Decreasing Free Radicals Level on High Risk Person After Vitamin C and E Supplement Treatment

by Dwi Rita Anggraini3
Decreasing Free Radicals Level on High Risk Person After Vitamin C and E Supplement Treatment

To cite this article: M S Sitorus et al 2017 IOP Conf. Ser.: Mater. Sci. Eng. 180 012093

View the article online for updates and enhancements.

Related content
- Skin Photocaging: Secondary prevention for skin photocaging
  R Yin, Q Chen and M R Hamblin
- Biochemistry of the lungs: Free radicals
  T D Presley
- Evaluation of free radical scavenging activity and radioprotective efficacy of
  Ginkgo biloba tincture
  Krishna V Sharma and Rashmi Sisodia
Decreasing Free Radicals Level on High Risk Person After Vitamin C and E Supplement Treatment

M S Sitotrim, D R Anggunia, Hidayat1
1Anatomy department, Medical Faculty, University of Sumatera Utara
2Biochemistry department, Medical Faculty, University of Sumatera Utara

Abstract. Has become a global issue that the increase in global warming mainly caused by high air pollution levels which are donated by motor vehicle emissions. As a rapidly developing country, Indonesia becomes vulnerable to health problems related to air pollution. Excessive free radicals that is produced by air pollution can initiate stress oxidative. Already known that, stress oxidative trigger many health problems. Vitamin C and E is a non enzymatic antioxidant that can neutralize free radicals. This study aims to investigate the decreasing free radicals level by administering vitamin C and E. This research using pre and post experimental design study. There are 24 operators gasoline station Pertamina as samples, with an average age of 26 years. The samples were divided into 4 groups. Group 1 (control), group 2, were given vitamin C doses of 500mg/day; group 3 was given vitamin E doses of 250 IU/day and the group 4 was given a combination of vitamins C and E. The treatment was given for 30 days. Free radicals level is obtained from malonaldehyde (MDA) level by spectrophotometer. Before treatment the average of MDA level is 5.540 μM. After the treatment, MDA is significantly decreased become 3.992 μM (t-test, sig<0.05). This result can be used as a sign of side effect of air pollutant in the operator SPBU. As reminding to protect the employee with safety aid and supplement.

1. Introduction
It has become a global issue that the global warming mainly caused by high air pollution levels. Air pollution is mainly caused by motor vehicle emissions. As a rapidly developing country, Indonesia using a motorized vehicle usage continues to increase. Air pollution caused by it has reached a crisis point. Poor air quality have led to 3 million people die every year in the world and millions of people suffer from asthma, upper respiratory tract disease, heart disease and blood vessels and lung cancer. In Indonesia, cases of respiratory tract infections reached 75,000 at 2015. One study reported that 57.8% of the population in Jakarta suffering from diseases caused by air pollution include asthma, bronchopneumonia and heart vessel disease.

Based on research conducted by Shrilatha et al (2011), showed hyper responsiveness neonatal rat lungs which exposed to pollution containing Environmentally Persistent Free Radicals (EPFRs). The ADB-funded regional Technical Assistance (2000) estimated emissions of motor vehicles in Jakarta, around 71% is nitrogen oxides (NOx), 15% are sulfur dioxide (SO2) and 70% of them are particulate matter (PM10). Monitor air quality in 30 cities in Indonesia shows the concentration of NO2 (0-30 ppm) and SO2 (0-50 ppm) are high. The number of vehicles on the road also increased by 12% in proportion.
to the increase in combustion. Alleged that inhaled particles cause inflammatory responses are ongoing will trigger the release of more free radicals and inflammatory mediators. This will trigger further occurrence of cellular effect. This situation causes Indonesian people, especially workers who work outdoors become susceptible to health problems related to air pollution.

