INHIBITORY EFFECT OF CAPSAICIN ON INTESTINAL THIAMINE ABSORPTION IN MICE

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ABSTRACT

The effect of capsaicin, a pungent principle of capsicum fruits, on intestinal thiamine absorption was examined in mice. In situ *study with*^{14} *C-thiamine hydrochloride by using intact jejunal loops, active thiamine absorption was found to reduce after exposed to capsaicin 7-42 mg% for 30 minutes. The maximum inhibition was observed approximately 45.82% at a concentration of capsaicin 42 mg%. The percent inhibition of thiamine absorption was related to capsaicin concentration. In contrast, capsaicin did not affect absorption of high concentration of thiamine in the intestinal lumen. The possible mechanism of capsaicin inhibitory action on thiamine absorption was also investigated. Capsaicin at concentrations of 3.5, 7, 14 and 21 mg% reduced the activity of* Na^+-K^+ *ATPase by 6.97%, 14.26%, 17.21% and 19.40% respectively while the mucosal ATP content was significantly inhibited by capsaicin only at 42 mg%. Moreover, no inhibitory effect of capsaicin on thiamine uptake in the intestinal brush-border membane vesicles was seen. These results suggest that there is a correlation between capsaicin inhibition of mucosal* Na^+-K^+ *ATPase activity and mucosal ATP content and the reduction of thiamine absorption in mouse jejunum* in situ.

INTRODUCTION

Thiamine deficiency is one of the public health problems among population in countries where the polished rice is the main dietary constituent. In Thailand, thiamine deficiency was found to be 21.6% and 25.0% in the northern and north-eastern respectively.[1] The factors that responsible for thiaminedeficiency are an inadequate intake of thiamine[2] or a decrease of the thiamine activity by antithiamine factors in food[3] or an intestinal absorption of thiamine may be impaired.[4] Recently, the reports of sudden death in sleep among Thai construction workers in Singapore suggested that low thiamine intake might be the cause of these deaths as well as those reported in the South-East Asian refugees.[2, 5] In Asia, especially in Thailand, it has been known for a long time that hot, green, red and yellow pepper are the most common spice used in cooking food. Capsaicin is the pungent material present in capsicum fruit[6] and the average capsaicin in diet that is consumed by Thai people is 14 mg% .[7] It was found that capsaicin impaired glucose absorption in rat and hamster both *in vivo* and *in vitro*.[8, 9] It is therefore of interest to see whether or not capsaicin can impair intestinal thiamine absorption.

MATERIALS AND METHODS

Materials

Capsaicin and all other reagents were obtained from Sigma Chemical Co. (St. Louis, MO). ^{14}C-thiamine HCl (specific activity 23 mCi/mmol) was purchased from Amersham Radiochemical Centre Ltd., (Bucks., UK). Membrane filter (pore size 0.45 μm, diameter 2.5 cm) was obtained from millipore Corporation (Bedford, MA).

Animals

Adult male Swiss albino mice (35-40 g) supplied from the Animal Production Center, Faculty of Science, Mahidol University, were caged in groups of six and given rat pellets, until the time of experiment, and water *ad libitumm*.

Effect of capsaicin on intestinal thiamine absorption in situ

Ligated loops of intestine were prepared by the method of Hoyumpa et al.[10] Mice were fasted overnight and weighed prior to perform the experiment. The animal was anesthetized with ether and the abdomen was opened. The luminal content in the upper jejunum (about 10 cm) was washed with Krebs-Henseleit-HCO_3 buffer, pH 7.4 containing 1.5 μM or 20 μM ^{14}C-thiamine HCl (specific activity 23 mCi/mmol) with various concentrations of capsaicin. For control group, the loop was filled with buffer containing ^{14}C-thiamine HCl and vehicle (absolute ethanol : Tween 80:0.9% NaCl; 10:10:80; V:V:V). The loop was replaced in the abdomen for absorption to proceed. After 30 min, the loop was excised and the content was flushed with 5 ml of buffer and the volume was mesured. The exact length in cm of the loop was measured. The loop was next opened lengthwise and shaken vigorously in the flask containing 5 ml of buffer. The opened loop was placed in the preweighed vial and oven-dried at 93°C overnight. 100 μl aliquots of both washings and the test solution were each counted in 7 ml of scintillation fluid by using the liquid scintillation counter (LKB Wallac, Rackbeta 1219). The rate of disappearance of labelled thiamine from the isolated loop was measured after subtracting backgroud counts. The results were expressed as μg/g tissue dry weight/30 min.

