Erratum: Distinct loops in arrestin differentially regulate ligand binding within the GPCR opsin

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In this Article, there are several instances where *all-trans*-retinal is incorrectly referred to as *all-trans*-retinol and *all-trans*-retinol is incorrectly abbreviated to ATR. The following sentences, and the revised figure below, are correct.

In the abstract: Here we show that rod arrestin induces uptake of the agonist *all-trans*-retinal in only half the population of phosphorylated opsin in the native membrane.

In the abstract: Such a mechanism would protect the rod cell in bright light by concurrently sequestering toxic *all-trans*-retinal and allowing regeneration with 11-*cis*-retinal.

In the Results: Incidentally, both *all-trans*-retinol and beta-ionone also induced arrestin binding to OpsP with an apparent $K_d \sim 40$ times higher than that for ATR (Fig. 3e).

On the x axis of Fig. 3a,c: *All-trans*-retinal.

In the legend to Fig. 3e: *All-trans*-retinol and beta-ionone were titrated against 4 μ M arrestin + 4 μ M OpsP (100% phosphorylated) in isotonic buffer, and arrestin binding was measured by pull down.

In the legend to Fig. 4: *All-trans*-retinol formation after addition of NADPH to samples of OpsP (2 μ M) and ATR (4 μ M) was monitored in the absence (dark red) or presence of 2 μ M unlabelled arrestin (red). Experiments were performed in 50 mM HEPES buffer pH 7 without salt, 35 °C.

In the legend to Table 1: ATR, *all-trans*-retinal; NA, not applicable; OpsP, phosphorylated opsin.

The correct version of Fig. 3 and its legend follows.

Figure 3 | Arrestin and retinoid titrations. (a) ATR was titrated against 4 μ M arrestin + 4 μ M OpsP. (b) Arrestin was titrated against 4 μ M OpsP + 40 μ M ATR. Arrestin binding (red), retinal Schiff base formation (blue) and blocked rhodopsin regeneration (green) were measured as described in the text. All experiments were performed in HEPES buffer pH 7 without salt, except for the pull-down experiment in (a) (red), where isotonic buffer was necessary to reduce agonist-independent arrestin binding. For the regeneration data (green), the fitted curves intercept the y axis at ~ 0.5 , reflecting the fact that not all OpsP was regenerated owing to experimental constraints (Methods). Both single-site (solid traces) and two-site (dashed traces) binding models were fit to the retinal titration data. (c) ATR was titrated against 4 μ M arrestin + 4 μ M OpsP using an OpsP preparation in which $\sim 70\%$ of receptors were able to bind arrestin as light-induced Meta II-P. The fitted curve reports an apparent K_d of 3.4 μ M and a stoichiometry of one arrestin per four receptors. Inset—time course of regeneration of OpsP (4 μ M) with 11-*cis*-retinal (20 μ M) in the absence or presence of arrestin (4 μ M) and ATR (40 μ M). (d) Arrestin titrations using mutants A366NBD (black symbols) or S344NBD (grey symbols) were performed against 4 μ M Meta II-P or 4 μ M OpsP + 40 μ M ATR using the same under-phosphorylated receptors as in (c). The fitted curves report that one arrestin bound for every 1.8 Meta II-P or 3.6 μ M OpsP/ATR (200 nM $< K_d < 300$ nM). For both (c) and (d), arrestin binding in isotonic buffer was measured by pull down. (e) ATR and beta-ionone were titrated against 4 μ M arrestin + 4 μ M OpsP (100% phosphorylated) in isotonic buffer, and arrestin binding was measured by pull down. The ATR data and fits from panel (a) are shown for reference. Note that beta-ionone caused scattering artefacts at higher concentrations. For all panels, data points from independent experiments are represented by differently shaped symbols, and the combined data points were used to fit the binding curves described in the Methods.