Supplemental Information

miRNA-129/FBW7/NF-κB, a Novel Regulatory Pathway in Inflammatory Bowel Disease

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Table S1. Basic characteristic of all subjects included in this study

| Parameter          | non-IBD | CD       | UC       |
|--------------------|---------|----------|----------|
| All (n)            | 189     | 172      | 147      |
| Male               | 107 (56.6%) | 114 (66.3%) | 95 (64.6%) |
| Age (years)        | 41.7 ± 9.5 | 49.4 ± 11.3 | 45.2 ± 8.2 |
| Location           |         |          |          |
| Colon              | 102     | 89       | 78       |
| Rectum             | 51      | 33       | 28       |
| Terminal ileum     | 22      | 28       | 26       |
| Ceca               | 14      | 22       | 15       |

IBD, inflammatory bowel disease; CD, Crohn’s disease; UC, ulcerative colitis.
Figure S1. Knockdown or overexpression of FBW7 had no effect on IκBα mRNA level. (A and B) RT-PCR analysis of IκBα mRNA expression in Caco-2 cells treated with FBW7 siRNA (A) or FBW7-GFP adenovirus (B). n=6.
Figure S2. The expression of FBW7-binding miRNAs in colon tissues of inflammatory bowel disease patients and trinitrobenzene sulphonic acid (TNBS)-induced mouse colitis model. (A) Western blotting analysis of the extracts from Caco-2 cells transfected with indicated miRNA mimics. (B) Quantification of FBW7 protein level normalized to GAPDH. **P<0.01 vs. control, n=6. (C-H) The expression of miR-363, miR-223 and miR-27a in non-IBD individuals (n=189), patients with Crohn’s disease (CD, n=172),
patients with ulcerative colitis (UC, n=147), control mice (n=12) and TNBS-treated mice (n=12) was determined by RT-PCR.