CytoSelect 24-well Wound Healing Assay Assay Kit

CytoSelect 24-well Wound Healing Assay

CATALOG #
AAA11414

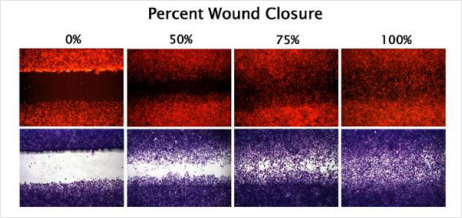
PRODUCT SYNONYMS

CytoSelect 24-well Wound Healing Assay; CytoSelect 24-well Wound Healing Assay assay kit

PRODUCT SPECIFICATIONS

Preparation and Storage	Upon receipt, transfer the DAPI Fluorescence Stain to -20 degree C. Store all other components at 4 degree C.

IMAGES



Application Data

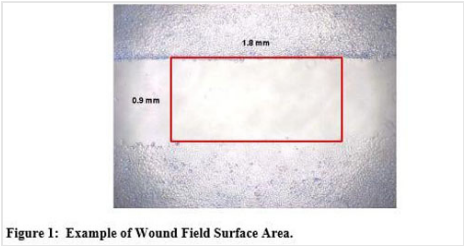
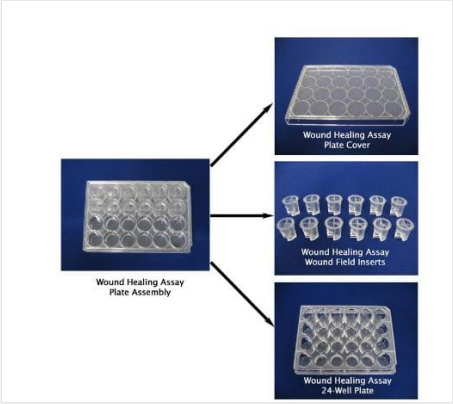


Figure 1: Example of Wound Field Surface Area.

Application Data



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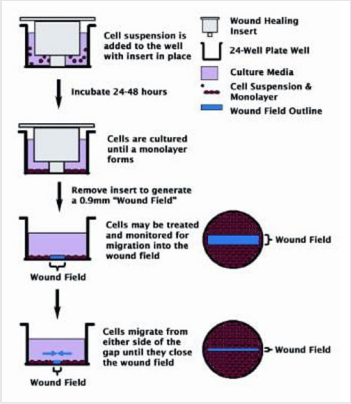

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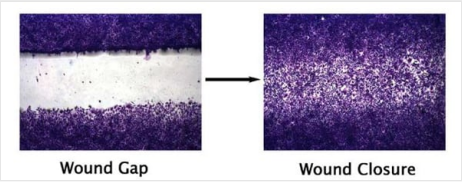

Application Data

ADDITIONAL INFORMATION

Related Product Information for CytoSelect 24-well Wound Healing Assay assay kit	Background: Wounded tissue initiates a complex and structured series of events in order to repair the damaged region. These events may include increased vascularization by angiogenic factors, an increase in cell proliferation and extracellular matrix deposition, and infiltration by inflammatory immune cells as part of the process to destroy necrotic tissue. The wound healing process begins as cells polarize toward the wound, initiate protrusion, migrate, and close the wound area. These processes reflect the behavior of individual cells as well as the entire tissue complex. Wound healing assays have been employed by researchers for years to study cell polarization, tissue matrix remodeling, or estimate cell proliferation and migration rates of different cells and culture conditions. Wound healing assays have been used to study cell polarity and actin cytoskeletal structure regulation through the role of Rho family GTPases, microtubule and Golgi apparatus orientation, the role of p53 in cell migration, as well as other physiological processes. These assays typically involve culturing a confluent cell monolayer and then displacing or destroying a group of cells by scratching a line through the monolayer. The open gap created by this "wound" is then inspected microscopically over time as the cells move in and fill the damaged area. This "healing" effect can take several hours to several days depending on the cell type, conditions, and the surface area of the "wounded" region. The disadvantage of these "scratch wound" assays is the lack of a defined wound surface area, or gap between cells. These wounds are varying sizes and widths, which inhibits consistent results and creates variation from well to well. In addition, the "scratch wound" assay often causes damage to the cells at the edge of the wound, which can prevent cell migration into the wound site and healing. Our CytoSelect™ Wound Healing Assay Kit overcomes this inconsistency by providing proprietary treated inserts that can generate a defined wound field or gap. Cells are cultured until they form a monolayer around the insert. The insert is removed, leaving a precise 0.9 mm open "wound field" between the cells. Cells can be treated and monitored at this point for migration and proliferation into the wound field. Progression of these events can be measured by imaging samples fixed at specific time points or time-lapse microscopy. CytoSelect™ Wound Healing Assay Kit includes proprietary "wound field" inserts to assay the migratory and wound healing characteristics of cells. This Trial Size kit contains sufficient reagents for the evaluation of 6 samples. The insert is optimal for use with most cell types and experimental conditions. The 0.9 mm wound field generated is compatible for use with most microscopes and imaging systems. Principle of the Assay: This Trial Size CytoSelect™ 24-well Wound Healing Assay Kit contains a 24-well plate containing 6 proprietary treated plastic inserts. The inserts create a wound field with a defined gap of 0.9mm for measuring the migratory and proliferation rates of cells. Migratory cells are able to extend protrusions and ultimately invade and close the wound field. Cell proliferation and migration rates can be determined using manual fixing and microscopic imaging. A fixing solution is provided for stopping cells at specific time points. Cell stain and DAPI stain are also provided for viewing results with light and fluorescence microscopy.
Product Categories/Family for CytoSelect 24-well Wound Healing Assay assay kit	Cell Based Assays; Cell Migration; Invasion and Wound Healing; Gap Closure Assays; Wound Healing Assays

