

(ODB) has been hypothesized to play a role in this process. However, there is a general lack of research on the long-term effects of acute kidney injury (AKI) on ODB and their total oxalate-degrading activity (ODA) in fecal microbiota. In this study, we evaluated whether renal dysfunction could affect intestinal ODB and their total ODA in a rat model of glycerol-induced AKI.

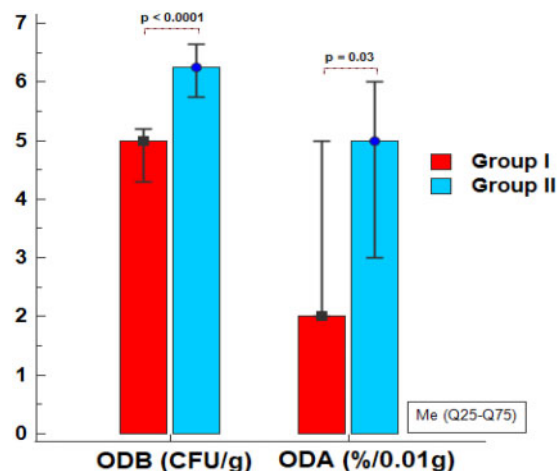
**METHOD:** The Male Wistar rats (200-300 g, n=20) on oxalate-free diet were randomly divided into 2 groups. After 24-h of water deprivation, Group 1 (n=10) received an intramuscular injection of 50% glycerol (10 ml/kg of body weight), and Group 2 (n=10) served as control. The numbers of ODB (incubated in a highly selective Oxalate Medium and determined using culture method) and total fecal ODA were measured after injection on days 7 and 70. The method of redoximetric titration with a KMnO4 solution was adopted to evaluate total ODA in fecal microbiota; the results were expressed as % of oxalate degradation per 0.01 g of feces. Renal injury was assessed by histopathological examination, serum creatinine and daily proteinuria levels after removing the animals from the experiment on day 70. Cortical interstitial fibrosis was measured by computerized image analysis on sections stained with picrosirius red. The median (Me) and the interquartile ranges (Q25; Q75) were calculated and compared using the nonparametric Mann-Whitney test. The Spearman correlation coefficient was used to evaluate association between the examined parameters.

**RESULTS:** The obtained results demonstrated: 1) after glycerol injection on day 7, no differences were found in the numbers of ODB and total fecal ODA between the experimental and control groups: 5.9 (5.4-6.0) vs 6.0 (5.4-6.4) CFU/g, p=0.65 and 2.0 (0.1-5.0) vs 2.5 (2.0-9.0) %/0.01g, p=0.24, respectively; 2) after AKI initiation on day 70, the numbers of ODB and total fecal ODA were significantly lower in Group I compared with control Group II (Fig. 1); 3) the higher percentage of renal interstitial fibrosis was, the higher total fecal ODA occurred in the experimental rats (Fig. 2). In addition, the number of ODB in feces in Group 1 had an inverse association with serum creatinine (r=-0.52, p=0.006) and 24-h proteinuria levels (r=-0.86, p<0.0001).

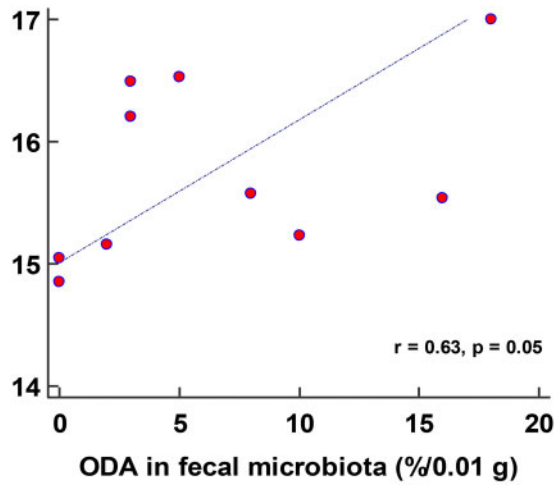
FC044 **THE LONG-TERM EFFECTS OF ACUTE KIDNEY INJURY ON INTESTINAL OXALATE-DEGRADING BACTERIA IN RATS**

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**BACKGROUND AND AIMS:** Gut microbiota is considered an important factor affecting oxalate handling in the intestine. It has been demonstrated that intestinal oxalate secretion provides a complementary route of excretion, and it becomes more evident when kidney function declines. A diversity of gut oxalate-degrading bacteria



FC044 Figure 1: The numbers of ODB and total fecal ODA in the rats of the experimental (Group I) and the control groups (Group II).



FC044 Figure 2: Association between renal interstitial fibrosis and total fecal ODA in the rats with glycerol-induced AKI.

**CONCLUSION:** AKI had the long-term negative effects on the quantitative and qualitative characteristics of ODB in fecal microbiota in rats. Moreover, the results of our study confirmed an increasing trend in total fecal ODA according to the aggravation of renal interstitial fibrosis in rats.