singh SV. Effect of essential oils of garlic and onion on alimentary hyperlipidemia. Atherosclerosis 1975; 21:15-19.
21. Jain RC and Vjas CR. Garlic in alloxan induced diabetic rabbits. Phytomedicine 2006; 13:624-629.
22. Trinder P. A colorimetric method for the determination of glucose. Ann Clin Biochem 1969; 6:24-26.
23. Van-Handle E. Estimation of glycogen in small amounts of tissue. Anal Biochem 1965; 11:256-262.
24. Reitman S and Frankel S. A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminase. Am J Clin 1957; 28:152-156.
25. Belfield A, Goldberg DM. Revised assay for serum phenyl phosphatase activity using 4-amino-antipyrine. Enzyme 1971;12:561-73.
26. Palton CJ and Crouch SR. Enzymatic determination of serum urea by modified Berthelot reaction. Anal Chem 1977; 49:464-469.
27. Henry RJ. Principles and Techniques. Clinical Chemistry, 2nd Ed. Harper and Row 1974:525.
28. Anil KB, Manju B, Giridhar S and Deepak B. Protective role of Vitamin E pre-treatment on N-nitrosodimethylamine induced oxidative stress in rat liver. Chem Biol Interact 2005; 20:101-102.
29. Liu CT, Hse H, Lii CK, Chen PS and Sheen LY. Effects of garlic oil and diallyl trisulfide on glyceric control in diabetic rats. Eur J Pharmacol 2005; 516:163-168.
30. Eidi A, Eidi M and Esmaeili E. Antidiabetic effect of garlic (Allium sativum L.) in normal and streptozotocin induced diabetic rats. Phytomedicine 2006; 13:624-629.
31. Eremine YN and Yocharina MG. Effect of nitrates on the state of thyroid gland in iodine deficiency and different diets. Vopr Pit. 1981; 5:60-62.
32. Holovakov P, Gordon D and Kulik T. Nitric oxide–generating compounds inhibit total protein and collagen synthesis in cultured vascular smooth muscle cells. Circ Res 1995; 76:305-309.
33. Kalantari H, Salehi M. The protective effect of garlic oil on hepatotoxicity induced by acetaminophen in mice and comparison with N-acetylcycteine. Saudi Med J 2001;22:1080-4.
34. Hikino H, Tohkkin M, Kiso Y, Namiki T, Nishimura S, Takeyama K. Antihypotoxic Actions of Allium sativum Bulbs1. Planta Med 1986; 52:163-168.
35. Eidi A, Hisam M and Esmaeili E. Antidiabetic effect of garlic (Allium sativum L.) in normal and streptozotocin induced diabetic rats. Phytomedicine 2006; 13:624-629.
50. Saravanan G and Prakash J. Effect of garlic (Allium sativum) on lipid peroxidation in experimental myocardial infarction in rats. J Ethnopharmacol 2004; 94:155-158.

51. Pedraza-Chaverri J, Maldonado PD, Barrera D, Cerón A, Medina-Campos ON, Hernández-Pando R. Protective effect of diallyl sulfide on oxidative stress and nephrotoxicity induced by gentamicin in rats. Mol Cell Biochem 2003; 254:125-130.

Author biography

Allal Ouhtit is a principal investigator and a member of the Stanley S Scott Cancer Center at the Louisiana State University (LSU) Health Sciences Center. He received his MPh in Neuroscience and his PhD in Biochemistry from the University Claude Bernard Lyon-I, France. He completed his postdoctoral work at the International Agency for Research on Cancer (IARC). In 2001, he became an Assistant Professor at the Queens University of Belfast, UK. He joined the LSU Health Sciences Center in 2005. His group focuses on two areas of research: 1) understanding the signaling mechanisms by which the cell adhesion receptors CD44 and CD146 regulate breast cancer metastasis; and 2) identification of powerful combinations of herbal antioxidants for chemoprevention of cancer and other diseases. Dr Ouhtit is an internationally respected scientist with awards for outstanding and innovative research.