Free radicals exist in air pollution will trigger the oxidative stress cells. Free radicals is molecules that have unpaired electrons. This molecule is very unstable because they will release and take electrons from other molecules in the vicinity.6 The free radicals also actually are generated internally by the body for human survival. The process of normal cell metabolism will produce a Reactive oxygen species (ROS) that is important for life and death of the cell.7 The main of them are ROS superoxide anion (O2−), hydroxyl radical (•OH) and hydrogen peroxide (H2O2).8,9 ROS are generated through enzymatic processes that in mitochondria, phagocytosis, the prostaglandin system, and the cytochrome P450 system. Through a non-enzymatic process, ROS are formed from the reaction of O2 with organic components that are initiated by the reactions of ions. In a state of equilibrium then it will be changed by the endogenous antioxidant: free radical superoxide anion (O 2−) become into O2 and H2O2 by superoxide dismutase. Furthermore H2O2 will be catalyzed into H2O and O2.8,9 External sources of ROS are exposure to X-rays, ozone, tobacco smoke, air pollution and chemicals industries.8,9

In condition where there is an imbalance of antioxidants with free radicals will lead to a state of oxidative stress. ROS can enter the nucleus and the cell membrane. ROS has a target to attack proteins, carbohydrates, fats and DNA. Among them the lipid peroxidation is more dangerous because lipid peroxidation will produce propagation chain of free radical reactions.8,9 When atoms remove from fatty acids by some reason, lipid radical (L •) will be formed, then the chain reaction of free radicals lipid starts. The release of hydrogen from lipid will form hydroxyl radical (HO •), hydroperoxy radical (HOO •), lipid peroxyl radical (LOO •), and alkoxy radical (LO •). As started, peroxidation chain reaction will continue on its own. Lipid radical rapidly reacts with the oxygen to form LOO •. It will attack other lipid and move the hydrogen atoms of the lipid. This situation led to the formation of lipid hydroperoxide (LOOH) and L • again. The new L • will also react with oxygen and will form LOO • back which will also attack lipid and produce lipid peroxide. Lipid peroxide becomes more as a result of lipid oxidation.11 It will damage the membrane lipid bilayer arrangement that causes the inactivation of membrane-bound receptors and enzymes that will increase tissue permeability. The end product of lipid peroxidation is malondialdehyde (MDA), propanal, hexanal, and 4-hydroxynonenal (4-HNE), which can make many cells protein inactive.10 MDA is a product of lipid peroxidation that highly mutagenic, and 4-HNE is the most toxic. MDA is a compound that can describe the activity of free radicals in the cells so that these compounds can be used as a biomarker of the lipid peroxidation.11,12

If the free radicals formed (can be from the body or excessive external) cannot be neutralized by antioxidants, oxidative stress occurred.13 Antioxidants is a substrate that is at low concentrations when compared with a substance that can be oxidized significantly delay or prevent the oxidation of the substance.14 There are two groups of antioxidants, enzymatic and non-enzymatic antioxidants. Enzymatic antioxidant defense subdivided into primary and secondary defense. The primary defense consists of: glutathione peroxidase, catalase and superoxide dismutase. This enzyme will prevent the formation and neutralize free radicals. Secondary defense consists of the enzyme glutathione reductase and glucose-6-phosphatase which are useful to support the work of the primary enzyme. The group includes non-enzymatic antioxidants are vitamins (A, E, C), an enzyme cofactor (Q10), minerals (zinc and selenium), peptide (glutathione), phenolic acid and nitrogen (uric acid compounds).13 Ascorbate (vitamin C) act directly as an antioxidant by reaction with aqueous peroxyl radicals, and indirectly by storing the components of the antioxidant vitamin E fat soluble.15 As a consequence of this will control the antioxidant activity of lipid peroxidation in cell membranes and surrounding areas. Giving sodium ascorbate can reduce the formation of malonaldehyde.16 The same condition also influence the intracellular free radicals that attack the non core lipid.15 Antioxidant activity of vitamin E as a scavenging reaction via α-tocopherol radicals. A-tocopherol radicals bond with the corresponding free radicals will form non radical product17, through a chain-breaking antioxidant.18
The outdoors workers such as gasoline station employee (Stasiun Pengisian Bahan bakar Umum/SPBU) are extremely exposed to free radicals from environment. If natural body antioxidant cannot neutralize free radicals, many health problem will occurred among these people. Can it be reduced by antioxidant supplement, such as vitamin C or E?

