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Brief Communication

Comparison of CXCL10 Secretion in Colorectal Cancer Cell Lines

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Established cancer cell lines are widely used for developing biomarkers for the patient-specific treatment of colorectal cancer and predicting prognoses. However, cancer cell lines may exhibit different drug responses depending upon the characteristics of the cell line. Therefore, it is necessary to select a tumor cell line suitable for the purpose of the study by considering the cell characteristics. This study investigated the levels of CXCL10, which were recently been reported to play an important role in the outcome of tumor treatment, secreted by colon cancer cells. 2×10^5 cells/mL of each colorectal cancer cell was seeded into a 35 mm cell culture dish. After 24 h incubation, culture supernatant was used to determine the secreted CXCL10 levels. Among six colorectal cancer cell lines (HT-29, HCT116, CaCo-2, SW620, SW480, and CT26), Caco-2 cells showed the highest level of CXCL10 secretion. HT-29 cells showed the second-highest level of CXCL10 secretion. No significantly measurable level of CXCL10 secretion was detected in HCT116 cells. These results will be helpful in investigating the molecular basis of colorectal cancer.

Key Words: Colorectal cancer, CXCL10, Chemotherapy, Chemokine, Biomarker

Colorectal cancer (CRC) accounted for 12.3% of the total cancer incidence in Korea in 2016, which is the second highest incidence in Korea after gastric cancer, with an incidence rate of 13.3%. Adjuvant chemotherapy, which is recommended to reduce the risk of recurrence and death by eliminating micrometastases (Gill et al., 2004), should be initiated in colorectal cancer patients within 6~8 weeks after surgical resection (André et al., 2004; André et al., 2009; Van Cutsem et al., 2009). However, surgery treatment, the most important method of colorectal cancer treatment, can cause complications such as intestinal obstruction, intra-abdominal infection, and changes in bowel movements. Patients experience high stress due to restrictions in daily life, low self-esteem, and discomfort from stoma formation (Kim et al., 2016). With recent developments in medical technology, the introduction of the Clinical Pathway [CP], and the hospital's efforts to increase the surgical bed turnover rate, the period of hospitalization and treatment for colorectal

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cancer patients is becoming shorter. Although the degree of uncertainty experienced by the patient is increasing due to this unimplemented treatment system, there is a lack of reliable, expert-based programs that can provide customized management and immediate feedback according to the treatment process of the target patient. Colorectal cancer dramatically impacts the patient's quality of life and various basic studies are being conducted to address this problem, which occurs partly because of the lack of biochemical, molecular, and biological biomarkers for early cancer detection and the monitoring of treatment progress.

Colorectal cancer cell lines are widely used for studying tumor biology, optimal therapy, and biomarker development (Mouradov et al., 2014). Colorectal cancer cell lines are the most commonly used non-clinical research model for colorectal cancer research and have provided diverse insight into tumor molecular and cell biology. These cell lines are fundamental tools used to discover novel anticancer drugs and drug sensitivity, resistance, and toxicity biomarkers and use molecular markers of response to existing chemotherapy and targeted agents that show clinical utility to patients (Bracht et al., 2010; Ashraf et al., 2012; Barretina et al., 2012; Weickhardt et al., 2012; Basu et al., 2013). However, the extent to which colorectal cancer cell lines represent and maintain the genetic diversity of primary cancer remains controversial.

