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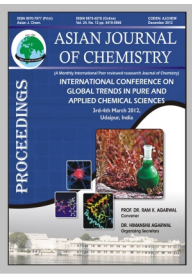

Bioavailability of Selenocystine Complex in Ten Healthy Volunteers Using Flameless Atomic Absorption Spectrophotometer†

WALEED ALI MAHMOUD

Chemistry Department, College of Science for Women, Baghdad University, Baghdad, Iraq

Corresponding author: E-mail: iraqwaleed@gmail.com

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The synthesis of selenocystine complex was formulated as 200 mcg tablet, to study the bioavailability of selenocystine complex in human sera of ten healthy volunteers investigated using flameless atomic absorption spectrophotometer. The selenocystine concentrations were measured at different intervals times from 0, 1, 1.5, 2, 2.5, 3, 3.5, 4 h. The concentrations time curve was constricted. The result observed that the maximum concentration was 135.3 ng/mL reached after 1.5 h of ingested tablet. The selenocystine complex was excreted through 4 h.

Key Words: Selenocystine, Bioavailability, Flameless atomic absorption spectrophotometer.

INTRODUCTION

Selenium is an essential trace metal for human and animals. The National Academy of Sciences recommends 200 µg of selenium per day in the diet of human adult. The role of selenium in the body has not been completely elucidated. However, selenium has been found to be essential for formation and activity of glutathione peroxides enzyme. This substance plays role in the immune system of the cell.

The enzyme is necessary to protect the body against inflammatory agents, mutagenic agents and carcinogens. It also protects the tissues from oxygen induced damage and is necessary for production of viable sperm in the male. Selenium has been found to be protective when the toxic heavy metals are consumed or breathed[1].

Research efforts have identified groups of people with low selenium intakes and presumed deficiency based on low blood selenium levels. Inhabitants of New Zealand, Finland[2] and China[3], often have low blood selenium.

Individuals with therapeutic diets low in selenium[4], people undergoing parenteral administration[5] and alcoholics with cirrhosis[6] can also be selenium deficient. Epidemiological studies have shown that low blood selenium levels have been linked to increased incidence of cancer[7,8] and heart disease[9-11].

Thus, it may be necessary to supplement the human diet with selenium in ideally the most bioavailability form of selenium. Approaches to the determination of bioavailability of physiologically important levels of selenium have been diverse and have yielded data of uncertain significance. One factor that contributes to this variation is the fact that selenium occurs in so many different chemical forms: SeO in elemental selenium, Se^{4+} in H_2SeO_3 2-(selenite) Se^{6+} in H_2SeO_3 2-(selenites) and Se^{2+} in selenomethionine complex or incorporated in protein be replacing sulphur in sulphur containing amino acids. Studies using species specific criteria such as oxidative diathesis or pancreatic fibrosis[12,13] in chicks have also yielded diverse and highly variable data. For instance, both criteria indicate a superior availability of plant over animal selenium source, but a very low availability of selenomethionine complex was found for protection against oxidative diathesis and a very high availability for protection against pancreatic fibrosis. Variation was wide in determining the effectiveness of a given selenium source fed at different levels (non-linear response) and fed at the same levels in different experiments. However, in all cases, selenite was the most available form of selenium. A good slope ratio assay has been developed relating plasma glutathione peroxidase activity to selenium intake in selenium depleted chicks. Selenite was the most available followed by selenomethionine complex, fish male, corn male and soybean male[14].

Comparatively less data occurs in the literature concerning the human bioavailability of different forms of selenium. A New Zealand study showed that selenomethionine showed more complete absorption, greater retention and smaller

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endogenous urinary and faecal losses than selenium from selenite or mackerel[15,16] human dietary study[17] which monitored urinary selenium concluded that the selenium in dairy products and eggs is more readily to treatment of selenium deficiency in men with low selenium status as measured by plasma[18]. Selenium in the present study was undertaken to investigate the available selenocystine complex in human supplementation.

