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Kidney TNF- α (unit/mL)

BACKGROUND & AIMS

Emerging evidence showed the important role of kidney-gut crosstalk in diverse pathological processes and alterations in intestinal barrier or dysbiotic gut microbiota have been demonstrated in chronic kidney disease (CKD).

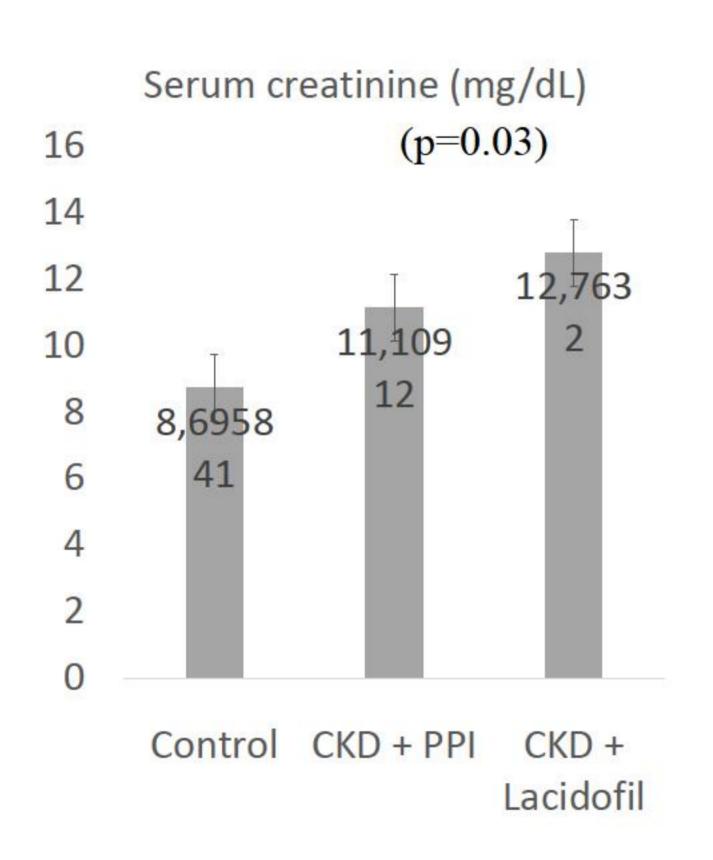
SUBJECTS & METHODS

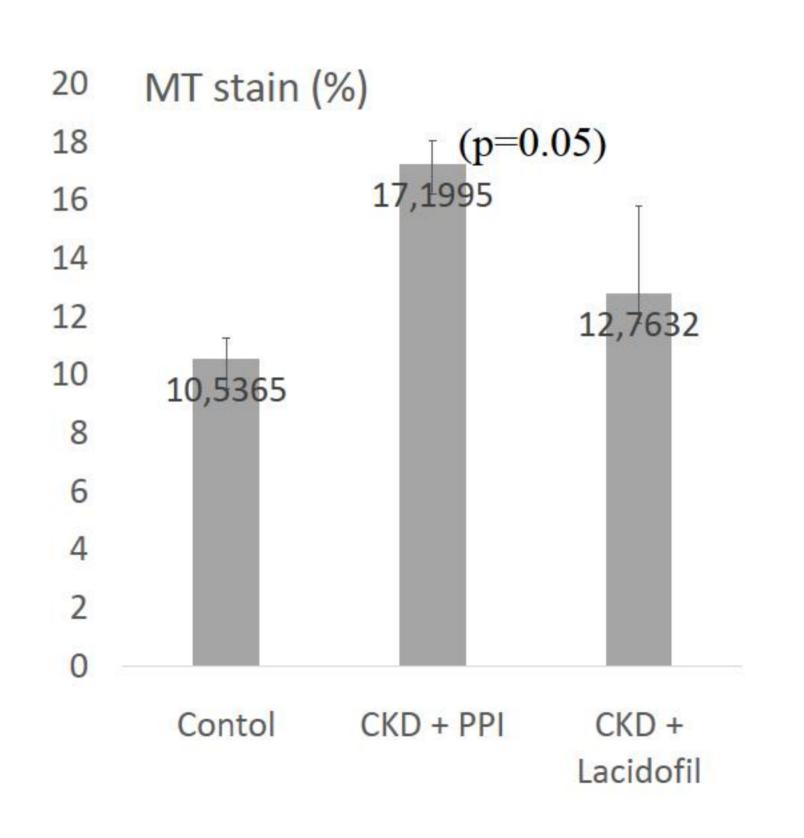
CKD was induced in 6 week old C57BL/6 mouse by 5/6 nephrectomy. Lactobacillus Rhamnosus R0011 and Lactobacillus Acidophilus R0052 and proton pump inhibitor (PPI) mixture were administered via oral gavage starting 1 day after the operation and continued for 8 weeks following intestinal decontamination. PPI only without probiotic served as control. Biochemical, histological analyses as well as immunological analyses were performed at 4 weeks after 5/6 nephrectomy.

