OPPOSITE EFFECTS OF ACUTE AND CHRONIC ADMINISTRATION OF ALCOHOL ON GASTRIC EMPTYING AND SMALL BOWEL TRANSIT IN RAT

FERENC IZBÉKI1*, TIBOR WITTMANN, SÁNDOR CSÁTI, ERZSÉBET JESZENSZKY2 and JÁNOS LONOVICS

University of Szeged, Faculty of Medicine, 1st Department of Internal Medicine, 2Institute of Forensic Medicine, Szeged, Korányi fasor 8 and
1Szent György Hospital of County Fejér, 1st Department of Internal Medicine, Székesfehérvár, Seregélyesi u. 3., Hungary

(Received 15 August 2000; in revised form 2 January 2001; accepted 29 January 2001)

Abstract — The effects of acute and chronic administration of a large dose of alcohol on gastric emptying and small bowel transit were studied in rats. The development of tolerance to the acute effect of alcohol on gastrointestinal motility during chronic alcohol administration was also investigated. Gastric emptying and small intestinal transit were assessed by the Phenol Red recovery method. Acutely, ethanol was given in a dose of 2.5 g/kg body wt by gavage 30 min before the test meal. Chronically, ethanol was administered by two different methods: (1) a dose of 2.5 g/kg body wt was administered by gavage daily for 10 days; (2) animals received 15% ethanol in their drinking water for 30 days. A single large dose of alcohol inhibited gastric emptying and small bowel transit. Treatment with a large dose of alcohol for 10 days did not change the gastric emptying significantly, but inhibited the small intestinal transit. Alcohol consumption in drinking water for 30 days accelerated gastric emptying and small bowel transit. Tolerance to the acute inhibitory effect of a single large dose of alcohol on gastrointestinal motility did not develop during chronic alcohol treatment.

INTRODUCTION

Conflicting reports are to be found in the literature as regards the effects of acute and chronic alcohol exposure on gastric emptying and small intestinal transit. Gastric emptying, both in humans and experimental animals, has been reported to be accelerated (Kaufman and Kaye, 1979), delayed (Barboriak and Meade, 1970; Jian et al., 1986; Scroggs et al., 1986; Wilson et al., 1990; Knight et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moor et al., 1992) or unchanged (Moo
were added to 0.5 ml of 20% aqueous trichloroacetic acid. After centrifugation at 10,000 g for 30 min, the supernatant was added to 4 ml of 0.5 N NaOH. The absorbance of the samples was read spectrophotometrically at 560 nm. The Phenol Red content of the samples was obtained from a calibration curve.

**Calculations and statistical analysis**

The gastric emptying and small bowel transit were characterized by the extent of Phenol Red recovery from the stomach and the intestinal segments. No dye was recovered from the most distal (fourth) intestinal segment; accordingly, only the data on three intestinal segments were evaluated. Data obtained from alcohol-treated rats were compared with those obtained from untreated animals or from control animals which received physiological saline.

Results are expressed as means ± SEM. The statistical significance of differences between groups was calculated by analysis of variance.

**Experiment I. Determination of normal rates of gastric emptying and small bowel transit in untreated animals**

Normal reference rates of gastric emptying and small bowel transit were determined in 20 rats, by the Phenol Red method described above.

**Experiment II. Examination of acute effects of alcohol on gastric emptying and small bowel transit in alcohol-naïve rats**

To test the acute effects of alcohol, a 2.5 g dose of ethanol per kg body wt (40% v/v ethanol solution) was administered to 10 rats by gavage 30 min before a test meal. Another 10 rats received an equal volume of physiological saline and served as controls. Gastric emptying and small bowel transit were determined as described above.

**Experiment III. Determination of effects of chronic alcohol treatment for different periods on gastric emptying and small bowel transit**

**Effects of 10-day alcohol treatment on gastric emptying and small bowel transit.** Ten rats received 2.5 g of ethanol per kg body wt (40% v/v ethanol solution) by gavage daily for 10 days. Seven control rats received an equal volume of physiological saline for 10 days. On day 11, the alcohol-treated group and the control group were tested for gastric emptying and small bowel transit as described above.