EXPERIMENTAL

Ten normal subject 6 males and 4 females, aged between 18-30 volunteered with informed consent. They collected a 24 h blank urine sample before the study. Each subject appeared 2 h after eating breakfast. The selenium (200 mcg) of selenocystine complex was drunk in l00 mL of a 10 % glucose solution. First one form was given, then 90 days later the other from was ingested. Capillary blood samples were taken at 0, 1.0, 1.5, 2.0, 2.5, 3.0, 3.5 and 4.0 h. A 24 h urine sample was collected following 500 ºC overnight ashing of blood or urine with magnesium nitrate ashing acid.

The samples were reconstituted nitric acid and the selenium measured by using flameless atomic absorption spectrophotometer according to the AOAC method[19]. Determination of model HGA2000. Argon gas (99.999 % purity) was used as a purge gas at flow rate of 50 mL/min, argon gas flow was maintained continuously through drying and charring step and gas stop mode was used during atomization stage. The auto sample procedure was used for dispensing samples into graphite furnace. Instruments setting and working condition for determination of Se are wavelength 196 nm, lamp current 16 mA, volume of sample 10 µL, argon as sheathing gas, dry time 30 min, drying, ashing temperature 1000 ºC, atomization temp., 2000 ºC and atomization time 3 s.

Spiked serum: Accuracy and precision of the analytical method were studied by spiking drug free serum with selenocystine complex dissolved in 0.01 M phosphoric acid to give final concentration of 20, 50 and 90 ng/mL.

Recovery: For recovery experiments, selenocystine complex dissolved in 0.01 M phosphoric acid was added to serum to give a final concentration of 20, 50 and 90 ng/mL. after incubation at 37 ºC for 30 min., selenocystine complex was extracted as described above.

The recovery of selenocystine complex in serum were between 86 % at concentration below 20 ng/mL and 96.5 % for concentration 90 ng/mL as shown in Table-1.

TABLE-1
RECOVERY AND PRECISION OF SELENOCYSTINE COMPLEX

Sample	Drug added (ng/mL)	Drug found (ng/mL)	Recovery (%)
Spiked serum	20	17.2	86.0 ± 8.6
	50	46.60	93.2 ± 3.2
	90	86.04	95.6 ± 3.8

RESULTS AND DISCUSSION

The average blood concentration before testing (o h) was 42.4 ± 23.0 for selenocystine complex. A plot of the average selenium concentration *vs*. time for selenocystine complex is shown in Fig. 1. The area under each subjects curve was measured by planimeter and the result are given in Table-2.

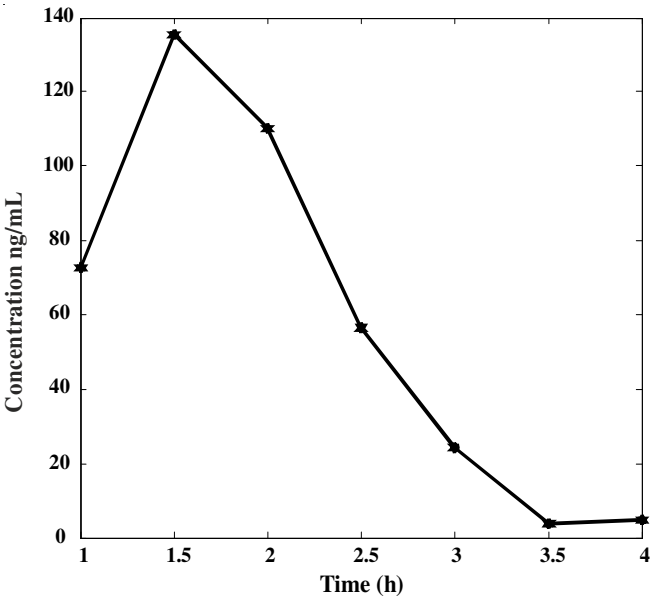

Fig. 1. Concentration time-curve of selenocystine complex in blood

TABLE-2
AREAS UNDER THE BLOOD CONCENTRATION TIME CURVES (ARBITRARY UNITS) SUPPLEMENT

Subject	Sex	Selenocystine complex
1	F	635
2	F	699
3	F	766
4	F	578
5	M	772
6	M	724
7	M	800
8	M	691
9	M	820
10	M	558
•P < 0.001		704 ± 90.4

The changes in blood selenium from the baseline (0 h) for each subject were measured and the average results in Table- 3. The selenocystine complex group had a significantly greater concentration after 1 h.

