GI inflammation Increases Sodium-Glucose Cotransporter Sglt1

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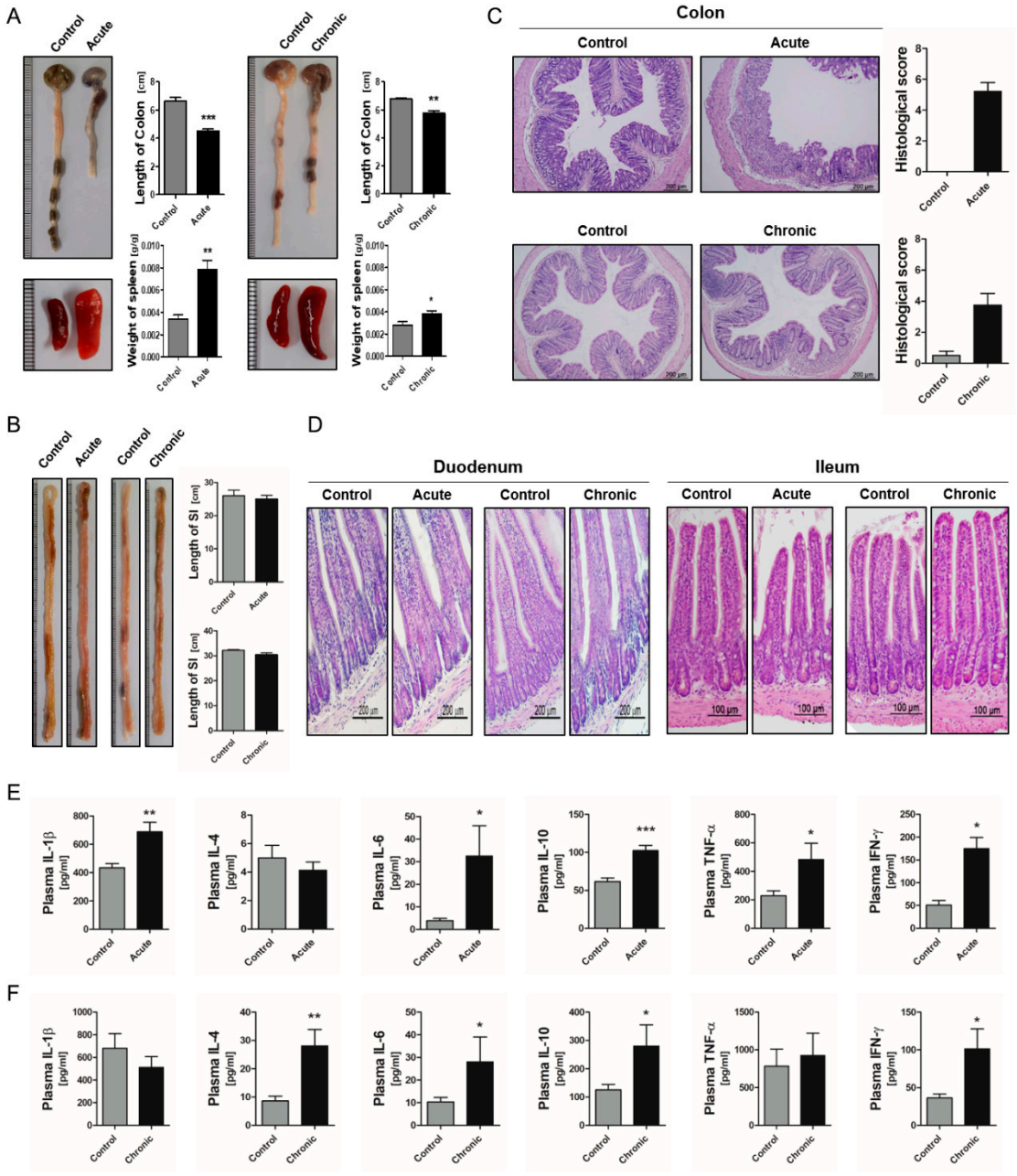
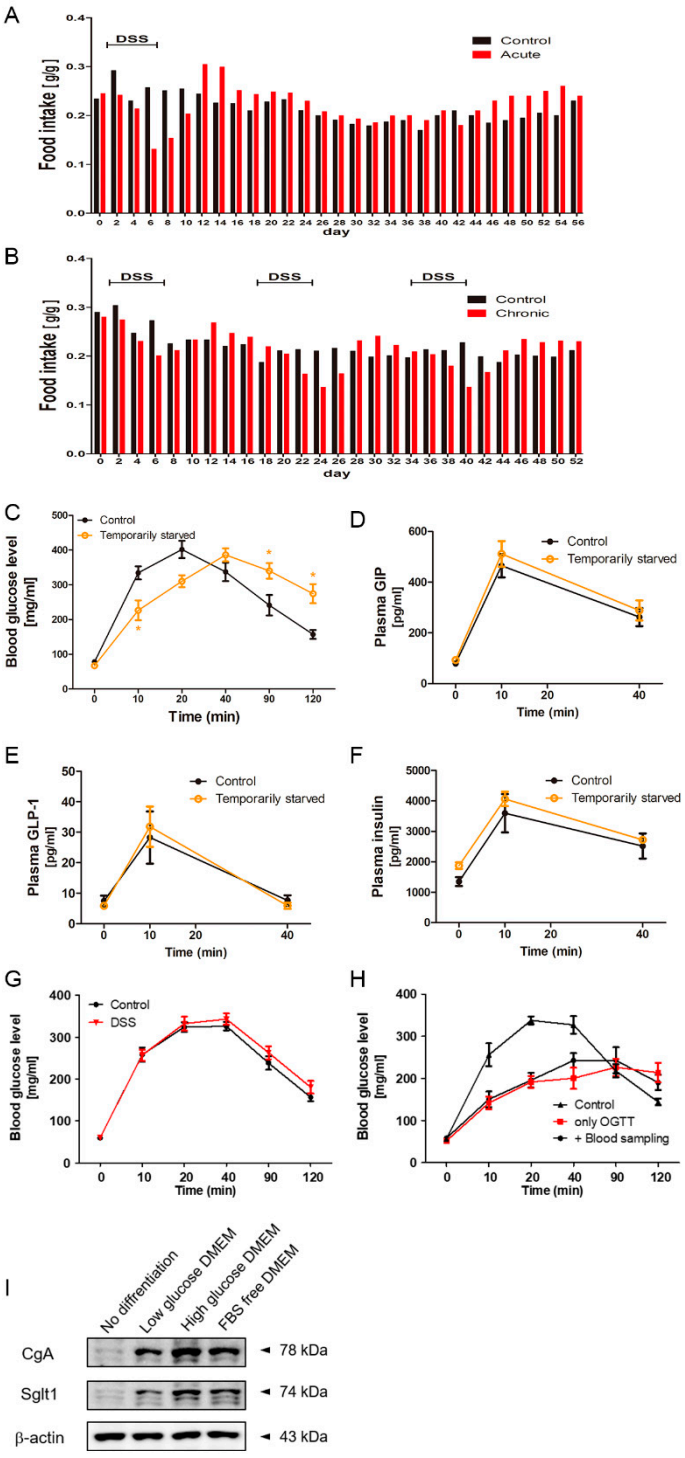
Supplementary Materials