Effect of capsaicin on intestinal mucosal Na^+-K^+ ATPase activity and ATP content in situ

Mice were fasted overnight and the intestinal loops were done as the method previously described. The loop (8 cm) was filled with 0.75 ml of 1.5 μM thiamine HCl with and without various concentrations of capsaicin ranging from 3.5-42 mg%. At the end of 30 min, the loop was excised, discarded its content and was flushed with ice cold saline solution. For determination of the mucosal ATPase activity, the mucosal tissue was gently scraped with a glass slide from the opened segment, weighed and then homogenized. The mucosal ATPase activity was determined according to the method of Kramer et al.[11] Mg^{2+} –ATPase activity was assayed in the presence of 1.0 mM ouabain, and the Na^+-K^+ ATPase activity was calculated as the difference between total ATPase

and Mg^{2+} –ATPase activities. Inorganic phosphate was determined by the method of Eibel and Lands[12] and proteins by that of Lowry et al.[13] ATPase activities were expressed as umole Pi released/mg protein/h. The intestinal mucosal ATP was extracted by the method of Dietrich and Friedland.[14] The ATP content was determined by Sigma Kit No. 366-UV.

Effect of capsaicin on thiamine uptake across intestinalbrush-border membrane (BBM)

Mice were sacrificed by decapitation and the BBM vesicles were prepared by the method previously described by Muir et al.[15] The pellets of BBM vesicles were suspended in buffer to obtain the protein concentration of 5-8 mg/ml. In order to determine the purity of BBM, biochemical assay of marker enzymes and electron microscopie examination were employed.

In the study of thiamine transport in BBM vesicles, vesicles (200 μg protein) were preincubated with and without capsaicin concentrations of 0.1-2.0 mM for 10 min at 37°C. Thiamine uptake was initiated by adding 1.25 μM ^{14}C-thiamine HCl (specific activity 24.2 mCi/mmol) and incubation medium provide for final concentrations of 100 mM D-mannitol, 2 mM $MgSO_4$, 10 mM Tris-Hepes, pH 7.5 and 100 mM NaCl to the final volume of 1 ml in each tube, then incubated at 37°C for 30 min. At the end of incubation time, thiamine uptake was stopped by adding 1.5 ml ice-cold stop solution (300 mM D-mannitol, 10 mM Tris-Hepes, pH 7.5 and 0.5 mM thiamine HCl) and then filtered through millipore filter (0.45 μm pore size), and immediately washed with 3 ml of ice-cold stop solution three times. The millipore filter paper with membrane vesicles was then transferred to 7 ml scintilltion fluid and counted in liquid scintillation counter. The thiamine uptake was expressed as pmole/mg protein/30 min.

RESULTS

The effect of various concentrations of capsaicin on intestinal active thiamine absorption in mouse jejunum *in situ* is summarized in Table 1. The results indicated that all concentrations of capsaicin (7-42 mg%) significantly inhibited thiamine absorption in mouse jejunum. Inhibition of thiamine absorption was detected at a concentration as low as 7 mg% capsaicin by 23.91% ($P < 0.01$) For higher concentrations of capsaicin at 14 mg%, 21 mg%, 35 mg% and 42 mg%, the degrees of inhibition of thiamine absorption were 32.2% ($P < 0.001$), 37.65% ($P < 0.001$), 43.06% ($P < 0.001$) and 45.82% ($P < 0.001$) respectively. A maximum inhibition was observed when a concentration of capsaicin at 42 mg%. It was found that active thiamine absorption was dose dependent. Additionally, it appeared that the percent inhibition of thiamine absorption increased from 7-42 mg% capsaicin with corresponding to the increment of the amount of thiamine left in the lumen.

