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High L-ascorbic acid content of diet modulates the non-specific immunity in rats

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This study was designed to examine the effect of high dose of L-ascorbic acid (L-AA) as a dietary supplement for rats on the phagocytic activity and oxidative burst of non-specific immune cells. Thirty Wistar rats (225-245 g initial body weight) were divided into three groups fed diet supplemented with 0.0, 0.3, and 0.6% L-AA (0, 187 and 375 mg L-AA/kg feed) for 41 days. At the end of the experiment, blood samples were analysed for selected indicators of non-specific immunity. The application of 0.6% of LAA, as compared to the remaining two groups reduced phagocytic activity measured as a number of opsonized *E. coli* cells consumed by neutrophiles and monocytes. However, 0.6% of dietary AA supplementation led to increased phagocytic monocytes and neutrophiles percentage, which produced reactive oxygen species after stimulation with opsonized bacteria (*E. coli*), phorbol 12-myristate 13-acetate (PMA) and the chemotactic peptide N-formyl-Met-Leu-Phe (fMLP). These results indicate that megadose (0.6%) of dietary AA can reduce an oxygen-independent phagocytosis, but increase the number of neutrophiles and monocytes representing the oxygen-dependent mechanism of killing.

KEY WORDS: ascorbic acid / oxidative burst / phagocytic activity / rats / vitamin C

Vitamin C is an essential compound of the diet and has been implicated for many years in boosting immune responses. Many investigations have been undertaken to elucidate the mechanism by which ascorbic acid enhances systemic immune reactions [Cummins and Brunner 1989, Gross 1992, Wu *et al*. 2000, Krause *et al*. 2001, Eo and Lee 2008]. Vitamin C plays an important role in the defense against oxidative damage,

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especially in leukocytes as the immune system is more sensitive than others to vit. C deficiency. Numerous studies have shown that vitamin C improves the phagocytic function of neutrophiles and monocytes in humans [Ciocoiu *et al*. 1998, Bergman *et a*l. 2004] and animals [Del Rio *et al*.1998, Victor *et al*. 2000].

Macrophages and neutrophiles are highly specialized phagocytes of the innate immune system. Phagocytosis, which is the characteristic function of neutrophiles and macrophages, is the act of engulfing with the aim to destroy an object, such as a microorganism [Galley *et al*. 2004]. After neutrophiles migrate into the inflammatory milieu, the production of reactive oxygen species (ROS) and the release of antimicrobial molecules such as cationic peptides, proteases and the iron-chelating molecule lactoferrin promote the elimination of the inciting agents [Appelberg 2007]. ROS kill invading microorganisms, but they also inflict damage on nearby tissue and are thought to be of pathogenic significance in many of diseases [Liu and Pope 2004].

Vitamin C is widely recognized for its ability to neutralize oxygen radicals. It protects phagocytes from damage by self-generated radicals [Krause *et al*. 2001]. According to Laggner *et al.* [2006] vitamin C protects the HL-60 cells, which are neutrophilic precursors, from damage by aggressive oxidants. It thereby increases cellular defense efficiency. However, at a high concentration this antioxidant can react with transition metals, such as iron, increasing oxidative damage to lipids, proteins and DNA [Cooke *et al*. 1998, Podmore *et al*. 1998].

The aim of the present study was to determine whether the high dose of L-ascorbic acid introduced into the diet stimulates non-specific immunity in rats.

Material and methods

Thirty male Wistar rats (225-245 g initial body weight) were divided into three groups of 10 kept in individual cages for 41 days at 22°C with 50-70% relative humidity and day/night lighting of 12/12 h. The rats had free access to water and feed and were fed a semi-purified diet according to NRC [1995] standards. The diet was supplemented with 0 (control group), 0.3 and 0.6% L-ascorbic acid (L-AA) – Table 1 – corresponding to 0, 187 and 375 mg of L-AA per kg body weight/day. The L-AA was obtained from ICN BIOMEDICALS, Aurora, Ohio, USA.

At the end of the experiment, the rats were fasted for 12 hours and then were sedated intramuscularly with ketamine (*Ketamini hydrochloricum* 5%, NARKAMON, SPOFA, Czech Republic). Blood was withdrawn from the heart into heparinized tubes and cooled to 4°C. The animals were euthanized by ketamine overdoses.

