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Effects of Antimetabolite 6-Aminonicotinamide on Levels of Free Amino Acids in Various Tissues of Quail

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The effects of antimetabolite 6-aminonicotinamide on levels of soluble proteins, free amino acids and protease activity in various tissues of quail have been in vestigated. The levels of soluble proteins in liver, heart and pectoral muscle were markedly lowered and the specific activity of protease in kidney and pectoral muscle was markedly increased. The concentrations of aspartic acid / asparagine, alanine, valine, methionine, isoleucine, leucine and lysine in the liver were markedly increased. In the kidney the concentrations of aspartic acid / asparagine, arginine, threonine, alanine, proline and lysine were markedly increased but those of glutamic acid / glutamine were decreased. The concentrations of glutamic acid / glutamine and serine in the heart were reduced but those in glycine and methionine were increased. In the pectoral muscle the concentration of arginine was decreased but the concentration of alanine and threonine was increased.

The overall results suggest that antimetabolite 6-aminonicotinamide may act to enhance concentrations of amino acids related to the generation of energy and to depress the biosynthesis of some specific amino acids.

KEY WORDS: Antimetabolite, 6-Aminonicotinamide, Free Amino Acid, Quail

6-Aminonicotinamide (6-AN), an analogue of nicotinamide, is substantially incorporated into NAD and NADP by glycohydrolase to form the antimetabolites 6-amino-NAD and 6-amino-NADP which are incapable of transferring hydrogens in oxido-reduction (Herken and Neuhoff, 1964). 6-Amino-NADP acts as an inhibitor for NADP-requiring enzymes such as 6-phosphogluconate dehydrogenase, glucose-6-phosphate dehydrogenase, glutathione reductase and malic enzyme as well as for ATP biosynthesis. (Dietrich *et al.*, 1958; Kohler *et al.*, 1980; Shin and Park, 1991).

6-AN not only interferes with the pentose phosphate shunt and glycolytic pathways, but also with the catecholamine (Jung and Park, 1992) and cholesterol synthesis (Hothersall *et al.*, 1981), gamma-aminobutyric acid synthesis (Bielicki and Kreiglstein, 1976), RNA synthesis (Knoll-Kohler *et al.*, 1980). poly (ADP-ribose), purine and pyrimidine nucleotides synthesis (Hunting *et al.*, 1985) and polyamine synthesis (Morris *et al.*, 1985).

In addition, 6-AN was demonstrated to cause a marked reduction in alkaline phosphatase activity but a marked increase in activities of creatine phsphokinase, glutamic oxaloacetate transaminase and glutamic pyruvate transaminase (Park *et al.*, 1990). Although no convincing evidence is available for the present, all these enzymatic alterations appeared to be associated with the adaptive metabolic response to the shortage of energy reserves as a result of 6-AN treatment

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(Griffiths *et al.*, 1981).

The information on the effects of 6-AN on amino acid metabolism is very scanty except Gaitondę's reports that 6-AN increased the level of aspartic acid and decreased the level of glutamate but didn't affect the level of glutamine.

Thus the present study was undertaken to determine directly the effects of 6-AN on the levels of soluble proteins, free amino acids and trypsin-like protease activity in various tissues of quail.

Materials and Methods

Materials

All chemicals used were of analytical grade and purchased from Sigma Chemical Co. (St. Louis, Mo., U.S.A.).

Treatment of animals

Female quails were housed in wooden cages at room temperature at 28 °C in humidity at 60-70% and maintained on a 12h-light/12h-dark cycles, with the light phase beginning at 09:00 h. They had free access to commercial chick diet and water until reaching a weight of 130 g. Then the diet adapted quail were divided into two groups, control group of 35 and 6-aminonicotinamide treated group of 40.

The quail in the test group received intraperitoneally 0.5 ml saline containing 6-aminonicotinamide (20 mg / kg of body weight) every other day for 20 days whereas those in the control group received equal volume of 0.9% saline only. After quail were killed by decapitation, heart, kidney, brain, pectoral muscle and liver tissues were promptly removed and then weighed and stored at −70°C until used for further analysis.

Preparation of soluble proteins

An appropriate portion of frozen tissues were homogenized in 1 ml of 0.1 M potassium phosphate buffer, pH 8.0 by glass-Teflon homogenizer for 3 min at 4°C (Kim and Park, 1995) After centrifuging the homogenate at 10,000 × g for 20 min at 4°C, the pellet was discarded and the supernatant was used for the determination of concentration of proteins.

