Rajani Kanth, V.; Uma Maheswara Reddy, P.; Raju, T. N.
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Nutrición Hospitalaria, vol. 21, núm. 5, septiembre-octubre, 2006, pp. 596-603
Grupo Aula Médica
Madrid, España

Available in: http://www.redalyc.org/articulo.oa?id=309226704006
Abstract
Protein-restriction or deficiency is associated with many pathological disorders. We have made an attempt to study the effect of marginal tryptophan deficiency and supplementation of adequate tryptophan on the activity of antioxidant enzymes in the liver and neuronal tissue of rats. Marginal tryptophan deficiency was created in the animals (group-C) by feeding them with diet consisting of casein (6%) and gelatin (12%). Control animals (group-A) received 20% casein in their diet. Another set of animals (group-B) received the marginal tryptophan deficient diet with 0.05% tryptophan. We have observed a decrease in body weight and organ development in the deficient animals. However, a protective mechanism has been observed in the tryptophan deficient animals that received 0.05% tryptophan. Biochemical studies have shown a decrease in protein content, reduced glutathione (GSH) levels, activities of catalase, glutathione-s-transferase (GSTs) and tryptophan-fluorescence in tryptophan deficient rats. There is an increase in lipid peroxidation and AGE-fluorescence suggesting the oxidative stress due to tryptophan deficiency. However, in deficient rats that received 0.05% tryptophan in diet there was an increase in protein content, glutathione levels, catalase, glutathione-s-transferase (GSTs) levels, tryptophan-fluorescence and inhibition in AGE-fluorescence and lipid peroxidation. Our findings suggest that adequate tryptophan administration to tryptophan deficient animals has a protective influence as revealed in the activity levels of antioxidant enzymes in relation to deficient animals.


Key words: Tryptophan. Reduced glutathione. Antioxidant enzymes. AGE-fluorescence. Tryptophan fluorescence. Protein carbonyls.

Original
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Nutr Hosp. 2006;21(5):596-603
ISSN 0212-1611 • CODEN NUHOEQ

S.V.R. 318

CAMBIOS CONDUCTUALES, MORFOLÓGICOS Y FISIOLÓGICOS EN LAS RATAS CON UN RÉGIMEN DEFICITARIO EN TRIPTÓFANO
Resumen
La restricción o deficiencia de proteínas se asocian a muchos trastornos patológicos. Hemos intentado estudiar el efecto de una deficiencia marginal de triptófano y la complementación adecuada con triptófano sobre la actividad de enzimas antioxidantes del hígado y tejidos neurales de la rata. Se consiguió una deficiencia marginal de triptófano en los animales (grupo C) alimentándolos con dieta consistente en caseína (6%) y gelatina (12%). Los animales control (grupo A) recibieron caseína al 20% en la dieta. Otro conjunto de animales (grupo B) recibió dieta con deficiencia marginal de triptófano al 0,05%. Hemos observado una disminución del peso corporal y del desarrollo en los animales con deficiencia. Sin embargo, se ha observado un mecanismo protector en los animales con deficiencia de triptófano que recibieron triptófano al 0,05%. Los estudios bioquímicos han mostrado una disminución del contenido en proteínas, una reducción de la concentración de glutatión (GSH), las actividades catalasa, glutatión-S-transferasa (GST), y fluorescencia de triptófano en las ratas con deficiencia de triptófano. Hubo un aumento de la peroxidación lipídica y la fluorescencia AGE, lo que sugiere un estrés oxidativo por una deficiencia de triptófano. Sin embargo, en las ratas deficientes que recibieron 0,05% de triptófano en la dieta, hubo una aumento del contenido proteico, las concentraciones de glutatión, la catalasa, las concentraciones de glutatión-s-transferasa, la fluorescencia de triptófano, y una inhibición de la fluorescencia AGE y de la peroxidación lipídica. Nuestros hallazgos sugieren que una administración adecuada de triptófano tiene una influencia protectora como viene mostrado por los niveles de actividad de enzimas antioxidantes en relación con los animales deficitarios.


