studies also demonstrated that the suppression of KLF-5 by silencing RNA resulted in a reduction of NFκB1 mRNA in IEC6 cells stimulated with lipopolysaccharide, indicating that KLF-5 is an upstream regulator for NFkB1 mRNA expression in IEC6 cells.⁷ Taken together, it is most likely that the upregulation of KLF-5 mRNA expression might lead to the enhanced expression of NFκB1 mRNA in BM CD34+ cells, but not vice versa, in RA. In addition, the upregulation of KLF-5 mRNA as well as NFkB1 mRNA in RA BM CD34+ cells might result in their abnormal capacities to differentiate into fibroblast-like cells.

In summary, we demonstrate that KLF-5 mRNA expression is upregulated in BM CD34+ cells independently of the systemic inflammation or treatment regimen in RA. Although it is strongly suggested that KLF-5 might be an upstream regulator of NF κ B1 mRNA in BM CD34+ cells, further studies to explore the mechanism of abnormal expression of KLF-5 mRNA in BM CD34+ cells would be important for a complete understanding of the pathogenesis of RA.

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Corrections

An author affiliation was incorrect in the an article published in March 2008 (Jónsdóttir T, Gunnarsson I, Risselada A, et al. Treatment of refractory SLE with rituximab plus cyclophosphamide: clinical effects, serological changes, and predictors of response. Ann Rheum Dis 2008;67:330-4). The correct affiliation for all authors is Department of Rheumatology, The Karolinska University Hospital, The Karolinska Institutet, Stockholm, Sweden.

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