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The Effects of Carvacrol and Thymoquinone on Cadmium-Induced Damage in Gastrointestinal Contractility and Histological Changes in Rats

Efectos del Carvacrol y la Timoquinona en el Daño Inducido por Cadmio en la Contractilidad Gastrointestinal y los Cambios Histológicos en Ratas

Emine Koç[1]; Hüseyin Sah[2]; Gözde Ögütçü[3]; Ülfet Farisoglu[4]; Tugçe Akyüz[3]; Ali Özant[5] & Aysel Kükner[3]

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SUMMARY: Cadmium (Cd) is an environmental pollutant found naturally in the world and is toxic to humans. Thymoquinone (TQ) is an aromatic ketone found in many medicinal plants. TQ has been reported for its therapeutic potential in many medical conditions. Carvacrol (Car) is a natural essential oil used in medicine. This study aims to investigate the effects of TQ and carvacrol on cadmium-induced damage to gastrointestinal contractility and histological changes. Thirty-six rats were divided into six groups (n:6); Control group, Cd group; Cadmium chloride was given 3.5 mg/kg by subcutaneous(sc). In the third group; the Cd+ carvacrol group; carvacrol was given 50 mg/kg orally by gastric lavage and in the fourth group Cd+ TQ group; TQ was given 5mg/kg orally by gastric lavage. In the fifth group, only carvacrol and only TQ were given orally for 14 consecutive days in the sixth group. The duodenal and ileal segments were isolated and suspended in a tissue bath. Acetylcholine (Ach) induced contractions were recorded. For histological changes, the ileum segments were examined by light microscopy. Ach-induced contractions were lower in Cd groups than in Control in all segments. Ach-induced contractions were increased in Cd+Car and Cd+TQ. On the other hand, Cd caused significant morphological damage in the duodenum. The administration of both carvacrol and thymoquinone alleviated this damage. This study has demonstrated that Cd reduced intestinal contractility and caused severe damage in morphology. TQ and carvacrol can relatively remove Cd-induced damage in intestinal contractility and histology.

KEY WORDS: Cadmium; Thymoquinone; Carvacrol; Contractility; Intestine.

INTRODUCTION

Cadmium is found naturally in the world and affects many organs with its toxic effect on humans. Cigarette smoke, and ingestion of food contaminated with Cd such as fish, organ meat grain, potatoes, leafy vegetables, and root vegetables can contain high levels of Cd. Humans are generally exposed to Cd ingestion of food and drinking water contaminated with Cd by inhalation and ingestion (Zalups & Ahmad, 2003). The main target organ of Cd is the gastrointestinal system where it can exert toxic effects. The local effects of Cd intake were evaluated in the homogenates of the duodenum, the intestinal region most reactive to Cd (Zhao *et al*., 2006).

Carvacrol is a mono-terpenic phenol compound found in many aromatic plants. It has many biological properties, such as antibacterial, anticancer, antifungal, vasorelaxant, anti-inflammatory, antioxidant, and antiplatelet. Carvacrol is also used at low concentrations in human food as a flowering ingredient (Suntres *et al*., 2015). Previous studies have shown an inhibitory effect of carvacrol on muscarinic receptors. It has been suggested that carvacrol exhibits its anticholinergic effect via inhibition of central nervous cholinergic system (Boskabady *et al*., 2011).

TQ is found in many medicinal plants known for its traditional therapeutic value in the disorder of the gastrointestinal tract. It is regarded as an antioxidant (Sayed-Ahmed *et al*., 2010). TQ has effects on the gut spasmolytic, tracheal, and airway relaxants, vasodilator and relaxant activities on the cardiac muscles by blockage of Ca^{++} influx

[1] Near East University, Faculty of Medicine, Department of Physiology, Nicosia, Cyprus.
[2] Near East University, Faculty of Veterinary Medicine, Department of Histology and Embryology, Nicosia, Cyprus.
[3] Near East University, Faculty of Medicine, Department of Histology and Embryology, Nicosia, Cyprus.
[4] Cyprus Health and Social Sciences University, Faculty of Medicine, Department of Physiology, Nicosia, Cyprus.
[5] Near East University, Faculty of Medicine, Department of General Surgery, Nicosia, Cyprus.