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Recibido: 8-XII-2005.
Introduction

It is known since long-time that carbohydrates, proteins and lipids are biologically active macromolecules involved in various metabolic and energy yielding processes of cellular systems. Metabolic disturbance of any of these molecules is known to contribute to several chronic pathological states. Amino acids, free as well as in the form of constituents of proteins, play an important role in maintaining the structural integrity and folding nature of proteins, and any imbalance or inadequacy of these components influence protein synthesis, thereby affecting the protein turnover. It has been reported that selective elimination of any one of the essential amino acids (i.e. L-tryptophan, L-phenylalanine and L-histidine etc.) is known to cause various pathologies. Tryptophan is an indispensable amino acid in biological systems and is involved in protein synthesis and is metabolized in mammals along the tryptophan-niacin or kynurenine pathway. Petzke, et al., have reported that chronic ingestion of high protein diets well above requirements (25.7 or 51.3% casein) does not lead to oxidative stress in adult rats when diets are adequate in antioxidants, in contrast to chronic feeding of an adequate protein diet (13.8% casein). And there were also studies indicating apoptotic tissue changes associated with depletion of essential nutrients.

Numerous investigations have been reported that the supplementation of essential nutrients such as methionine, zinc, riboflavin etc. restored the intact protein synthesis as well as redox cycle in protein restricted or deficient animals which help in retarding several pathological states. It is well documented that deficiency or accumulation of tryptophan has been implicated in various age-related complications like renal-insufficiency, cataract and AIDS dementia complex etc. Bel and Artigas have reported that the administration of tryptophan-free diet to rats resulted in a drastic reduction in the synthesis of 5-hydroxy tryptophan. Force-fed feeding of rats with elevated level of tryptophan (1%) with respect to control (0.2% tryptophan) showed a significant rise in hepatic protein synthesis, cytochrome P-450 and b5 activity.

In this study we have investigated the effect of marginal tryptophan deficiency and adequate tryptophan administration on the antioxidant enzymes of hepatic and neuronal tissues. We have also observed behavioural, morphological and physiological alterations due to tryptophan deficiency. The data related to ocular disorders and tryptophan-kynurenine and tryptophan-serotonin pathways will be discussed elsewhere.

Materials and methods

Materials: L-Tryptophan, 2-thiobarbituric acid (TBA), 1, 1, 3, 3-tetraethoxy propane (TEP), O-pthalaldehyde (OPT), reduced glutathione (GSH), 2,4-dinitrophenylhydrazine, guanidine hydrochloride and Bovine serum albumin (BSA) were obtained from Sigma (St. Louis, MO, USA). All other chemicals and solutions are of analytical grade.

Animals and diet: Male Wistar-NIN rats of weanling age (21-24 days) having an average body weight of 35-40 g, were obtained from National Centre for Laboratory Animal Sciences (NCLAS) NIN, Hyderabad and randomly assigned into three groups with twelve to fifteen animals in each group. The dietary composition was in accordance with John and Bhat with necessary modifications as per the experimental protocol.

Diet composition of each group is as follows: group A: control group (n = 12); group B: adequate-tryptophan group (0.05% trp./100gm diet) (n = 15); group C: marginal tryptophan-deficient group (n = 20). The diet composition of each group was tabulated in table I.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Dietary components</th>
<th>Control</th>
<th>Tryptophan administered (0.05%)</th>
<th>Tryptophan Deficiency (0%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Sucrose</td>
<td>71.6</td>
<td>71.6</td>
<td>71.6</td>
</tr>
<tr>
<td>2.</td>
<td>Vitamin Free casein</td>
<td>20</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>3.</td>
<td>Gelatin</td>
<td>–</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>4.</td>
<td>Refined Pea nut oil</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>5.</td>
<td>Salt mixture*</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>6.</td>
<td>Vitamin Mixture#</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>7.</td>
<td>Choline chloride</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>8.</td>
<td>L-Methionine</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>9.</td>
<td>L-Alanine</td>
<td>–</td>
<td>0.022</td>
<td>0.022</td>
</tr>
<tr>
<td>10.</td>
<td>L-Tryptophan</td>
<td>–</td>
<td>0.05</td>
<td>–</td>
</tr>
<tr>
<td>11.</td>
<td>Vitamin-A</td>
<td>10,000 IU</td>
<td>10,000 IU</td>
<td>10,000 IU</td>
</tr>
<tr>
<td>12.</td>
<td>Vitamin-D</td>
<td>800 IU</td>
<td>800 IU</td>
<td>800 IU</td>
</tr>
<tr>
<td>13.</td>
<td>Vitamin-E</td>
<td>1 mg</td>
<td>1 mg</td>
<td>1 mg</td>
</tr>
</tbody>
</table>