Some of the latest research in Palembang and Jakarta showed that MDA level of gasoline station employee is higher than non gasoline station employee. But there is no further research to reduce that level among this population although they are vulnerable to suffer chronic diseases caused by air pollution. This research will obtain MDA level of SPBU employee and to find out whether it can be reduced by taking vitamin C and E supplement. Thus, evidence of decreased levels of free radicals, gives information to take vitamin C and E as the protection of workers.

2. Method
This research uses pre and post-experimental study. The research sample is 24 operators refueling at gasoline stations, Stasiun Pengisian Bahan Bakar Umum (SPBU) COCO Polonia, Nusa Tiga, and Setia Budi. All samples are male, who worked for more than six months, and has levels of MDA more than normal. Samples were randomly divided into 4 groups. Group K is the control (no treatment), the treatment group 1 (P1) is given supplements of vitamin C dose of 500 mg/day (Vitamin C), group 2 (P2) is given supplements of vitamin E 200 IU/day (Eve-E) and the treatment group 3 (P3) is given the combination vitamin C dose of 500 mg/day and vitamin E 250 IU/day. Before treatment all samples levels of malonaldehyde (MDA) pre-test was measured. The venous blood was aspirated as much as 3 ml. Next, treatment groups were given supplements of vitamin C and E to be consumed for 30 days. After 30 days, venous blood samples were aspirated back as much as 3 ml to measure malonaldehyde levels post test.

MDA level is determined from an examination of the blood sample with spectrophotometer with thioarbituric acid method (TBA). Malonaldehyde is the end product of lipid peroxidase which reacts with TBA in acidic conditions will become pink. Interpretation of MDA sample compared to the normal value is 1.04 ± 0.43 mol/L. The significance of decreased levels of MDA was tested statistically by T-test. T-test value is significant if p < 0.05. Measurement of MDA conducted at the Laboratory of Biochemistry Faculty of Medicine, University of Sumatera Utara.

Prior to all samples has given an explanation about the purpose of research, treatment performed and side effects. Then the samples require approval in writing. This study has received permission from the research ethics committee at Medical Faculty, University of Sumatera Utara. A subsection

3. Result
The distribution of sample characteristics (age, duration of work, smoking habits, drinking habits) showed in Table 1. Age distribution of samples ranging from 17 to 47 years, with the mean age is 26 years. Only one person at the age of 47 samples (4.17%). Based on the duration work at the pump, the average time to work about 3 years (1-3 years, 70.83%). There are 11 persons have the habit of smoking for 5-10 years(45.83%). And only one person (4.17%) who has a habit of drinking alcohol.
Table 1. Characteristic distribution of the operator gasoline station

| Characteristics       | Frequency | Mean   | Percentage |
|-----------------------|----------|--------|------------|
| Classification of age |          |        |            |
| <20 years             | 2        | 26     | 8.33       |
| 20-30 years           | 15       |        | 62.5       |
| 30-40 years           | 6        |        | 25         |
| 40-50 years           | 1        |        | 4.17       |
| Duration of work      |          |        |            |
| <1 years              | 1        | 2 years| 4.17       |
| 1-3 years             | 17       |        | 70.83      |
| 3-5 years             | 1        |        | 4.17       |
| 5-7 years             | 3        |        | 12.5       |
| >years                | 2        |        | 8.33       |
| Smoking habitual      |          |        |            |
| No - <1 years         | 6        |        | 25         |
| 1-5 years             | 3        | 10 years| 12.5       |
| 5-10 years            | 11       |        | 45.83      |
| >10 years             | 4        |        | 16.67      |
| Alcohol consumption   |          |        |            |
| No drink              | 23       |        | 95.83      |
| <1-2 a month          | 1        |        | 4.17       |
| Always (1x a month)   | 0        |        |            |

On the first day, and after 30 days of supplementation, blood samples were aspirated for MDA measurement. MDA levels were showed in Table 2. All MDA level samples are showed above normal, 1.04 ± 0.43 μmol/L, with mean 5.2588 μmol/L. After treatment the average levels of MDA decreased to 3.9921 μmol/L.

