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 NFκB1 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 NFκB1 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.

S Hirohata,[1] Y Miura,[2] T Tomita,[3] H Yoshikawa[3]

[1] Department of Rheumatology and Infectious Diseases, Kitasato University School of Medicine, Kanagawa, Japan; [2] Faculty of Health Sciences, Kobe University School of Medicine, Kobe, Japan; [3] Department of Orthopedic Surgery, Osaka University Medical School, Osaka, Japan

Correspondence to: Dr S Hirohata, Department of Rheumatology and Infectious Diseases, Kitasato University School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228–8555 Japan; shunsei_tenpoint@yahoo.co.jp

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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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An author name and affiliation was incorrect in the an article published in October 2008 (Lu TY-T, Jónsdóttir T, van Vollenhoven RF, *et al.* Prolonged B-cell depletion following rituximab therapy in systemic lupus erythematosus: a report of two cases. *Ann Rheum Dis* 2008;**67**:1493–4). The second author's name is spelt Jónsdóttir, not Jonsdottir as given in the article. The correct affiliation for all authors is Department of Rheumatology, The Karolinska University Hospital, The Karolinska Institutet, Stockholm, Sweden.

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