CXCL10 is a chemokine ligand mainly induced by IFN-γ, also called IFN-γ-induced protein 10 (IP-10). It is induced in various cells including endothelial cells, monocytes, fibroblasts, and keratinocytes (Aronica et al., 2009). CXCL10 is generated not only by IFN-γ but also by IFN-α/β and weakly by TNFα. CXCL10 is a ligand that binds to the G protein-coupled receptor CXCR3 (Lambert and Paulnock, 1987; Liu et al., 2011). When CXCL10 binds to CXCR3, naïve T cells differentiate into Th1 cells (T helper 1 cells) (Aronica et al., 2009). After Th1 cells are differentiated, CTLs, NK cells, and NKT cells are activated through IFN-γ (Ohmori et al., 1993). CXCL10 is also known to be a substance that can affect cancer treatment. Depending upon the secretion method of CXCL10, it may inhibit or promote cancer. CXCL10 exhibits cancer-suppressing properties when acting as a paracrine substance. Paracrine means that the CXCL10 ligand, which binds to the CXCR3 receptor, was secreted from immune cells, not from cancer cells. One of the cancer-suppressing functions of CXCL10 is that it can activate immune cells. As an example, CXCL10 has a positive correlation with tumor-infiltrating lymphocytes (TILs), lymphocytes that selectively migrate to the location of cancer cells. Thus, if TILs are detected in the patient's body, whether or not the patient's body will respond to checkpoint inhibitors can be predicted (Ohmori et al., 1993). Since CXCL10 is an ELR (glutamic acid-leucine-arginine)-negative CXC chemokine, it inhibits tumor angiogenesis when it acts as a paracrine substance and generally inhibits tumor growth. A previous study showed that CXCL10 inhibited cancer angiogenesis and tumor growth in animals transplanted with lung lymphoma, pulmonary squamous cell carcinoma, and lung adenocarcinoma (Qian et al., 2007). Another study reported that CXCL10 reduced high-risk HPV (human papillomavirus) oncoproteins E6 and E7 in cervical cancer cells. In addition, CXCL10 suppressed tumor growth factors in immunodeficient mice and reduced the expression of proliferating cell nuclear antigen (PCNA) in tumor tissues (Tokunaga et al., 2018). Vascular endothelial growth factor (VEGF) is known to increase tumor angiogenesis, and estrogen promoted VEGF expression in mammals (Wang et al., 2009). When CXCL10 was injected into female C3H mice, CXCL10 inhibited the effects of estrogen. In this way, when CXCL10 acts as a paracrine substance, it attracts immune cells to tumors and inflammatory sites, promotes the differentiation of immune cells, and enhances the biological functions of these immune cells. Previous studies showed that CXCL10 had a negative effect on cancer treatment when it was an autocrine substance in cancer cells. Cancer cells increase metastasis by activating tumor-derived ligands through autocrine signaling. Autocrine chemokines also influence the activation of Th2 cells, Tregs, and MDSCs, which form the tumor microenvironment (Umansky and Sevko, 2013). Studies to understand the function of CXCR3 in the metastatic process confirmed that lung metastasis was significantly reduced in mice injected with 4T1 cells in which CXCR3 had been removed. This also suggests that CXCR3 plays an important role in promoting tumor metastasis (Zhu et al., 2015). The autocrine CXCL10/CXCR3 loop has pro-

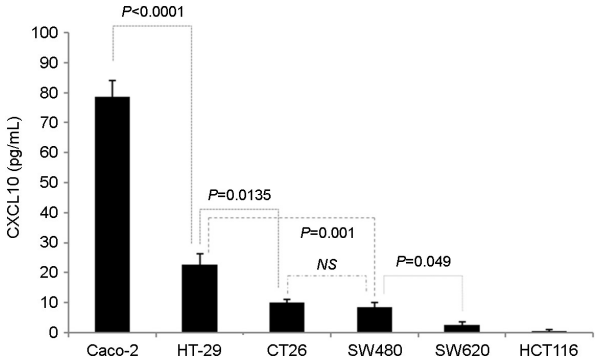

Fig. 1. Comparison of CXCL10 Secretion by Colorectal Cancer Cell Lines. The indicated colorectal cancer cells (2×10^5 cells/mL) were seeded into 35-mm cell culture dishes and incubated at 37℃ for 24 h. The supernatant was recovered to determine the CXCL10 production levels in the cell culture medium.

tumoral effects in adenocarcinoma patients (Wightman et al., 2015; Duruisseaux et al., 2017). Indeed, it has been reported that CXCL10 promotes tumor growth by activating Raf and PI3K signaling pathways in breast cancer cells (Datta et al., 2006). In this way, CXCL10 induces cancer cell proliferation and metastasis, making cancer treatment difficult. In this study, we analyzed the levels of CXCL10 secreted by widely used colorectal cancer cell lines.

Six colorectal cancer cell lines (HT-29, HCT116, CaCo-2, SW620, SW480, and CT26) were purchased from the American Type Culture Collection (ATCC, Manassas, VA, USA). Cells (2×10^5 cells/mL) were seeded into 35-mm cell culture dishes and incubated at 37℃ for 24 h. The supernatant was recovered to determine CXCL10 production levels in the cell culture medium. CXCL10 levels were quantified according to the manufacturer's procedure (R&D Systems, Minneapolis, MN, USA). In brief, cell culture supernatant of each colorectal cancer cells were loaded into a CXCL10 specific capture antibody coated plate and followed by detection antibody, horseradish peroxidase (HRP) enzyme, and 3,3',5,5' Tetramethylbenzidine (TMB) solution step. Between each experimental step, the plate was washed with 0.05% triton x-100 in phosphate-buffered saline (PBS). In this study, the data from at least three independent experiments were analyzed and statistical significance was determined using SPSS ver. 18.0 (SPSS Inc., Chicago, IL, USA). A *P*-value of less than 0.05 was considered significant.