Table 2. MDA Levels

| No Sample | Group | Pre | Mean  | Post1 | Mean  | Sig (2-tailed) |
|-----------|-------|-----|-------|-------|-------|----------------|
| 1         | P1    | 6.65| 5.258 | 5.29  | 3.992| .001           |
| 2         | P1    | 6.96| 5.37  |       |       |                |
| 3         | P1    | 5.25| 4.26  |       |       |                |
| 4         | P1    | 9.26| 4.99  |       |       |                |
| 5         | P1    | 7.51| 3.43  |       |       |                |
| 6         | P1    | 7.50| 5.51  |       |       |                |
| 7         | P2    | 2.97| 1.94  |       |       |                |
| 8         | P2    | 2.95| 3.49  |       |       |                |
| 9         | P2    | 4.96| 2.29  |       |       |                |
| 10        | P2    | 7.35| 5.51  |       |       |                |
| 11        | P2    | 5.13| 3.78  |       |       |                |
| 12        | P2    | 5.17| 4.28  |       |       |                |
| 13        | P3    | 9.08| 3.90  |       |       |                |
| 14        | P3    | 5.33| 3.36  |       |       |                |
| 15        | P3    | 2.34| 3.16  |       |       |                |
| 16        | P3    | 5.44| 2.82  |       |       |                |
| 17        | P3    | 3.08| 5.03  |       |       |                |
| 18        | P3    | 5.23| 4.86  |       |       |                |
| 19        | K     | 2.35| 2.41  |       |       |                |
| 20        | K     | 5.88| 5.16  |       |       |                |
| 21        | K     | 4.07| 3.50  |       |       |                |
| 22        | K     | 2.96| 2.01  |       |       |                |
| 23        | K     | 4.19| 5.24  |       |       |                |
| 24        | K     | 4.90| 4.22  |       |       |                |
Kolmogorov-Smirnov analysis shows the whole group has a normal distribution. The t-test pre and post treatment showed t value = 3.682 and p = 0.001 (p < 0.05). Group 1 (P1) MDA level decreased the most, which is 2.3800 mol/L as seen in chart 1.

![Chart 1. Mean decreasing MDA level in groups](image)

Chart 1 shows the decreasing of MDA level in all groups samples. The vitamin C group is decreased the most. While the control group decreased the minimum is 0.2517 μmol/L. Furthermore, the Anova test to compare each group to control is found p = 0.187.

4. Discussion

Indonesia is a rapidly growing country, especially in the transportation sector. This sector contributes to a great source of free radicals for the population, mainly outdoor’s workers such as gas station operators. The mean age of the gas station operator in this study were 26 years of age, with the highest age range of 20-30 years. This age is the young productive age. The population of Indonesia in 2010 by the agency Bureau of Statistics (BPS) as much as 237.6 million people and a population median age of 27.2 years, making Indonesia as a country with a population of intermediate category. About 11 million people are males aged 25-29. These data underlie why the number of samples to be highest at the age of 26 years.

There are 17 people (70.83%) who have worked for 1-3 years. The duration of the study sample had worked at the gas station also contributes the availability of free radicals sources constantly at work, which exposed to the scent of gasoline or diesel fuel, motor vehicle fumes, sunlight. Gasoline containing more than 150 chemicals, including small amounts of benzene, toluene, and xylene. A small number of these chemicals will evaporate in the air when refueling vehicles and will be inhaled through breathing. People who work at the gas pump, as operator, clerk reloading oil, tanker truck drivers have a greater risk expose to gasoline vapor.

