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This supplementary material has been provided by the authors to give readers additional information about their work.

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Selection of the Sample of participants from the ABIDE database

Datasets were selected from the Autism Brain Imaging data exchange (ABIDE) repository (http://fcon_1000.projects.nitrc.org/indi/abide/) according to the criteria described in the Methods section in the main text and detailed below. The flowchart in eFigure 1 provides a graphical representation of the selection procedure, reporting the number of participants included at each step. In the final sample, participants from 8 sites were included: Leuven (sample 1), NYU, OLIN, PITT, Stanford, SDSU, USM, Yale. Additional information about the final sample are reported in Table 1 in the main text, and in eTable 1.

Sample selection: gender and diagnosis.
We included only Male participants since they represent 80% of our initially included sample of 749 participants. Analogously, in the Autism Spectrum Disorder (ASD) group, we included only participants with a diagnosis of either Autism or Asperger’s syndrome, according to the Composite Phenotypic File available on the ABIDE website, since patients with a diagnosis of PDDNOS accounted to only 4% of the initially selected sample. As reported in the ABIDE website, http://fcon_1000.projects.nitrc.org/indi/abide/, the diagnosis of ASD was supported by as the Autism Diagnostic Observation Schedule (ADOS ¹) and the Autism Diagnostic Interview-Revised (ADI-R ²) in 7/8 Sites, often combined with clinical judgment based on expertise and DSM-IV-TR diagnostic criteria. The Social Responsiveness Scale ³ was also administered to an informant known to the participant in 4/8 Sites. Medical conditions such as tuberous sclerosis, fragile-X syndrome, epilepsy, Tourette’s syndrome were also reported by several 4/8 sites as exclusion criteria. Details of image acquisition for each site can be found on the ABIDE website http://fcon_1000.projects.nitrc.org/indi/abide/.

Preprocessing of structural (T1-weighted) and resting-state fMRI images.
Structural (T1-weighted) images were skull stripped (and the results visually checked) after inhomogeneity correction. Resting-state fMRI (rsfMRI) data were limited to 180 time points (see below for rationale) and preprocessed with motion correction ⁴; slice-timing correction; spatial smoothing (Gaussian kernel, FWHM=6mm); highpass temporal filtering (100 sec). Each subject’s rsfMRI image was then linearly registered ⁴ to the MNI152 template via the high-resolution structural (T1-weighted) image, and resampled to a voxel dimension of 4x4x4x4mm. The resulting preprocessed data were subsequently used for Independent Component Analysis (ICA).

Sample selection: length of resting-state fMRI acquisition.
For the current study, we included only resting-state fmri (RS-fmri) scans of at least 180 time points (4.5-6 minutes, depending on the TR). A similar choice was pursued in a previous study ⁵ on a dataset of 1093 participants, aggregated from 24 acquisition sites, in order to perform Independent Component Analysis (ICA) with a very similar methodology to the one adopted in our work. Our decision to include only sites with RSfmri image of at least 180 time points, and to truncate all included datasets to 180 time points, was motivated by the following reasons: (1) One study ⁶ suggests that almost asymptotic reproducibility of RS networks obtained by seed-based correlation is reached after 5’ of scan time. (2) There are both technical restrictions (in the employed FSL MELODIC software) and theoretical concerns which prevent performing a temporally-concatenated ICA on participants whose RS fmri scan has a different number of time points. (3) Shorter scan time would increase the degree of partial correlation between sub-systems, reducing the ability to delineate them ⁷.

Independent Component Analysis
Temporal filtering: Consistent with previous works where ICA was performed on RS-fMRI data ⁵, ⁸, we did not low-pass filter the data. High frequencies (above 0.1 Hz) are usually filtered out from RSfmri data in seed-based correlation studies because they can reflect physiological parameters of little or no interest such as heartbeat or respiration, and
because early reports suggested that frequencies in the range 0.009-0.08 Hz mostly contribute to the spontaneous BOLD fluctuations in the brain. However, it has been recently shown that the spectral range of resting-state networks extends above 0.1Hz. Also, many artefactual signals have spectral peaks that are either truly within similar low frequency ranges seen with RSNs, or are aliased by the fMRI temporal sampling into these ranges. Instead, several studies showed that ICA can isolate high-frequency components associated with motion, respiratory and cardiovascular signal fluctuations. In the first stage of the subsequent dual regression, network-specific summary time courses are estimated using all the spatial modes extracted by the ICA. In this way, artefactual signal from confounding components is regressed out from the estimation of the summary time course for a specific functionally relevant network. Nevertheless, in the subsequent functional network connectivity analysis, we chose to filter the summary time course in the frequencies between 0.009 and 0.08 Hz. This choice was operated for the following reasons: (1) an optimal procedure to remove signal from confounding high-frequency components would involve performing ICA for every subject separately, followed by a qualitative evaluation of each component. For a large dataset such as the one employed in this study, this choice would be practically unfeasible, and likely to bias the results by experimenter-dependent qualitative choices. This limit will likely be removed when reliable automated method for discriminating artefactual components will be available. (2) Limiting the frequencies under examination in the range between 0.009 and 0.08 Hz would facilitate the comparison of our results with previous seed-based functional connectivity analysis, which represents one of the implicit scope of our study.