As shown in Fig. 1, among the six colorectal cancer cells, Caco-2 cells showed the highest level of CXCL10 secretion. HT-29 cells showed the second-highest level of CXCL10 secretion, and statistically significantly higher levels of CXCL10 secretion than the remaining SW480, SW620, HCT116, and CT26 cell lines except for CaCo-2 cells. CT26 and SW480 cells showed the next-highest CXCL10 secretion levels, and there was no statistical significance between the levels. Finally, no significantly measurable levels of CXCL-10 secretion were detected in HCT116 cells.

Previous studies reported changes in chemokine production in various pathophysiological situations in human colorectal cancer cell lines. In HT-29 cells, the gene expression of CXCL10 by *Bifidobacterium* infection was increased more than three times compared to the control group (López et al., 2012). However, since that study compared the expression at the mRNA level, the control level of CXCL10 production was not directly measured. SW480 and SW620 are cell lines derived from human colon cancer (Duranton et al., 2003). SW480 is a hypoxic human isogenic non-metastatic colorectal cancer cell originating from a primary tumor. SW620 is a metastatic colorectal cancer cell derived from a metastatic site of the same patient as SW480 cells (Nakurte et al., 2018; Siekmann et al., 2019). The combination of glycolysis, glutamine degradation, and novel fatty acid synthesis inhibitors showed that the expression of nine chemokines was decreased in the human-derived colorectal cancer SW480 cell line [interleukin-9, C-X-C motif chemokine ligand (CXCL) 10, eotaxin, chemokine ligand (CCL) 9, CXCL5, CCL20, CXCL1, CXCL11, and CCCL4] (Schcolnik-Cabrera et al., 2019). The basal level of CXCL-10 production in control SW480 cells was reported to be 250 pg/mL or more, and CXCL10 was secreted into the cell supernatant at a much higher level than in the experiments in this study. This difference is considered to be a difference in the experimental conditions, such as the number of cells cultured for the experiment and the size of the culture dishes. However, the above report and this study detected a measurable level of CXCL10 secreted from the control SW480 cell line. In addition, it has been reported that TNFα, a pro-inflammatory cytokine, increased CXCL10 gene expression and CXCL10 production in SW480, SW620, and HCT116

colon cancer cell lines (Shin et al., 2011; Wang et al., 2021). The expression level of CXCL10 in tumor tissue was also reported to be a factor predicting tumor prognosis (Jiang et al., 2010). Caco-2 cell lines were isolated from colorectal cancer but showed the morphological and biochemical properties of small intestine cells upon differentiation (Hidalgo et al., 1989). The Caco-2 cell line is a cell line that induces tumor cell-induced platelet aggregation (Medina et al., 2006; Zarà et al., 2018). In addition, the production of CXCL10 by Caco-2 cells was increased by SARS-CoV-2 (CoV-2) infection (Poeta et al., 2021). Finally, CT26 cells are undifferentiated tumor cells induced by N-nitroso-N-methylurethane-(NNMU) in BALB/c mice and have the form of fibroblasts. *In vivo*, CT-26 cells have high migration and invasion rates, unlike *in vitro* (de Both et al., 1999). SW480 and SW620 are cell lines derived from human colon cancer (Duranton et al., 2003). It was reported that liver metastasis was reduced when CXCL10 was overexpressed in CT26 cells (Kikuchi et al., 2019). In addition, tumor cell growth was inhibited in CT26-transplanted mice by treatment with CXCL10 alone or in combination with CXCL11 (Wang et al., 2010). Summarizing the above, the regulation of the generation of CXCL10 secreted by tumor cells or their surrounding environment can be an important target for anticancer therapy. Accordingly, it is very important to comprehensively measure the levels of CXCL10 secreted by tumor cells. However, although there have been observations of CXCL10 expression or secretion in various pathophysiological conditions in each cell line, there are no data comparing the levels of CXCL10 secreted level by each colorectal cancer cell line in one study, as was done here. Some direct comparisons of the degree of malignancy of the tumor cells used in this study have been made. In an experiment comparing the sphere-forming ability of HCT116 and HT-29 cell lines, the HCT116 cell line formed relatively larger spheres than the HT-29 cell line (Olejniczak et al., 2018). In an experiment using an orthotopic mouse model, the HCT116 cell line showed higher rate of liver metastasis than the HT-29 cell line (Xu et al., 2020).

The results of this study will be helpful in investigating the molecular basis of colorectal cancer pathogenesis, congenital and acquired drug resistance, and the search for new therapeutic modalities for this malignancy. In the future, we plan to investigate the secreted levels of CXCL9 and CXCL11, which share CXCR3 receptors with CXCL10, and changes in CXCL10 expression in tumor cells in various pathophysiological experimental models.

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CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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