Cellular Physiology	Cell Physiol Biochem 2021;55:235-237	
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Erratum

In the original article by Liao, et al., entitled "CXCR4 Accelerates Osteoclastogenesis Induced by Non-Small Cell Lung Carcinoma Cells Through Self-Potentiation and VCAM1 Secretion" [Cell Physiol Biochem 2018;50(3):1084-1099, DOI: 10.1159/000494533], due to unclear file naming, incorrect representative images were included in Fig. 3C 24hr Control, Fig. 3C 48hr Control, Fig. 3E panel 2, Fig. 4B GAPDH, Fig. 4E panel 2, Fig. 5A panels 2 and 4, Fig. 5D panel 2, Fig. 5G panel 3.

The authors confirm that all of the results and conclusions of the article remain unchanged, as well as the figure legends.

The corrected Figure 3, 4 and 5 can be found below.

Fig. 3. Effects of CXCR4 downregulation on the proliferation, migration and invasive ability in H1299 cell lines. A. CXCR4 was knocked down via siRNA transfection. The mRNA levels were measured with qRT-PCR (n=3, *p<0.05, compared with the control group or siRNA negative control group; NS, not significant). B. The CXCR4 protein levels were significantly decreased in the group transfected with siR-NA against CXCR4 compared with that in the negative control group (n=3, *p<0.05, compared with the siRNA negative control group). C. The migration ability of H1299 cells in the presence or absence of siRNA CXCR4 was evaluated via the wound-healing assay. Representative images were recorded at 0, 24 and 48 h post-wound time points. D. Quantification of healing distance (n=3, *p<0.05, compared with the control group or siRNA negative control group). E. Matrigel assay of H1299 cells transfected with siRNA against CXCR4 or control siRNA. Migratory cells were counted in 10 non-overlapping areas and quantified using a bar graph (n=3, *p<0.05, compared with the control group or siRNA negative control group). F. H1299 cell proliferation was measured using a CCK-8 kit. The OD values



were measured at 24, 48 and 72 h in the control group and CXCR4-knockdown group (n=5, *p<0.05, compared with the siRNA negative control group).

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Fig. 4. Effects of CXCR4 overexpression on the proliferation, migration and invasive ability in H1975 cell lines. A. CXCR4 was overexpressed in H1975 cells via an adenovirus carrying CXCR4. The mRNA levels were measured using qRT-PCR (n=3, [#]p<0.01 compared with negative control adenovirus transfection group). B. The CXCR4 protein levels were measured with Western blot in H1975 cells transfected with a CXCR4-carrying adenovirus (n=3, [#]p<0.01 compared with the negative control adenovirus transfection group). C. The migration ability of H1975 cells in the presence or absence of the CXCR4 adenovirus were measured with the wound-healing assay. Representative images were recorded at 0, 24 and 48 h post-wound time points. D. Quantification of healing distance (n=3, *p<0.05, compared with the adenovirus negative control group). E. Matrigel invasive assay of H1975 transfected with the CXCR4 adenovirus or control vector. Migratory cells were counted in 10 non-overlapping areas and quantified as shown in the bar graph (each group n=3, with 10 visions, *p<0.05, compared with the negative control adenovirus transfection group). F. H1975 cell proliferation was measured using a CCK-8 kit. The OD values were measured at 24, 48 and 72 h in the



control group and the CXCR4 adenovirus transfection group (n=3, *p<0.01 compared with the negative control adenovirus transfection group).

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Fig. 5. Effects of CXCR4 manipulation in NSCLC on osteoclast progenitor cell differentiation. A. TRAP staining was performed to evaluate the formation of multinucleated cells. RAW264.7 cells were treated with 10 ng/mL RNAKL with or without H1299 culture medium (CM) harvested from the CXCR4-knockdown group or the control transfection group. B. Ouantification of TRAP-positive cells (n=3, *p<0.05 compared with the negative control siRNA group). C. The mRNA levels of the osteoclast differentiation marker genes cathepsin K and TRAP in treated RAW264.7 cells were measured via RT-PCR at 48 h after treatment (n=3, *p<0.05 compared with the negative control siRNA group). D. TRAP staining of osteoclast progenitor cells when CXCR4 was overexpressed. RAW264.7 cells were treated with 10 ng/mL RNAKL with or without H1975 CM harvested from the CXCR4-overexpression group compared with the adenovirus or control group. E. Quantification of TRAP-positive cells upon stimulation with cell culture media from CXCR4-overexpressing H1975 (n=3, *p<0.05 compared with the adenovirus negative control group). F. mRNA levels of the osteoclast differentiation marker genes cathepsin K and TRAP in treated RAW264.7 cells were measured via RT-PCR at 48 h after treatment (n=3, [#]p<0.01 compared with the negative control adenovirus transfection group). G. TRAP staining was performed to detect RAW264.7 cell differentiation following treatment with 10 ng/mL RNAKL and culture medium from CXCR4-overexpressing H1975 cells in the presence or absence of 10 ng/ml AMD3100, as indicated. H. Quantification of TRAP-positive cells upon stimulation with culture media from CXCR4-overexpressing H1975 cells in the presence or absence of AMD3100 (n=3, *p<0.05). I. mRNA levels of osteoclast differentiation marker genes cathepsin K and TRAP in treated RAW264.7 cells were measured via RT-PCR at 48 h in the indicated groups (n=3, *p<0.05).

