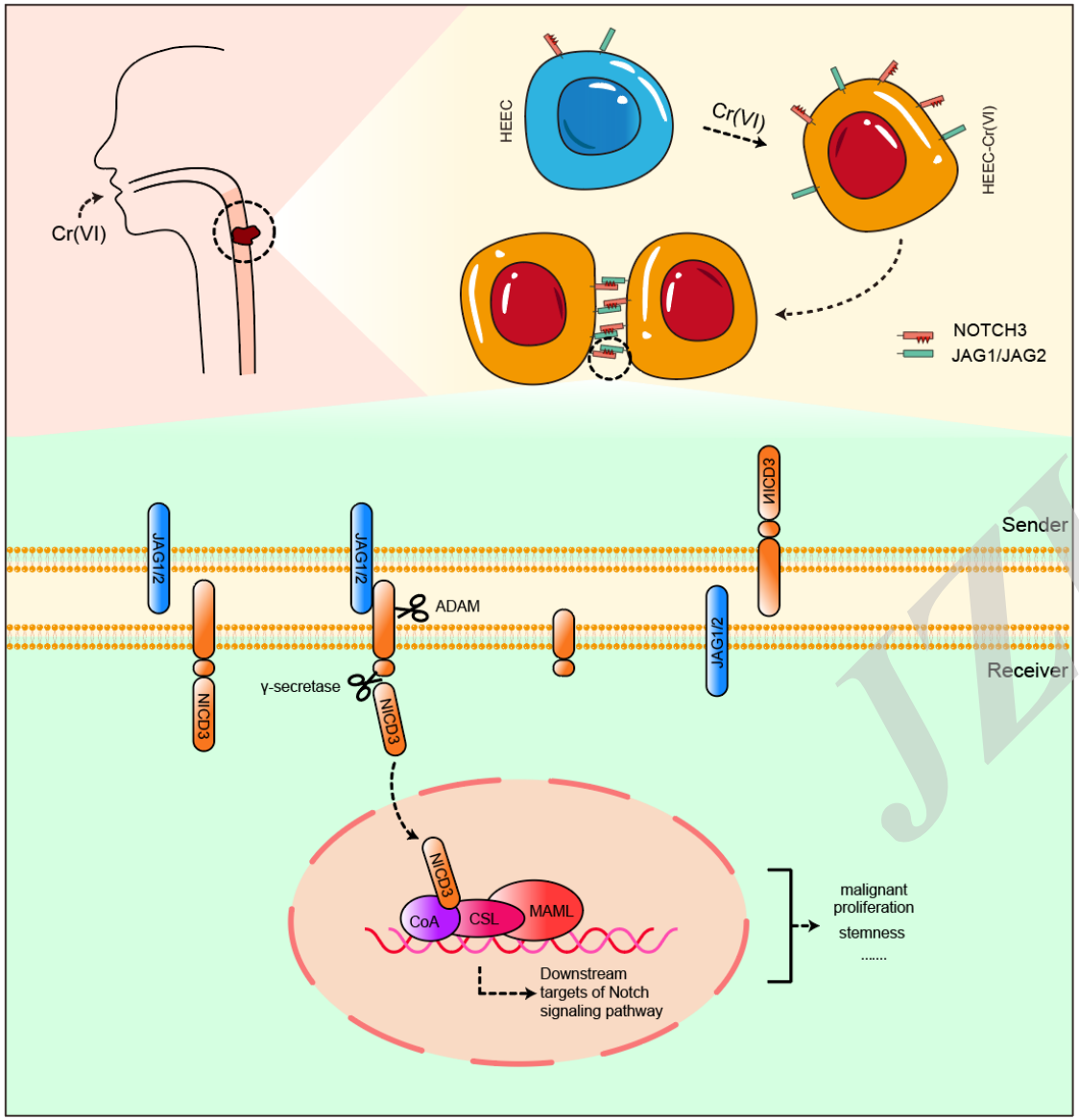
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Chronic exposure to hexavalent chromium induces esophageal tumorigenesis via activating the Notch signaling pathway

Key words: Hexavalent chromium [Cr(VI)]; Esophageal tumorigenesis; Malignant proliferation; Stemness; Notch signaling pathway

