

磁性附連體使用之基礎與臨床問題研究-由非侵入性穩固度偵測至促進骨母細胞分化之生物學探討

Investigations on the basic and clinical problems of magnetic attachment – from non-invasive stability detection to osteoblastic maturity effects

中文摘要

本研究建構一個「釘板系統-黏著劑-牙根-骨塊」的三維有限元素模型，並以模型中釘板系統與黏著劑間的邊界條件為變數，計算不同邊界結合強度下釘板系統的第一個自然頻率與模態形狀。體外模態測試的結果顯示，當釘板系統的邊界條件改變時，所測得的自然頻率會有不同 ($p < 0.01$)。三維有限元素模型的模擬結果則顯示，當介面彈簧元素的彈性係數小於 104 Nm^{-1} 時，釘板系統是處於完全鬆脫的狀態。而彈性係數介於 $104 \sim 107 \text{ Nm}^{-1}$ 時，模型的自然頻率 ($6.16 \text{ kHz} \sim 15.52 \text{ kHz}$) 會有增加的趨勢。當彈性係數大於 107 Nm^{-1} 之後，則模型的自然頻率的數值達一高原期。由以上之初步結果可知，以自然頻率的變化來評估釘板系統的邊界穩定狀態，是具有可行性的。本研究亦進行細胞實驗來探討靜磁場對骨母細胞的分化促進作用。首先將 MG63 似骨母細胞株以強度 $0.1\text{-}0.4 \text{ T}$ 的靜磁場連續暴露 0 、 12 、 24 、 48 與 72 小時，靜磁場影響細胞增殖的作用以流式細胞技術法分析，細胞形態與基質小泡的分泌利用掃描式與穿透式電子顯微鏡加以觀察。此外靜磁場對 MG63 細胞表現 TGF- β 、第一型膠原蛋白、造骨素、鹼性磷酸酉每活性的影響亦被加以分析。細胞暴露於 0.4 T 的靜磁場之後造成的生長因子促進功能的影響，與細胞膜物理性質改變則被用以探討靜磁場作用機轉。本研究結果發現，MG63 細胞接受靜磁場暴露之後會比控制組細胞表現出更為成熟的細胞形態與更多的分化調控因子。此外， 4 小時的靜磁場暴露使得 MG63 細胞表現出更高的螢光等向性，並在 12 小時後減少生長因子的促進增殖功能。這些發現顯示靜磁場會經由減少細胞膜流動性而降低生長因子促進增殖的作用，並因而改變分化早期調控因子而影響骨母細胞的成熟狀態。

英文摘要

A 3-D finite element model of the Root Keeper-cement-dentin system was established for modal analysis. Natural frequency (NF) values of the first vibration mode of the model with various boundary conditions were calculated and compared. On the other hand, in vitro results showed that the measured NF values changed significantly ($p < 0.01$) under various surrounding material conditions. Results obtained from finite element (FE) simulations demonstrated that Root Keeper would fully loosen when the constant values of the spring elements were lower than 104 .

Furthermore, the NF values of the model increased significantly ($p < 0.01$) when the constant was increased from 104 Nm^{-1} (6.16 kHz) to 107 Nm^{-1} (15.52 kHz), and then reached a plateau. These results demonstrate that the NF has the potential to be used as a parameter for monitoring the stability of Root Keeper. Cell culture studies were carried out to examine the hypothesis that static magnetic fields (SMF) affect osteoblastic differentiation. MG63 osteoblastlike cells were continuously exposed to 0.1-0.4 T SMFs for 12, 24, 48, and 72 hours. The proliferation effects of SMF were tested by flow cytometry. The morphology change and matrix vesicles release were observed by scanning and transmission electron microscopy. The effects of SMFs on levels of TGF- β 1, Type I collagen, osteopontin, and alkaline phosphatase were compared between the exposed and unexposed cells. Growth factors binding assay and membrane fluidity were used to evaluate the alternations in biophysical properties of cellular membranes after 0.4 T SMF simulation. The data suggest MG63 cells treated with SMF exhibit a more differentiated morphology. The local regulatory factors produced by SMF treated cells were higher than the control cultures. Furthermore, MG63 cells exposed to SMF exhibited a significant increase in fluorescence anisotropy at 4 hrs, then significant reduction in the proliferation effects of growth factors noted at 12 hrs. These findings provide evidence that SMF affect osteoblastic maturation by upregulating early local factors. Our results also suggest that SMF affect osteoblastic maturation by increasing the membrane fluidity and reducing the proliferation-promoting effects of growth factors at the membrane domain.