Table 2 shows the effect of capsaicin on intestinal active and passive thiamine absorption in mouse jejunum *in situ*. The rate of passive thiamine absorption at high concentration of thiamine (20 μM) was higher than that of active thiamine absorption at low concentration of thiamine (1.5 μM). Capsaicin at a concentration as high as 21

TABLE 1 Effect of capsaicin on active thiamine absorption in ligated loops of mouse jejunum incubated in Krebs-Henselit HCO_3 buffer (pH 7.4) containing 1.5 μM ^{14}C-thiamine HCl with and without capsaicin *in situ*

Capsaicin (mg%)	Rate of Thiamine disappearance from lumen (μg/g tissue dry wt./30 min)	Inhibition[a] (%)	Amount of thiamine left in Lumen (ng)
0	20.58 ± 1.51	–	12.53 ± 2.51
7	15.15 ± 0.64**	23.91 ± 4.49	18.78 ± 3.62
0	19.57 ± 1.00	–	12.56 ± 2.69
14	12.96 ± 0.87***	32.20 ± 4.58	24.55 ± 3.48***
0	19.41 ± 1.31	–	12.69 ± 2.43
21	11.99 ± 0.77***	37.65 ± 3.53	36.13 ± 1.22***
0	19.38 ± 1.75	–	12.69 ± 3.07
35	10.79 ± 0.77***	43.06 ± 4.65	48.61 ± 2.42***
0	19.44 ± 0.96	–	12.83 ± 2.96
42	10.51 ± 0.55***	45.82 ± 2.56	51.38 ± 93***

[a] The degree of inhibition is derived from a comparison of the control and capsaicin treated experiments of the rate of thiamine disappearance from lumen.

Values are means ± S.E.M. of 6 mice.

Significant values (Student's t-test) between the control and capsaicin groups are indicated by asterisks ; **P < 0.01, ***P < 0.001.

TABLE 2 Effect of capsaicin on passive thiamine absorption in ligated loops of mouse jejunum incubated in Krebs–Henseleit–HCO_3 buffer (pH 7.4) containing 1.5 μM or 20 μM ^{14}C–thiamine HCl with and without capsaicin *in situ*

Capsaicin (mg%)	Thiamine (μM)	Rate of Thiamine disappearance from Lumen (ng/cm/5 min)	Inhibition[a] (%)
0	1.5	6.56 ± 0.11	–
21	1.5	5.65 ± 0.07***	13.90 ± 1.04
0	20	32.62 ± 2.18	–
21	20	31.82 ± 2.30	2.46 ± 0.14

[a] The degree of inhibition is derived from a comparison of the control and capsaicin treated experiments of the rate of thiamine disappearance from lumen.

Values are means ± S.E.M. of 6 mice.

Significant value (Student's t-test) between the control and capsaicin groups is indicated by asterisks : ***$P < 0.001$.

TABLE 3 Biochemical contents in purified brush-border membrane vesicles from mouse jejunum

Biochemical Content	Cell Fraction	
	Homogenate	Vesicle
Protein (mg/g tissue)	133.12 ± 7.60	1.20 ± 0.26
DNA (mg/g tissue)	136.53 ± 9.28	0.39 ± 0.04
Sucrase (IU/mg protein)	0.03 ± 0.00	0.52 ± 0.02
Na^+–K^+ ATPase (IU/mg protein)	0.002	0.002
NADH cytochrome C reductase (nmol/mg protein/min)	86.02 ± 9.31	13.26 ± 1.41
NADPH cytochrome C reductase (nmol/mg protein/min)	3.46 ± 0.22	0.92 ± 0.07

Values are means ± S.E.M. of 6 preparations.

TABLE 4 Effect of capsaicin on thiamine uptake across brush-border membrane of mouse jejunum *in vitro*

Capsaicin Conc. (mM)	Thiamine Uptake (pmol/mg protein/30 min)	% Change from control
0	1.94 ± 0.28	–
0.1	1.97 ± 0.68	↑ 1.54
0	2.32 ± 0.27	–
0.25	2.37 ± 0.38	↑ 2.15
0	2.68 ± 0.37	–
0.5	2.38 ± 0.28	↓ 11.20
0	2.46 ± 0.17	–
1.0	2.10 ± 0.24	↓ 14.64
0	2.31 ± 0.27	–
2.0	2.08 ± 0.44	↓ 9.96

Values are means ± S.E.M. of 6 preparations.

mg% was found to inhibit the intestinal active thiamine absorption by 13.90% ($P < 0.001$) whereas capsaicin at this concentration could not inhibit the intestinal passive thiamine absorption.