Based on research conducted by Ramtej Jayram Verma, there was increased levels of lipid peroxidation, significantly (p < 0.05) in rats exposed to gasoline. The study showed that an increase in lipid peroxidation caused by a significant decrease in antioxidant enzymatic activities such as catalase, superoxide dismutase and glutathione peroxidase as well as non-enzymatic antioxidants such as glutathione and total ascorbic acid in the liver of mice exposed to gasoline. Employee in the gasoline station as samples continuously expose to free radicals from the fuel, and make them susceptible to the occurrence of chronic diseases. This situation can be seen from the MDA sample exceeds the normal value as in chart 1.
Sources of free radicals that also recorded from the sample is the smoking habit. Most of the samples smoked for 10 years. Cigarette smoke contains a mixture of large amounts of chemicals that not only have the potential toxic and carcinogenic, but also contain free radicals, ROS and free radical species gas.\(^2\) Smoke-free radicals have two populations, one in tar and another in the gas phase. Radicals in tar are quinone / hydroquinone complexes are capable to reduce molecular oxygen, producing superoxide, hydrogen peroxide and subsequently become hydroxyl radicals.\(^2\) Hydroxyl radicals is generated by cigarette tar of aqueous can oxidize DNA damage. Cigarette smoke and chemicals in it will synergy to increase the production of hydroxyl radical.\(^2\) Free radicals of materials resulting from the radical reaction is responsible for the inactivation of alpha 1-protease inhibitor with new smoke.\(^3\) The longer the smoking habits will contribute many free radicals for himself.

Alcohol gives the formation of ROS and disrupts the normal body defense mechanisms against ROS separately through various processes, especially in the liver. In the liver, alcohol is broken down into molecules with subsequent cell metabolism, that produce ROS. Alcohol also stimulates the activity of cytochrome P450s enzyme, who play the role to produce ROS. Further alcohol can affect the levels of certain metals in the body that facilitates the formation of ROS. Finally alcohol will also reduce substances which serves to inhibit ROS. As a result there will be a state of oxidative stress that leads to cell injury until alcoholic liver disease occurred.\(^2\) In this study, found only one person (4.17%) who have drinking alcohol habit. Donations of free radicals from alcohol in this study is not so instrumental.

Level MDA samples are 100% higher than normal. This means that there is an oxidative stress state that leads to lipid peroxidation. MDA is the end product of lipid peroxidation, derived from the reaction between the hydroxyl radical and polyunsaturated fatty acids, cross-linking occurs in the fat, protein and nucleic acid.\(^3\) High MDA levels can be found in people at high risk, such as filling station operator workers.\(^6\) Samples were continuously exposed to the scent of fuel that comes out of the hose pump oil, fumes, sunlight. This situation, if allowed to continue will cause chronic diseases such as asthma, atherosclerosis, cancer, inflammatory joint disease, diabetes and senile dementia.\(^7\) By nature, humans can inhibit free radicals with antioxidants, including ascorbic acid (vitamin C), alpha-tocopherol (vitamin E), beta carotene, coenzyme Q10, enzyme metal, selenium and zinc.\(^8\)

One sample is given vitamin C supplements at a dose of 500 mg/day for 30 days. After 30 days of MDA re-examination, it was found the levels of MDA decreased as much as 2,300 \(\mu\)mol/L. Among those groups, the MDA level of vitamin C group decrease the lowest. According to Singshal S study found a significant decrease of MDA with vitamin C and vitamin E. The largest reduction was in the administration of vitamin E 400 units/day (36.4 \(+/-\) 17.7%).\(^7\) A study showed the usage of vitamin C and E can help and treat cancer.\(^8\) This is the reason the largest decline in the group with 500 mg of vitamin C should be investigated further. As considered that risk factors such as smoking, long work, diet also needs to be uniform. In terms of diet, this sample is not restricted to diet, this can affect the levels of free radicals.

5. Conclusion
There is a significant decrease in MDA levels after giving the gas station operator worker with vitamins C, E and the combination compared to controls. This decreasing indicates the role of antioxidant vitamins C and E in preventing chronic diseases. Therefore, gas station workers are particularly vulnerable exposed to free radicals, and antioxidants is recommended to consume regularly for eliminating it.

