

Effect of Distal Small Bowel Resection on Bile Salt Absorption in Caecum

It is well known that surgical resection of the small bowel results in adaptive changes in the residual intestine (7, 13, 14). The entero-hepatic circulation of bile salts, which is responsible for maintaining constant bile salt pool, is also impaired by intestinal resection. Bile salts are largely reabsorbed by an active transport mechanism in the ileum (3, 8), and to a lesser extent by passive diffusion in the jejunum, caecum and colon (2).

Although the effects of intestinal resection on bile salt absorption in jejunum (10, 12) and colon (5, 10) have been widely studied, little attention has been paid to bile salt absorption in the caecum, so that we have investigated the role of the caecum in cholic acid absorption.

In the current work, two groups of rats have been employed: 1) Sham operated, and 2) Rats with 80 % distal small bowel resection. Operative details have been previously described (9).

One month after the surgical operation, animals were starved overnight (with access to water only), anaesthetized with subcutaneous injections of sodium pentobarbital (4.5 mg/100 g b. w.), and placed on a heated operating table to maintain rectal temperature at 38°C. A conventional intestinal loop technique described by SOLS and PONZ (11) was employed to study cholic acid absorption in the caecum. Briefly, laparotomy was performed and the caecum was isolated between

two glass cannulae. The caecum was carefully squeezed, its contents weighed and the caecum returned to the peritoneal cavity. The entry glass cannula was connected to a reservoir (reservoir A). The caecum was washed by filling the reservoir A with physiological saline solution (0.9 % NaCl) at 38°C. When the effluent fluid was translucent, the liquid in the reservoir A was allowed to descend until a fixed level was reached. At this point the exit cannula was clamped, introduced into a 50 ml volumetric flask (reservoir B) and 10 ml of Ringer's solution at 38°C, containing 4 mM cholic acid together with tracers of (¹⁴C) Cholic acid, was placed in the reservoir A. The exit cannula was then opened and the liquid in the reservoir A was allowed to descend to the fixed level. The exit cannula was then clamped and the time was noted. After 1 h absorptive period, the caecum was drained by adding 30 ml of physiological solution to the reservoir A and the effluent was collected in reservoir B. Cholic acid absorption was estimated as luminal loss, and was determined by liquid scintillation counting.

At the end of the experiment the caecum was removed, blotted carefully on both sides to remove excess of moisture, weighed wet, dried for 24 h at 80°C and the (wet-dry weights)/wet weight ratio determined.

Table I. Effect of intestinal resection on caecal tissue, wet and dry weights and bile salts in the caecal content.

Analysis were made 1 month after 80 % intestinal resection. Data are given as means \pm S.E. Number of animals per data 10.

Animal	Caecal tissue		Caecal bile salt	
	Wet weight (g)	Dry weight (g)	Total content (μ moles)	Concentration (mM)
Sham	1.44 \pm 0.11	0.19 \pm 0.02	17.19 \pm 2.67	12.45 \pm 1.25
Resected	3.12 \pm 0.27*	0.34 \pm 0.03*	66.53 \pm 8.66*	6.94 \pm 0.70*

Comparisons between sham and resected animals (t-Student Test), * $p < 0.001$.

Bile salts in the caecal content were extracted according to the method described by DE WAEL *et al.* (1), and determined by the Fausa and Skalhogg enzymatic method (4).

Caecal tissue weights (Table I) were significantly increased in resected animals. However, tissue water content was not significantly modified by intestinal resection (85.76 \pm 1.66 % in sham animal v.s. 88.50 \pm 0.8 % in resected rats), indicating a caecal enlargement.

Since 80 % distal small bowel resection deprives the animal of those parts of the small intestine (ileum and jejunum) where bile salts are preferentially absorbed (3, 8), an increase in caecal bile salts content could be expected. Total bile salts content in the caecum was significantly increased by 80 % intestinal resection (Table I). However the caecal bile salt concentration was less in resected animals due to a higher caecal content. The observed increase in caecal content hydration, expressed as a percentage of wet weight (79.53 \pm 3.1 % in sham animal v.s. 88.64 \pm 1.06 % in resected rats, $p < 0.025$) could be due to increased bile salts input into the caecum (6).

