Erratum

Erratum to “Ethanol Extracts of Fruiting Bodies of Antrodia cinnamomea Suppress CL1-5 Human Lung Adenocarcinoma Cells Migration by Inhibiting Matrix Metalloproteinase-2/9 through ERK, JNK, p38, and PI3K/Akt Signaling Pathways”

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(1) Would you please change the Abstract section with this following?

Metastatic cancer attributes to a major cause of cancer death. In this pioneer study, we aimed to investigate how Antrodia cinnamomea (A. cinnamomea), indigenous to Taiwan, affects migration ability of highly metastatic human adenocarcinoma lung cancer cells CL1-5. Our result demonstrated that noncytotoxic ethanol extract of fruiting bodies of A. cinnamomea (EEAC) exhibited a dose-dependent inhibitory effect on motility and migration of the highly metastatic CL1-5 cells. Results of a gelatin zymography assay illustrated that A. cinnamomea repressed the activities of matrix metalloproteinase- (MMP-) 2 and 9 in a dose-dependent manner. A. cinnamomea administration decreased MMP-9 and MMP-2 protein expressions from Western blotting assay, whereas the expression of the tissue inhibitors of MMP (TIMP-1 and TIMP-2) increased. Additional study disclosed that A. cinnamomea suppressed FAK, ERK1/2, p38, AKT, and JNK1/2 phosphorylation, and also PI3K and Rac-1 were found decreased. Further, treatment of CL1-5 cells with inhibitors specific for PI3K (LY294002), ERK1/2 (PD98059), JNK (SP600125), and p38 MAPK (SB203580) decreased the expression of MMP-2 and MMP-9. Taken together, EEAC induced FAK phosphorylation and exhibited its antimigration activities via the PI3K/AKT and MAPK signalings in CL1-5 cells. This is the pioneer study verifying the antimigration activity of A. cinnamomea against human lung adenocarcinoma CL1-5 cancer cells.

(2) Would you please change the Introduction section with this following?

Lung cancer develops as the number one cancer cause of death in several countries, and it accounts for more fatalities than total of prostate cancer, breast cancer, and colorectal cancer [1]. Human lung adenocarcinoma cell lines CL1-0, L1-1, CL1-5, and CL1-5-F4 are a series of sublines with progressively invasive ability established by in vitro invasion screening. CL1-5 cells are a human lung adenocarcinoma cell line derived from the parental CL1 cells by five successive matrigel selections. CL1-5 cells showed a 4- to 6-fold higher invasive ability than the parental cells, and their production of 92-kDa MMP-9 also exhibited a drastic increase over that of their parental cells [2].

Overgrowth and metastasis are the two major characteristics of malignancy with poor clinical outcome. Malignant tumor progression depends upon the capacity to invade,
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3.2. EEAC Inhibited Cell Mobility with Wound Healing Assay

Wound healing assay was conducted to study the effect of EEAC on the mobility/migration of CL1-5 cells. Under wound healing assay, the confluent monolayer was scraped with a sterile micropipet tip to create a scratch wound. Our result demonstrated that EEAC inhibited the migration of CL1-5 cells in a dose-dependent manner, with 21.85% and 32.03% inhibition at 0.5 and 1.0 µg/mL after incubation for 36 h, respectively (Figures 2(a) and 2(b)).

(4) I would like to replace Figure 2(a) with the new one shown earlier.