Asian Journal of Chemistry; Vol. 25, No. 12 (2013), 6991-6992

ASIAN JOURNAL OF CHEMISTRY

http://dx.doi.org/10.14233/ajchem.2013.15110

NOTE

Theoretical Study for Antioxidant Activity of Vitamin C Beside C_{16} Cluster as a Novel Carrier

T. ARDALAN*, M. MONAJJEMI, H. AGHAIE and K. ZAREH

Department of Chemistry, Science and Research Branch, Islamic Azad University, Tehran, Iran

*Corresponding author: E-mail: tooran_ardalan_1363@yahoo.com

(*Received*: 9 January 2013; *Accepted*: 10 June 2013) AJC-13620

Theoretical calculations were carried out to interaction between vitamin C, as a good antioxidant compound and C_{16} cluster using density functional theory method. Thermodynamic binding parameters such as binding energy, enthalpy, entropy and free Gibbs energy have been calculated. Also HOMO, LUMO and HOMO-LUMO Gap energies have been performed for C_{16} inside vitamin C. The results show that HOMO- LUMO gap energy of vitamin C decreases after connecting to C_{16} cluster and by decreasing of HOMO- LUMO gap energy for vitamin C beside C_{16} cluster, vitamin C can act better as an electron donor and antioxidant.

Key Words: Vitamin C, Antioxidant properties, Thermodynamic parameters, C_{16} Cluster.

Vitamin C (ascorbic acid) is a six-carbon lactone that is synthesized from glucose in the liver of most mammalian species, but not by humans, non-human primates and guinea pigs. These species do not have the enzyme gulonolactone oxidase, which is essential for synthesis of the ascorbic acid immediate precursor 2-keto-l-gulonolactone. The DNA encoding for gulonolactone oxidase has undergone substantial mutation, resulting in the absence of a functional enzyme[1,2]. Consequently, when humans do not ingest vitamin C in their diets, a deficiency state occurs with a wide spectrum of clinical manifestations. Clinical expression of vitamin C deficiency, scurvy, is a lethal condition unless appropriately treated. Thus, humans must ingest vitamin C to survive. Vitamin C is an electron donor and therefore a reducing agent. All known physiological and biochemical actions of vitamin C are due to its action as an electron donor. Ascorbic acid donates two electrons from a double bond between the second and third carbons of the 6-carbon molecule.

A reactive and possibly harmful free radical can interact with ascorbate. The reactive free radical is reduced and the ascorbyl radical formed in its place is less reactive. Reduction of a reactive free radical with formation of a less reactive compound is sometimes called free radical scavenging or quenching. Ascorbate is therefore a good free radical scavenger due to its chemical properties[3,4].

Vitamin C can be oxidized by many species that have potential to be involved in human diseases[5-7]. Some of the relevant species, which receive electrons and are reduced by vitamin C, are superoxide, hydroxyl radical, peroxyl radicals, sulphur radicals and nitrogen-oxygen radicals.

Oxidant damage might cause or exacerbate common human diseases, such as atherosclerosis[8-10] and type II diabetes mellitus[11,12]. It might also have a role in the pathophysiology of type I diabetes mellitus[13], diabetic complications[14], chronic renal failure[15,16], complications of end stage renal disease and hemodialysis[17], rheumatoid arthritis[18], neurodegenerative diseases and pancreatitis[19]. Oxidants are thought to cause further damage to organ systems during acute illnesses such as myocardial infarction, acute pancreatitis, sepsis and inflammatory disorders and play an important role in the long term damage from cigarette smoking.

Due to the importance of vitamin C as an antioxidant in this study we used C_{16} cluster as a novel carrier for vitamin C.

Computational details: The C_{16} cluster and vitamin C inside C_{16} cluster were geometrically optimized using 6-311G, 6-311G* and cc-pvdz basis sets with the Gaussian 03 by the B3LYP method (Figs. 1 and 2). From the optimized structure, quantum-mechanical descriptors were calculated and compared. Also HOMO-LUMO gap energy were calculated using DFT method by using the Gaussian 03 software[20]. The binding energies were calculated using the following equation[21]:

$$\Delta E_b = E(C_{16}/\text{Vitamin C}) - E(\text{Vitamin C}) - E(C_{16})$$

where $E(C_{16}/\text{vitamin C})$ is the total electronic energy of the C_{16} cluster with the attached vitamin C, $E(C_{16})$ is the electronic energy of the C_{16} cluster and E(vitamin C) is the electronic energy of the vitamin C (Table-1).

TABLE-1
HOMO, LUMO AND HOMO–LUMO GAP ENERGIES VITAMIN C AND C_{16} CLUSTER INSIDE VITAMIN C

	Vitamin C			C_{16} cluster inside vitamin C		
Basis set	B3LYP/6-311G	B3LYP/6-311G*	B3LYP/cc-pvdz	B3LYP/6-311G	B3LYP/6-311G*	B3LYP/cc-pvdz
HOMO (eV)	-0.26814	-0.25948	-0.24934	-0.24356	-0.23359	-0.22793
LUMO (eV)	-0.09013	-0.07601	-0.06535	-0.18262	-0.18401	-0.17806
HOMO-LUMO Gap (eV)	0.17801	0.18348	0.18339	0.06094	0.04958	0.04987

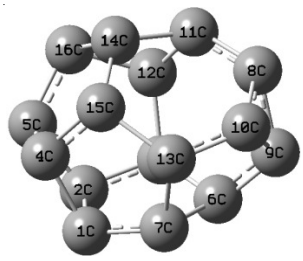

Fig. 1. Optimized structure C_{16} cluster

Fig. 2. Optimized structure C_{16} cluster inside vitamin C

Vitamin C is considered the most important water-soluble antioxidant in extracellular fluids. It is capable of neutralizing free radicals in the aqueous phase before lipid peroxidation is initiated. Vitamin C is an electron donor and therefore a reducing agent. All known physiological and biochemical actions of vitamin C are due to its action as an electron donor. It donates two electrons from a double bond between the second and third carbons of the 6-carbon molecule. It is also called an antioxidant because, by donating its electrons, it prevents other compounds from being oxidized. Therefore in this study effect of C_{16} cluster on antioxidant activity of vitamin C have been investigated.