Number of participants in each of the 25 temporally-concatenated ICA
The spatial maps of resting-state brain networks used to calculate their respective summary time course were obtained in a metaICA procedure similar to the one described in Biswal BB et al. (2010). This procedure was preferred to performing a temporally-concatenated ICA on all included datasets, in order to prevent the potential impact of initialization values on the final results of the ICA, and also to prevent the spatial maps to be biased by sites including more subjects than others. The metaICA was run on the results of 25 temporally-concatenated ICAs carried out on randomised subsets of 112 participants. In each subset, 7 ASD + 7 TD participants were randomly selected from each site. This number corresponds to the smallest available number of included ASD participants in 2/8 sites (SDSU and Yale).

Details about ICA implementation
Preprocessing for the ICA analysis included masking out non-brain voxels, voxel-wise de-meaning and variance normalization of the RSfMRI data. For each of the 25 temporally-concatenated probabilistic ICA (tcPICA), as well as for the metaICA, preprocessed data were whitened and projected into an n-dimensional subspace using Principal Component Analysis, where the number of dimensions was estimated using the Laplace approximation to the Bayesian evidence of the model order. Spatially independent components (IC) were extracted by optimising for non-Gaussian spatial source distributions using a fixed-point iteration technique. Estimated Component maps were divided by the standard deviation of the residual noise and thresholded by fitting a mixture model to the histogram of intensity values.

The number of spatially independent components (ICs) estimated in the 25 tcPICA ranged from 22 to 30 (median=27). The subsequent metaICA, performed on these results, extracted 52 ICs. This difference in the estimated number of ICs was expected because of the different nature of the input data, that is: the preprocessed fMRI time courses for the 25 tcPICA, and the results of these tcPICA for the metaICA.

Components selection
To identify RSNs out of the 52 components extracted by the metaICA, we employed a semi-supervised procedure including both quantitative measures and qualitative comparison with previous works. First, using visual inspection, we labelled as potentially relevant for FNC analysis 16 spatial components with the following features: (i) high spatial consistency with RSfMRI networks described in previous ICA studies; (ii) highest thresholded Z-scores, estimated from mixture modelling inference in the metaICA, located mostly in cortical/subcortical gray matter; (iii) spatial
distribution of the component map compatible with cortical sulcal/gyral morphology or with the location of subcortical structures.

We then quantified for each component (1) the proportion of thresholded spatial map lying inside/outside gray matter (GM) and (2) the reproducibility of that component across the 25 tcPICA. The first quantity was calculated on the mean GM partial volume estimate maps thresholded at 0.4, obtained from the segmentation of the T1-weighted image of all included participants using FSL FAST 26. The reproducibility of each metaICA component was quantified by looking at its spatial correlation with every component extracted in each of the 25 tcPICA, and retaining the maximum spatial correlation score for each tcPICA. The median of these 25 maximum spatial correlation values was used to estimate the reproducibility of each metaICA component across randomizations. This approach was chosen under the hypothesis that RSNs should be reliably extracted with high spatial consistency across the randomizations carried out in the 25 tcPICA, independently from the particular subset of ASD + TD participants, and from ICA initialization parameters.

The 16 labelled components featured significantly higher reproducibility ($T_{(50)}=3.07$, $p<.0035$) and GM proportion ($T_{(50)}=2.23$, $p<.03$) compared to the remaining 36 components. Plotting all the 52 components according to these two variables revealed other seven previously discarded components featuring levels of reproducibility and GM proportion comparable to those of the initially selected 16 components (see eFigure 4). An additional visual examination of the spatial distribution of these components on the basis of the anatomical criteria defined above led us to include IC9, IC10, IC15, IC30 in the following analysis, and to exclude IC11, IC14, IC45. Also, one of the initially selected component (IC7) was excluded from further analysis due to relatively low levels of reproducibility. We anticipate that the methodological choices about the 7 initially discarded ICs did not affect the results of the FNC analysis, in the sense that none of the subsequently included components showed significant group difference. The inclusion of those components led to an FDR 27 corrected threshold of $p<.0008$, with respect to the $p<.001$ threshold of the analysis without the additional components, however this did not affect the results of the subsequent FNC analysis in terms of surviving group differences (See eFigure 5).