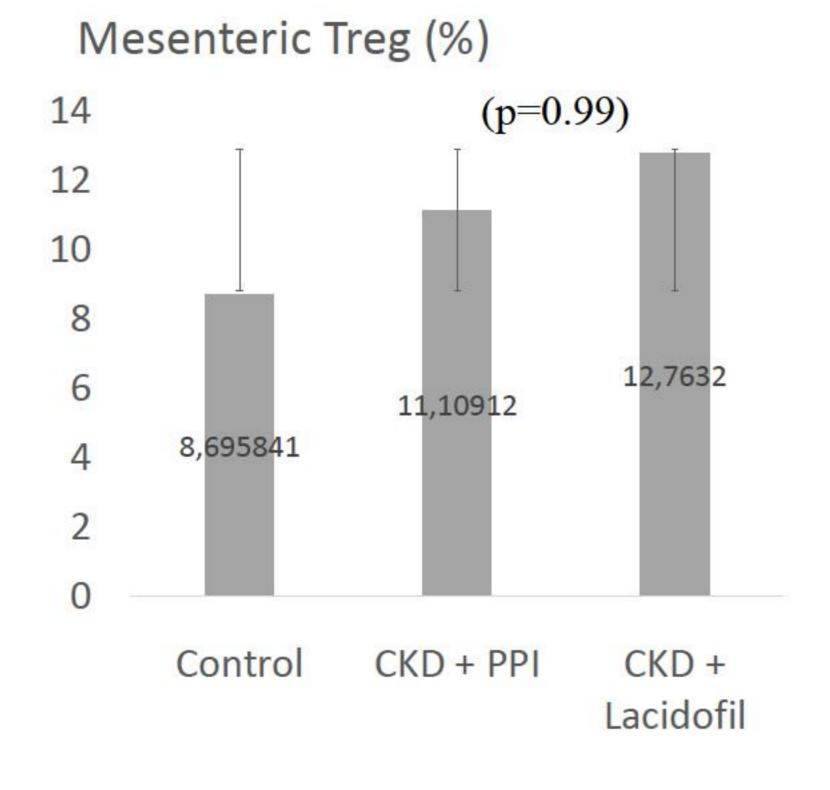
RESULTS

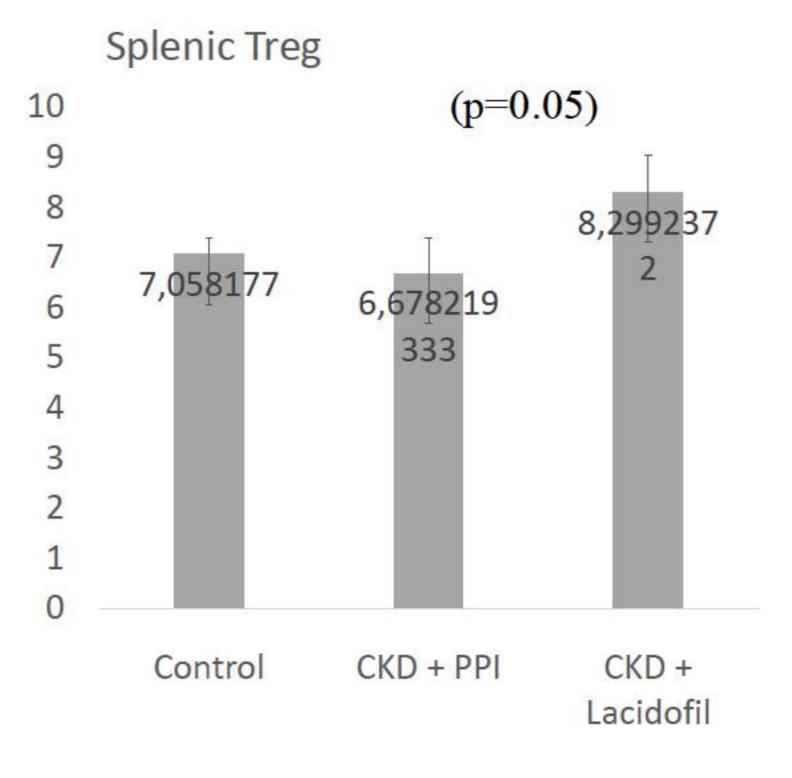
At 8 weeks, serum creatinine decreased in probiotics treated group compared to CKD or sham group (p=0.03). Probiotics group shows decreased fibrosis (Masson's trichrome staining, semiquantitative fibrosis score 17.19 Vs 12.76, p=0.05). Urine protein creatinine ratio showed no statistical significance but we confirmed the declining trend in probiotics group (p=0.13).

