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Spatial Negative Priming, but Not Inhibition of Return, with Central (Foveal) Displays

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ABSTRACT

The view persists that the inhibition of return (IOR) and the spatial negative priming (SNP) phenomena may be produced by a common "orientation inhibition" mechanism (e.g., Christie & Klein, 2001), held to arise during the processing of peripherally delivered (parafoveal) visual events. Both IOR and SNP effects are present when responding to recently to-be-ignored distractor events is delayed. Since an SNP effect has been produced using centrally located distracters (visual angle of about 2.5? or less), a common mechanism view would require that these locations generate orientation inhibition, which then cause of the SNP effect. We report past results and an experiment that reject the common mechanism view. Subjects completed four tasks; two, 1-response tasks, using either central (Task 1) or peripheral (Task 2: IOR) event locations, and two, 4-response tasks, again, using central (Task 3: SNP-central) or peripheral (Task 4: SNP-peripheral) locations. Trials occurred in pairs; first the prime (a target or a distractor), then the probe (target only). Critically, neither distractor- nor target-occupied prime locations produced either inhibitory (SNP effect) or positive after-effects, respectively, in Task 1. Seemingly, centrally located events do not generate orientation inhibition and so, unlike the IOR effect, this inhibition does not cause the SNP-central phenomenon.

KEYWORDS

Centrally Positioned Events; Orientation Inhibition; Spatial Negative Priming

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