Marginal tryptophan deficiency was created by feeding the rats with 6% Casein and 12% gelatin. Casein consists of 0.15% L-tryptophan which is about half the minimal requirement reported for growing rats. Gelatin comprises the entire range of amino acids in the form of peptides except tryptophan. The Departmental Animal Ethics Committee has approved the animal care. The experiment was carried out for three months. All the animals had free access to food and water and were caged separately with similar ambience and the diurnal rhythm. Food intake and body weights were monitored daily and weekly respectively.

Morphological and Behavioral studies
During the study period daily food intake and weekly body weights were recorded. Morphological (hair loss) and behavioral changes were observed periodically.

Tissue extraction and processing:
At the end of the experiment animals were sacrificed by CO2 asphyxiation and tissues were harvested and stored at -80°C till further analysis. The liver and neuronal tissues were extracted from each animal and liver was perfused with normal saline (0.9% NaCl) to reduce red blood cell contamination. A 10% homogenate of hepatic and neuronal tissues was prepared and MDA levels were estimated in total homogenate. The rest of the biochemical parameters were carried out with 25,000x g supernatant and necessary centrifugations depending on the assays.

Biochemical estimation of oxidative stress markers and antioxidant enzymes:
Malondialdehyde (MDA) production was estimated by thiobarbituric acid-reactive substances (TBARS) as described by Buyan et al. Reduced glutathione was estimated by the spectrofluorometric method of Hissin et al. Total Superoxide dismutase (SOD, E.C 1.15.1.2) activity was assayed by monitoring the rate of inhibition of pyrogallol reduction. One unit of SOD represents the amount of enzyme required for 50% inhibition of pyrogallol reduction/min. Catalase (CAT, E.C 1.11.1.6) activity of tissues was measured by the method of Aebl by monitoring the disappearance of H2O2. One unit of catalase represents the decrease of 1 µmol H2O2/min. The activity of Glutathione-s-transferase (GST, E.C 2.5.18) was assayed spectrophotometrically using CDNB (1-Chloro-2, 4-dinitrobenzene) as substrate. Protein carbonyl content of soluble protein of tissues was measured spectrophotometrically using the 2, 4-dinitrophenyl-hydrazine.

Advanced glycation related fluorescence:
The Maillard reaction products i.e. advanced glycation end products (AGE) fluorescence was measured in soluble protein (0.3 mg/2 ml in 0.05 M sodium phosphate buffer, pH 7.4). Fluorescence spectra were obtained from 400-500 nm with excitation at 370 nm in a spectrofluorometer.

Tryptophan fluorescence:
Tryptophan fluorescence was measured in the soluble protein fraction (0.15 mg/ml in 0.05 M sodium phosphate buffer, pH 7.4) to determine the protein oxidation and conformational changes in different groups of animals. The fluorescence was monitored at 295 nm (excitation) and 340 nm (emission).

Protein estimation:
The protein content of tissues was estimated by the method of Lowry et al. using BSA as the standard.

Statistical analysis:
The differences between the control and treated groups were analyzed using one-way ANOVA, followed by Post Hoc test (multiple comparison). The differences were considered significant if p was at least < 0.05.

