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Data Article

Data on human neutrophil activation induced by pepducins with amino acid sequences derived from β2AR and CXCR4

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ABSTRACT

The data described here is related to the research article titled (Gabl et al., 2016) [1]. Pepducins with peptide sequence derived from one of the intracellular domains of a given G-protein coupled receptor (GPCR) can either activate or inhibit cell functions. Here we include data on human neutrophil function induced by pepducins derived from β2AR (ICL3-8) and CXCR4 (ATI-2341), respectively. ICL3-8 exerts neither direct activating effect on the NADPH-oxidase as measured by superoxide release nor inhibitory effect on FPR signaling. ATI-2341 dose-dependently triggers neutrophil activation and these cells were subsequently desensitized in their response to FPR2 specific agonists $F2Pal_{10}$ and WKYMVM. Moreover, the ATI-2341 response is inhibited by PBP_{10} and the peptidomimetic Pam-(Lys-betaNSpe)6-NH2 (both are FPR2 specific inhibitors), but not to the FPR1 specific inhibitor cyclosporine H.

Specification Table

Subject area	*Biology*
More specific subject area	*G-protein coupled receptor signaling*

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Type of data	*Figures*
How was data acquired	*Isoluminol amplified chemiluminescence; luminometer*
Data format	*Processed*
Experimental factors	*Isolated human neutrophils were activated by pepducin or receptor specific agonists and the responses induced were determined*
Experimental features	*The effects of receptor specific inhibitors and desensitization profiles were determined*
Data source location	*Gothenburg, Sweden*
Data accessibility	*Data are within this article*

Value of the data

- Receptor specific neutrophil responses can be determined by the sensitive assay to measure superoxide production.
- The precise receptor involved can be identified by the desensitization profile and by the use of defined receptor specific antagonists.
- The data provide insights into the highly variable effects of pepducins which includes receptor hijacking.

1. Data

Data describes human neutrophil activation, measured by isoluminol-enhanced chemiluminescence systems, with two pepducins derived from β2AR (ICL3-8) and CXCR4 (ATI-2341), respectively. Direct neutrophil activation by ICL3-8 and its modulatory effect on FPR signaling are shown (Fig. 1). In addition, data on dose-dependent neutrophil activation induced by ATI-2341 and the effects on this response of FPR specific agonists as well as antagonists are provided (Fig. 2).

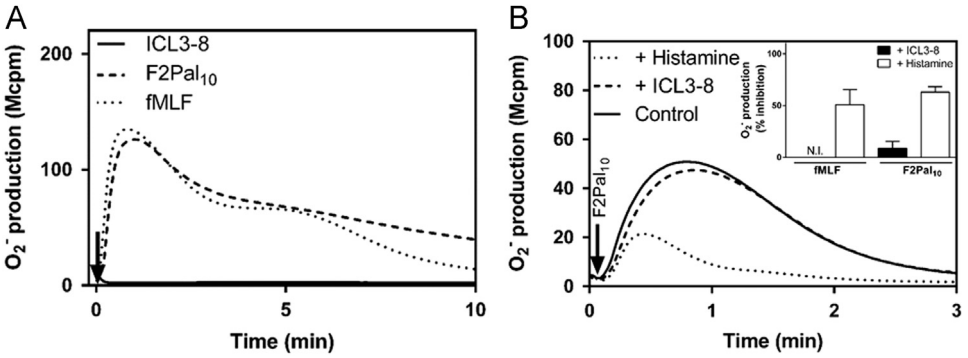

Fig. 1. No direct activating or inhibitory effect on the FPR-mediated NADPH-oxidase response is induced by the Gs modulating pepducin ICL3-8. A) Human neutrophils (10^5 cells) were pre-incubated with latrunculin A (25 ng/ml) at 37 °C for 5 min before the addition of the Gs modulating pepducin ICL3-8 (1 μM, solid line), the FPR1 specific agonist fMLF (100 nM, dotted line) and the FPR2 specific agonist $F2Pal_{10}$ (1 μM, dashed line). Superoxide production was continuously measured by isoluminol-amplified chemiluminescence. The inset shows the response induced by the ICL3-8 pepducin with higher resolution. Representative curves out of at least five independent experiments using individual blood donors are shown. The arrows indicate the time points for addition of stimuli. B) Human neutrophils (10^5 cells) were pre-treated with either ICL3-8 (1 μM) or histamine (10 μM) at 37 °C for 5 min before activation with $F2Pal_{10}$ (1 μM; main figure and inset) or fMLF (100 nM; only inset). In the main figure, representative curves out of at least five independent experiments are shown. The arrow marks the time point for addition of $F2Pal_{10}$. Inset: the inhibitory effect of ICL3-8 (black bars) and histamine (white bars) on FPR-mediated superoxide production (induced by the FPR1 agonist fMLF or the FPR2 agonist $F2Pal_{10}$) expressed as percent inhibition compared to the activity induced in cells incubated without any inhibitor (mean ± SD, $n=4$). N.I. equals no inhibition.