Determination of free amino acids by HPLC

The reverse-phase HPLC with fluorometric detection (Waters model 510, Milford, MA) was employed to measure free amino acids (Lee and Park, 1991). The deproteination involved the filteration of the tissue samples through the membrane filter (0.45 μm) followed by centricon treatment. The detection system was a 420-AC fluorometer equipped with a monochromator at the excitation side and a filter at the emission side. The mobile phase, consisting of buffer B (methanol-CH_3CN-H_2O in 45 : 10 : 45 ratio), was pumped at a flow rate of 2 ml/min (4,000 psi) at 45°C.

Protein determination

Protein content was determined by the method of Bradford (1976) with bovine serum albumin as standard.

Determination of protease activity

The activity of trypsin-like protease was determined by the spectrophotometric method of Jackman (1990). The enzymatic reaction involved 100 mM sodium phosphate buffer (pH 7.0), 2.5 mM TAME (p-tosyl-l-arginine methylester hydrochloride) and 10 μl of enzyme solution. The enzyme activity was assayed at 246.5 nm.

Statistical analysis

Statistical significance of differences was assessed by Student's unpaired t-test. The Student t-test was employed for the determination of statistical significance (Smith, 1962). Differences between means which give probability value (P) smaller than 0.05 are considered to be significant.

Results

Changes in body weight

The effect of administration of an antimetabolite 6-aminonicotinamide (20 mg / kg body weight) on body weight of quail for 20 days is illustrated in Fig. 1. The 6-aminonicotinamide treated animals started to show the reduction in body weight at the

fourth day of the experiment and their body weights were reduced to about 62% of the initial body weight at the twentieth day of the experiment. However, the body weights in the control animals remained virtually unchanged.

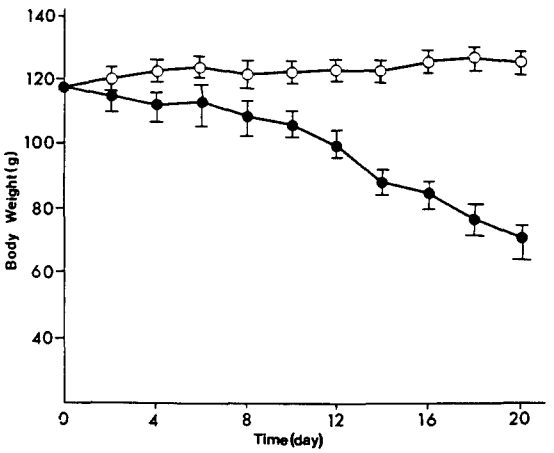

Fig. 1. Effects of 6-aminonicotinamide administration on body weight of quail. Each point represents the average body weight of 35 to 40 animals ± S.E.M. The control group (○-○) was administered saline solution whereas the 6-aminonicotinamide treated group (●-●) was administered saline containing 6-aminonicotinamide (20 mg/kg body weight) every other day for 20 days.

Table 1. Effects of 6-aminonicotinamide on levels of soluble proteins in various tissues of quail.

Organ	Treatment	
	Control	6-Aminonicotinamide
	mg/g tissue	
Liver	416±70	335±23*
Heart	172±30	118±26**
Kidney	329±32	345±74
Pectoral muscle	105±17	74±13*

Values are mean ± S.E. for 5 samples. Each sample consisted of tissues pooled from 6 animals. The control animals received 0.5 ml of 0.9% saline whereas 6-aminonicotinamide treated animals received intraperitoneally 0.5 ml saline containing 6-aminonicotinamide (20 ml/kg of body weight) every other day for 20 days.

Means in the same row not showing a common superscript letter are significantly different (*$P < 0.05$ and **$P < 0.01$).

Changes in concentrations of soluble proteins

The effect of 6-aminonicotinamide on concentrations of soluble proteins of quail was presented in Table 1. The concentrations of soluble proteins in liver ($P<0.05$), heart ($P<0.01$) and pectoral muscle ($P<0.05$) were significantly lowered as compared to those of the control animals. However, there were no significant differences in concentrations of soluble proteins in the kidney.

