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Author Correction: CXCR7 ameliorates myocardial infarction as a β -arrestin-biased receptor

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This Article contains an error in Figure 3F, where the x-axis labels ‘CM’ and ‘FB’ were reversed.

Furthermore, the legend of Figure 3,

“ERK is activated through CXCR7 in cardiomyocytes. (a) β -Arrestin recruitment assay of CXCR7 showing that CXCL12 and TC14012 induce coupling of CXCR7 with β -arrestin in a dose-dependent manner (EC_{50} : 14.8 nM and 47.4 nM, respectively). Replicate samples are derived from independent HEK293 cells ($n = 3$). Data are shown as the mean \pm SEM. (b) Immunoblot analysis of phosphorylated ERK (pERK) and total ERK (tERK) in HEK293 cells transfected with a CXCR7 expression plasmid at various time points after stimulation with CXCL12 (100 nM). (c) Quantitative data of the results shown in (b). $n = 4$. Data are shown as the mean \pm SEM. Significance was calculated by ANOVA followed by the Bonferroni procedure; $***P < 0.001$. Note that ERK was activated upon stimulation with CXCL12, and activity peaked at 5 min. (d) Immunoblot analysis of pERK/tERK in HEK293 cells transfected with various amounts of a CXCR7 expression plasmid with CXCL12 (100 nM) or vehicle (veh). Note that ERK was activated in a CXCR7 expression plasmid-dose-dependent manner under stable CXCL12 stimulation. (e) Quantitative data of the results shown in (d). $n = 3$. Data are shown as the mean \pm SEM. Significance was calculated by one-way analysis of variance (ANOVA) followed by the Bonferroni procedure; $*P < 0.05$, $**P < 0.01$, $***P < 0.001$. (f) *Cxcr7* mRNA expression in cardiomyocytes and fibroblasts from primary culture of neonatal rat hearts. Data are shown as the mean \pm SEM. Significance was calculated by an unpaired *t*-test. $**P < 0.01$. (g) Immunoblot analysis of pERK and tERK in primary culture of NRCMs at various time points upon stimulation with the CXCR7-specific agonist, TC14012. (h) Quantitative data of the results shown in (e). $n = 3$. Data are shown as the mean \pm SEM. Significance was calculated by ANOVA followed by the Bonferroni procedure; $**P < 0.01$, $***P < 0.001$.”

should read:

“ERK is activated through CXCR7 in cardiomyocytes. (a) β -Arrestin recruitment assay of CXCR7 showing that CXCL12 and TC14012 induce coupling of CXCR7 with β -arrestin in a dose-dependent manner (EC_{50} : 14.8 nM and 47.4 nM, respectively). Replicate samples are derived from independent HEK293 cells ($n = 3$). Data are shown as the mean \pm SEM. (b) Immunoblot analysis of phosphorylated ERK (pERK) and total ERK (tERK) in HEK293 cells transfected with a CXCR7 expression plasmid at various time points after stimulation with CXCL12 (100 nM). (c) Quantitative data of the results shown in (b). $n = 4$. Data are shown as the mean \pm SEM. Significance was calculated by ANOVA followed by the Bonferroni procedure; $***P < 0.001$. Note that ERK was activated upon stimulation with CXCL12, and activity peaked at 5 min. (d) Immunoblot analysis of pERK/tERK in HEK293 cells transfected with various amounts of a CXCR7 expression plasmid with CXCL12 (100 nM) or vehicle (veh). Note that ERK was activated in a CXCR7 expression plasmid-dose-dependent manner under stable CXCL12 stimulation. (e) Quantitative data of the results shown in (d). $n = 3$. Data are shown as the mean \pm SEM. Significance was calculated by one-way analysis of variance (ANOVA) followed by the Bonferroni procedure; $*P < 0.05$, $**P < 0.01$, $***P < 0.001$. (f) *Cxcr7* mRNA expression in cardiomyocytes and fibroblasts from primary culture of neonatal rat hearts. Data are shown as the mean \pm SEM. Significance was calculated by an unpaired *t*-test. $**P < 0.01$. (g) Immunoblot analysis of pERK and tERK in primary culture of NRCMs at various time points upon stimulation with the CXCR7-specific agonist, TC14012. (h) Quantitative data of the results shown in (g).

