null distributions of influence terms (F-values) and their differences (22). A causal connectivity graph was constructed using the thickness of connecting arrows to indicate the strengths of the causal influences (Fig. 3A, “raw” F-values normalized by the maximum F-value; raw F-values reported in Table S4). Only links that showed significant directed connectivity (influence terms) at the group-level (Mann-Whitney U test, $P < 0.01$; Bonferroni corrected for multiple comparisons) are shown (gray arrows, Fig. 3A); a subset of these links that showed a dominant directional influence (difference of influence terms) are highlighted in red in the same figure (Mann-Whitney U test, $P < 0.05$).

Fig. 2. Onset latencies of the event-related responses in the six key nodes of the SN (blue bars), CEN (green bars) and DMN (yellow bars) in the auditory event segmentation task. The rFIC onset significantly earlier than each of the nodes in the CEN and DMN (two-sample t-test, $q < 0.05$, indicated by *), FDR corrected for multiple comparisons. Error bars denote standard error of the mean (SEM) across subjects.

predictability of signal changes in one brain region based on the time-course of responses in another brain region (28). We performed GCA using a bivariate model (22) on the time-courses extracted from the six key regions used in the onset latency analysis. We used bootstrap techniques (29) to create