**Effects of 30-day alcohol treatment on gastric emptying and small bowel transit.** Ten rats weighing 200–220 g were kept on normal chow and received ad libitum 15% (v/v) aqueous ethanol solution for drinking for 30 days. The rats weighed 300–350 g at the end of the study. Animals were Starved for 48 h prior to experiments. Alcohol was taken away 2 h before the beginning of the experiments.

Fluid consumption was recorded daily. The average daily consumption was 33 ± 3 ml per rat, which corresponds to 4.4 g of ethanol per rat. Calculated relative to the average weight of the rats during the study period, this is equivalent to a daily dose of 15–17 g of ethanol per kg body wt. The gastric emptying and small bowel transit were studied by the Phenol Red content assay as described above.

**Experiment IV. Examination of development of tolerance to acute effects of alcohol during chronic alcohol treatment**

Examination of tolerance after a 10-day alcohol treatment. Twenty rats received 2.5 g of ethanol per kg body wt by gavage daily for 10 days. On day 11, 10 rats were intubated with 2.5 g of ethanol per kg body wt, followed by 2 ml of test material containing Phenol Red, and gastric emptying and small bowel transit were determined as described above. The remaining 10 rats, which served as controls, received the same volume of physiological saline per kg body wt instead of alcohol before testing on day 11.

Examination of tolerance after a 30-day alcohol treatment. Twenty rats weighing 200–220 g were kept on normal chow and received 15% aqueous ethanol solution ad libitum for drinking for 30 days. Daily alcohol consumption was recorded and calculated relative to body wt as described above. The rats weighed 300–350 g at the end of the study.

At the end of the 30-day period, 10 rats received the acute dose of 2.5 g of ethanol per kg body wt by gavage and were tested 30 min later for gastric emptying and small bowel transit as described above. The other 10 rats were given an equal volume of physiological saline by gavage instead of alcohol and served as controls.

**RESULTS**

**Acute effects of alcohol on gastric emptying and small bowel transit**

The distribution of Phenol Red recovered from the stomach and the three intestinal segments of untreated rats is detailed in Table 1. The amount of Phenol Red recovered from the stomach following acute alcohol loading was significantly greater than that observed in untreated rats or in control rats

<table>
<thead>
<tr>
<th>Groups of rats</th>
<th>Recovered Phenol Red (% ± SEM)</th>
<th>Intestinal segments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stomach</td>
<td>1st</td>
</tr>
<tr>
<td>Untreated controls (n = 20)</td>
<td>34.8 ± 4.1</td>
<td>8.5 ± 1.4</td>
</tr>
<tr>
<td>Physiological saline-treated</td>
<td>19 ± 0.9¹</td>
<td>12.2 ± 2</td>
</tr>
<tr>
<td>controls (n = 10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol-treated (n = 10)</td>
<td>56.4 ± 5.8bc</td>
<td>21 ± 4.2</td>
</tr>
</tbody>
</table>

Untreated control vs physiological saline-treated control: ¹P < 0.02; alcohol-treated vs physiological saline-treated control: ²P < 0.001, ³P < 0.05; alcohol-treated vs untreated control: ⁴P = 0.001.
receiving physiological saline (Table 1). This indicates that this dose of alcohol inhibits gastric emptying. Following an acute alcohol challenge, the percentage of recovered Phenol Red was significantly highest in the first intestinal segment and lowest in the third intestinal segment as compared to the levels in the controls that received physiological saline and in the untreated rats. This points to the inhibitory effect of alcohol on small bowel transit. Physiological saline alone, given 30 min prior to the test material, lowered the amount of Phenol Red recovered from the stomach and increased that found in the distal intestinal segment.