Activities of protease in tissues

The effect of 6-aminonicotinamide on activities of trypsin-like protease in various tissues of quail was given in Table 2. The specific activity of protease in kidney and pectoral muscle in 6-aminonicotinamide treated group was significantly higher ($P<0.025$) as compared to those in the control group. However, no significant differences in protease activity were observed in liver and heart.

Changes in concentrations of free amino acids

The effect of 6-aminonicotinamide on concentration of free amino acids in the liver of

Table 2. Effects of 6-aminonicotinamide on activities of protease in various tissues of quail.

Organ	Treatment	
	Control	6-Aminonicotinamide
	$\times 10^{-3}$mU/mg protein	
Liver	13.5±2.0	15.7±2.3
Heart	19.8±2.6	21.1±1.7
Kidney	18.0±2.9	22.1±1.0**
Pectoral muscle	8.5±1.8	11.1±0.7**

Values are mean ± S.E. for 5 experiments. Each experiment consisted of tissues pooled from 4 animals. The activity of trypsin-like protease was determined using TAME (p-tosyl-1-arginine methylester hydrochloride) as substrate.

Means in the same row not showing a common superscript letter are significantly different (*$P < 0.025$).

quail was presented in Table 3. The concentrations of aspartic acid/asparagine, alanine, valine, methionine, isoleucine, leucine and lysine in the liver of 6-aminonicotinamide treated group were significantly higher ($P<0.05$) than those in the control group. However, the concentrations of other amino acids were not significantly affected. As shown in Table 4 the concentrations of glutamic acid/glutamine and serine in the heart of 6-aminonicotinamide treated quail were reduced markedly ($P<0.05$) whereas those of glycine and methionine were increased and of other amino acids remained unchanged. In the Table 5 the concentrations of aspartic acid/ asparagine, arginine methionine, alanine, proline and lysine in the kidney of 6-aminonicotinamide treated group were markedly increased ($P<0.05$) but those of glutamic acid/glutamine were markedly reduced ($P<0.05$). In the pectoral muscle of 6-aminonicotinamide treated quail there was noticeable decrease ($P<0.05$) in the concentration of arginine but was marked increase ($P<0.05$) in the concentration of alanine and threonine.

Discussion

It has been known that 6-aminonicotinamide causes various physiological disorders such as paralysis, blindness, tremors and motor disabilities in rats, mice and cats (Johnson and McColl, 1955). Very similar phenomena were observed in our previous studies (Park *et al.*, 1990: Jung and Park, 1992). In addition, the body weight was markedly reduced. This is indicative of metabolic disturbances created by 6-aminonicotinamide (Shin and Park, 1991).

6-Aminonicotinamide was also found to reduce

Table 3. Effects of 6-aminonicotinamide on levels of free amino acids in the liver of quail.

Amino acids	Control	6-Aminonicotinamide
	μ mol/ml	
Aspartic acid/ asparagine	81±15	142±20*
Glutamic acid/ glutamine	364±35	314 ±30
Serine	76±10	80±12
Glycine	265±23	277±20
Histidine	55±8	63±11
Arginine	99±10	119±13
Threonine	76±15	98±18
Alanine	157±18	237±20*
Proline	58±8	65±10
Tyrosine	27±5	21±5
Valine	39±6	71±8*
Methionine	22±5	46±7*
Isoleucine	20±5	31±6*
Leucine	29±6	64±9*
Phenylalanine	11±5	15±4
Lysine	47±13	108±19*

All values are expressed in terms of mean ± S.D. of 4 experiment. Each experiment consisted of liver samples pooled from 5 animals. Aspartic acid/asparagine represents the sum of aspartic acid and asparagine. Glutamic acid/glutamine represents the sum of glutamic acid and glutamine. *$P<0.05$

Table 4. Effects of 6-aminonicotinamide on the levels of free amino acids in the heart of quail.

Amino acids	Control	6-Aminonicotinamide
	μ mol/ml	
Aspartic acid/ asparagine	208±30	219±28
Glutamic acid/ glutamine	388±37	295±31*
Serine	25±6	18±5*
Glycine	147±34	198±26*
Histidine	68±15	80±20
Arginine	2002±201	2193±190
Threonine	52±16	60±11
Alanine	42±12	64±17
Proline	28±7	21±8
Tyrosine	15±6	13±4
Valine	10±3	12±4
Methionine	11±7	25±13*
Isoleucine	6±1	8±3
Leucine	32±8	38±9
Phenylalanine	237±48	278±54
Lysine	22±8	21±8

All values are expressed in terms of mean ± S.D. of 4 experiments. Each experiment consisted of heart samples pooled from 5 animals. Aspartic acid/ asparagine represents the sum of aspartic acid and asparagine. Glutamic acid/glutamine represents the sum of glutamic acid and glutamine. *$P<0.05$

Table 5. Effects of 6-aminonicotinamide on the levels of free amino acids in the kidney of quail.