TABLE-2
BINDING ENERGY, ENTHALPY, ENTROPY AND FREE GIBBS FOR C_{16} CLUSTER INSIDE VITAMIN C

ΔE_b (J)	3404117.756
ΔS_b (J/K)	-448.9469386
ΔH_b (J)	3401601.72
ΔG_b (J)	3535387.908

The results shown The HOMO energy for vitamin C increases and HOMO-LUMO gap energy of vitamin C decreases after connecting to C_{16} cluster. Also by decreasing of HOMO-LUMO gap energy for vitamin C beside C_{16} cluster, it can act better as an electron donor and antioxidant. Thermodynamic analyses show C_{16} cluster beside vitamin C have positive values of relative energies (ΔE), enthalpies (ΔH) and free Gibbs energies (ΔG) in gas phase. Our results also show that entropy (ΔS) for C_{16} cluster beside vitamin C system has negative values (Table-2).

Conclusion

The interaction between vitamin C and C_{16} cluster have been investigated with density functional theory using B3LYP method. We analyze the binding parameters, HOMO, LUMO and HOMO-LUMO gap energies for this cluster. HOMO-LUMO gap energy for vitamin C after connection to C_{16} cluster was decreased and vitamin C can act better as an electron donor and antioxidant. The thermodynamic analyses also show C_{16} cluster beside vitamin C have positive values of relative energies (ΔE), enthalpies (ΔH) and free Gibbs energies (ΔG) in gas phase. Also, our results show that entropy (ΔS) for C_{16} cluster beside vitamin C system has negative values. Therefore we arrive at the prediction that the C_{16} cluster can be implemented as a novel carrier for vitamin C.

REFERENCES

1. M. Suhail, N. Bilal, H. Khan, S. Hasan, S. Sharma, F. Khan, M. Mansoor and N. Banu, *J. Clin. Pharm. Ther.*, **37**, 22 (2012).
2. M. Nishikimi and K. Yagi, *Subcell Biochem.*, **25**, 17 (1996).
3. M. Naziroglu, F. Kilinc, A. Uguz, O. Celik, R. Bal, P. Butterworth and M. Baydar, *Cell Biochem. Funct.*, **28**, 300 (2010).
4. B. Ceylan, M. Naziroglu, A. Uguz, C. Barak, B. Erdem and L. Yavuz, *Biol. Trace Element Res.*, **10**, 8712 (2010).
5. D. Ratnam, D. Ankola, V. Bhardwaj, D. Sahana and M. Kumar, *J. Control. Rel.*, **20**, 189 (2006).
6. K. Lee, J. Hyong, S. Young and Y. Chang, *Am. J. Clinic. Nutr.*, **78**, 1074 (2003).
7. J. Neuzil, S. Thomas and R. Stocker, *Free Radic. Biol. Med.*, **22**, 57 (1997).
8. S. Haffner, *Metabolism*, **49**, 30 (2000).
9. J. Baynes and S. Thorpe, *Free Radic. Biol. Med.*, **28**, 1708 (2000).
10. S. Maxwell, *Basic Res. Cardiol.*, **95 (Suppl 1)**, I65 (2000).
11. B. Lipinski, *J. Diabetes Complicat.*, **15**, 203 (2001).
12. J. Anderson, M. Gowri, J. Turner, L. Nichols, V. Diwadkar, C. Chow and P. Oeltgen, *J. Am. Coll. Nutr.*, **18**, 451 (1999).
13. E. Ho and T.M. Bray, *Proc. Soc. Exp. Biol. Med.*, **222**, 205 (1999).
14. N. Cameron and M. Cotter, *Diabetes*, **46**, S31 (1997).
15. T. Miyata, K. Kurokawa and C. van Ypersele de Strihou, *Kidney Int.*, **58S**, S120 (2000).
16. B. Descamps-Latscha and V. Witko-Sarsat, *Kidney Int.*, **59**, S108 (2001).
17. T. Nguyen-Khoa, Z. Massy, J. De Bandt, M. Kebede, L. Salama, G. Lambrey, V. Witko-Sarsat, T. Drueke, B. Lacour and M. Thevenin, *Nephrol. Dial. Transplant*, **16**, 335 (2001).
18. D. Blake, P. Winyard and R. Marok, *Ann. NY Acad. Sci.*, **723**, 308 (1994).
19. A. Argyriou, E. Chroni and A. Koutras, *Neurology*, **64**, 26 (2005).
20. M. Frisch, GAUSSIAN 03, Revision C.02, Gaussian, Inc., Wallingford, CT (2004).
21. M. Veloso, A. Souza Filho, J. Mendes Filho, S. Fagan and R. Mota, *Chem. Phys. Lett.*, **430**, 71 (2006).