ACKNOWLEDGEMENTS
We would like to acknowledge the financial support of Kementrian Riset, Teknologi, and Pendidikan Tinggi and also the Rector of University of Sumatera Utara.
References
[1] Haryanto B, Franklin P. Air Pollution: a tale of two countries. Rev Environ Health. 2011;26(1):53-59.
[2] Birandra K.S. Roles of free radicals in the toxicity of environmental pollutants and toxicants. J Clin Toxicol. 2013.
[3] Yudha SW. Air Pollution in Indonesia. Challenges and Imperatives for Change. The National Bureau of Asian Research. NBR Commentary; 2016.
[4] Balakrishna S, Saravja J, Thevenot P et al. Environmentally persistent free radicals induce airway hyperresponsiveness in neonatal rat lungs. Particule and Fibre Toxicology. 2011;8:11.
[5] Marjamaki M, Keskinen J. Particulates Characterisation of Exhaust Particulate Emissions from Road Vehicles. European Commission. January 2001.
[6] Lobo V, Patil A, Phataik A and Chandra N. Free radicals, antioxidants and functional foods impact on human health. Pharamcogn Rev. 2010;4(8):118-126.
[7] Rahaman A, Ghosh AK, and Sarma AD. Free Radicals and Their Role in Different Clinical Conditions: An Overview. International Journal of Pharma Sciences and Research (IJPSR). 2010;1(3):185-192.
[8] Noori S. An Overview of Oxidative Stress and Antioxidant Defensive System. Open access scientific reports. 2012.
[9] Esra B, Umit MS, Cansin S, Serpil E and Omer K. Oxidative Stress and Antioxidant Defense. WAOJOURNAL. 2012.
[10] Young WK, Taitian VB. Oxidative stress in angiogenesis and vascular disease. Blood. 2014;1235.
[11] Yoshikawa T, Naito Y. What is Oxidative Stress? The Journal of the Japan Medical Association. 2002;45(7):217-276.
[12] Antonio A, Mario FM, sandro A. Oxidative Medicine and Cellular Longevity; 2014.
[13] Yevgenia S, David H, Yaal KT, Zhi D, Yaron Y. Natural Antioxidants: Function and Sources. Food and Nutrition Sciences. 2013;4:643-649.
[14] Halliwell BB, Poulson HE. Biochemistry of oxidative stress. Biochem Soc trans. 2007;35:1147-1150.
[15] Bendich A, Machlin LJ, Scandurra O et al. in Abstract: The antioxidant role of vitamin C. Advancements in Free Radical Biology & Medicine. 1989;2(2):419-444.
[16] Berardo A, De Maere H, Stavropoulos DA, Rymser T, Leroy F, De Smet S. Effect of sodium ascorbate and sodium nitrite on protein and lipid oxidation in dry fermented sausages. Meat Sciences. 2016;121:3-364.
[17] Yamouchi R. Vitamin E: mechanism of Its Antioxidant Activity. Food Sci Technol. Int. 1997;3(4):303-309.
[18] Burton GW. Vitamin E: Antioxidant Activity, Biokinetics, and Availability. Annu. Rev. Nutr. 1990;10:357-382.
[19] Badan Pusat Statistik. Indonesia. Available at: www.bps.go.id.
[20] Satcher D. Toxicological Profile for Automotive Gasoline. Agency for Toxic Substances & Disease Registry. Toxic Substances Portal Gasoline, Automotive; 1995. Available at: https://www.atsdr.cdc.gov.
[21] Verma RJ, Manjeet D, Neeta M. A Study on Toxicity of Gasoline and GM10 on Liver of Mice and Its Amelioration by Black Tea Extract. Acta Poloniae Pharmaceutica – Drug Research. 2008;65(5):601-605.
[22] Valavanidis A, Tomais V, Konstantinos F. Tobacco Smoke: Involvement of Reactive Oxygen Species and Stable Free Radicals in Mechanisms of Oxidative Damage, Carcinogenesis and Synergistic Effects with Other Respirable Particles. Int. J. Environ. Res. Public Health. 2009;6:445-462.
[23] Church DF, Prvor WA. Free-radical chemistry of cigarette smoke and its toxicological implications. Environ Health Perspect. 1985;65:111-126.
[24] Wu D, Arthur I C. Alcohol, Oxidative Stress and Free Radical Damage. Free Radical Biology Medicine. 2002;32:11-16.