**Effects of chronic alcohol treatment for different periods on gastric emptying and small bowel transit**

Effects of 10- and 30-day chronic alcohol treatment on gastric emptying and small bowel transit. The amount of Phenol Red recovered from the stomach in the 10-day alcohol-treated group was somewhat, though not significantly, higher than that found in the untreated control group (Table 2). In the 10-day alcohol-treated group, the percentage of Phenol Red recovered from the proximal intestinal segment was significantly increased, whereas that from the distal intestinal segment was significantly decreased relative to the levels in the untreated group of rats. The distribution of recovered Phenol Red in the control group which received physiological saline for 10 days did not differ from that observed in the untreated control group (31.4 ± 2.7, 10.1 ± 2.6, 27.5 ± 2.5 and 30.7 ± 3.6% vs 34.8 ± 4.1, 8.5 ± 1.4, 30.1 ± 4.6 and 26.1 ± 3.8% respectively for the stomach and first, second and third intestinal segments).

The amount of Phenol Red recovered from the stomach of the 30-day alcohol-treated group was significantly lower, while the amount recovered from the distal intestinal segment was significantly higher than the corresponding values for the untreated group. This indicates an accelerated gastric emptying and an enhanced small bowel transit after 30 days of alcohol administration.

**Examination of tolerance after 10 and 30 days of alcohol treatment.** The distribution of recovered dye in the stomach and in the small intestinal segments following an acute alcohol challenge in the 10-day alcohol-treated group of rats was very similar to that found in the alcohol-naive group after the same acute alcohol load (Table 3). As compared to the levels found in the control group that received physiological saline, which acted as a control group for the 10-day alcohol-treated rats, a significantly higher amount of dye was observed in the stomach (26 ± 2.3 vs 59.4 ± 3.8%, P < 0.001) and a significantly decreased amount in the third intestinal segment (32.3 ± 3.8 vs 8.6 ± 2%, P < 0.001) following an acute alcohol load.

The amount of dye recovered from the stomach and the small intestinal segments in the 30-day alcohol-treated group of animals following an acute alcohol challenge was almost exactly the same as that found in the alcohol-naive rats after an acute alcohol load (Table 3). As compared to the control group of the 30-day alcohol-treated animals (the one that received physiological saline), the acute alcohol challenge resulted in a significantly higher recovery of Phenol Red from the stomach (57.9 ± 4.2 vs 12.7 ± 3.2%, P < 0.001) and led to a significantly lower recovery from the distal intestinal segment (8.4 ± 2.5 vs 42.5 ± 11.6%, P < 0.001).

**Blood-ethanol levels**

The levels of blood ethanol 1 h after the oral administration of a 2.5 g dose of ethanol per kg body wt were 1.1 ± 0.52 g/l in the alcohol-naive rats, 1.27 ± 0.68 g/l in the 30-day and 1.79 ± 0.78 g/l in the 10-day alcohol-treated animals. These data indicate a significantly higher blood alcohol level in the

---

### Table 2. Effects of chronic alcohol treatment on gastric emptying and small bowel transit in rats

<table>
<thead>
<tr>
<th>Groups of rats</th>
<th>Recovered Phenol Red (% ± SEM)</th>
<th>Intestinal segments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stomach</td>
<td>1st</td>
</tr>
<tr>
<td>Untreated controls (n = 20)</td>
<td>34.8 ± 4.1</td>
<td>8.5 ± 1.4</td>
</tr>
<tr>
<td>10-day alcohol treatment (n = 10)</td>
<td>42.9 ± 4.8</td>
<td>20.1 ± 2.5</td>
</tr>
<tr>
<td>30-day alcohol treatment (n = 10)</td>
<td>11.8 ± 1.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.4 ± 2.5</td>
</tr>
</tbody>
</table>

Alcohol-treated for 10 days vs untreated control: <sup>a</sup>P < 0.001; alcohol treated for 30 days vs untreated control: <sup>b</sup>P < 0.001.

