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ACRs (atypical chemokine receptors) were initially referred to as 'silent' receptors on the basis of a lack of signalling and functional activities that are typically observed with conventional chemokine receptors. Although ACRs do not directly induce cell migration, they indirectly control leucocyte recruitment by shaping chemokine gradients in tissues through degradation, transcytosis or local concentration of their cognate ligands. Recent evidence also suggests that these biological activities are supported by G-protein-independent, β-arrestin-dependent signalling events. In the present article, we review current knowledge on structural and signalling properties of ACRs that are changing our view on this entire class of receptors from silent to endogenous β-arrestin-biased signalling receptors.