Fig. 1 shows the effect of various concentrations of capsaicin on intestinal mucosal Na^+-K^2 ATPase in mouse jejunum *in situ*. Capsaicin (3.5-21 mg%) significantly inhibited mucosal Na^+-K^2 ATPase but no effect on Mg^{2+} −ATPase activity. Inhibition of Na^+-K^+ ATPase activity was detected at a concentration as low as 3.5 mg% capsaicin by 6.97% ($P < 0.05$). Higher concentrations of capsaicin at 7 mg%, 14 mg% and 21 mg%, the degrees of inhibition of Na^+-K^+ ATPase were 14.26% ($P < 0.01$), 17.21% $P < 0.01$) and 19.40% $P < 0.001$) respectively. A maximum inhibition was observed when a concentration of capsaicin at 21 mg%. It appeared that the percent inhibtion of mucosal Na^+-K^+ ATPase activity was progressively increased with the concentration of capsaicin up to 7 mg% and then slightly increased from 7-21 mg%.

The effect of various concentrations of capsaicin on intestinal mucosal ATP content in mouse jejunum *in situ* is shown in Fig. 2. There were no significant difference in mucosal ATP content between control and capsaicin-treated animals at concentratins from 3.5-21 mg% during thiamine absorption. The amount of ATP content was significantly inhibited by 42.86% ($P < 0.001$) in mice treated with high concentration of capsaicin 42 mg%.

The activity of enzymes markers, DNA and protein contents in membrane vesicles are shown in Table 3. The specific activity of NADH cytochrome C reductase and NADPH cytochrome C reductase in BBM vesicle was reduced from the homogenate preparation. However, Na^+-K^+ ATPase activity was not altered whilst the sucrase activity increased by 17 folds. The amounts of protein and DNA were reduced by 130 and 350 folds respectively. The purity of BBM of mouse jejunum, however, was also supported by electron microscopic examination in which the membranes were mostly vesiculated with diameters about 0.10 μm (Fig. 3).

Table 4 summarizes the effect of various concentrations of capsaicin on thiamine up take across BBM vesicles of mousejejumum. These was no changes in thiamine uptake across BBM after 30 min exposed to capsaicin at concentrations of 0.1 mM and 0.25 mM. Higher concentrations of capsaicin at 0.5 mM, 1.0 mM and 2.0 mM, thiamine uptake was slightly inhibited by 11.20%, 14.64% and 9.96% respectively. However, there were no statistical significance.

DISCUSSION

The results present here demonstrated that capsaicin has the ability to inhibit intestinal active thiamine absorption (Table 1). In contrast, it has no effect on passive thiamine absorption (Table 2). Capsaicin inhibit active thiamine absorption in a dose dependent manner similar to the results from other investigation on the inhibitory effect of capsaicin on intestinal glucose absorption in both rat and hamster.[9, 16] The mechanisms by which capsaicin interfered with active transport of thiamine across the intestine were investigated.

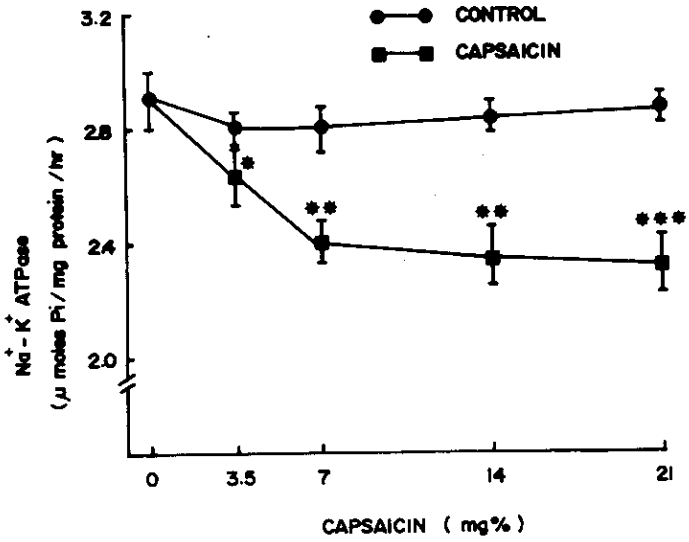

Fig. 1 Effect of various concentrations of capsaicin on mucosal Na^+-K^+ ATPase activity of mouse jejunum *in situ*. Each value is mean ± S.E.M. of 6 mice. Significant values (Student's t-test) are indicated by asterisks : *P < 0.05, **P < 0.01, ***P < 0.001.