---

### Table 3. Effects of an acute dose of alcohol on gastric emptying and small bowel transit in chronic alcohol-treated rats

<table>
<thead>
<tr>
<th>Groups of rats</th>
<th>Recovered Phenol Red (% ± SEM)</th>
<th>Intestinal segments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stomach</td>
<td>1st</td>
</tr>
<tr>
<td>Acute effects of alcohol in alcohol-naive rats (n = 10)</td>
<td>56.4 ± 5.8</td>
<td>21 ± 4.2</td>
</tr>
<tr>
<td>Acute effects of alcohol in 10-day alcohol-treated rats (n = 10)</td>
<td>59.4 ± 3.8</td>
<td>15.3 ± 2.8</td>
</tr>
<tr>
<td>Acute effects of alcohol in 30-day alcohol-treated rats (n = 11)</td>
<td>57.9 ± 4.2</td>
<td>13.8 ± 2.9</td>
</tr>
</tbody>
</table>
10-day alcohol-treated rats ($P < 0.02$). A significant amount of alcohol was not detected in animals treated for 10 or 30 days in the absence of an acute alcohol challenge.

**DISCUSSION**

Alcohol has been shown to produce a variety of macroscopic, light and electron microscopic structural alterations in the gut, depending on the dose and the duration of exposure (Dinoso *et al*., 1970, 1976; Gottfried *et al*., 1978; Fox *et al*., 1979; Parl *et al*., 1979; Ivey *et al*., 1980). Nausea, vomiting and diarrhoea, especially after episodes of heavy drinking, are commonly observed in connection with acute and chronic alcohol consumption. Though the morphological changes caused by alcohol may contribute to the disturbances in the gastric and small bowel motor activity, these symptoms of acute and chronic alcohol consumption can be manifestations of disturbed functions either of the central nervous system or of gastrointestinal motility. In rats, an inhibition of the synthesis of gastric and small bowel motor activity, these symptoms of alcohol consumption. Though the morphological changes caused by alcohol may contribute to the disturbances in the gastric and small bowel motor activity, these symptoms of acute and chronic alcohol consumption can be manifestations of disturbed functions either of the central nervous system or of gastrointestinal motility. In rats, an inhibition of the synthesis of the smooth muscle contractile proteins has been observed after acute and chronic alcohol administration (Preedy *et al*., 1988; Preedy and Peters, 1990), which may also alter the motor activity of the gut. In the present study, we investigated the effects of acute and chronic alcohol exposure on the motor function in the stomach and small bowel.

In the first set of experiments, we investigated the acute effects of a large dose of alcohol that has been shown to have an analgesic effect in rats (Fidecka *et al*., 1986; Malec *et al*., 1987; Bell *et al*., 1998; Gatch and Lal, 1999). Comparable doses of alcohol have been demonstrated to inhibit the gastric emptying and small bowel transit in mice (Scroggs *et al*., 1986) and lower doses inhibited gastric emptying of food in dogs (Knight *et al*., 1992) and humans (Jian *et al*., 1986). In agreement with these observations, our data revealed that a single dose of 2.5 g of ethanol per kg body wt, given intragastrically, inhibited gastric emptying and slowed the small bowel transit. In contrast, alcohol at much lower doses has been reported to accelerate gastric emptying in healthy volunteers (Kaufman and Kaye, 1979). In that study, alcohol was mixed into a test meal and postprandial gastric emptying was measured against a normal caloric loading. In our experiments, in order to evaluate the effect of alcohol on gastric emptying, a non-caloric test material was used to establish the normal rate of gastric emptying in untreated animals; another group of control rats received physiological saline instead of alcohol.

Chronic alcohol consumption has been found to delay gastric emptying in chronic alcoholic patients (Barboriak and Meade, 1970; Jian *et al*., 1986), whereas no gastric emptying abnormality was detected in chronic alcoholics after 3–10 days of abstinence (Keshavarzian *et al*., 1986). Only one study has assessed the effects of chronic alcohol administration on the gastric emptying in experimental settings; a delayed gastric emptying was found in cats after a month of alcohol treatment (Wilson *et al*., 1990).