References	1. Ridley AJ, Schwartz MA, Burridge K, Firtel RA, Ginsberg MH, Borisy G, Parsons JT, Horwitz AR. (2003) Science 302, 1704-9. 2. Horwitz R, Webb D. (2003) Curr Biol. 13, R756-9. 3. Lauffenburger DA, Horwitz AF. (1996) Cell 84, 359-369. 1. Otsubo, T. et al. (2015). Aberrant DNA hypermethylation reduces the expression of the desmosome-related molecule periplakin in esophageal squamous cell carcinoma. Cancer Med.. doi: 10.1002/cam4.369. 2. Kawai, T. et al. (2015). Secretomes from bone marrow-derived mesenchymal stromal cells enhance periodontal tissue regeneration. Cytotherapy. doi: 10.1016/j.jcyt.2014.11.009. 3. Niu, F. et al. (2014). Tat 101-mediated enhancement of brain pericyte migration involves platelet-derived growth factor subunit B homodimer: implications for human immunodeficiency virus-associated neurocognitive disorders. J Neurosci. 34:11812-11825.4. Cui, X.G. et al. (2013). Response gene to complement 32 deficiency causes impaired placental angiogenesis in mice. Cardiovasc Res. 10.1093/cvr/cvt121. 5. Gutschner, T. et al. (2013). The noncoding RNA MALAT1 is a critical regulator of the metastasis phenotype of lung cancer cells. Cancer Res. 73:1180-1189. 6. Xing, C. et al. (2013). Reversing effect of ring finger protein 43 inhibition on malignant phenotypes of human hepatocellular carcinoma. Mol. Cancer Ther. 12:94-103. 7. Mao, R. et al. (2012). Circadian gating of epithelial-to-mesenchymal transition in breast cancer cells via melatonin-regulation of GSK3beta. Mol Endocrinol. 26:1808-1820. 8. Walsh, E.M. et al. (2012). Importance of interaction between nerve growth factor and alpha9beta1 integrin in glial tumor angiogenesis. Neuro Oncology. 10.1093/neuonc/nos119. 9. Hirata, H. et al. (2012). MicroRNA-1826 targets VEGFC, Beta-catenin (CTNNB1) and MEK1 (MAP2K1) in human bladder cancer. Carcinogenesis 33:41-48. 10. Wang, J.N. et al. (2011). Response gene to complement 32 promotes vascular lesion formation through stimulation of smooth muscle cell proliferation and migration. Arterioscler Thromb Vasc Biol. 31:e19-e26. 11. Oommen, S. et al. (2011). Vascular Endothelial Growth Factor A (VEGF-A) induces endothelial and cancer cell migration through direct binding to integrin alpha9beta1: identification of a specific alpha9beta1 binding site. J. Biol. Chem. 286:1083-1092. 12. Buckley, S. et al. (2011). The milieu of damaged alveolar epithelial type 2 cells stimulates alveolar wound repair by endogenous and exogenous progenitors. Am. J. Respir. Cell Mol. Biol. 45:1212-1221. 13. Tao, Y. et al. (2011). Treatment of breast cancer cells with DNA demethylating agents leads to a release of Pol II stalling at genes with DNA-hypermethylated regions upstream of TSS. Nucleic Acid Res. 10.1093/nar/gkr611.

For Research Use Only. Not for use in diagnostic or therapeutic procedures. Small volumes of product may become entrapped in the seal of the vial during shipment and storage; if necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the cap.

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Online datasheet: https://www.aaabiotech.com/assay-kits/cytoselect-24-well-wound-healing-assay/11414