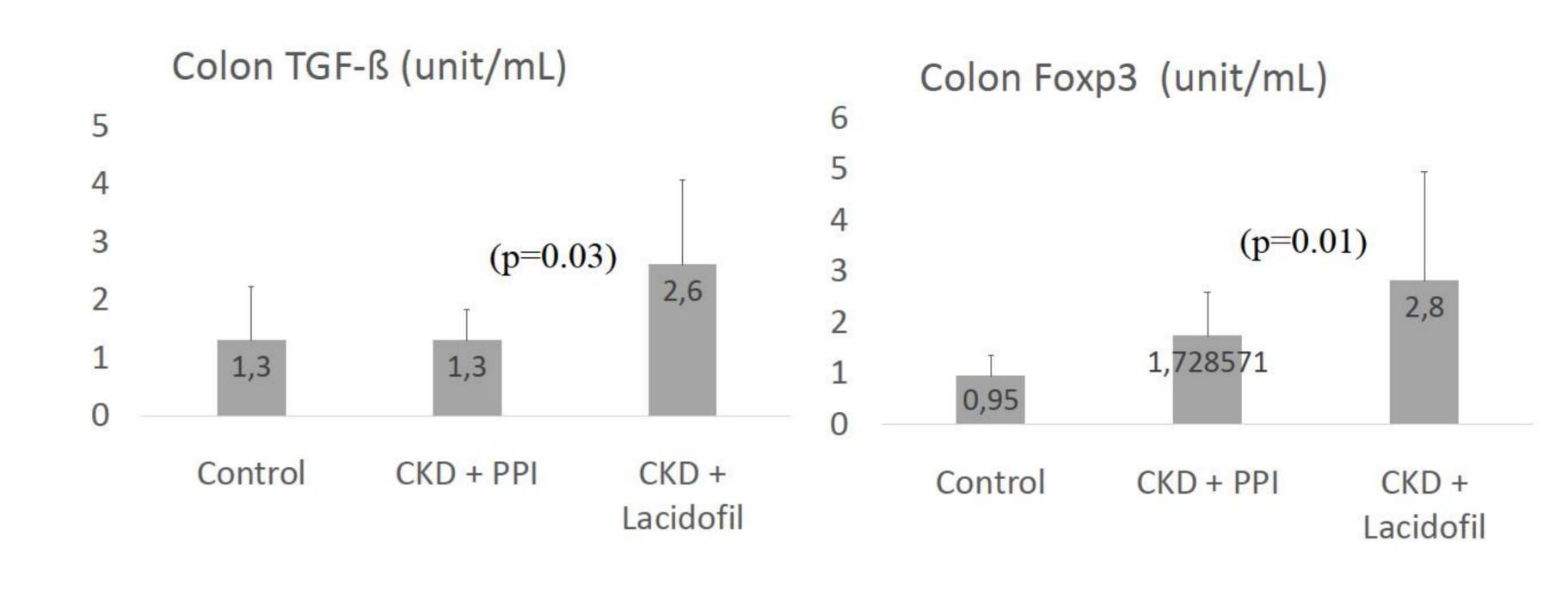
Serum IL-6 concentration (p=0.02), as well as kidney TNF- α , MCP-1 (p<0.01) concentration significantly decreased in CKD+Lacidofil group compared to CKD+PPI group. In addition, percentage of splenic Tregs and colonic Foxp3 mRNA expression level showed a significant increase in CKD+Lacidofil group (p<0.05, p=0.03).