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into the cells through voltage-operated Ca++ channels (Ghayur *et al*., 2012).

There is not enough research on the damage, caused by Cd, in the gastrointestinal contractility and histological changes and the effects of carvacrol and TQ. Therefore, this study aimed to examine the protective effects of carvacrol and TQ on Cd-induced damage on gastrointestinal contractility and histological structure.

MATERIAL AND METHOD

This study received approval from the local ethics committee of Near East University (Ethics No: 2023/157). Male Wistar rats were used in this study, and they were housed under standard conditions. They were maintained using a 12 hr light/dark cycle and provided with commercially available rat chow and tap water ad libitum. Rats were randomly divided into six experimental groups. The first group was the control group (C), and the second experimental group was the Cd group, which received CdCl2 3,5 mg/kg sc, the third experimental group was the Cd+carvacrol group, Which Cd was given 3.5 mg/kg sc and carvacrol (50 mg/kg) was given orally gastric lavage. In the fourth group; Cd+TQ group; in addition to Cd the animals received TQ (5mg/kg) orally by gastric lavage and in the fifth group, only carvacrol was given orally by gastric lavage and in the sixth group; only TQ was given orally. All experimental procedures were applied every day for 14 days. At the end of the 14 days, the animals were anesthetized with 90 mg/kg of ketamine and 10mg/kg of xylazine (i.p). The duodenal and ileal tissue segments (0.3-0.5 mm long pieces) were surgically removed and then placed in a petri dish containing Krebs solutions. Then the strips were suspended in tissue baths containing 20 ml of the Krebs-Henseleit solution (mM NaCl 118.9, KCl 4.6, $CaCl_2$ 2.5, KH_2PO_4 1.2, $NaHCO_3$ 25, $MgSO_4$ 1.2 and glucose 11), and a 95 % O_2, 5 % CO_2 mixture at 37 ºC, pH 7.4, was passed into the solutions. The segments were brought into equilibrium for 60 min under an optimal resting tension of 1g. After equilibration, the duodenal and ileal segments were contracted with Ach. The Ach ($3x10^{-7}$ M) doses were considered maximal doses, after the cumulative addition of Ach into the control group. The acetylcholine (Sigma A6625), and TQ were obtained from Near East University Pharmacology Laboratory, Cd (Sigma Aldrich, USA), and carvacrol (Sigma Aldrich, USA) were obtained from Sigma Chemical. Contraction responses were recorded in the organ bath (May-ITBS 08). The results were evaluated with the Graphpad Prism 8.3.1. One-way ANOVA was used to compare differences between groups, and Tukey's test was used for multiple comparisons. Evaluations $p \leq 0.05$ were considered significant.

Histological preparation. The ileal segment was first fixed in 10% formalin and was embedded in paraffin, then sectioned (5 μm), and stained with Hematoxylin and Eosin (H&E) for histological analysis under light microscopy (Leica LAS EZ Version 3.00). Villous lengths were evaluated for each group in the ileum. Villous lengths were recorded using the Fiji/ImageJ program by measuring 5 different areas in x10 objectives from each group and evaluated statistically.

RESULTS

Contraction results. As seen in Figures 1 and 2, the Ach-induced contractions in the Cd groups of both duodenum (p=0,0005) and ileum (p=0.003) segments were significantly decreased compared to the control groups.

Ach-induced contractions were significantly higher in the Cd+Carvacrol group when compared to the Cd group in the duodenum (p=0.02) and ileum (p=0.02) segments (Figs. 1A, B).

Ach-induced contractions were significantly higher in the Cd+TQ groups when compared to the Cd group in the duodenum (p=0.003) and ileum(p=0.03) segments (Figs. 2A, B).