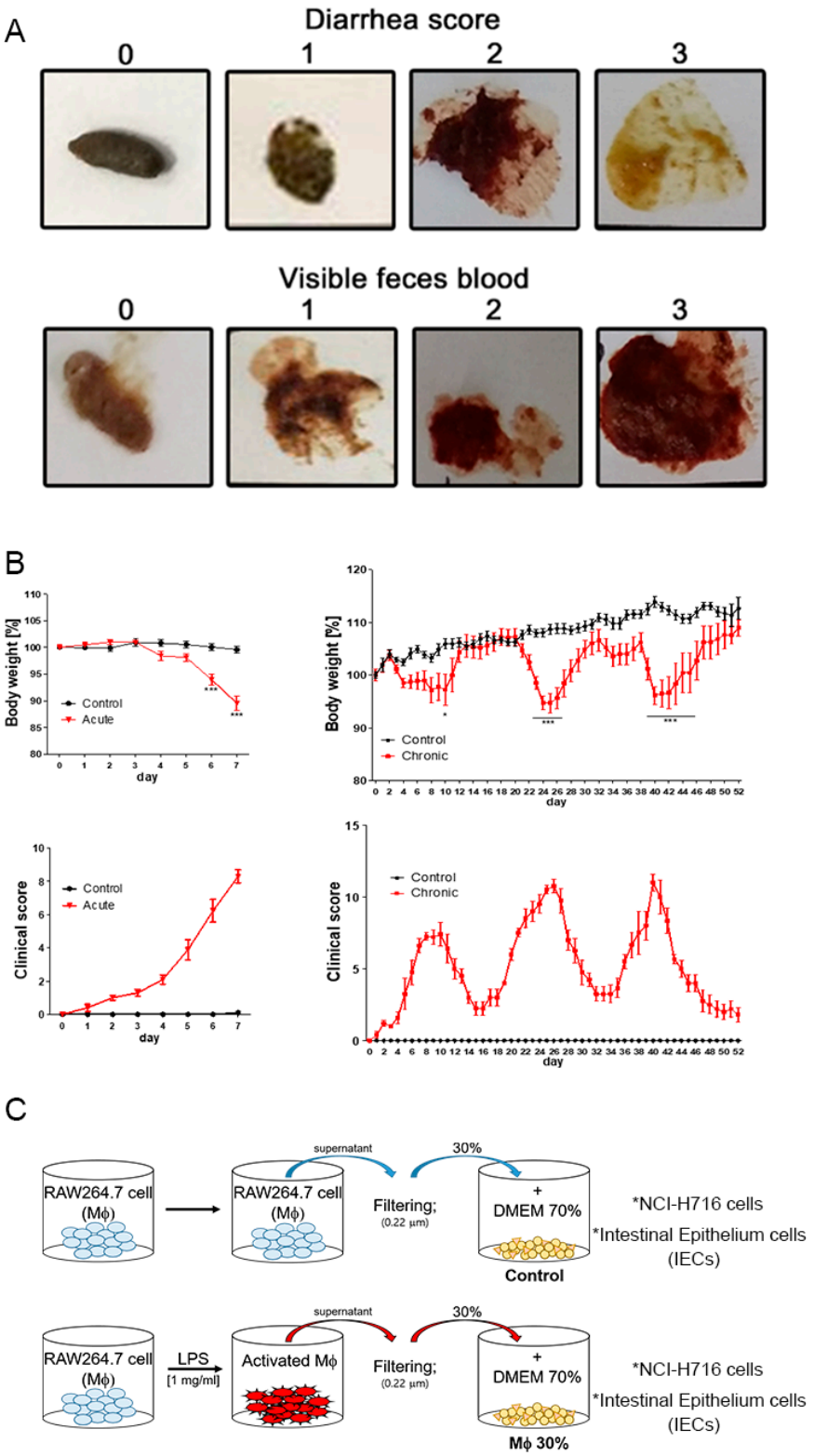
Supplementary Figure 1. Gastrointestinal tract (GI) inflammatory condition induced by dextran sodium sulfate (DSS) administration. The index of the GI-inflamed mice. The shortened length of the colon in the acute and chronic groups and the overgrown spleen (right) compared to the control group (left) (A). The small intestine did not show significant changes in its outward appearance (B). In the colon, one-third of the crypts areas was destroyed, and lymphocyte infiltration into the submucosa was observed in the acute group as opposed to the chronic group (C). Scale bar represents 200 μm. The villus of the small intestine was not destroyed by DSS administration and was not directly