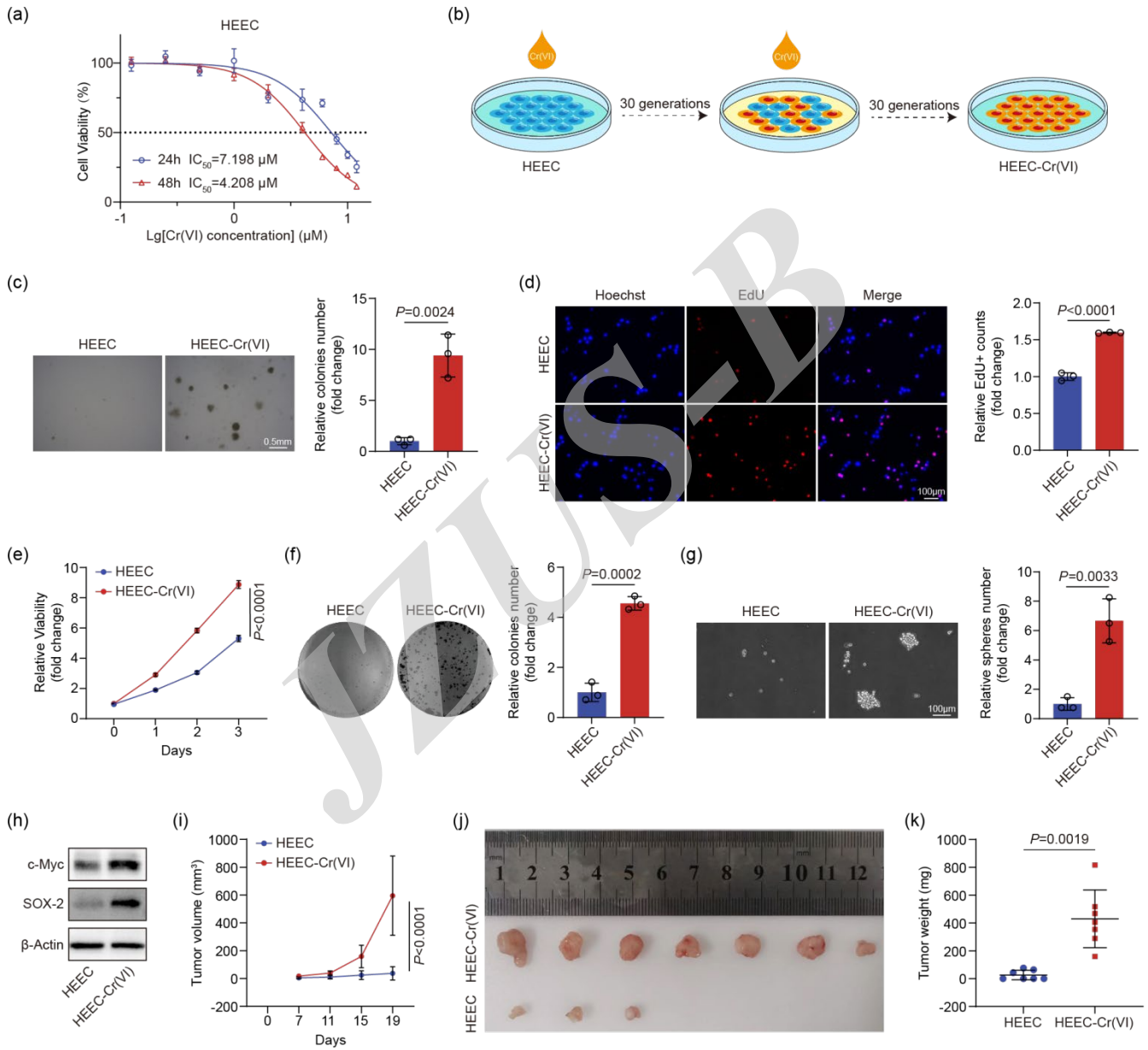
Research Summary



Our study mainly focused on illuminating the molecular mechanism underlying Cr(VI)-induced esophageal tumorigenesis. Here, immortalized human esophageal epithelial cells (HEECs) were induced to be malignantly transformed cells, termed HEEC-Cr(VI) cells, via chronic exposure to Cr(VI). We found that HEEC-Cr(VI) cells obtain the ability of anchorage-independent growth, greater proliferative capacity, cancer stem cell properties, and capacity to form subcutaneous xenografts in BALB/c nude mice when compared to its parental cells, HEECs. Mechanistically, chronic exposure to Cr(VI) induced abnormal activation of Notch signaling, which is crucial to maintaining the capacity for malignant proliferation and stemness of HEEC-Cr(VI) cells. In summary, our study provides novel insights for further basic research and clinical therapeutic strategies about Cr(VI)-associated esophageal cancer.

