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 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 strength 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, FDR corrected links shown in Table S4) (see SI Materials and Methods for details). GCA on the time-courses extracted from the key regions revealed statistically significant direct or indirect causal influences from the rFIC to all of the regions in the CEN and DMN (Fig. 3A). To quantify the causal interactions of each node of the network, we performed network analyses on key graph metrics (see Materials and Methods), and constructed a distribution of these metrics, across subjects (for each node). Network analysis on the causal flow network identified with GCA revealed that the rFIC had the highest number of causal outflow connections (out-degree), the lowest number of causal inflow connections (in-degree), and the shortest path length among all regions (means and standard errors of these metrics are reported in Table S5A). The rFIC also had a significantly higher net causal outflow (out-in degree) than all of the nodes of the CEN and DMN (two-sample t test, P < 0.05). Differences in (out-in) degree between the rFIC and the rDLPC, rPPC, and PCC remained significant after FDR correction for multiple comparisons (q < 0.05) (Fig. 4A). Similarly, the rFIC had a significantly

![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 errors of the mean (SEM) across subjects.](image)

![Fig. 3. Granger causality analysis (GCA) of the six key nodes of the Salience (blue nodes), Central-Executive (green nodes) and Default-Mode (yellow nodes) networks during (A) auditory event segmentation, (B) visual oddball attention task, and (C) task-free resting state. GCA revealed significant causal outflow from the rFIC across tasks and stimulus modalities. In each subfigure, the thickness of the connecting arrows between two regions corresponds to the strength of directed connection (F-value) normalized by the maximum F-value between any pair of regions for that task ("raw" F-values reported in Table S4). Only links that showed significant directed connectivity at the group-level (Mann-Whitney U test, P < 0.01; Bonferroni corrected for multiple comparisons) are shown (gray arrows); a subset of these links that showed a dominant directional influence (difference of influence term) are highlighted in red (Mann-Whitney U test, P < 0.05).](image)