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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 ± 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 ± 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 ± 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 ± 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 ± 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 ± 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 ± 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 ± 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 ± 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**).

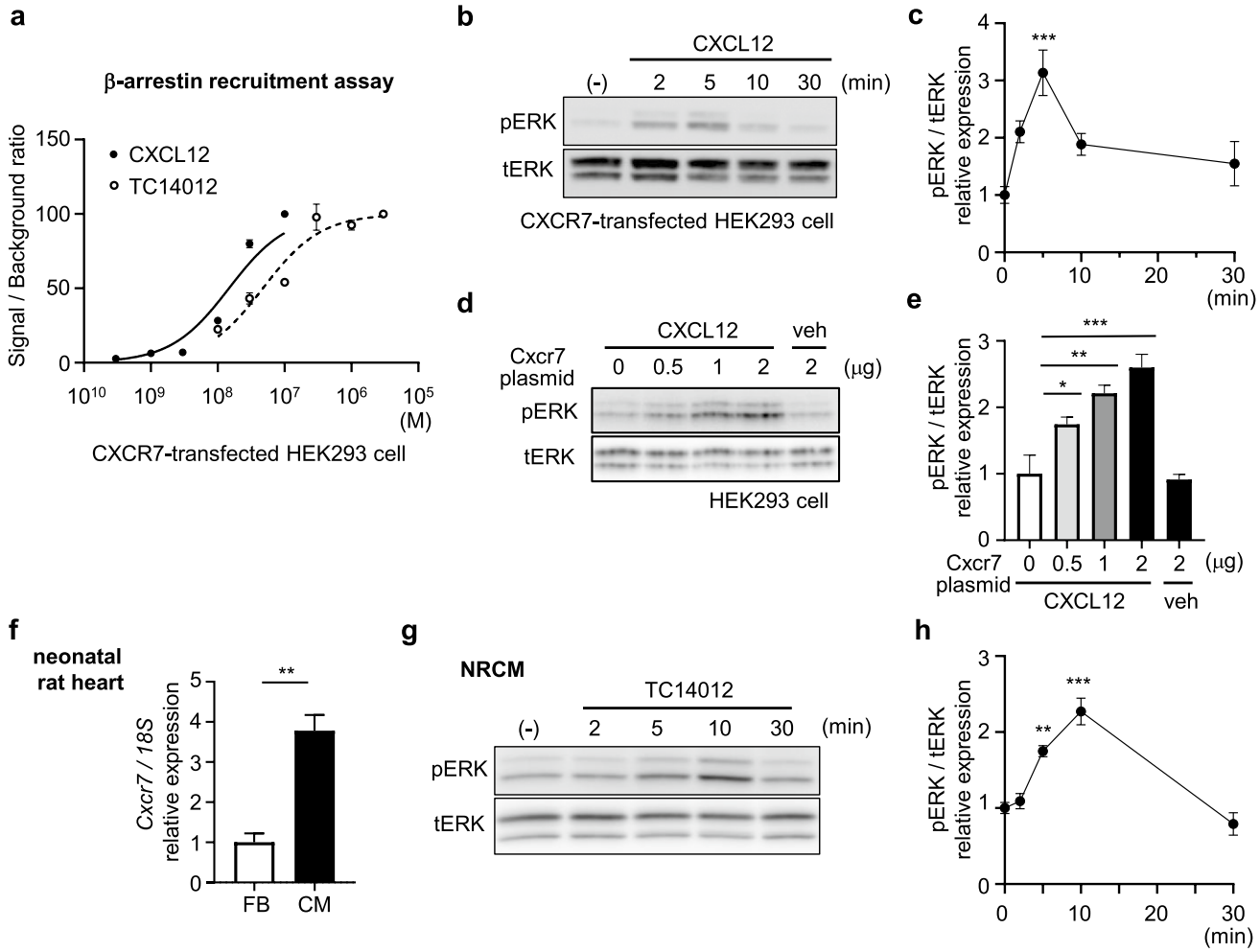

Figure 1. ERK is activated through CXCR7 in cardiomyocytes. (**a**) β-Arrestin recruitment assay of CXCR7 showing that CXCL12 and TC14012 induce coupling of CXCR7 with β-arrestin in a dose-dependent manner (EC_{50}: 14.8 nM and 47.4 nM, respectively). Replicate samples are derived from independent HEK293 cells (n = 3). Data are shown as the mean ± 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 ± 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 ± 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 ± 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 ± 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.