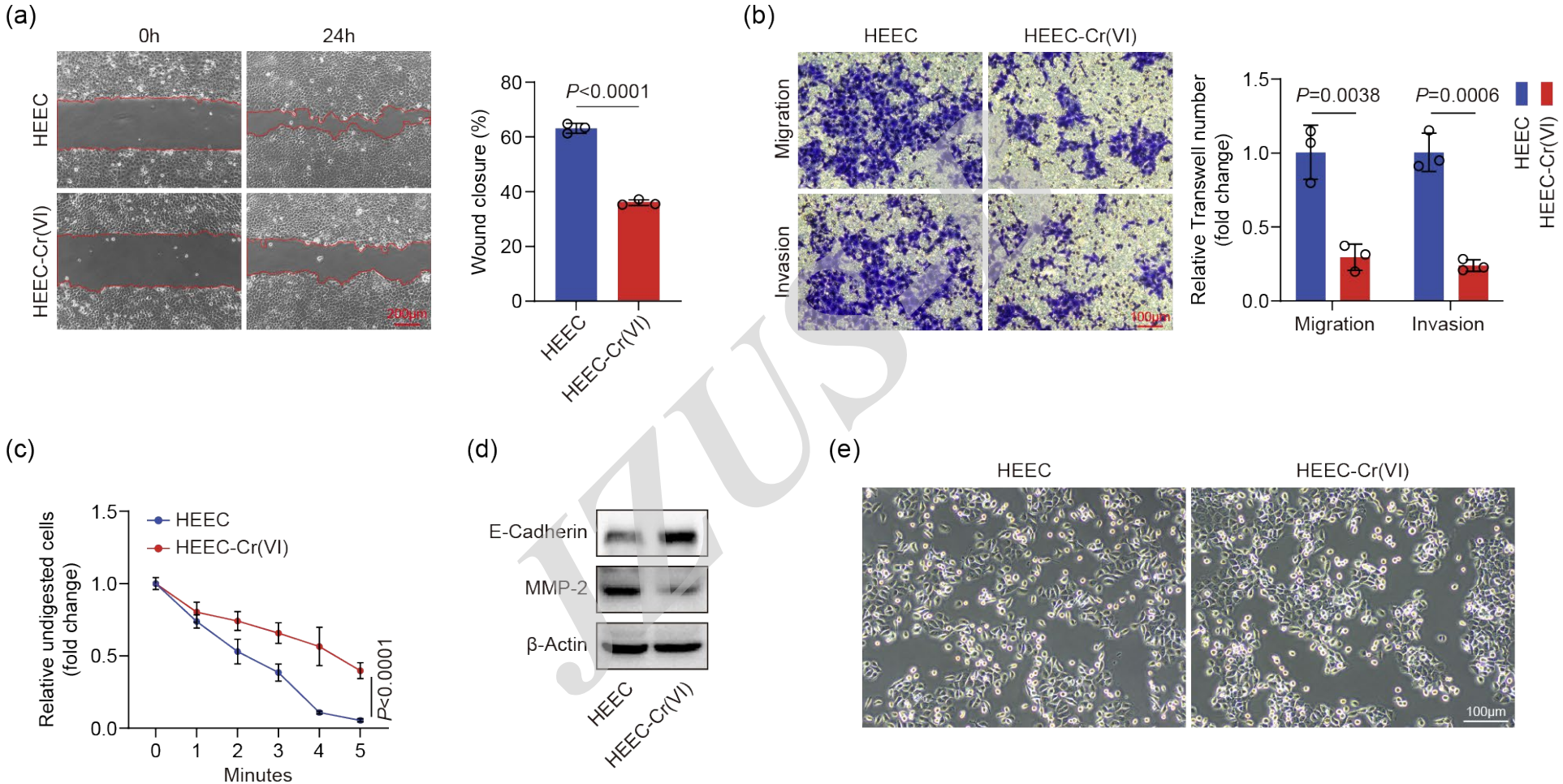
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1. HEECs with chronic exposure to Cr(VI) acquire the phenotypes of malignant proliferation and stemness