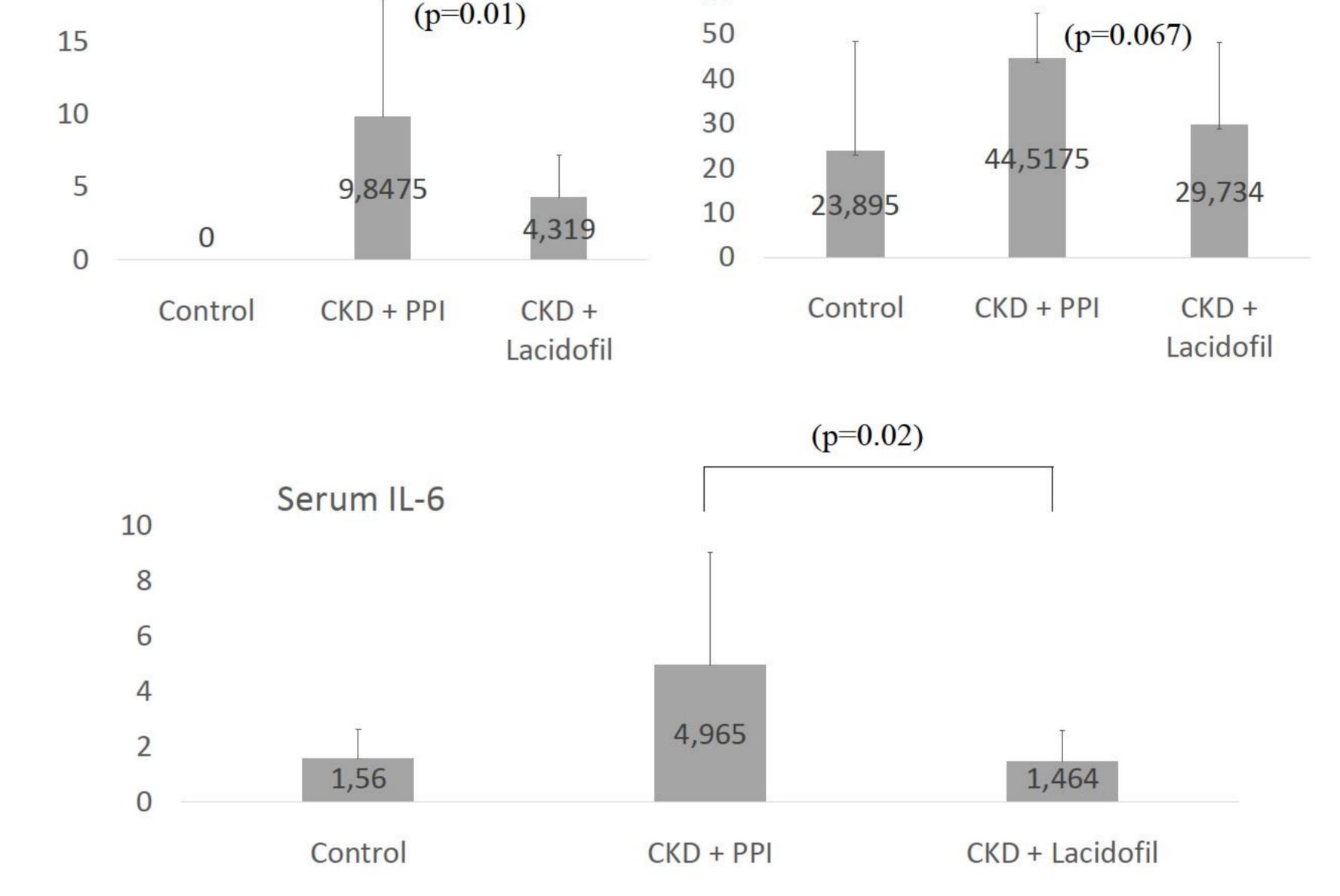






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Kidney MCP-1 (unit/mL)