Non-specific immune indicators were assayed in the heparinized blood. Quantification of phagocytic activity of monocytes and neutrophiles stimulated by *E. coli* was performed following the method based on a commercial kit standard procedure PHAGOTEST®, Orpegen Pharma, Heidelberg, Germany [Hoffman-Jagielska *et al*. 2005]. The percentage of phagocytes which had ingested bacteria (FITC-labelled) and their activity (number of bacteria per cell) were determined with a FACStrak

Table 1. Composition of diets (% fresh matter)

Ingredient	Diet		
	0.0% L-AA (control)	0.3% L-AA	0.6% L-AA
Casein	14.0	14.0	14.0
Corn starch	62.2	61.9	61.6
Saccharose	10.0	10.0	10.0
Cellulose	5.00	5.00	5.00
Rapeseed oil	4.00	4.00	4.00
L-cystine	0.18	0.18	0.18
Choline chloride	0.10	0.10	0.10
Mineral mixture	3.50	3.50	3.50
Vitamin mixture	1.00	1.00	1.00
L-ascorbic acid	-	0.3	0.6

L-AA – L-ascorbic acid.

flow cytometer with CellQuest software (BECTON-DICKINSON, Erembodegem, Belgium).

The oxidative burst stimulated by *E. coli*, fMLP (N-formyl-Met-Leu-Phe), and PMA (phorbol 12-myristate 13-acetate) was caused in accordance with the standard procedure based on the commercial kit BURSTTEST-PHAGOBURST, Orpegen Pharma, Heidelberg, Germany [Hoffman-Jagielska *et al*. 2005]. The percentages of monocytes and neutrophiles producing reactive oxygen species (converting dihydrorhodamine to rhodamine) as well as the mean fluorescence intensity (enzymatic activity) were analysed by FACStrak flow cytometer [Hoffmann-Jagielska *et al*. 2005].

The results were evaluated with monofactorial ANOVA and Duncan's multiple range test using the Statgraphic 4.1 Plus software package (StatPoint, Inc., USA). The differences at $P < 0.05$ were considered significant.

Results and discussion

Group means for feed intake during 41 days or for initial and final body weights, were not found significantly different (Tab. 2).

Table 2. Means and standard errors (SE) for body weight and feed intake over 41 days of experimental feeding

Indicator	Diet			SE (pooled)	Significance (P-value)
	0.0% L-AA (control)	0.3% L-AA	0.6% L-AA		
Initial body weight (g)	235.0	235.1	235.1	1.82	NS
Final body weight (g)	426.7	426.3	423.5	4.12	NS
Feed intake (g/day)	20.9	21.1	21.2	0.21	NS

L-AA – L-ascorbic acid.

Supplementing the rats' diet with L-AA did not significantly affect the percentage of neutrophiles or monocytes phagocytosing *E. coli* (Tab. 3). However, the phagocytosing cells content of blood in rats kept on diet supplememted with 0.6% of L-AA was higher than in the other two groups, although it did not reach statistical significance. Furthermore, the addition of 0.6% L-AA to the diet reduced the

Table 3. Means and standard errors (SE) for phagocytic activity indicators of neutrophiles and monocytes stimulated by *E. Coli*

Indicator		Diet			SE (pooled)	Significance (P-value)
		0.0% L-AA (control)	0.3% L-AA	0.6% L-AA		
Neutrophiles	Phagocytosing cells (%)	52.84	53.96	65.53	6.781	ns
	Mean fluorescence intensity – FU[1]	408.0[b]	404.1[b]	283.8[a]	23.25	0.004
Monocytes	Phagocytosing cells (%)	38.91	39.63	53.48	5.772	ns
	Mean fluorescence intensity – FU[1]	176.6[ab]	241.5[b]	114.4[a]	24.34	0.010

[1]Fluorescence units (4 decades, 1025 channels, log).
[ab]Within rows means bearing different superscripts differ significantly at P given in the last column.
L-AA – L-ascorbic acid.

Table 4. Means and standard errors (SE) for oxidative burst activity indicators of neutrophiles

Indicator		Diet			SE (pooled)	Significance (P-value)
		0.0% L-AA (control)	0.3% L-AA	0.6% L-AA		
E. coli	Oxidizing cells (%)	57.19[a]	39.98[a]	79.22[b]	5.81	0.002
	Mean fluorescence intensity – FU[3]	23.25	22.2	21.2	1.37	ns
fMLP[1]	Oxidizing cells, %	8.22[a]	3.41[a]	11.70[b]	1.625	0.001
	Mean fluorescence intensity – FU	27.30	19.3	18.7	3.70	ns
PMA[2]	Oxidizing cells (%)	30.22[a]	22.45[a]	72.70[b]	2.438	0.001
	Mean fluorescence intensity – FU	26.52	18.2	20.8	2.64	ns

[1]N-formyl-Met-Leu-Phe.
[2]Phorbol 12-myristate 13-acetate.
[3]Fluorescence units (4 decades, 1025 channels, log).
[ab]Within rows means bearing different superscripts differ significantly at P given in the last column.
L-AA – L-ascorbic acid.

individual cellular phagocytic activity of monocytes and neutrophiles, measured as a fluorescence intensity (FU) of FITC-labelled bacteria consumed by monocytes and neutrophiles after stimulation with *E. coli*. In neutrophiles FU was significantly lower than in both the control and 0.3% L-AA, while in monocytes it was significantly lower than in the 0.3% L-AA group.