[25] IC Ozturk, Kadir B, Metin G. Comparison of Plasma Malonaldehyde, Glutathione, Glutathione Peroxidase, Hydroxyproline and Selenium Levels in Patients with Vitiligo and Health Controls. Indian Journal of Dermatology. 2008;53(3):106-110.

[26] Florence TM. The Role of Free Radicals in Disease. Abstrac in: Aust N Z J Ophthalmol. 1995;23(1):3-7.

[27] Singhal S, Gupta R, Goyle A. Comparison of Antioxidant efficacy of vitamin E, vitamin C, vitamin A and fruits in coronary heart disease: a controlled trial. J. Assoc Physicians India. 2001;49:327-331.

[28] Ian DC, Mary LH, Paul GS. Antioxidant Vitamin C and Vitamin E for the Prevention and Treatment of Cancer. J Gen Intern Med. 2006;21(7):735-744.
Decreasing Free Radicals Level on High Risk Person After Vitamin C and E Supplement Treatment

**ORIGINALITY REPORT**

| Percentage | Source Type | Source |
|------------|-------------|--------|
| 16%        | Similarity Index |        |
| 14%        | Internet Sources  |        |
| 7%         | Publications     |        |
| 8%         | Student Papers   |        |

**PRIMARY SOURCES**

1. china.iopscience.iop.org
   - Internet Source
   - 7%

2. research.aalto.fi
   - Internet Source
   - 2%

3. Mohammed Hossain. "In Vitro Assessment of Tobacco Smoke Toxicity at the BBB: Do Antioxidant Supplements Have a Protective Role?", BMC Neuroscience, 2011
   - Publication
   - 1%

4. Colin D. Kay, Penny M. Kris-Etherton, Sheila G. West. "Effects of antioxidant-rich foods on vascular reactivity: Review of the clinical evidence", Current Atherosclerosis Reports, 2006
   - Publication
   - 1%

5. Sabrine Sellimi, Abdelkarim Benslima, Ghada Ksouda, Veronique Barragan Montero, Mohamed Hajji, Moncef Nasri. "Safer and healthier reduced nitrites turkey meat sausages
   - Percentage
   - 1%
using lyophilized Cystoseira barbata seaweed extract", Journal of Complementary and Integrative Medicine, 2018
Amir M. Al Hroob, Mohammad H. Abukhalil, Omnia E. Hussein, Ayman M. Mahmoud. "Pathophysiological mechanisms of diabetic cardiomyopathy and the therapeutic potential of epigallocatechin-3-gallate", Biomedicine & Pharmacotherapy, 2019

DONKIN, A.J.M.. "Gender and Living Alone as Determinants of Fruit and Vegetable Consumption among the Elderly Living at Home in Urban Nottingham", Appetite, 199802

Choudhary, A.. "Ameliorative effects of black tea extract on aflatoxin-induced lipid peroxidation in the liver of mice", Food and Chemical Toxicology, 200501

Sushil K. Jain. "Curcumin Supplementation
Lowers TNF-α, IL-6, IL-8, and MCP-1 Secretion in High Glucose-Treated Cultured Monocytes and Blood Levels of TNF-α, IL-6, MCP-1, Glucose, and Glycosylated Hemoglobin in Diabetic Rats", Antioxidants and Redox Signaling, 02/2009

Goran Bjelakovic. "Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases", Cochrane Database of Systematic Reviews Reviews, 04/23/2008