modified morphologically (D). In acute group, macrophage-derived Th1 cytokines (IL-1β, IL-6, TNF-α and IFN-γ) and the anti-inflammatory cytokine IL-10 were increased (E). In contrast, cytokines related to the Th2 response were increased in the chronic group (F). Grey bar: Control, Black bar: GI-inflamed mice groups. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, Student's *t*-test.



Supplementary Figure 2. Incretin secretion in response to glucose administration did not affect fasting and blood loss. Food intake (feed / mice body weight) during recovery in acutely inflamed group (A) and chronic group (B). We provided limited feed to construct a condition similar to decreased food intake in acute group. Compared with starved mice with limited feed for 3 days, during which

dietary intake decreased in acutely inflamed mice. After oral glucose gavage (5 g/kg), blood glucose levels (C), GIP levels (D), GLP-1 levels (E), and insulin levels (F) in fasting mice. n=5/group, *$p < 0.05$, one-way ANOVA. Before DSS administration, the blood glucose level did not differ in the control and GI-inflamed groups (G). The damaged groups were performed only OGTT experiment without blood loss (red square) and with blood loss (circle) compared to the control group (H). No significance between without blood sampling and with blood sampling group. n=10/group. In NCI-H716 cells, differentiation with FBS free media and low glucose DMEM did not increase of Sglt1 protein (I).



Supplementary Figure 3. Assessed for the score of diarrhea and feces blood. For diarrhea score, 0 points were assigned for well-formed pellets, 2 points for pasty and semi-formed stools that did not adhere to the anus, and 3 points for watery stools that did smeared to the anus (A). For visible feces

blood, feces were squashed and scored for the degree of feces blood. Changes in body weight and daily monitoring of the clinical score graph (n=17-20/group) (B). Briefly described summary about preparation of conditioned media (C).

Supplementary Table 1. Primary antibodies used in immune-reactive experiments

Antibodies	Company	Cat.	Dilution range
Sglt1	Abcam	ab14686	WB, 1:1000 IF, 1:200
GLUT2	Abcam	ab54460	WB, 1:1000
T1R2	Novus Biologicals	NB110-74920	WB, 1:1000
T1R3	Bioss antibodies	bs-9113R	WB, 1:1000
Gnat3	LifeSpan BioSciences	LS-C406431	WB, 1:1000
β-actin	Santa cruz	sc -47778	WB, 1:5000
Chromogranin A	Abcam	Ab45179	WB, 1:1000 IHC, 1:200
GLP-1	Santa Cruz	sc-47778	IHC, 1:200
GIP	Santa Cruz	sc-57162	IHC, 1:100
Phosph-ERK	Cell signalling	9101S	WB, 1:3000
Total-ERK	Cell signalling	9102S	WB, 1:3000