The oxidative burst of neutrophiles and monocytes in rats receiving mega-doses of L-AA was measured after stimulation with *E.coli*, PMA and fMLP (Tab. 4 and 5). Feed supplemented with 0,3% of ascorbic acid did not alter any of all measured indicators. Dietary L-AA given as 0.6% of the diet increased the content of phagocytic neutrophiles, which produced ROS after stimulation with opsonized bacteria (*E. coli*), phorbol 12-myristate 13-acetate (PMA) and the chemotactic peptide N-formyl-Met-Leu-Phe (fMLP). Moreover, 0.6% of L-AA increased the content of phagocytic monocytes, which produced reactive oxidants after stimulation with opsonised *E. coli* and fMLP.

No significant effect of applied doses of L-AA was identified on the individual cellular phagocytic activity measured as a number of FITC-labelled bacteria per cell (FU).

Compared to other cell types, leukocytes have an enriched complement of ascorbate, which is important for antioxidant balance occurring in oxygen-dependent mechanisms of phagocytosis [Gerber *et al*. 2002]. Suprisingly, high concentrations of vitamin C can exert a pro-oxidant effect *via* Fenton reactions when the most harmful hydroxyl radical is formed [Rehman *et al*. 1998]. This leads to major interrelated disruptions of cell metabolism, including protein oxidation [Bartsch and Nair 2006].

Table 5. Means and standard errors (SE) for oxidative burst activity indicators of monocytes

Indicator		Diet			SE (pooled)	Significance (P-value)
		0.0% L-AA (control)	0.3% L-AA	0.6% L-AA		
E. coli	Oxidizing cells (%)	10.19 [a]	4.15 [a]	21.68 [b]	3.306	0.009
	Mean fluorescence intensity – FU[3]	16.7	18.2	15.6	2.40	ns
fMLP[1]	Oxidizing cells (%)	4.80[a]	0.78[a]	15.01[b]	3.830	0.057
	Mean fluorescence intensity – FU	30.2	29.8	21.7	7.80	ns
PMA[2]	Oxidizing cells (%)	19.82	11.30	48.48	10,742	Ns
	Mean fluorescence intensity – FU	18.2	16.9	17.4	1.61	ns

[1]N-formyl-Met-Leu-Phe.
[2]Phorbol 12-myristate 13-acetate.
[3]Fluorescence units (4 decades, 1025 channels, log).
[ab]Within rows means bearing different superscripts differ significantly at P given in the last column.
L-AA – L-ascorbic acid.

Phagocytosis plays an essential role in host-defense mechanisms through the uptake and destruction of infectious pathogens. Phagocytes use a broad array of oxygen-dependent and oxygen-independent antimicrobial weapons to destroy and remove infectious agents. Oxygen-dependent mechanisms involve the production of reactive oxygen species (ROS), which can be antimicrobial. ROS generation contributes to inflammation and the immune response [May and Machesky 2001]. In the present study, 0.6% of dietary L-AA significantly reduced phagocytic efficiency evaluated as a number of consumed bacteria per neutrophile or monocyte cell, compared to the control group (Tab. 3). We can suppose that the mechanism of release and/or activation of antimicrobial molecules (mainly protein) could be hampered. Neutrophile granules contain a wide spectrum of antimicrobial and potentially cytotoxic substances that are delivered to the phagosome or to the extracellular environment, following degranulation [Appelberg 2006]. These compounds can be very sensitive to oxidation-reduction (redox) state of the cell environment. Imbalace of redox homeostasis in the reduction direction can inhibit antibacterial molecules impact. Loss of bioactive properties by enzymes decreases the oxygen-independent activity of neutrophiles. Consequently, phagocytosis based on oxygen-independent mechanisms could be reduced in rats fed the diet supplemented with 0.6% of L-AA. Literature reports concerning the effect of vitamin C on the engulfing capacity of the phagocytes are rather controversial. Del-Rio *et al*. [1998] reported an enhancing effect of vitamin C on phagocytic activity of mouse macrophages. However, Andreasen *et al*. [1999] did not observe any stimulatory effect of ascorbic acid on the phagocytic activity of chicken heterophiles *in vitro*.