Results
Body weights and Food intake
Significant loss in body weight was observed in the animals that received tryptophan deficient diet (group-C) than that of group-A&B animals (fig. 1). The daily food consumption was significantly less in the animals, which received tryptophan deficient diet (group-C) than the groups A and B (data not shown). The food consumption in tryptophan deficient rats was reduced considerably in relation to adequate tryptophan and control groups by the end of 1.5-2 months and, deficiency became life threatening by this time. The mortality rate was 50% in tryptophan deficient group while there was no mortality in group A & B respectively.

Behavioral and morphological studies
Throughout the course of our study, animals of control and adequate tryptophan groups were healthy. Interestingly, tryptophan deficient group showed severe retardations in growth and development of muscular tissues and behavioral changes were observed.

Fig. 1.—Body weights of all the experimental animals during the course of study. The values are Mean ±SD.
system. Spinal cord of deficient animals resembled a bow in appearance. Severe hair-loss and skin lesions have been observed. However, there was no indication of dementia in tryptophan deficient animals. Ocular problems have been observed. The data related to ocular problems will be published elsewhere.

Organ development
Retardation in organ development was observed in group C with respect to their controls. However, adequate tryptophan rats (group-B) did not show any retardation of organ development (data not shown).

Protein content
There is a significant decrease in the protein content of tryptophan deficient animals (group-C) with respect to their controls (group-A) (table II). Interestingly, adequate tryptophan administration at 0.05% level to the tryptophan deficient rats (group-B) enhanced the protein levels significantly in relation to controls both in hepatic and neuronal tissues (table II).

Oxidative stress markers: TBARS and protein carbonyls content.
Tissue MDA (measured as TBARS) levels were significantly higher in tryptophan deficient rats (group-C) compared to the control group (table III). A significant inhibition of peroxidation was observed in the group-B (table III).

Carbonyl content: A significant inhibition of carbonyl formation was observed in tryptophan deficient rats (group-C) with respect to controls (table III). However, there is no significant variation in carbonyl formation in group-B with respect to controls (table-III).

Antioxidant enzymes
In tryptophan deficient rats (group-C), reduced glutathione (GSH) level had significantly decreased by 43.53% and 36.26% in hepatic and neuronal tissues respectively with respect to controls (table IIIa, b). However, glutathione content was significantly increased by 70.64% and 67.87% in hepatic and neuronal tissues of group-B animals in comparison to that of controls (group-A) (table IIIa, b).

Tryptophan deficient rats showed a significant decrease in the activity of catalase and glutathione-S-transferase in hepatic and neuronal tissues in relation to groups A and B (table IIIa, b). And there was a significant rise in these enzymes in the tryptophan-administered animals (group-B) in relation to group C (table IIIa, b).

Superoxide dismutase (SOD) activity had significantly increased in tryptophan deficient animals (group-C) compared to adequate tryptophan and control groups (table IIIa). Tryptophan administration at 0.05% had significantly restored the enzyme activity in relation to controls (table IIIa). There was no significant change in SOD activity in neuronal tissue (table IIIb).

Behavioral, morphological and physiological shift in the rats administered with tryptophan deficient regimen

<table>
<thead>
<tr>
<th>Group</th>
<th>LPO (nmol g⁻¹ wet weight)</th>
<th>GSH (µmol of H₂O₂ µmol decomposed conjugated)</th>
<th>SOD (IU µmol H₂O₂ µmol GSH protein)</th>
<th>Catalase (IU µmol H₂O₂ µmol GSH protein)</th>
<th>GST (IU µmol H₂O₂ µmol GSH protein)</th>
<th>Protein Carbonyl (µmoles mg⁻¹ protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gr-A</td>
<td>317.48 ±60.75</td>
<td>7.12 ±1.05</td>
<td>26.386 ±2.85</td>
<td>1.67 ±0.309</td>
<td>205.90 ±21.36</td>
<td>4.77 ±1.26</td>
</tr>
<tr>
<td>Gr-B</td>
<td>507.12 ±120.26</td>
<td>5.03 ±0.87</td>
<td>35.948 ±5.52</td>
<td>1.43 ±0.085</td>
<td>154.66 ±14.19</td>
<td>3.84 ±1.11</td>
</tr>
<tr>
<td>Gr-C</td>
<td>907.41 ±48.42*</td>
<td>3.10 ±1.24*</td>
<td>57.278 ±3.70*</td>
<td>1.22 ±0.247*</td>
<td>89.75 ±24.27*</td>
<td>2.28 ±0.41*</td>
</tr>
</tbody>
</table>

