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Static magnetic fields (SMFs) dynamics application for the exacerbated DNA damage-induced apoptosis in renal tubule injury

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Objectives: Static magnetic fields (SMFs) are constant fields created by magnets or charges with no intensity or direction changes over time and are investigated as apoptosis mediators associated with DNA damage. In this experiment, multiple SMFs platforms were designed to mimic the pathophysiological events in renal tubule injury and to evaluate non-invasive renal cell apoptosis with low cytotoxicity. Here, our study demonstrates the effect of multiple SMFs platforms as a physical method to elevate the magnetic forces-induced ROS stress *in vitro*.

Methods: Primary human tubular epithelial cells (HTECs) were incubated on multiple static magnetic field slots (Single center slot, North/South (N/S) full slot between like poles, and mixed full slot between unlike poles) and employed to produce vertically orientated magnetic fields. The diameter of the cell culture plate with height (60 x 15 mm) and digital gauss meter (mT; Tesla) was used to measure magnetic field strength. After 48 hours, gene regulation and DNA damage were analyzed and quantified.

Results: Single center slot, N/S full slot, and mixed slot magnetic field strength were obtained as follows, 6.6mT, 4.9mT, 0.3mT. The effect of the SMFs force on HTECs showed that higher strength in N/S full slot (4.9mT) was associated with increased DNA damage (OGG) and cell death (P21). Furthermore, incubating HTECs with an SMFs platform for 48 increased inflammatory markers (IL-6 & IL-17) but decreased ROS stress tolerance markers (SOD-1 & NQO-1) expressions. In addition, E-cadherin and AQP-1 levels were significantly reduced by SMFs. Finally, the level of NGAL and Bax increased, highlighting that the SMFs culture system suppressed HTECs function and proliferation via the apoptosis signaling pathway.

Conclusions: These findings suggest that SMFs-driven damage at the cellular level is a powerful tool for resembling magnetic-causing renal tubular injury.