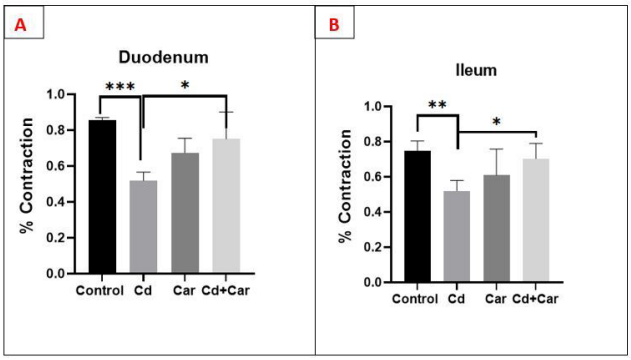

Fig. 1. The responses to the Ach-induced contractions in duodenal and ileal segments in Control, Cd, and Cd + Car groups. *** P<0.001, ** p<0.002, * p<0.05

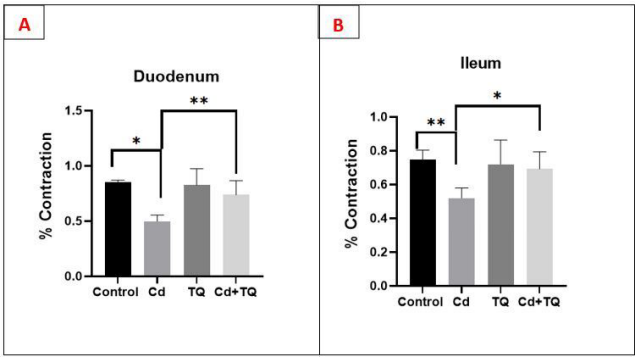

Fig. 2. The responses to the Ach-induced contractions in duodenal and ileal segments in Control, Cd, and Cd + TQ groups. *** P<0.001, ** p<0.002, * p<0.05

Histological results. There is no histological damage observed in the control groups (Fig. 3A). In the Cd group, in the ileum, intestinal tissue morphologies were severely damaged and the epithelial cells in the villus degenerated, congestion and inflammatory cell infiltration were observed (Fig. 3B). In the Cd + Carvacrol and Cd + TQ groups, it was observed that deterioration in villus structure, epithelial cell degeneration, congestion, and inflammatory cell infiltration was reduced in the treatment groups when compared to the Cd group (Figs. 3C, D). Villus length was significantly lower in the Cd group compared to the control group in the ileum ($p<0.05$). In the treatment groups, both Cd + Carvacrol ($p<0.05$) and Cd + TQ ($p<0.05$) groups, villous lengths were significantly higher when compared to the Cd group (Fig. 4).