Total cholic acid absorption in the caecum (μ mol/h) and caecal cholic acid absorption per gram of wet tissue were also significantly enhanced after 80 % bowel resection (Table II). PERRY (10),

by measuring isotope excretion in fistule bile, also found an increase in caecal chololate absorption after distal small bowel resection.

That the increase in cholic acid absorption per hour (3-fold increase) was higher than that per gram wet tissue (2-fold increase) might be due to the caecal enlargement. On the other hand, the increased cholic acid absorption per gram of wet tissue could be explained by either an increase in epithelial permeability or/and an increase in the bile salts subepithelial clearance.

In conclusion, the present study shows that rat-caecum undergoes morphological and functional adaptations following major small bowel resection.

Table II. Effect of intestinal resection on cholic acid absorption in the caecum in vivo.

Analysis were made 1 month after 80 % intestinal resection. Data are given as means \pm S.E.

Number of animals per data 10.

$p =$ Comparisons between sham and resected animals (t-Student Test).

Animal	Caecal cholic acid absorption	
	μ moles/h	μ moles/g w.w./h
Sham	4.17 \pm 0.35	2.09 \pm 0.39
Resected	13.14 \pm 1.60	4.11 \pm 0.59
p	< 0.001	< 0.025

Key words: Bile salt absorption.

References

1. DE WAEL, J., RAAYMAKERS, C. E. and ENDEMAN, H. J.: *Clin. Chim. Acta*, **79**, 465-470, 1977.
2. DIETSCHY, J. M.: *J. Lipid. Res.*, **17**, 572-576, 1976.
3. DIETSCHY, J. M., SALOMON, H. S. and SIPERSTEIN, M. D.: *J. Clin. Invest.*, **45**, 832-846, 1966.
4. FAUSA, O. and SKALHEGG, B. A.: *Scand. J. Gastroent.*, **9**, 249-254, 1974.
5. GERSON, C. D., COHEN, T. and JANOWITZ, H. D.: *Gastroenterology*, **64**, 907-912, 1973.
6. GORDON, S. J., KINSEY, M. D., MAGEN, J. S., JOSEPH, R. E. and KOWLESSAR, O. D.: *Gastroenterology*, **77**, 38-44, 1979.
7. HANSEN, W. R., OSBORNE, J. W. and SHARP, J. G.: *Gastroenterology*, **72**, 701-705, 1977.
8. LACK, L. and WEINER, I. M.: *Biochim. Biophys. Acta*, **135**, 1065-1068, 1967.
9. MURILLO, M. L., CAMPOS, M. S., MURILLO, A. and VARELA, G.: *Rev. esp. Fisiol.*, **34**, 365-370, 1978.
10. PERRY, M.: *Ann. Roy. Coll. Surg. Engl.*, **57**, 139-147, 1975.
11. SOLS, A. and PONZ, F. A.: *Rev. esp. Fisiol.*, **3**, 207-211, 1947.
12. TILSON, M. D., BOYER, J. L. and WRIGHT, H. K.: *Surgery*, **77**, 231-234, 1975.
13. URBAN, E., STARR, P. E. and MICHEL, A. M.: *Dig. Dis. Sc.*, **28**, 265-272, 1983.
14. URBAN, E. and WESER, E.: *Adv. Intern. Med.*, **26**, 265-291, 1980.

C. M. VÁZQUEZ *, A. ILUNDAIN, M. L. MURILLO and J. BOLUFER

Departamento de Fisiología Animal
Facultad de Farmacia
41012 Sevilla (Spain)

(Received on April 22, 1985)

* To whom all correspondence should be addressed.

