Direct Cell-Cell Communication Controls the Division of the PC-3 Human Prostate Cancer Cell Line's Stem Cells

Department of Applied Biological Science, Tokyo University of Agriculture and Technology, 3-5-8 Saiwaicho, Fuchu, Tokyo, 183-8509, Japan

Disease undi erentiated cells (CSCs) are a subpopulation that can drive repeat and metastasis. In this way, treatments focusing on CSCs are required. The precise molecular mechanism by which non-CSCs regulate CSC proliferation and di erentiation in the tumor microenvironment is largely unknown, despite previous ýndings suggesting this. In the PC-3 human prostate cancer cell line, we discovered that a direct interaction between CSCs and non-CSCs decreased CSC division [1]. When non-CSC-rich parental PC-3 cells were present in a culture, the proliferation of PC-3-derived CSCs (PrSCs) was signiýcantly lower (47%) than when they were absent. When PrSCs were indirectly cocultured with PC-3 cells across a Transwell insert, there were no di erences in PrSC proliferation, and PrSCs that were transiently bound to immobilized PC-3 cells proliferated more slowly than PrSCs. The recurrence of cell division with earlier PrSC contact was 2.8 times higher in the PrSC monoculture contrasted and that in the coculture with PC-3 cells [2]. A cell proximity assay revealed that the PrSCs were approximately 1.3 times more closely associated in the monoculture than in the coculture with PC-3 cells. In the coculture with PC-3 cells, the frequency of asymmetric PrSC division was 1.0%, while it was 6.5% in the monoculture (P 0.045). We discovered that PrSC ï non-CSC contact regulates PrSC division frequency and mode through data analysis. The treatment of cancer may have a useful target in this regulation [3].

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