TABLE-3
CHANGES IN AVERAGE BLOOD CONCENTRATION (ng/mL) FROM BASELINE

Time (h) suppl.	1	1.5	2.0	2.5	3.0	3.5	4.0
Concentration (ng/mL) of selenocystine	72.7	135.3	110.1**	56.4*	24.2	3.8	4.9
Standard deviation (±)	31.0	40.3	49.0	36.1	15.4	8.6	4.8

*Significantly greater P < 0.05; **Significantly greater P < 0.01

The results are shown in the Table-3. The changes in selenocystine complex level in blood were decreased sharply for selenocystine complex after 1.5 h of maximum concentration selenocystine complex was an amino acid chelate has good bioavailability study. If the selenium is in a chelate form, then it must be very stable and is in competition with chelating cellular acceptor sites on the mucosa or other tissues[20] or it may be the +4 oxidation state as selenium dioxide, which is

the commonly used and least expensive form of selenium. The selenocystine complex in which the selenium is probably covalently bound to amino acid (cystine) in the -2 state had the most bioavailability.

REFERENCES

1. C.D. Thompson and M.F. Robinson, *Am. J. Clin. Nutr.*, **33**, 303 (1980).
2. T. Westermack, P. Rouna, M. Kirjinta and L. Lappalianen, *Acta. Pharmacol. Toxicol.*, **40**, 463 (1977).
3. X. Chen, G. Yang, Z. Wan and K. Ge, *Biol. Trace Elem. Res.*, **2**, 91 (1980).
4. I. Lombeck, K. Kasperek, H.D. Harbish, K. Becker, E. Schuman, W. Schroter, L.E. Feinenden and H.J. Bremer, *Eur. J. Pediatr*., **128**, 213 (1978).
5. A.M. Van Rij, C.D. Thomson, J.M. McKenzie and M.F. Robinson, *Am. J. Clin. Nut*., **32**, 2076 (1976).
6. M.J. Valimake, K.J. Harju and R.H. Ylikahi, *Clin. Chim. Acta*, **130**, 291 (1983).
7. G.N. Schrauzen, D.A. White and C.J. Schneider, *Bioinorg. Chem.*, **7**, 23 (1973).
8. K.P. McConnell, M.J. Rama, K.I. Bland and A.J. Blotcky, *J. Surg. Oncol.*, **15**, 67 (1980).
9. R. Jetmund, K.J. Bjarne, T. Yngvar and S.T. Dag, *J. Epidemiol. Commun. Health*, **41**, 329 (1987).
10. X. Forceville, R. Vitoux, A. Combes, P. Lahilaire and P. Chappuis, *Crit. Care Med.*, **26**, 1536 (1998).
11. D.K. Hayland, R. Dhaliwal, U. Suchner and M.M. Berger, *Intensive Care Med.*, **31**, 327 (2005).
12. A.H. Cantor, M.L. Langevin, T. Noguchi and M.L. Scott, *J. Nut.*, **105**, 106 (1975).
13. A.H. Cantor, M.L. Scott and T. Noguchi, *J. Nut.*, **105**, 96 (1975).
14. B.O. Gabrielson and J. Opstvedt, *J. Nut.*, **110**, 1096 (1980).
15. M.F. Robinson, H.M. Rea, R.D.H. Stewart, P.C. Snow and C.D. Thomson, *Br. J. Nut*., **39**, 589 (1978).
16. J.D. Peter, A. Alison, W. David, K. Marion, L. Bernard, G. William, D. Luke, G. Claire and Jonathan, *BMJ*, **342**, d1542 (2011).
17. I.S. Palmer, O.E. Olsen, M. Larose, R.D. Kettering and C.E. Shank, *J. Am. Diet. Assoc.*, **82**, 511 (1983).
18. G.S. Doig and F. Simpson, *Intensive Care Med.*, **36**, 1089 (2010).
19. W. Horwitz, Official methods of Analysis. Association of official Analytical Chimsts, Washington D.C., edn. 13, p. 409 (1980).
20. B.G. Shah, *Nut. Res.*, **1**, 617 (1983).