This procedure led to the identification of 19 RSN which were selected for FNC analysis (see Figure 1 and Table 2 for included components and eFigure 3 for discarded components). As an additional procedure, we ran Classic Multidimensional Scaling 28 on feature vectors quantifying for each of the initial 52 components their GM proportion, reproducibility, dynamic range of the spectrum 29 and fALFF 30. The results of this more complex approach converged with the one considering only proportion of GM and reproducibility (See eFigure 6). This led us to use the simpler approach using only GM proportion and reproducibility to aid the selection of components to be considered for FNC analysis.

Since we were also interested in focusing on components previously described in the literature, we computed the spatial correlation with the ICs reported in previous ICA studies. The 19 spatial maps selected for FNC featured on average high spatial similarity with the 20 ICs reported by Biswal BB et al. (2010)5 (median correlation $= .72$) and the 70 ICs reported by Smith SM et al. (2009)23 (median correlation $= .70$) (See also eFigure 7). However, this measure was not used as a discriminating criterion for component selection, given the different model order (NIC=20 or NIC=70 established a priori, versus our freely estimated model order of 52) and the different composition of the sample, which in our case included also patients with ASD.

Additional analyses aimed at validating the detected group differences in FNC against potential contamination from participants' motion in the scanner.

Recent works 31-33 have shown that participants' motion in the scanner can have a substantial impact on measures of resting-state functional connectivity (FC). This has raised the concern that some differences of FC between ASD and TD participants might be due or enhanced by artifactual effects linked to motion 34, 35. To address these concerns, in the analyses of group differences in FNC - as well as in the subsequent correlation analyses between FNC and either SRS or age - we used mean framewise displacement (FD) 32 as a covariate of no interest to limit the potential contamination of our results by participants' motion in the scanner, as suggested by the current literature 31, 33. Yan et al. (2013)36 recently showed that correcting for mean FD at the group level yields benefits comparable to those yielded by...
scrubbing. The high similarity of seed-based functional connectivity results obtained by either scrubbing or group-level regression of mean FD was shown also in the paper introducing the ABIDE dataset 37.

In addition, we further explored the likelihood that our results were contaminated by a residual effect of motion by repeating the analyses of group differences in FNC using two additional preprocessing methods. Specifically:

(i) we de-spiked the data using AFNI 3dDespike prior to any other preprocessing step, as indicated by Jo HJ et al. 38. The results obtained after despiking are virtually identical to those from our initial analysis without despiking (see eFigure 8 (middle matrix) and eTable 4). Most importantly, the basic pattern of hyperconnectivity between subcortical and primary sensory networks remains fully significant after despiking.

(ii) we included only participants within one standard deviation of the whole-sample mean FD (i.e. FD<0.227mm) instead of the original two standard deviations. This threshold is consistent with the apriori FD and RMS threshold values used in several current works, notably in Satterthwaite et al. (2013) for spike identification and in Power JD (2012a) for scrubbing data points in seed-based functional connectivity. We then additionally excluded TD participants below the 20th percentile of the mean FD distribution and ASD participants above the 80th percentile of the mean FD distribution. The final sample resulting from these procedures consisted of 329 participants (151 ASD + 178 TD) matched for FD at p=0.36. We then repeated our FNC analysis on this sample, which replicated the results obtained on our sample of 359 participants (eFigure 8 - bottom matrix).

**Effect of sample size - Power analysis**

FNC differences between ASD and TD yielded medium effect sizes (Cohen d ranging from 0.36 to 0.41). This suggests that large samples of participants are required to detect these differences. In order to investigate the effect of sample size on our results, we estimated FNC group differences in randomized subsets varying in size from 10 participants per group to the whole sample, and did this 200 times. For each subset of TD or ASD, participants were selected from the whole sample of ASD (N=166) and TD (N=193), irrespective of the acquisition site, which was modeled, as in the analyses described in the main text, with a covariate of no interest. In each subset, we matched groups on age, FIQ and mean FD. Subsampling allowed us to explore the impact of sample size on (i) the ability to detect differences that are significant in the whole group (‘Hits’ in eFigure 9) and (ii) the risk to detect differences that are clearly rejected in the whole sample (‘False alarms’ in eFigure 9). The results of this analysis showed that at least ~140 participants per group are required to detect the effects we found in our whole sample using a false discovery rate of q(FDR)=.05 with 80% probability. These results fit a power analysis performed with G*Power 3.1 showing that at least 123 ASD + 123 TD participants would be required to have an 80% chance to detect an effect size d=0.36 at using alpha level of .05 in a two-tailed independent t test (see picture below).
Comparison of our results with those obtained from the ABIDE dataset in the inaugural ABIDE paper