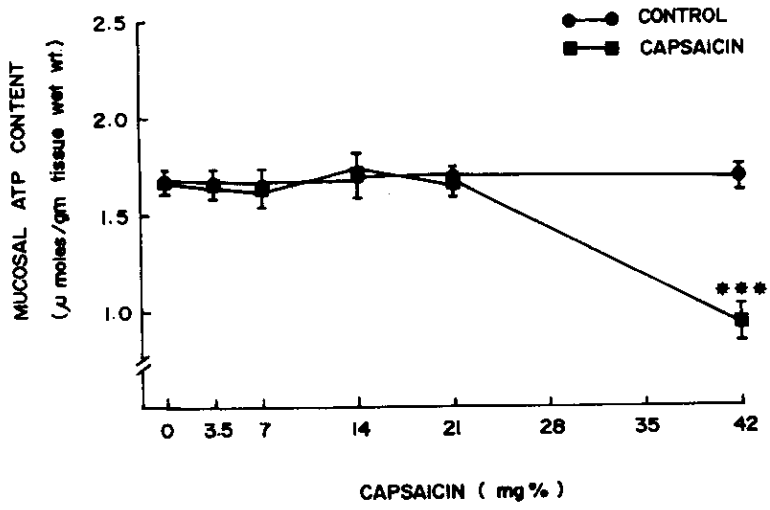

Fig. 2 Effect of various concentrations of capsaicin on mucosal ATP content off mouse jejunum *in situ*. Each value is mean ± S.E.M. of 6 mice. Significant value (Student's t-test) is indicated by asterisks : ***P < 0.001.

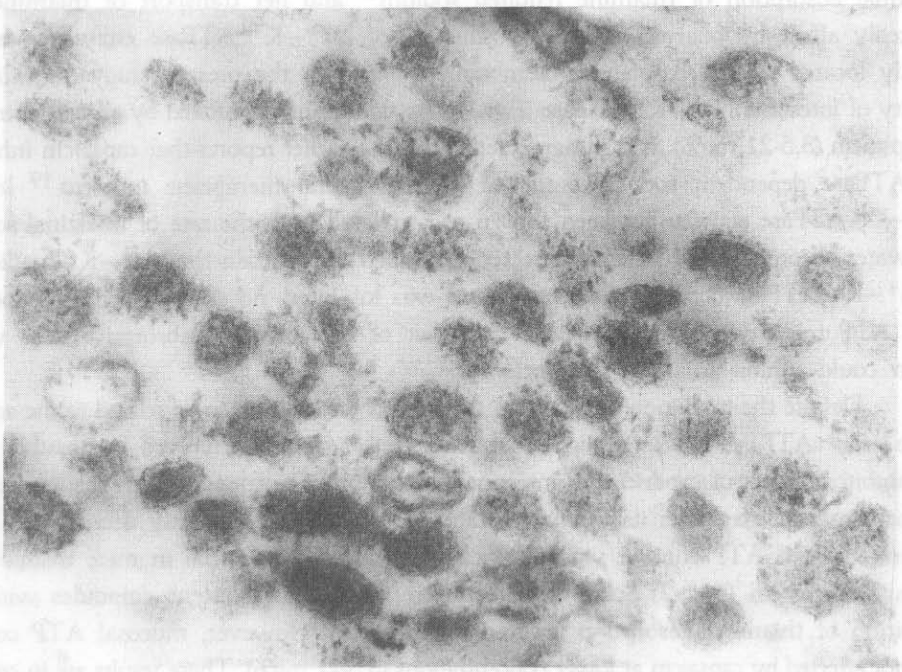

Fig. 3 Electron micrograph of brush-border membrane vesicles of mouse jejunum (85,000X)

Intestinal absorption of thiamine required sodium[17] and net transport of thiamine was markedly affect by ouabain, a known inhibitor of Na^+-K^+ ATPase enzyme which is mainly located on the basolateral membrane.[10, 17, 18] In the present study *(in situ)*, the activity of intestinal Na^+-K^+ ATPase (Fig. 1) was significantly inhibited by all concentrations of capsaicin (3.5-21 mg%). It is in agreement with the earlier reports that capsaicin inhibited the ATPase dependent sodium pump of the basolateral membrane *in vitro*.[19] Indeed, Na^+-K^+ ATPase activity has been shown to correlated with the rate of intestinal sodium and water absorption and potassium secretion needed to maintain the Na^+-K^+ gradient.[20] If Na^+-K^+ ATPase dependent sodium pump was inhibited, no energy would be liberated from ATP for active transport of Na^+ ion out of the intestinal absorptive cells which inturn could inhibit thiamine absorption.

