EFFECTS OF DIETARY MUTTON ON NITRIC OXIDE, NITRIC OXIDE SYNTHASE, LIPID PEROXIDATION IN RATS

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***Abstract*—Thirty SD rats were allotted to two groups randomly, which were subjected to the following treatments: feeding with the basal diet containing 20% protein as control group(n=15), supplementation with 20% mutton powder to the basal diet at a final concentration of 20% protein as mutton group(n=15).After four weeks, the rats were killed and effects of nitric oxide(NO), nitric oxide synthase(NOS), superoxide dismutase (SOD)and malondialdehyde(MDA) in plasma were examined. Results indicated that NO、MDA and SOD of serum in mutton group were significantly higher than control group. Comparing with control group, NOS in serum was significantly decreased. Thus, it was demonstrated that mutton could increase the produce of NO, raise excessive oxide free radicals and lipid peroxidation lesion induced by NO and other free radicals.**

***Index Terms*—mutton, Nitric Oxide, Nitric Oxide Synthase, Lipid Peroxidation**

I. INTRODUCTION

Mutton is the major source of meat in human life. Currently, some studies assumed that meat, especially red meat, enhances risk for cancer, particularly of the colon, breast and prostate. Antioxidant capacity plays an important role in the formation of cancer. Proto-oncogene ras carcinogenic fibroblasts confirmed, the level of superoxide radical $\bullet O_2^-$ significantly increased in cancer cells[Irani K et al,.(1997), Penndsi E(1997)].Thus, tumor cells could produce superoxide[Szatrowski TP &Nathan CF(1991)].And Cancer-causing free radical theory presumed that two stages of cancer related reactive oxygen species. Lipid peroxidation in tumor tissue was significantly higher, but antioxidant enzyme activities decreased and the balance of Free Radicals was broken[Zima T et a1,.(1996), Slaga TJ,(1995)].With this background, In this paper, mutton was used to be experimental materials .We investigated the effects of long term use of dietary mutton on Nitric Oxide，Nitric Oxide Synthase, Lipid Peroxidation in rats.

II. MATERIALS AND METHODS

2.1. Materials

Synthetically prepared mutton powder was provided by Frozen mutton(White goat leg)from ShangDong, dried to 10% moisture content, then supplementation with 20% mutton powder to the basal diet at a final concentration of 20% protein..

2.2. Animals and diet

Thirty 1-week-old male Sprague Dawley rats, weighed 100-105g, purchased from Nanjing

Qinglong Animal Breeding Centre. Animals were housed in wire-bottomed individual cages in a room on naturally light/dark cycle, under protocols approved by the College of Food Science and Technology of Nanjing Agricultural University. Animals were divided into two groups based on body weight and fed either control or mutton containing diet for 4 weeks.

The diet was as follows(ingredient, %): corn 55, mutton powder 20, Soybean meal 5.3, wheat 17, bone meal 2.0,salt 0.2, premix 0.5 (DE 15.9 kJ/kg，CP20%). Control diet was pet rat chow without mutton powder(DE 15.8 kJ/kg，CP 20%).Diet was stored at -20° C until use. Diet and water, available ad libitum, were freshly provided twice a week.

2.3. Sacrifice and other analyses

At the end of the feeding period, animals were sacrificed by stunned and decapitation blood. After centrifugation, serum sorted at -20° C until use.

2.4 indexes

NO and total NOS Activity Assay were used Enzymatic; the total SOD activity assay was used Xanthine oxidase; MDA was used TBA. All indexes were followed as kit's instructions and standards.

2.5. Statistical analysis

Data are shown as means and standard errors. All analyses were carried out using SAS software (Version 9.1.0, SAS institute Inc) by the generalized linear model procedure and least square means options. Significance was defined at $p < 0.05$, extremely Significance was defined at $p < 0.01$.

III. RESULTS AND DISCUSSION

There was extremely significant difference in T-NOS、i-NOS and NO between control and mutton groups.(Table.1). Comparing with control group, the levels of T-NOS and i-NOS in serum were extremely significantly decreased($p<0.01$). But NO in serum was significantly increased than control groups ($p<0.01$). The level of NO in serum, control 4.00±0.49μmol/L, and mutton group 18.89±0.62μmol/L(n=30).

Table.1Effects of dietary mutton on NO, T-NOS and i-NOS in rats' serum (n=30)

Diet	T-NOS(U/ml)	i-NOS(U/ml)	NO（μmol/L）
control	47.03 ± 1.23^{A}	8.32 ± 0.24^{A}	4.00 ± 0.49^{A}
mutton	40.20 ± 1.68^{B}	7.28 ± 0.19^{B}	18.89 ± 0.62^{B}

Note:1 means with different capital letters are extremely significantly different（$P<0.01$）, the number is mean±SE, the same below.

There was extremely significant difference in SOD and MDA between control and mutton groups.(Table.2). The SOD and MDA in mutton group were both extremely significantly increased than control groups ($p<0.01$). The level of SOD in serum, control 172.62±4.76U/mL, mutton group 206.64±2.81U/mL, and MDA in serum, control 9.01±0.60nmol/ml, mutton group 12.50±0.91 nmol/ml (n=30).

Table.2 Effects of dietary mutton on SOD and MDA in SD rats' serum(n=30)

Diet	SOD(U/ml)	MDA(nmol/ml)
control	172.62 ± 4.76^{A}	9.01 ± 0.60^{A}
mutton	206.64 ± 2.81^{B}	12.50 ± 0.91^{B}

NO was generated by the vascular endothelial cells and released as a relaxing factor, it was an important cytokines to adjust and participate in physiological and pathological processes in the body. NO as a free radical and transmitter, plays an important role in transferring information, participate in inflammation and tissue damage and cell proliferation, regulating gastrointestinal motility and mucosal protection. When the balance of NO in body was broken, too much or too less, would induce the disease [Syder SH (1992), Nguyen T, Saberg HD(1992)].NO biosynthesis through a branch of ornithine cycle to complete, L-arginine and molecular oxygen generated in the three different NOS-catalyzed, such as endothelial NOS, neuronal NOS and inducible NOS[Knowles RG(1994), Vincent SR(1994)]. The result shows that, for mutton group, NO in serum was extremely significantly increased, but NOS was extremely significantly decreased. Excess of NO could cause tissue damage, because of NO with superoxide anion can be to form a powerful cytotoxic substances-peroxynitrite(ONOO-)[Beckman JS, Koppenol WH(1996)]. Under normal circumstances, the free radicals were in this balance. In this study, after long-term consumption of mutton, rats' body produced and accumulated excess of free radicals, but the cleaning ability of SOD was limited in serum. Thus, the balance between generation and control was broken, and accumulated a large number of free radicals. Resulting in a large number of MDA in serum, lead to lipid peroxidation eventually. The level of MDA reflects attacks of the body cells severity and the degree of cell damage by free radicals [Tatsumi N,Fujisawa M,Kanzakim,et al,.(1997)]. The study indicated that mutton could raise rats body lipid peroxidation lesion.

IV. CONCLUSION

Results from this report indicate that after long-term consumption of mutton, could increase the produce of NO, raised excessive oxide free radicals and lipid peroxidation lesion. This could enhance risk for cancer.

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