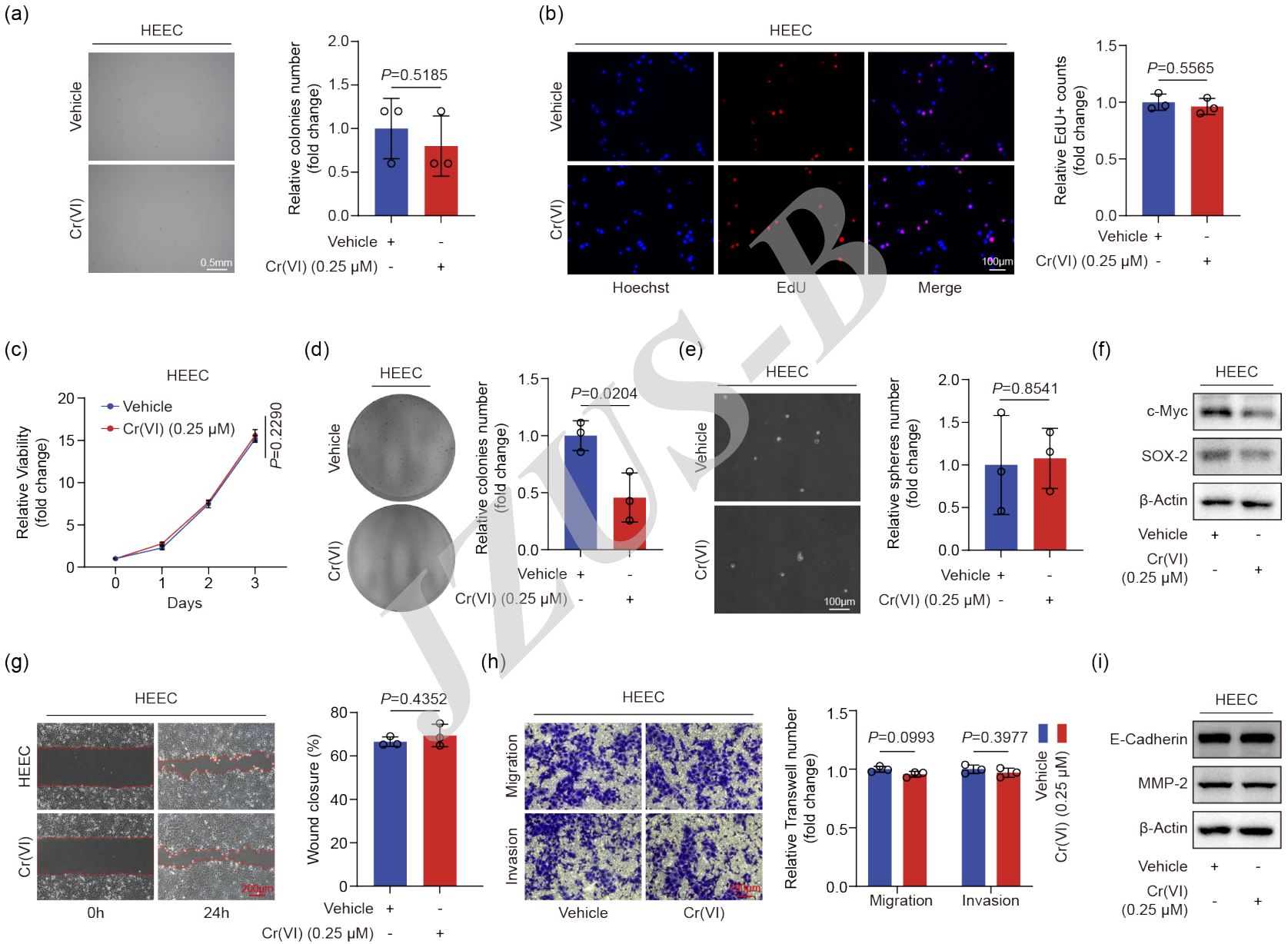


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2. HEEC-Cr(VI) cells display attenuated cell motility and enhanced cell adhesion