Amino acids	Control	6-Aminonicotinamide
	μ mol/ml	
Aspartic acid/ asparagine	72±16	102±18*
Glutamic acid/ glutamine	507±38	367±25*
Serine	129±34	164±26
Glycine	406±42	441±50
Histidine	47±13	60±20
Arginine	245±34	319±48*
Threonine	30±12	84±22*
Alanine	150±56	241±72*
Proline	41±13	92±20*
Tyrosine	17±6	24±8
Valine	34±15	58 ±11
Methionine	57±14	44±12
Isoleucine	13±7	17±8
Leucine	21±10	39±13
Phenylalanine	14±7	17±9
Lysine	43±16	98±20*

All values are expressed in terms of mean ± S.D. of 4 experiments. Each experiment consisted of kidney samples pooled from 5 animals. Aspartic acid/ asparagine represents the sum of aspartic acid and asparagine. Glutamic acid/glutamine represents the sum of glutamic acid and glutamine. *P<0.05

Table 6. Effects of 6-aminonicotinamide on the levels of free amino acids in the pectoral muscle of quail.

Amino acids	Control	6-Aminonicotinamide
	μ mol/ml	
Aspartic acid/ asparagine	23±5	24±5
Glutamic acid/ glutamine	54±13	36±12
Serine	35±10	40±13
Glycine	95±18	84±13
Histidine	11±3	14±3
Arginine	648±40	474±31*
Threonine	340±25	394±23*
Alanine	804±42	916±40*
Proline	44±12	38±11
Tyrosine	8±2	7±3
Valine	8±3	9±2
Methionine	12±4	13±2
Isoleucine	7±2	6±1
Leucine	14±3	16±5
Phenylalanine	301±35	312±40
Lysine	39±12	41±13

All values are expressed in terms of mean ± S.D. of 4 experiments. Each experiment consisted of pectoral muscle samples pooled from 5 animals. Aspartic acid/ asparagine represents the sum of aspartic acid and asparagine. Glutamic acid/glutamine represents the sum of glutamic acid and glutamine. *P<0.05.

the growth of leukemia L1210 and CHO cells (Hunting *et al.*, 1985) and cause a spongy state of the gray matter and neuronal chromatolysis in central nervous system and the preferential destruction to glial cells of rats (Sternberg and Philips, 1958). It has been suggested that these morphological neuropathogenecity may be directly or indirectly associated with the interference with glycolytic flux rate (Krieglstein and Stock, 1975), block of pentose phosphate shunt pathway (Kohler *et al.*, 1970) and reduction of concentrations of putative neurotransmitters (Bielicki and Krieglstein, 1976).

The lower concentrations of soluble proteins in liver, heart and pectoral muscle could be due to either the degradation of some specific proteins (Park *et al.*, 1991) or the depression of protein biosynthesis (Fong *et al.*, 1989).

SDS-polyacrylamide gel electrophoresis showed that the soluble proteins with molecular weights corresponding to 70, 115 and 146 kDa were absent in the liver of 6-aminonicotinamide treated group (Park's unpublished results). However, there were no noticeable differences between the control group and 6-aminonicotinamide treated group in the electrophoretic patterns of the soluble proteins in other tissues. The biological significance of absence of soluble proteins of higher molecular weight in the liver of 6-aminonicotinamide treated group is not clear yet. Further research should be carried out to identify individual missing proteins and this may provide a basis for a better understanding of their metabolic significances in protein metabolism.

The higher protease activity in kidney and pectoral muscle could be accounted for the fact that the semi-starvation created by the administration of an antimetabolite 6-

aminonicotinamide caused the substantial portions of proteins in kidney and pectoral muscle broken down to amino acids (Gan *et al.*, 1967). These amino acids may be transported from the muscle to the liver where they may be utilized for gluconeogenesis (Owen *et al.*, 1979). The mode of how 6-aminonicotinamide is involved in the enhancement of protease activity still needs to be clarified but it may be related to the adaptive metabolic response to the shortage of energy reserves as a result of the rapid decline of glycolytic flux and ATP (Krieglstein and Stock, 1975).