In the present study, the chronic effects of administration of two different doses of alcohol for two different lengths of time were also investigated. In one experiment, the rats repeatedly received the acute dose of alcohol for 10 days. Gastric emptying was not significantly influenced by administration of 2.5 g of ethanol per kg body wt for 10 days, though a tendency toward an inhibited gastric emptying was observed. Our experiments in which a larger amount of alcohol was administered over a longer period of time demonstrated an accelerated gastric emptying. The difference between the effects of alcohol on gastric emptying in the 10-day and the 30-day alcohol-treated rats could not be a consequence of a difference in blood-ethanol levels, because significant amounts of ethanol were not detected in these groups of rats before acute alcohol loading. Both acute and chronic administration of alcohol affect gastric emptying. Accelerated gastric emptying may reflect a disturbed regulation. A high daily alcohol intake for a long period of time seems to be the more damaging to the neuro-hormonal regulation of gastric emptying.

The effects of alcohol on small bowel motility have been poorly investigated. In clinical settings, the oro-coecal time has been studied in chronic alcoholic patients (Addolorato *et al*., 1997; Papa *et al*., 1998); a delay was found. In experimental animals, acute administration of alcohol has been found to inhibit the small bowel transit in mice (Scroggs *et al*., 1986), whereas an acceleration was observed in rats (Krishnamra and Limlomwongse, 1987). In the latter study, alcohol was intubated directly into the duodenum, which might have influenced motility. Prolonged consumption of moderate doses of alcohol in rats impaired the spontaneous and tonic contractility of duodenal muscle contraction in vitro, whereas, in the ileal intestinal muscle, it was left unaffected (Palasciano *et al*., 1995).

The chronic effect of alcohol on small bowel transit has not been investigated previously. In our experiments, we assessed the effects of alcohol on small bowel motility via the distribution of Phenol Red recovered from the small intestinal segments. The distribution of the recovery was shifted to the left in the small bowel segments following a single large dose of alcohol, which points to the inhibitory effect of acute alcohol administration on small bowel transit. In contrast, a shift in the recovery to the right occurred in the 30-day alcohol-treated animals, which may reflect an accelerated small bowel transit in these rats. After 10 days of alcohol treatment, the small bowel displayed a relatively inhibited state of intestinal transit.

Tolerance to the various actions of alcohol has been reported to develop (Ivey *et al*., 1980; Bennet *et al*., 1993; Holloway *et al*., 1993; Gatch and Lal, 1999). It has also been reported that tolerance develops to the analgesic effect of 2.5 g of ethanol per kg body wt by day 10 (Gatch and Lal, 1999). The development of tolerance to the acute effects of alcohol on gastrointestinal motility has not been described. We therefore investigated this question in experiments involving chronic alcohol exposure for 10 or 30 days. Acute administration of 2.5 g of ethanol per kg body wt caused an additional inhibition of the gastric emptying and the small bowel transit in rats treated with the same amount of alcohol for 10 days. We found that the inhibitory effect of an acute alcohol challenge on gastric emptying in the 30-day alcohol-treated rats was equal to the action of the same dose of alcohol in the alcohol-naive rats. An inhibitory effect of acute alcohol administration on small bowel transit in the 30-day alcohol-treated rats was likewise observed, though the effect was not as pronounced as in the alcohol-naive group. These data furnish evidence that tolerance to the acute effects of alcohol on gastrointestinal motility does not develop even after long-term treatment with a high dose of alcohol. Our finding regarding the persistence
of the inhibitory effect of acute alcohol administration on gastrointestinal motility without the development of tolerance during chronic alcohol exposure is in accord with observations of the inhibitory effects of opiates on gastrointestinal motility (Bell et al., 1998; Yuan et al., 1998a,b).

In conclusion, a single large dose of alcohol inhibits gastric emptying and small bowel transit. Chronic administration of a large dose of alcohol accelerates gastric emptying and small bowel transit in rats. Tolerance to the inhibitory effect of an acute alcohol challenge on gastrointestinal motility does not develop during chronic alcohol treatment.

Acknowledgements — The authors thank Zoltán Kocsisptér for excellent technical assistance. This work was supported by Hungarian Ministry of Health research grants 140-02/2000/ETT and 543-02/2000/ETT.

REFERENCES