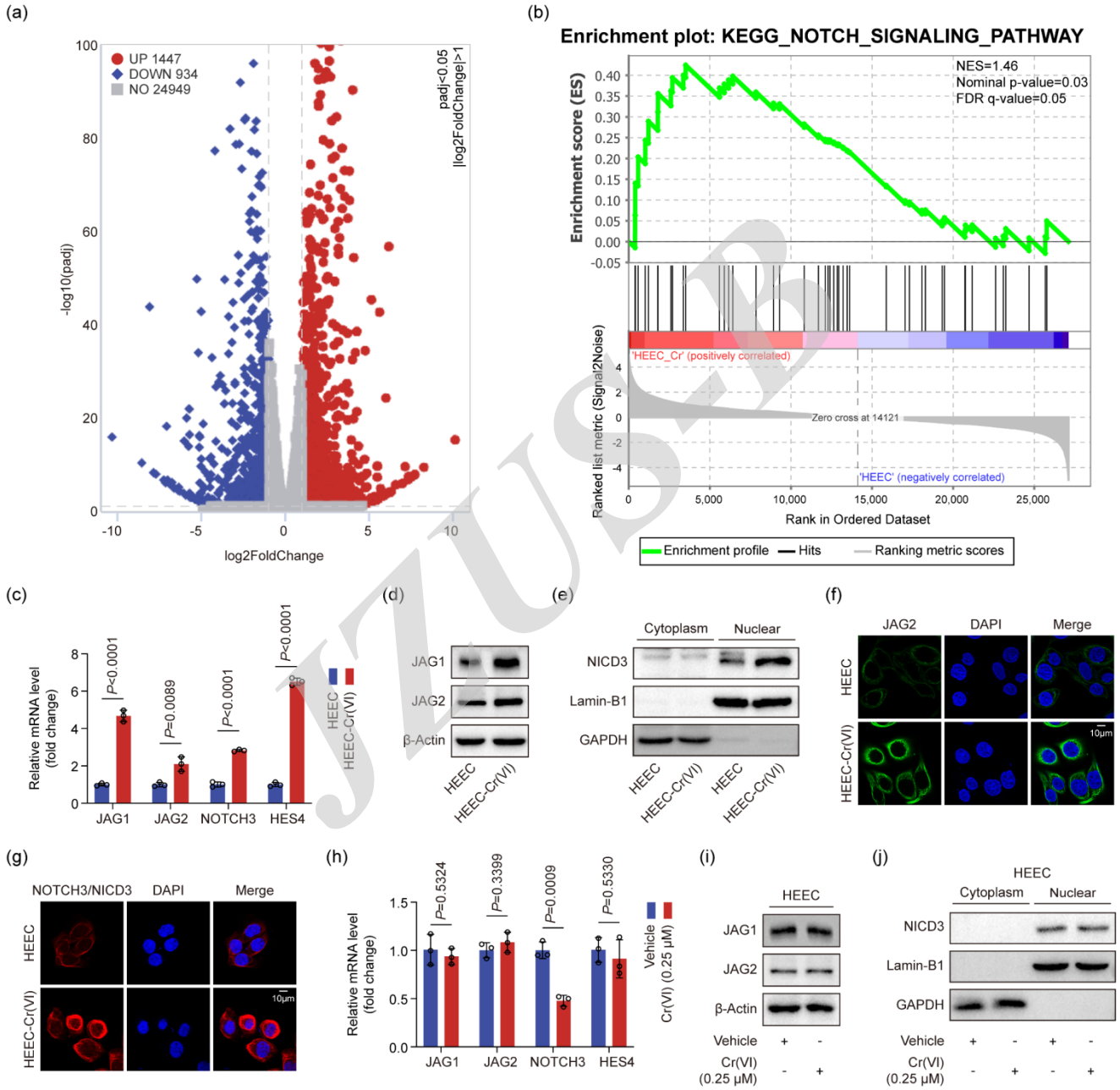


3. Acute exposure to Cr(VI) failed to confer HEECs with the characteristics of HEEC-Cr(VI) cells



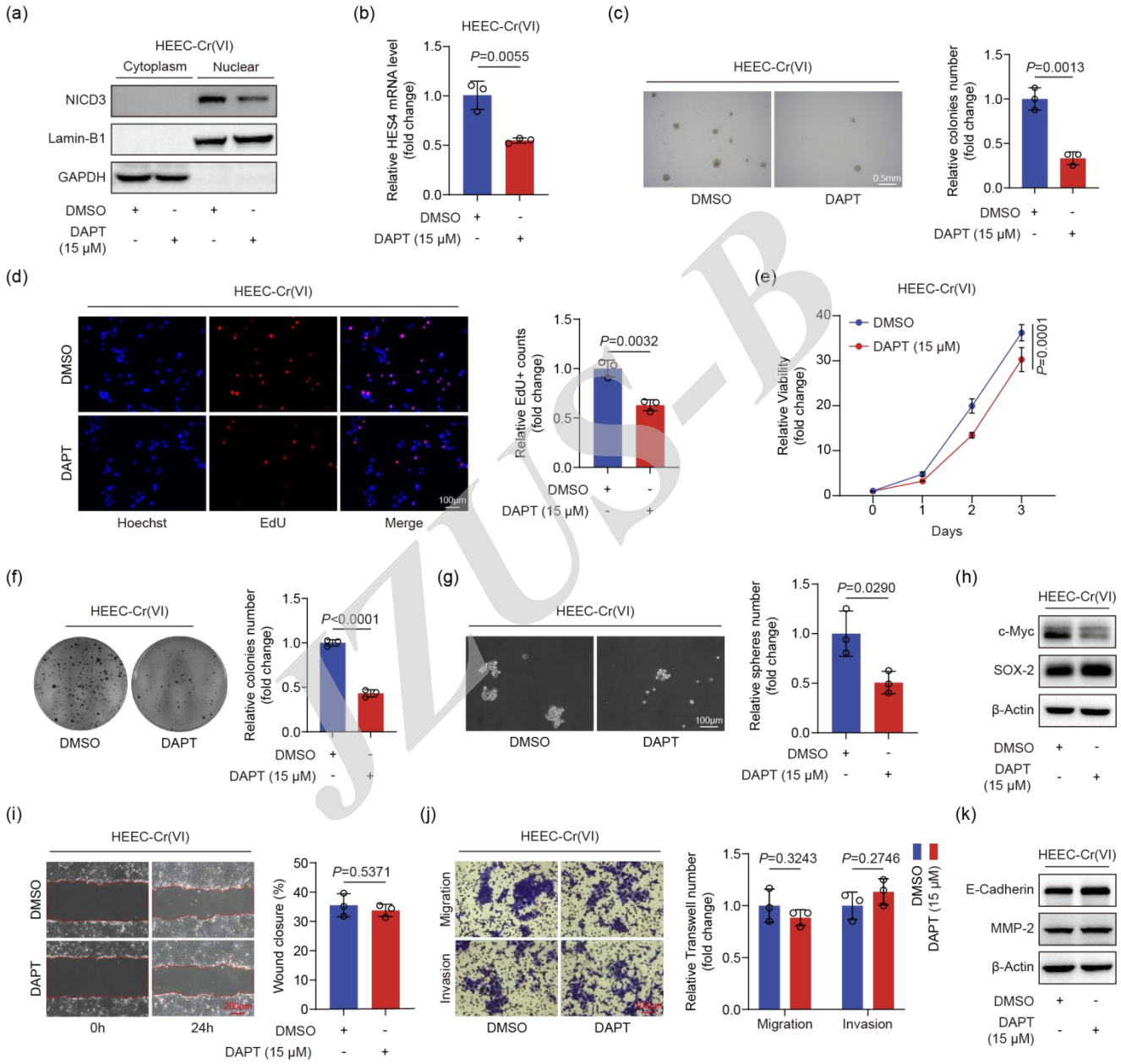
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4. Notch signaling pathway is abnormally activated in HEEC-Cr(VI) cells



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5. Blockage of the Notch signaling pathway impairs the malignant proliferation and stemness of HEEC-Cr(VI) cells



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