The most noticeable change in free amino acids in liver was the increased concentrations of aspartic acid/asparagine, alanine, valine, methionine, isoleucine, leucine and lysine. The increased concentration of aspartic acid formed as a result of the enhanced degradation of proteins by the metabolic stress (Park *et al.*, 1991) may be employed to serve as a precursor of quinolinic acid for the biosynthesis of NAD (Nasu *et al.*, 1978). Or aspartic acid, alanine, valine, methionine, isoleucine and asparagine may be converted to oxaloacetate by aspartate aminotransferase which will be eventually transformed into glucose through phosphoenolpyruvate by phosphoenolpyruate kinase (Voet and Voet, 1991). Although the reason for the increased concentration of lysine and leucine is not clear yet, it can be suggested that these ketogenic amino acids may be catabolized to acetyl-CoA which may be involved in the production of ketone body (Linder, 1991). In severe nutritional stress, ketone body may become as important as glucose as a source of energy (Voet and Voet, 1991). Similar explanation can be given to the increased concentrations of lysine and threonine in the kidney. Likewise, arginine, glutamic acid/glutamine and proline are converted into α-ketoglutarate which may be involved in the formation of amino acids essential for the urgent metabolic demands.

In contrast, concentrations of glutamic acid/glutamine in the kidney and of both glutamic acid /glutamine and serine in the heart were decreased markedly. The similarly decreased concentration of glutamate was also reported in the rat brain following 6-aminonicotinamide treatment (Gaitonde *et al.*, 1988). It has been suggested that 6-aminonicotinamide not only interferes with glucose metabolism but also with biosynthesis of some specific amino acids.

In the pectoral muscle the increased alanine could be derived from the transamination of pyruvate, carrying amino-N from the muscle to the liver (Felig, 1975). Thus the concentration of alanine may reflect predominantly changes in the overall pattern of amino acid oxidation and also of protein-dependent gluconeogenesis. It can be speculated that some amino acids produced from the degradation of proteins by protease were converted to keto acids or essential amino acids among which some of them were utilized as intermediates in TCA cycle to eventually generate glucose to maintain the basal metabolism of animals (Vander *et al.*, 1985). The metabolic significance of the increase in threonine concentration is uncertain and requires further investigations for better understanding. Whether the decrease in arginine concentration resulted from a depression of biosynthesis or a stimulation of oxidation of arginine also remains to be determined.

Although the underlying mechanisms which cause the changes in free amino acids in various tissues is unclear, it is possible that 6-AN may serve to enhance activities of protease in some tissues which lead to the formation of some glucogenic and ketogenic amino acids ultimately to generate energy essential for the maintenance of basal metabolism in animals.

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항 대사물질 6-Aminonicotinamide가 메추리 조직 내 유리 아미노산에 미치는 영향

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항 대사물질 6-aminonicotinamide가 메추리 조직의 수용성 단백질, 유리 아미노산 및 단백질 가수분해효소에 미치는 영향을 조사하였다. 간($P<0.05$), 심장($P<0.01$), 흉부근육 조직($P<0.05$)의 수용성 단백질의 함량은 대조군에 비해 현저히 감소하였다. 신장과 흉부근육 조직의 단백질 가수분해효소의 비활성도는 대조군에 비해 현저히 증가하였다($P<0.05$). 간($P<0.05$)의 aspartic acid/asparagine, alanine, valine, methionine, isoleucine, leucine, lysine의 농도는 대조군에 비해 증가하였다. 신장($P<0.05$)의 aspartic acid/asparagine, arginine, threonine, alanine, proline과 lysine은 증가하였으나 glutamic acid/glutamine의 농도는 감소하였다. 심장에서는 glycine과 methionine 농도는 증가하였으나 glutamic acid/glutamine의 농도는 감소하였다. 흉부근육 조직에서는 arginine의 농도는 감소하였으나 ($P<0.05$) alanine과 threonine의 농도는 증가하였다($P<0.05$). 본 연구 결과는 항 대사물질인 6-aminonicotinamide가 유리 아미노산의 농도를 증가시키므로써 기초 물질 대사에 필요한 에너지를 유지하는 것으로 사료된다.