Despite the inhibition of thiamine, absorption was shown to be related to the activity of Na^+-K^+ ATPase. It was thought that other factors may be involved. A possible factor for inhibitory effect of capsaicin on intestinal absorption of nutrients which required energy is that capsaicin may alter its cellular metabolism with a consequently decreased in ATP content.Mucosal ATP content was significantly reduced by 42.86% in mice treated with 42 mg% capsaicin (Fig. 2) Such[a] decrease in mucosal ATP content coindides with the inhibition of thiamine absorption induced by capsaicin. However, mucosal ATP content was not affected by capsaicin at lower concentrations (3.5-21 mg%). These results are in contrast to the previous report that mucosal ATP content was significantly decreased in rat and hamster after exposed to 14 mg% capsaicin for 60 min *in vitro*.[9] The time of exposure to capsaicin and the difference in techniques may have to be take, into an account for this event. The reduction of mucosal ATP content may be due to the decrease in the ATP synthesis because capsaicin was shown to inhibit ATP oxidative phosphorylation in isolated rat liver mitochondria.[21] It seemed that the reduction of Na^+-K^+ ATPase activity was a contributing factor for the inhibtion of intestinal thiamine uptake with low concentrations of capsaicin whereas the reduction of Na^+-K^+ ATPase activity and ATP content is responsible for the inhibition of thiamine absorption when exposed to high concentration of capsaicin.

In the morphological studies, it was found that the villi of small intestine became shortened and the lumens of glandular portions were enlarged after exposure to capsaicin (data not shown). The severity of morphological alteration increased with capsaicin concentration. These structural alterations could interfere with the intestinal thiamine absorption since the shortened villi would reduce the surface area for intestinal absorption of nutrients.

Other possible mechanism that would lead to the inhibition of thiamine absorptin is that capsaicin may alter the thiamine uptake across the brush-border membrane of the intestine. The results of this study found that capsaicin (0.1-2.0 mM) had no inhibitory effect on thiamine uptake across BBM vesicles (Table 4). It was slightly inhibited but not statistical significant at high concentration of capsaicin.

In conclusion, the reduction of intestinal mucosal Na^+-K^+ ATPase activity and ATP content and the structural alteration of intestinal absorptive cells should be responsible for the inhibition of thiamine absorption in isolated jejunal loop of mice after capsaicin administration.

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บทคัดย่อ

การศึกษาถึงผลของแคพไซซิน, สารเผ็ดในพริก, ต่อการดูดซึมไทอะมีนในลำไส้ของหนูถีบจักร์ โดยการฉีดสารละลาย ^{14}C-thiamine HCl ก็แคไซซินเข้าไปในโพรงลำไส้ *(in situ)* พบว่าการดูดซึมไทอะมีนชนิดที่ต้องใช้พลังงาน (1.5 μM) ลดลงหลังจากได้รับแคพไซซินที่ความเข้มข้นตั้งแต่ 7-42 mg% นาน 30 นาที การยับยั้งการดูดซึมของไทอะมีนจะมากขึ้นเมื่อความเข้มข้นของแคพไซซินในโพลงลำไส้เพิ่มขึ้นและผลการยับยั้งสูงสุด (45.82%) เกิดขึ้นที่ความเข้มข้น 42 mg% ในทางตรงกันข้ามแคพไซซินไม่มีผลต่อการดูดซึมไทอะมีนที่ความเข้มข้นสูง (20 μm) การทำงานของ Na^+-K^+ ATPase และ ปริมาณ ATP ในลำไส้ชั้นมิวโคซาลดลงเมื่อได้รับแคพไซซิน การทดลองนี้แสดงให้เป็นถึงความสัมพันธ์ระหว่างผลการยับยั้งการทำงานของ Na^+-K^+ ATPase และการลดปริมาณ ATP ในมิวโคซาของลำไส้และการเปลี่ยนแปลงโครงสร้างของเซลล์ดูดซึมบริเวณลำไส้เล็กกับการยับยั้งการดูดซึมไทอะมีนในลำไส้เล็กของหนูถีบจักร์