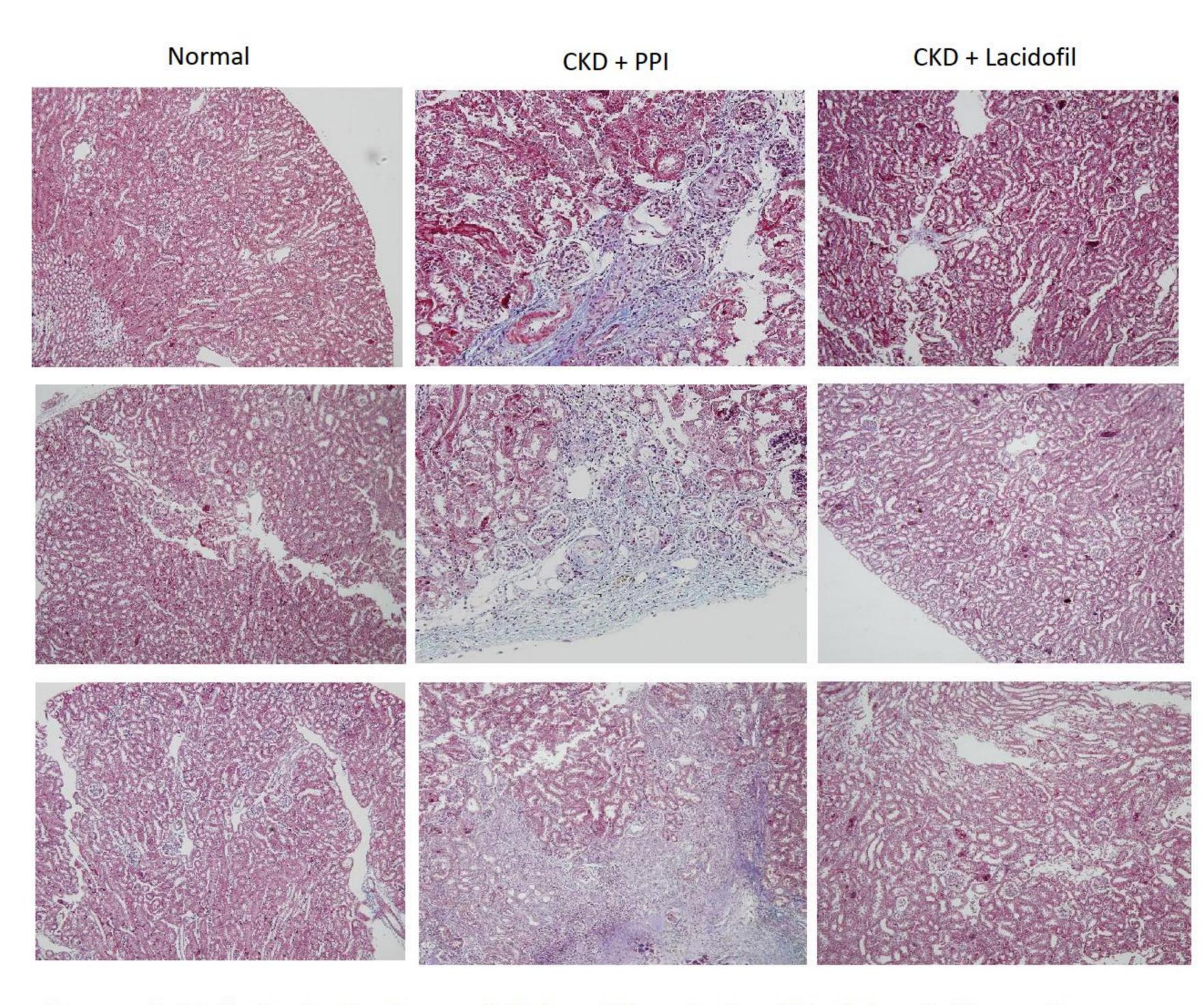


Figure 1. Pathologic findings of kidney fibrosis by MT stain at 14 weeks old 5/6 nephrectomy mice (x100)

CONCLUSION

These data demonstrated the possibility that altered microbiota in CKD might be partially responsible for CKD associated inflammation, CKD progression and suggest that probiotic treatment might have therapeutic potential.

