

MEETING ABSTRACT

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# Effects of aging on endothelial progenitor cells (EPCs) subpopulations in peripheral blood: a possible rationale for age-associated vascular dysfunction

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## Background

Some evidence supports vascular aging as an independent risk factor for cardiovascular morbidity and mortality. Endothelial progenitor cells (EPCs) are a subset of bone-marrow cells that are thought to play an important role in improving the endothelial function. In particular, it has been shown that EPCs recruited into ischemic tissues promote angiogenesis and thereby contribute to tissue regeneration. The recruitment of EPCs to promote neovascularization or repair damaged endothelium is diminished by aging. Therefore, it is possible that aging can alter the process of differentiation from immature early-EPCs to mature and ready for homing late-EPCs. This process is complex and proceeds through several steps. Early-EPCs express markers of hematopoietic stem cells (CD34 and CD133), then these cells lose CD133 and express endothelial markers, such as VEGFR2 and Ve-Cadherin.

## Material and methods

Following approval of the study by the local ethics committee 26 healthy subjects were enrolled. Participants were classified in two groups, accordingly to their age: 13 young subjects (8 men, 5 women; age,  $43 \pm 0.65$ ) and 13 aged subjects (6 men, 7 women; age,  $56 \pm 1.5$ ). We analyzed peripheral blood not later than 5h after collection, using a high performance flow cytometer BD FACS-Canto. We identified after setting a live gate on cells: early-EPCs as  $CD34^+/CD133^+/CD117^+$  mononuclear

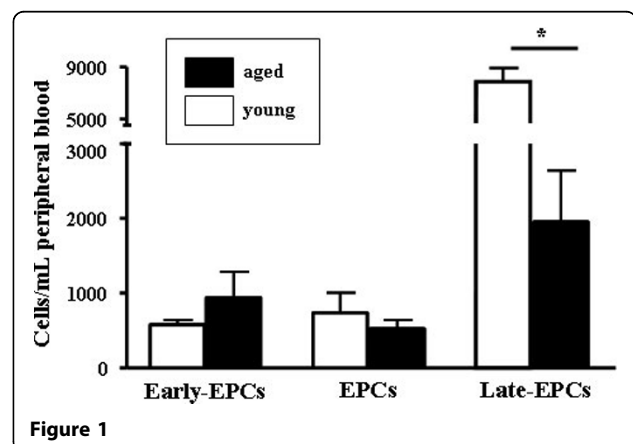
cells; EPCs as  $CD34^+/CD133^+/VEGFR2^+$  mononuclear cells; late-EPCs as live  $CD31^+/VEGFR2^+/Ve-Cadherin^+$  mononuclear cells. For each sample we acquired from  $0.5$  to  $1.0 \times 10^6$  events and fluorescent beads were used for the absolute count.

## Results

We found that there were not significant differences in the numbers of early-EPCs and EPCs between young and aged people. In contrast, aged people showed a dramatic decrement in the number of late-EPCs with respect to the young group ( $*P < 0.001$ ; Fig. 1).

## Conclusions

Our results suggest that one of the mechanisms that underlies the age-associated alterations in vascular



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structure and function may be related to a combination of alterations in homing and maturation of EPCs.

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