The Consortium on Interplay of Genes and Environment Across Multiple Studies (IGEMS): Does Sleep Duration Moderate Genetic and Environmental Contributions to Cognitive Performance?

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The present study tested whether sleep duration moderates the genetic and environmental variances of cognitive performance using twin samples from the Interplay of Genes and Environment Across Multiple Studies (IGEMS) consortium spanning mid- to late-life: Mage = 57.6, range 27 to 91 years, N = 7052, Female = 43.70%, 1525 complete MZ pairs, 2001 complete DZ pairs. Measures included sleep duration, and four cognitive tests tapping verbal fluency, spatial reasoning, processing speed, and episodic memory (i.e., Animal Naming, Block Design, Symbol Digit, and Word List). Phenotypically, the correlations between sleep duration and cognitive performance were modest (r = -.01 to -.10). Twin correlations for sleep duration were rMZ = .36, rDZ = .21. Extended univariate ACE moderation models were fitted with sleep duration as the primary moderator, accounting for age moderation. Model comparisons suggested significant overall moderation of ACE components for fluency by sleep duration and AC components for memory, but no significant moderation of ACE components by sleep for speed. Results were inconclusive for spatial reasoning. For fluency, as sleep duration increased, genetic variance (a²) in cognition decreased (a²4hours = .45, a²10hours = .19) and shared common environment (c²) showed a slight U-shaped pattern (c²4hours = .12, c²7hours = .001, c²10hours = .11). For memory, as sleep duration increased, a² decreased (a²4hours = .35, a²10hours = .003) and c² increased (c²4hours = .003, c²7hours = .37). For speed, as sleep duration increased, a² decreased (a²4hours = .63, a²10hours = .26) and c² increased (c²4hours = .01, c²10hours = .37). Patterns of unique environmental contributions (e²) differed. Notably, genetic variance was highest at short sleep, thus aligning with current literature regarding the association between short sleep and ineffective beta-amyloid clearance which may point to mechanisms underlying poor cognitive performance.

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