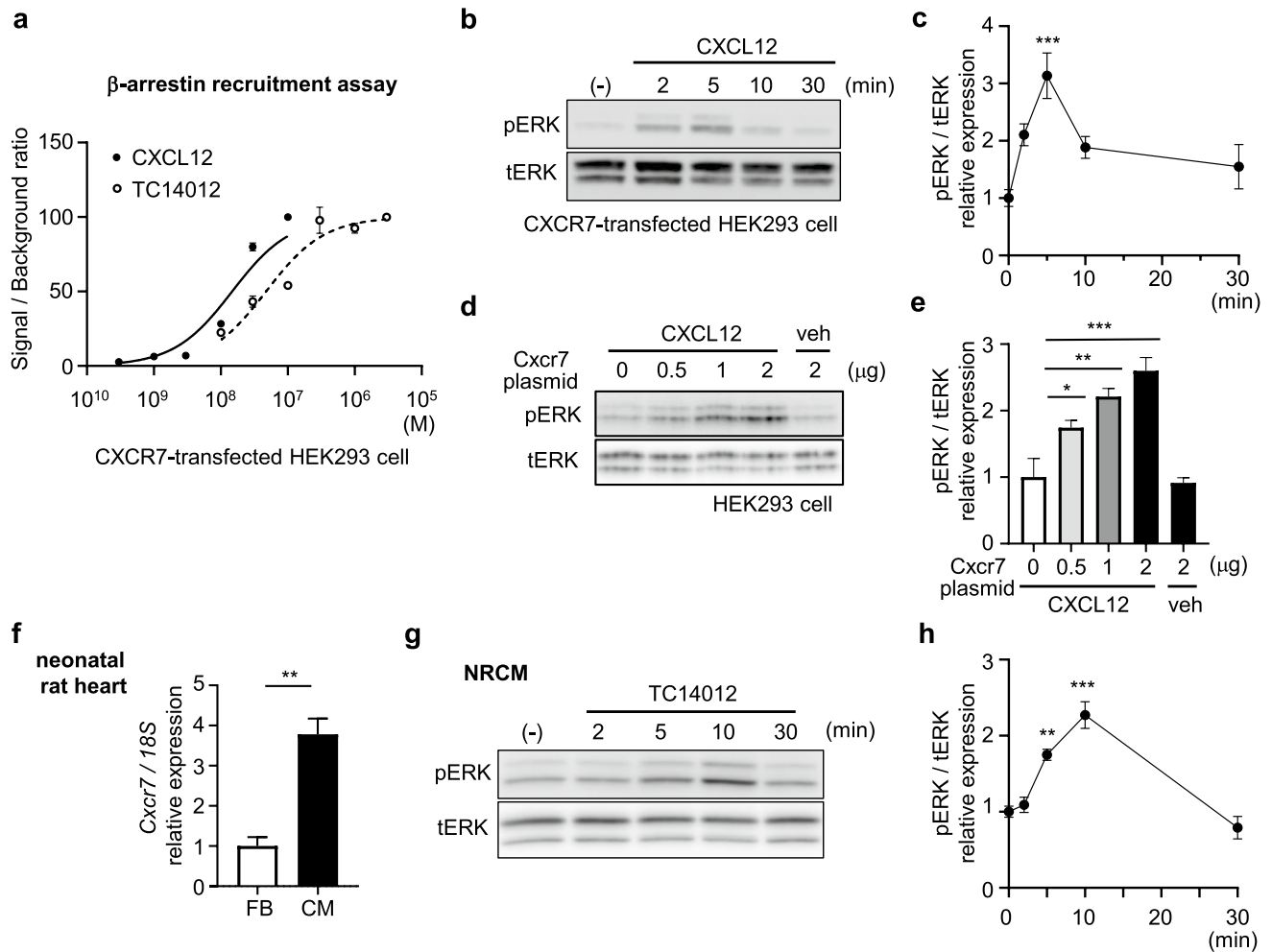


Figure 1. ERK is activated through CXCR7 in cardiomyocytes. **(a)** β -Arrestin recruitment assay of CXCR7 showing that CXCL12 and TC14012 induce coupling of CXCR7 with β -arrestin in a dose-dependent manner (EC_{50} : 14.8 nM and 47.4 nM, respectively). Replicate samples are derived from independent HEK293 cells ($n = 3$). Data are shown as the mean \pm SEM. **(b)** Immunoblot analysis of phosphorylated ERK (pERK) and total ERK (tERK) in HEK293 cells transfected with a CXCR7 expression plasmid at various time points after stimulation with CXCL12 (100 nM). **(c)** Quantitative data of the results shown in **(b)**. $n = 4$. Data are shown as the mean \pm SEM. Significance was calculated by ANOVA followed by the Bonferroni procedure; $***P < 0.001$. Note that ERK was activated upon stimulation with CXCL12, and activity peaked at 5 min. **(d)** Immunoblot analysis of pERK/tERK in HEK293 cells transfected with various amounts of a CXCR7 expression plasmid with CXCL12 (100 nM) or vehicle (veh). Note that ERK was activated in a CXCR7 expression plasmid-dose-dependent manner under stable CXCL12 stimulation. **(e)** Quantitative data of the results shown in **(d)**. $n = 3$. Data are shown as the mean \pm SEM. Significance was calculated by one-way analysis of variance (ANOVA) followed by the Bonferroni procedure; $*P < 0.05$, $**P < 0.01$, $***P < 0.001$. **(f)** *Cxcr7* mRNA expression in cardiomyocytes and fibroblasts from primary culture of neonatal rat hearts. Data are shown as the mean \pm SEM. Significance was calculated by an unpaired *t*-test. $**P < 0.01$. **(g)** Immunoblot analysis of pERK and tERK in primary culture of NRCMs at various time points upon stimulation with the CXCR7-specific agonist, TC14012. **(h)** Quantitative data of the results shown in **(g)**. $n = 3$. Data are shown as the mean \pm SEM. Significance was calculated by ANOVA followed by the Bonferroni procedure; $**P < 0.01$, $***P < 0.001$.

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The correct Figure 3 and accompanying legend appear below as Figure 1.



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