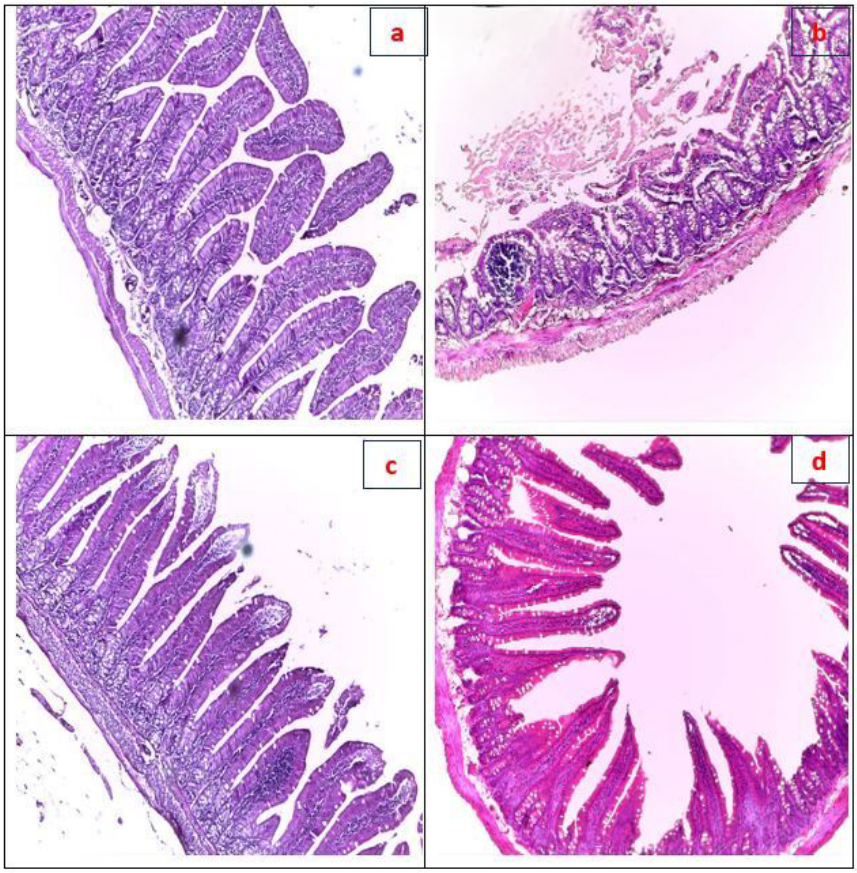

Fig. 3. Light microscopic images of ileum tissues from experimental groups. It is seen that villi structures were normal in the ileum of the control group, there was no degenerative change (a), villi structures were disrupted in the Cd group, epithelial cells were shed into the lumen, congestion,+ and inflammatory cell infiltration were observed (b), and villi degeneration was decreased in the Cd groups treated with Carvacrol (c) and thymoquinone (d).

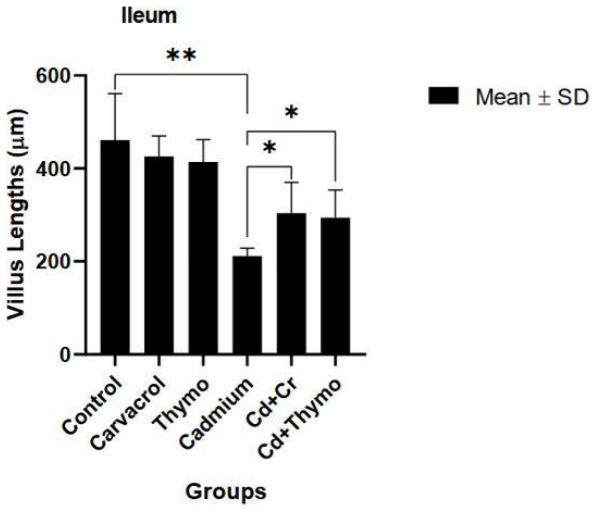

Fig. 4. Effect of carvacrol and thymoquinone on the villus length in the experimental groups of the ileum segment. *** $P<0.001$, ** $p<0.002$, * $p<0.05$

DISCUSSION

Cd is a naturally toxic element that is found as a mineral in combination with oxygen, chlorine, or sulfur (Cuypers *et al.*, 2010) and is highly soluble in water. This experiment gives new insight into the protective effects of carvacrol and TQ in Cd-induced damage about gastrointestinal contractility and morphological changes. An important finding of our study is the protective effect of carvacrol and thymoquinone on subchronic Cd-induced intestinal toxicity.

Our results showed that in Cd-ingested groups the Ach-induced contractions decreased intestinal motility as Liu *et al.* (2020) showed. They presented that Cd exposure decreased the contractile response of colonic muscle strips (Liu *et al.*, 2020). At the cellular level, Cd induces oxidative stress in many organisms (Thévenod, 2009), which results in physiological damage in different organs (Järup & Akesson, 2009). It was shown that the effects of Cd on oxidative capacity are dual. First, Cd induces oxidative stress via the inhibitions of antioxidants, and second, it also activates several antioxidative components because of disturbed redox balance and a consecutively induced signal transduction cascade (Cuypers *et al.*, 2010).

