The increase in these populations in the B27 rats may contribute to the bone marrow and blood of rats over-expressing HLA-B27. CD43\textsuperscript{lo} (CD172a\textsuperscript{+}CD43\textsuperscript{lo}CD11b\textsuperscript{-} and CD172a\textsuperscript{+}CD43\textsuperscript{lo}CD11b\textsuperscript{+}) were present. No differences in osteoclastogenesis were observed between B27 and controls rats. Finally, evaluation of circulating monocytes demonstrated that all blood monocytes express CD11b and that the CD43\textsuperscript{lo}CD11b\textsuperscript{+} population was significantly increased in B27 rats. This corresponded with an increase in CCL2 plasma levels in the B27 rats.

Conclusions We have identified a previously unreported CD11b\textsuperscript{-}monocyte population in the bone marrow of rats, which can differentiate into mature osteoclasts. Along with the numbers of total CD43\textsuperscript{lo} monocytes, this population is significantly increased in the bone marrow and blood of rats over-expressing HLA-B27. The increase in these populations in the B27 rats may contribute to enhanced inflammation and bone loss.

**Results**

A previously unidentified CD172a\textsuperscript{+} CD43\textsuperscript{lo} CD11b\textsuperscript{-} population of monocytes was observed in the bone marrow. These are bona fide monocytes, expressing CDC115 and CCR2, as determined by uptake of fluorescent M-CSF and CCL2 respectively. Interestingly, this new monocyte population was significantly increased in B27 rats. Assessment of the osteoclastogenic potential of bone marrow monocyte subsets revealed that CD172a\textsuperscript{+} CD43\textsuperscript{lo} CD11b\textsuperscript{-}, but not CD172a\textsuperscript{-} CD43\textsuperscript{lo} CD11b\textsuperscript{-} monocytes, can differentiate into mature osteoclasts. Furthermore, although CD172a\textsuperscript{-} CD43\textsuperscript{lo} CD11b\textsuperscript{-} have osteoclastogenic potential, optimal osteoclastogenesis was observed only when all CD43\textsuperscript{lo} (CD172a\textsuperscript{+} CD43\textsuperscript{lo} CD11b\textsuperscript{-} and CD172a\textsuperscript{-} CD43\textsuperscript{lo} CD11b\textsuperscript{+}) were present. These are bona fide monocytes, expressing CD115 and CCR2, and with the possibility of being a potential therapeutic target.

**Background and objectives**

HSCs represent a cell population that gives rise to every lineage of blood cells, including granulocytes, lymphocytes, monocytes and endothelial cells, all of which have been implicated in the pathogenesis of Systemic Lupus Erythematosus (SLE). Although HSCs are thought to exist in a dormant state in bone marrow niche, recent evidence suggests that in lupus mice they over-predominate due to both intrinsic and extrinsic factors. Whether such alteration in the HSC compartment contributes to lupus pathogenesis, remains elusive. Furthermore, the molecular identity of this cell type after the inflammatory impact is unknown. Our working hypothesis is that the fundamental immune aberrations in SLE - genetic or epigenetic - may be easier to be traced back to the HSC population.

**Materials and methods**

Hematopoietic stem cell progenitors were isolated from either healthy or NZB x NZW F1 lupus prone mice bone marrow. The selection markers used are Lin\textsuperscript{-}Sca-1\textsuperscript{-}c-Kit\textsuperscript{-} (LSK) cells, including long and short term HSCs. Cell sorting of the aforementioned populations was performed for enumeration and RNA extraction. RNA-seq analysis was performed.

**Results**

Significantly increased frequencies as well as absolute numbers of HSCs were observed in the BM of lupus mice with established disease as compared to young NZB x NZW F1 mice or to C57BL/6 control mice. Importantly, our results show that bone marrow populations such as HSPCs, lymphoid and myeloid lineages seem to differ in homogeneity, correlating with either age or disease, indicating an alteration in HSC potential under inflammatory conditions. In depth bioinformatics analysis of the RNA-seq results of HSCs is in process.

**Conclusions**

Our results will characterise the global gene expression of HSCs originating by lupus mice. Simultaneously, we plan to map SNPs in the genetic background of lupus mice and pinpoint the important ones for HSCs integrity. Acquired knowledge will provide insights into the contribution of HSC in lupus pathogenesis as well as will demonstrate how the respective environment could shape the HSC compartment function.