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Advanced glycation end product (AGE) fluorescence:

AGE fluorescence (fig. 2) reveals an increase in AGEs formation in tryptophan deficient rats (group-C) compared to controls (group-A). Interestingly, the tryptophan-deficient animals which received 0.05% tryptophan had shown significant inhibition of AGE fluorescence (fig. 2).

Tryptophan fluorescence:

Tryptophan fluorescence spectra (fig. 3) showed a decrease in tryptophan fluorescence in tryptophan deficient rats (group-C) in relation to controls (group-A). However, adequate tryptophan administration has been found to inhibit the decrease in tryptophan fluorescence in comparison to group-C (fig. 3).

Discussion

It is well documented that protein deficiency / restriction will affect the morphological, physiological status of the cellular systems possibly through the generation of free radicals / reactive oxygen species, which damage the integrity of biological systems leading to several pathological states24,25. In our present study, we have noticed a significant decrease in food intake, body weight, organ development and retardation in muscular system in the rats that were fed on tryptophan deficient diet and this is in agreement with the previous observations of protein deficiency5,24,26-28.

However, these features were not seen in adequate tryptophan group and this is suggestive of a clear protective influence of tryptophan in maintaining the physiological status with respect to controls. In an earlier study carried out by John and Bhat6 no significant variation in the protein content, tryptophan levels and antioxidant enzymes between the control and pair-fed control groups was reported. In a pilot experiment carried out by us we didn't notice significant variation in the control and pair-fed control groups (data not shown). Therefore, in order to minimize the number of animals due to ethical constraints, we could not maintain pair-fed control groups in our investigation.

Table IIIb

<table>
<thead>
<tr>
<th>Group</th>
<th>LPO (nmol g-1 wet)</th>
<th>GSH (µmol of µmol H2O2)</th>
<th>SOD (IU)</th>
<th>Catalase (µmol H2O2 decomposed conjugated)</th>
<th>GST (µmol CDNB)</th>
<th>Protein Carbonyl (µmol mg-1 protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gr-A Control</td>
<td>281.54 ±64.07</td>
<td>3.86 ±0.66</td>
<td>4.00 ±1.99</td>
<td>0.801 ±0.135</td>
<td>96.91 ±30.85</td>
<td>2.72 ±0.34</td>
</tr>
<tr>
<td>Gr-B Tryptophan administered (0.05%) (67.87)</td>
<td>378.22 ±72.27</td>
<td>2.62 ±0.85</td>
<td>3.27 ±1.13</td>
<td>0.618 ±0.060</td>
<td>68.00 ±24.86</td>
<td>2.48 ±0.52</td>
</tr>
<tr>
<td>Gr-C Protein-deficient</td>
<td>730.08 ±54.26 *</td>
<td>1.40 ±0.60 *</td>
<td>2.95 ±0.75</td>
<td>0.455 ±0.050 *</td>
<td>33.57 ±14.43 *</td>
<td>0.90 ±0.12 *</td>
</tr>
</tbody>
</table>

Fig. 2.—AGE fluorescence of hepatic and neuronal tissue, which were fed on different protein concentration with supplementation of tryptophan. Data are average of five values.
with tryptophan deficient regimen physiological shift in the rats administered Behavioral, morphological and synthesis.

Tryptophan, an essential amino acid, plays a crucial role in protein content to a moderate extent (70.17% and 0.05%) to tryptophan-deficient animals restored the values.

Fig. 3.—Tryptophan fluorescence of soluble protein of hepatic systems. In the present study we have observed peroxides mediated through enzymatic and non-enzymatic systems. In the present study we have observed a decrease in protein content up to 54.69% and 45.73% in hepatic and neuronal tissues respectively in relation to controls which is in agreement with the earlier reports. Interestingly, administration of tryptophan in tryptophan deficient rats and this indicates a higher level of peroxidation in these rats. Van Pilsum et al., had observed a decrease in catalase activity in tryptophan-deficient animals increased the activity in- dicating an inhibition or detoxification of H2O2. However, tryptophan administration at 0.05% raised the levels of glutathione. However, tryptophan administration at 0.05% raised the levels of glutathione.