Our histological results showed that Cd-induced damage in intestinal tissue morphologies. Early investigations showed that orally administrated Cd degenerated the architecture of villi and caused intestinal cell necrosis (Andersen *et al*., 1988). Our histological results also showed that the villous epithelial cells were degenerating (Zhao *et al*., 2006), suggested that the intestinal inflammation response could be caused by chemotactic cytokine MIP-2 and neutrophil infiltration. However, Cd induces oxidative stress which results in physiological damage to many organs (Järup & Akesson, 2009). As we showed in our histological study, it was shown that Cd exposure causes significant damage to the gut barrier, including the toxicity of enterocytes, induction of inflammatory response, death of epithelial cells, and damage to the tight junctions in the intestine (Blais *et al*., 1999). There are also findings in the literature that are contrary to our findings. It was shown that when cadmium selenite was administrated for 28 days in rats, there were no histopathological findings related to cadmium selenite injury except for one case of focal hepatic inflammations in high dose (1000 mg/kg) (Kim *et al*., 2009).

Our results showed that in the Cd-treated group, the contractile responses induced with Ach were significantly decreased in duodenal and ileal segments. Since the absorption of Cd mainly occurs in the small intestine (Zhai *et al*., 2016), it is expected to be effective on duodenal and ileal contractility as in our findings. Anderson *et al*., 1994 showed that absorption of Cd occurs primarily in the duodenum and early jejunum. It was observed that Cd ions effectively inhibit the movements of Ca ions through certain types of Ca channels in excitable cells (Taylor, 1988). Even though they use the same channels Cd ions move through the channels much slower than calcium ions (Friedman & Gesek, 1994). It was also shown that intestinal uptake of Cd depends on its chemical form. The most effective form of Cd is CdCl (Groten *et al*.,1992), so we used CdCl in our experiments. It was shown that chronic administration by drinking water Cd reduces the Ach-induced contraction in duodenal segments (Koç *et al*., 2008). There are studies in the literature that support our findings that Cd exposure impairs neurogenic and myogenic contractile activity in rat detrusor muscle (Bayazıt *et al*., 2002). In the Cd group in the ileum segment; our histological results showed that there were degenerative changes in the villus. Results are like our findings which showed that ingested Cd resulted in inflammation and damage in the intestine (Ninkov *et al*., 2015).

Although many studies have shown that Cd also causes damage at the cell level, many factors affect cellular injury induced by Cd such as dose, route of exposure, and duration of exposure.

In our experiments to investigate the effects of carvacrol on Ach-induced contraction and histological effects in the ileum segment, we designed a group that was given Cd+carvacrol and only carvacrol. In these groups, we showed that in the Cd+Carvacrol group, the contraction amplitude was significantly higher as compared to Cd. Histological results showed that the administration of carvacrol to the Cd group, relatively improved the degenerative effect of Cd in intestinal tissue morphology.

Our results demonstrated that carvacrol decreased the Ach-contraction as compared to control groups. It was reported by some investigators that carvacrol extracts inhibited acetylcholine-induced contraction in the guinea pig ileum. They thought that carvacrol decreases calcium by blocking the release of intracellular bound calcium and extracellular calcium influx in the smooth muscle cell (Van der Broucke & Lemni, 1982). Peixoto-Neves *et al*. (2010) showed that carvacrol at low concentrations blocked the Calcium influx through the membrane in rat aortic segments. It has been shown that carvacrol modulates calcium homeostasis by transient receptor potential (TRP) channels in smooth muscle (Earley *et al*., 2010). In skeletal muscles, carvacrol inhibits sarcoplasmic reticulum calcium-ATPase and activates ryanodine receptors (Sárközi *et al*., 2007). The findings of Aydın & Seker (2005) are contrary to our findings, in that they showed that carvacrol did not inhibit ACh-induced contraction in isolated rat fundus.

