>50% reduction in ALC. For each of these cases, however, we did not observe a significant change in the proportionate representation of the mutant *SF3B1* allele, indicating that such treatments did not have selective activity or inactivity for subclones with *SF3B1* mutations.

Taken together, this study reveals subclonal evolution involving cells with *SF3B1* mutations in CLL. Our data indicate that the proportionate representation of cells harboring *SF3B1* mutations can increase independent of therapy or loss of functional p53. Finally, the data presented here suggest that subclones with *SF3B1* mutations do not necessarily have a selective advantage or disadvantage in the setting of effective cytoreductive therapy. Nevertheless, the prevalence of *SF3B1* mutations appears higher among patients who already have undergone therapy. This might reflect the fact that treated patients more commonly have had longer disease histories, potentially providing greater time for emergence and subclonal evolution of CLL cells harboring *SF3B1* mutations. Further studies with additional cases will be required to address the therapeutic implications of the *SF3B1* mutations and subclonal evolution in this disease.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

MS, EG, LR and TJK designed, analyzed the data and wrote the manuscript. LZR and TJK provided patients samples and clinical data. MO performed DNA extractions and edited the paper. MLD'A examined the cytogenetics. JFF analyzed the data and edited the paper.

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Supplementary Information accompanies the paper on the Leukemia website (http://www.nature.com/leu)

OPEN

Call for case histories of BMT in patients with coincident schizophrenia

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Recently, the case for an immune component in the etiology of schizophrenia has regained support,[1,2] leading to randomized controlled trials to explore treatment with immunosuppressants and anti-inflammatory drugs.[3] Both *post-mortem*[4] and *in-vivo* studies[5,6] provided indications for an increased pro-inflammatory status in the brain of patients with recent-onset schizophrenia. A common characteristic of most if not all autoimmune diseases (AD) is their favourable response to immunoablation and rescue with bone marrow transplantation. It was established in radiation chimaeras more than 50 years ago that the immunological and hematological systems have a common stem cell. In the wake of this discovery came a strain of investigations into the role of the bone marrow in AD demonstrating that both hereditary AD and

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the susceptibility to induced AD could be transferred by hematopoietic stem cell transplants and that both forms of AD in experimental animals could be cured by an allogeneic BMT from healthy donors.[7] Accordingly, the records of long-term survivors of an allogeneic bone marrow transplant (BMT) were searched for patients with a coexisting AD at the time of the transplant. Up to 1998, case histories of a total of 22 such patients were retrieved, all of whom except one went into complete remission of their AD.[8] These findings did not initiate treatment of AD patients with allogeneic BMT because of the high risks of this procedure. However, after it was demonstrated both in rats with induced systemic arthritis and in rats suffering from experimental allergic encephalomyelitis (model for multiple sclerosis) that autologous bone marrow transplants were potentially equally effective as allogeneic transplants, this modality has been widely explored for treating refractory AD of all sorts.[9] The European Group for Blood and Bone Marrow Transplantation (EBMT) estimates that, worldwide, around 3000 AD patients have since been treated with autologous BMT; 1200 cases were entered in their database by June 2011.[10] Overall 5-year survival rate of the first 900 cases analysed was 85%, with 43% progression-free survival.

At present, around 50 000 hematopoietic stem cell transplants are registered annually. Considering a conservative prevalence estimate for schizophrenia of 8 per 1000, the registries can be expected to contain data on many survivors with coincident schizophrenia at the time of transplantation. Information on the clinical course of schizophrenia after BMT would greatly enhance our understanding of the role of immune processes in schizophrenia. We have therefore addressed EBMT and the Medical College of Wisconsin Clinical Cancer Center (CBMTR) concerning the feasibility of initiating such research. Simultaneously, we call upon haematologists and psychiatrists to inform us directly of their relevant case histories by submitting this appeal to their respective professional journals and websites. We venture that this way of crowd sourcing may not only save time and money, but may also bring to light information that has not been put into the databases of the BMT registries.

Please send the information about your cases, including identification numbers if registered, to >i.sommer@umcutrecht.nl<

Comments on this Letter to the Editor from the Editor-in-Chief Dr Nicole Muller-Berat Killmann:

I support wholeheartedly the move of Drs I E Sommer and D W van Bekkum to go directly to the core of the problem and to unearth first of all more cases of schizophrenia in the wake of the transplantation process.

Submitted to the journals: *Bone Marrow Transplantation, Leukemia, Stem Cells, Blood, Schizophrenia Research and Schizophrenia Bulletin*.

Submitted to the websites: Schizophrenia Forum, International Society for Hematology and Stem Cells, American Society of Hematology, European Hematology Association, Japanese Society of Hematology and Hematological Society of Taiwan.

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