The healthy status of living cells can be assessed by their structural integrity and in assessing oxidative damage. As a result, proteins undergo rapid oxidation leading to the alterations in balance in this ratio thereby making macromolecules vulnerable to oxidative damage. As a result, proteins undergo rapid oxidation leading to the alterations in balance in this ratio thereby making macromolecules vulnerable to oxidative damage. In normal physiological state, oxidants generated by metabolism play a significant role in maintaining the redox cycle tripeptide i.e. reduced glutathione since it is the coenzyme for several enzyme-catalyzed reactions and participates in the detoxification processes besides its role in a wide variety of substitution reactions and de-sulphonic enzymes, conjugate with GSH and play a crucial role in the maintenance of cellular integrity. It is also a major source for the nicotinamide and coenzymes, NAD and NADP.

The levels of catalase, an ubiquitous hydrogen peroxide detoxifying enzyme, had decreased in tryptophan-deficient rats. This observation is in accordance with the previous reports. Inclusion of 0.05% tryptophan to rat brains didn't show significant variation in the levels of catalase activity in hepatic tissue and neuronal tissue in different groups. Data are average of five values.

Animals subjected to the deficiency of essential amino acids. There were also reports suggesting that decreased in protein content and this could be the underlying mechanism for the observed decrease in carbonyl content in tryptophan-deficient rats. This observation is in agreement with the previous report of Youngman et al., who had observed a decrease in carbonyl formation in the aging rats which were fed on protein-restricted diet. However, it cannot be construed that the tryptophan administration inhibited the peroxidation and oxidative stress in these animals. However, adequate tryptophan administration restored the levels of catalase activity and glutathione levels in the tryptophan deficient rats indicating an inhibition or detoxification of H2O2. In pathological state, glutathione will decrease in pathological states the- erby making the cells prone to oxidative injury. In pathological state, glutathione will decrease in pathological states thereby making the cells prone to oxidative injury.
behavioral with respect to tryptophan administration and control groups. This might possibly be due to the high oxidative stress deficiency in methionine, tryptophan, isoleucine, or choline deficiency in methionine, tryptophan, isoleucine, or choline. The probable mechanism in the increase of SOD activity in hepatic tissue of tryptophan deficient animals indicating the possible glycation, AGE fluorescence has been increased in tryptophan deficient animals suggesting the tissue.

This work was supported by University Grant Commission (UGC) (No. F. No. 4-27/2001 (BSR)) for providing fellowship to Dr. G. Bhanu Prakash Reddy, Senior Research Officer, NIPER, with an additional grant from the State Government of Tamil Nadu. The authors would like to thank Dr. S. Subramanian, Professor in Microbiology, for the valuable comments on the manuscript. The authors declare that there is no conflict of interest.

In summary, our observations reveal that the administration of tryptophan protects the tissues from oxidative damage induced through tryptophan deficiency. Our findings also suggest that administration of tryptophan protects the tissues from oxidative insult. Tryptophan administration at 0.05% raised the SOD levels in relation to tryptophan deficient animals. The probable mechanism in the increase of SOD activity in hepatic tissue of tryptophan deficient animals may be attributed to decreased levels of tryptophan and increased IDO levels as some of the tryptophan metabolites are neurotoxic.

Acknowledgments:

We have not observed any significant change in the protein-bound sulphydryl level in the hepatic tissue of tryptophan deficient rats and due to low concentration of GSH, this could be due to variation in IDO levels as some of the tryptophan metabolites are neurotoxic. We have not observed any significant change in the protein-bound sulphydryl level in the hepatic tissue of tryptophan deficient animals indicating the possible glycation, AGE fluorescence has been increased in tryptophan deficient animals suggesting the tissue.


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