Also in this study, we investigate the potential protective effects of TQ on the Cd-induced damage on gastrointestinal contractility and histological damage. We demonstrated that Ach-induced contraction was reduced insignificantly with the administration of TQ in the duodenum and ileum as compared to control groups. The findings of Ghayur *et al*. (2012) results support our findings, which showed that TQ was suppressed spontaneously contacting rabbit jejunum and guinea pig ileum with dose dependent. They suggested that the reduction in spontaneous contraction was due to the possible ability of the compound to block the entry of calcium via voltage-operated calcium channels with nonspecific mechanisms (Ghayur *et al*., 2012).

We showed that when Cd and TQ were used together there was a significant increase in contraction as compared to Cd groups. In our study, we indicated that there were repairing effects of TQ in gastrointestinal contractility and histological damage.

CONCLUSION

In conclusion, although it was known that the effects of Cd are dependent on many factors such as application

dose, route, and duration of exposure, our findings suggest that Cd plays an important role in intestinal contractility and histological damage and may have clinical implications for people who exposed to cadmium. Also, our study indicates that treatment with carvacrol and TQ can alleviate Cd-induced toxicity in gastrointestinal dysmotility and histological damage.

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KOÇ, E.; SAH, H.; ÖGÜTÇÜ, G.; FARISOGLU, Ü.; AKYÜZ, T.; ÖZANT, A. & KÜKNER, A. Efectos del carvacrol y la timoquinona en el daño inducido por cadmio en la contractilidad gastrointestinal y los cambios histológicos en ratas. *Int. J. Morphol., 43(4)*:1196-1201, 2025.

RESUMEN: El cadmio (Cd) es un contaminante ambiental presente de forma natural en el mundo y es tóxico para los humanos. La timoquinona (TQ) es una cetona aromática presente en muchas plantas medicinales. Se ha descrito el potencial terapéutico de la TQ en diversas afecciones médicas. El carvacrol (Car) es un aceite esencial natural utilizado en medicina. Este estudio tuvo como objetivo investigar los efectos de la TQ y el carvacrol en el daño inducido por cadmio en la contractilidad gastrointestinal y los cambios histológicos. Treinta y seis ratas se dividieron en seis grupos (n: 6); grupo de control, grupo Cd; cloruro de cadmio se administró 3,5 mg/kg por vía subcutánea (sc). En el tercer grupo; el grupo Cd + carvacrol; carvacrol se administró 50 mg/kg por vía oral por lavado gástrico y en el cuarto grupo Cd + grupo TQ; TQ se administró 5 mg/kg por vía oral por lavado gástrico. En el quinto grupo, solo carvacrol y solo TQ se administraron por vía oral durante 14 días consecutivos en el sexto grupo. Los segmentos duodenal e íleon se aislaron y suspendieron en un baño de tejido. Se registraron las contracciones inducidas por acetilcolina (Ach). Para los cambios histológicos, los segmentos del íleon se examinaron por microscopía óptica. Las contracciones inducidas por Ach fueron menores en los grupos Cd que en el control en todos los segmentos. Las contracciones inducidas por Ach aumentaron en Cd + Car y Cd + TQ. Por otro parte, el Cd causó un daño morfológico significativo en el duodeno. La administración de carvacrol y timoquinona alivió este daño. Este estudio ha demostrado que el Cd redujo la contractilidad intestinal y causó graves daños morfológicos. La TQ y el carvacrol pueden reducir relativamente el daño inducido por el Cd en la contractilidad e histología intestinal.

PALABRAS CLAVE: Cadmio; Timoquinona; Carvacrol; Contractilidad; Intestino.

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Corresponding Author
Gözde Ögütçü (M.Sc.)
Near East University
Faculty of Medicine
Department of Histology and Embryology
99138, Nicosia
CYPRUS

ORCID https://orcid.org/0009-0002-3381-5543

Email: gozde.ogutcu@neu.edu.tr