During the phagocytosis, the reduced nicotinamide adenine dinucleotide phosphate (NADPH) oxidase complex is activated to deliver ROS into the phagosome by producing superoxide from the oxidation of NADPH and reduction of molecular oxygen [El-Benna *et al*. 2005]. In the present experiment the supplement of 0.6% of L-AA caused a significant increase in number of neutrophiles and monocytes with oxygen-dependent mechanisms, regardless of activators (Tab. 4 and 5). Probably, high doses of vitamin C, taken up into the neutrophile and monocyte cells, activate oxidative burst (*via* NADP reduction) and use this mechanism as a compensatory mechanism to stimulate defense when protection via oxygen-independent mechanisms of phagocytosis is reduced. Tal *et al*. [1998] reported inhibition of cell-free activation of the neutrophile superoxide-generating NADPH-oxidase by cationic polypeptides and proteins exhibiting enzymatic or nonenzymatic antibacterial activity (lysozyme, bactericidal permeability-increasing protein, proteases, azurocidin, myeloperoxidase, defensins) of neutrophile granules. A soluble extract of neutrophile granules negatively interfered with activation of the NADPH oxidase in a cell-free system [Aviram and Faber 1990]. Consequently, the low level of protein molecules stored within neutrophiles, as an outcome of L-ascorbic acid's effect, might increase the number of cells in which oxidative burst was observed. Furthermore, accumulation of NADPH protected by L-ascorbic acid can lead to stimulation of NADPH-oxidase during phagocytosis.

On the other hand, activation of the phagocyte NADPH oxidase requires the participation of cytosolic proteins (p67-phox, p47-phox, p40-phox and Rac ½) with the membrane cytochrome b558 [Paclet *et al*. 2007]. Proteins p67-phox and p46-phox could play a role in the regulation of NADPH oxidase by phosphorylation/dephosphorylation reactions [Benna *et al*. 1997]. These enzymatic reactions are modulated by superoxide and hydrogen peroxide. However, Afanasev [2006] suggested that mechanisms of action of superoxide differ from those of hydrogen peroxide action. Hypothetically, an excess of vitamin C in the cell, by reduction of superoxide to hydrogen peroxide, can alter activity of NADPH oxidase.

Earlier Niemiec *et al*. [2005] has documented that 0.3% of L-AA in a diet increased the vitamin C concentration of blood serum in rats, but surprisingly 0.6% of L-AA had no effect on vitamin C level of serum. L-AA can be synthesized *de novo* following the hexuronic acid pathway in the liver of rats. However, a high intake of L-AA can elevate or reduce ascorbate level in murine tissues *via* a feedback mechanism [Banhegey 1998]. The present results might suggest that the addition of 0.6% of L-AA to the diet may increase vitamin C level in neutrophiles, monocytes and other cells, as a mechanism of vitamin C level homeostasis in blood serum.

It can be concluded that supplementation of 0.3% of L-ascorbic acid had no effect on neutrophile and monocyte phagocytic activities. However, the application of 0.6% of L-AA increased the content of neutrophiles and monocytes, which produced ROS after stimulation, but the quantity of consumed FITC-labelled *E. coli* cells by immune cells was reduced. These results indicate that megadose (0.6%) of dietary L-AA can suppress phagocytosis by following an oxygen-independent mechanism, with simultaneous raise in the number of neutrophiles and monocytes with an oxygen-dependent mechanism of killing.

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Wysoki poziom kwasu L-askorbinowego w diecie moduluje nieswoistą odporność szczurów

S t r e s z c z e n i e

Celem pracy była ocena wpływu wysokiej zawartości kwasu L-askorbinowego w diecie na aktywność fagocytarną i wybuch tlenowy neutrofili i monocytów krwi obwodowej szczurów. Zwierzęta trzech grup (po 10 osobników) żywiono przez 41 dni mieszanką półsyntetyczną o różnej zawartości (0,0; 0,3 i 0,6%) kwasu L-askorbinowego (L-AA). Poziom 0,3% L-AA w mieszance nie wpływał na aktywność badanych komórek odpornościowych. Poziom 0,6% L-AA w mieszance wpłynął na zwiększenie liczby neutrofili i monocytów z uruchomionymi mechanizmami wybuchu tlenowego, a zarazem spowodował zmniejszenie liczby bakterii sfagocytowanych przez te komórki. Uzyskane wyniki wskazują, że megadawka (0,6%) kwasu L-askorbinowego w mieszance może hamować beztlenowe mechanizmy zabijania drobnoustrojów w procesie fagocytozy, zwiększając zarazem liczbę neutrofili i monocytów z uruchomionymi procesami bakteriobójczymi.