University of California, Irvine Statistics Seminar

Uncertainty & Invariance in Causal Inference

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A critical concept in causation is that of *invariance* – the extent to which a putative cause acts "the same" across the many compounds and contexts in which it occurs. The notion of invariance is central to the reproducibility of scientific results, as well as to the generalization of human causal knowledge from learning contexts to novel situations. For example, a pharmaceutical drug found to reduce hypertension in adult males may, or may not, have a different effect on females, or on male adolescents, or may behave differently when combined with a different release mechanism - implications for clinical, commercial and personal decision making are profound. Notably, psychological theories of human causal induction differ on how invariance is defined, as well as on whether occlusion of invariance by confounding precludes causal discovery. In particular, causal invariance may be defined with respect to *unobservable* causal influences, formalized as their noisy-logical integration, or on the observable, additive, difference made to the state of the effect. Moreover, while Bayesian models of human causal cognition predict that occlusion of invariance by confounding will generate high levels of uncertainty in judgments, associative, error-driven, models do not. I will discuss a series of behavioral and neuroimaging studies aimed at assessing how naïve human reasoners define independent causal influence, and how deviations from the independent influence and independent occurrence of putative causes modulate uncertainty in causal inferences. I will show that, when asked to make judgments about the influences of a set of fictitious putative causes, reasoners predominantly adopt a noisy-logical definition of independent influence, and report high levels of uncertainty for both interacting and confounded causes. At the neural level, activity in dissociable substrates scales with Bayesian and associative uncertainty signals, respectively. I will argue, based on these results, that human reasoners make tacit assumptions that align with normative accounts of causal induction and basic principles of scientific inference.