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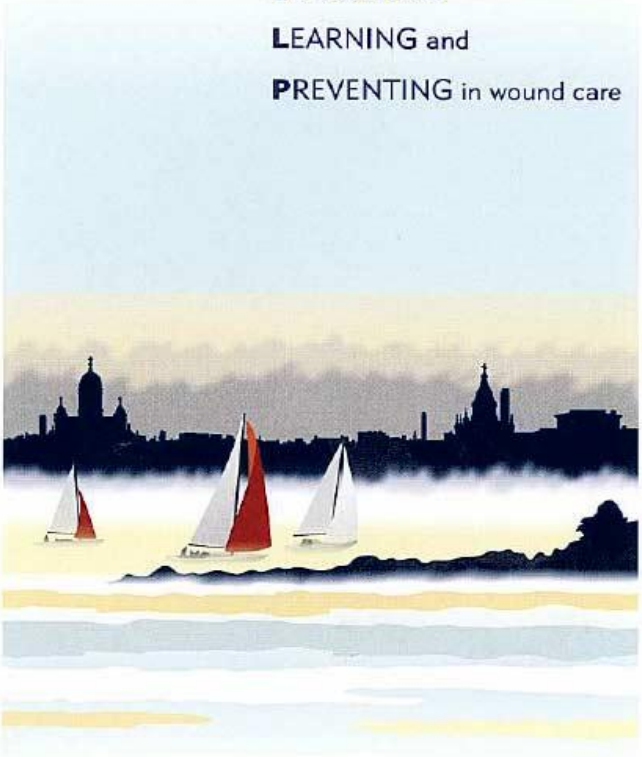

ABSTRACTS

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INHIBITION OF TIBIAL FRACTURE HEALING IN SMOKERS: CELLULAR AND MOLECULAR ASPECTS

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Tobacco smoking has a detrimental impact on fracture healing, and has been implicated in the non-union and delayed union of bone. Whilst previous studies have concentrated on the clinical manifestations of smoking, little work has been undertaken biochemically.

Aim: To study the effects of smoking at the cellular and molecular level in vitro.

Methods: *Cell-culture assay:* Fracture haematomas were collected from anaesthetised patients (n=16; 5 smokers vs. 11 non-smokers) that had sustained a tibial fracture. The semisolid material was explanted into tissue culture flasks and allowed to clot. Complete culture media was introduced into the flasks, which were placed in an incubator (37°C; humidified CO_2). Cultured cells were characterised via immunofluorescence and immunophenotyping using known mesenchymal stem cell antibody markers (CD29, CD44, and CD166); CD34 was applied as a negative control. A flow cytometer was used to count cell populations at the end of each passage. *ELISA assays:* Serum and cells obtained from the fracture haematomas were subjected to an ELISA to compare the amount of VEGF-A (serum) and IL-6 (cells) between smokers and non-smokers.

Results: Cell counting showed a reduction in the rate of proliferation of cells in smokers over 3 passages (~-200%). The VEGF-A (serum) and IL-6 (cells) ELISA revealed a reduction of these acute phase proteins in patients who were smokers (VEGF-A ~-10%; IL-6 ~-15%).

Conclusion: Fracture haematoma mesenchymal stem cells proliferate at a slower rate in vitro in smokers than non-smokers. The amounts of VEGF-A and IL-6 are reduced in smokers' serum and cell cytoplasm respectively.