The presence of subcortico-cortical overconnectivity between *apriori*-defined brain regions represents one of the main results in the inaugural ABIDE paper. In our work we show that this overconnectivity is preserved when examining the interaction between RSNs' summary time courses. However, another main result of the ABIDE paper was the presence of extensive underconnectivity between cortico-cortical regions. We also detected underconnectivity in ASD participants in cortico-cortical RSNs at *p*<.05 uncorrected (see eFigure 12), however, with the exception of the interaction between auditory (IC16) and ventral somatosensory networks (IC29) (see Fig. 2), these group differences did not survive FDR correction. With respect to these differences between ours and previous analyses on the ABIDE, two considerations must be done: (i) because of how the summary time course of each RSN is estimated - by means of a multiple spatial regression including also the spatial components of all other RSNs - FNC allows to focus on the specific signal of each RSN unmixed from that of other RSNs. Therefore the comparison between our and previous results on the ABIDE dataset would suggest that underconnectivity effects in ASD are more pronounced when considering the amount of variability in the fMRI signal which is shared between RSNs, or between brain regions encompassed by the same functional network: such is the analysis carried out by seed-based functional connectivity, where the total (preprocessed and denoised) fMRI signal in each seed region of interest is used to estimate functional connectivity. (ii) Previous rs-fMRI studies in schizophrenia have shown that decreased inter-regional connectivity can coexist with increased between-network connectivity in largely overlapping datasets of participants. Therefore, while our results on subcortico-cortical overconnectivity in ASD at the network level are consistent with previous seed-based studies at the inter-regional level, it is possible that the degree of cortico-cortical underconnectivity varies between different anatomical scales. Both these considerations highlight the complementary nature of ICA-based FNC studies and seed-based functional connectivity studies.


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eFigure 1 - Selection of the Sample of participants from the ABIDE database

Image quality inclusion criteria
(1) dataset includes a T1-weighted and RS-fmri scan with at least 180 time points (~5 min)
(2) absence of excessive artifacts (visual inspection on RS-fmri mean and stdev image over time - see eFigure 2)
(3) near-full brain coverage and successful registration to T1-weighted image and MNI template

N = 545

Meeting basic phenotypical inclusion criteria
Phenotypical inclusion criteria, based on the information provided by the Composite Phenotypic File available on the ABIDE website (Phenotypic_V1.0b.csv)
Sex=Male
Full IQ (FIQ) > 70
Age at scan available
Eye-status during scan available
Control participants (TD group) or participants with diagnosis of either Autism or Asperger's syndrome (ASD group)

N = 407
192 ASD
215 CON

Matching for Age, FIQ, Eye-status at scan
Participants are matched by Age and Eye-status at scan in 8/8 sites, and by FIQ in 7/8 sites. The significant difference in FIQ in USM site (CON > ASD: p<.8E6) drives a significant difference in the FIQ in the entire group (CON > ASD: p<.004). To match participants by FIQ, a subsample of CON/ASD participants from the USM site with FIQ in the highest/lowest 20-th percentile are discarded.

N = 387
181 ASD
206 CON

Exclusion based on mean Framewise Displacement (mean FD)
Mean FD is calculated according to Satterthwaite TD et al. (2012 Neuroimage) for each participant. Participants with mean FD higher than 0.34, corresponding to two standard deviations (2*0.1127) above the whole sample mean (0.1146) were excluded from further analysis. Specifically, 9 ASD and 5 CON participants were excluded.

N = 373
172 ASD
201 CON

Matching for mean FD
Due to a residual difference in mean FD in the whole group (ASD > CON: p<.013), driven by participants in the NYU site (p<.043), CON/ASD participants from the NYU site with mean FD in the lowest/highest 5-th percentile are discarded (6 ASD and 8 CON). After this exclusion, remaining participants are matched by Age (p=.39), FIQ (p=.35) Eye-status at scan (p=.81) and mean FD (p=.09). In subsequent analyses, all these parameters, plus 7 vectors coding for the acquisition site (8 sites minus one to prevent rank deficiency), are used as covariates of no interest.

N = 359
166 ASD
193 CON

Data were selected from the ABIDE database (http://fcon_1000.projects.nitrc.org/indi/abide/). Specifically, we selected the participants for this analysis from the sites that featured all the basic inclusion criteria, described in the top two steps of the flowchart: Leuven (sample 1), NYU, OLIN, PITT, Stanford, SDSU, USM, Yale. Additional information about the final sample are reported in Table 1 in the main text, and in eTable 1.

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All the participants' dataset including one T1-weighted and one RSfmri scan of at least 180 time points underwent standard image preprocessing (see the relevant section in the eMaterials). The image of the standard deviation over time, calculated on the RSfmri scan after linear registration to the MNI template, was qualitatively assessed for evident problems in the quality of the images. Some of these images are reported in this picture to exemplify the cases in which a participant was excluded. Exclusion criteria included image distortion and excessive lack of parts of the brain, especially at the dorsal crest of the hemispheres, and in the temporal poles.
eFigure 3 - Excluded components from the metaICA
eFigure 3 (Continued) - Excluded components from the metaICA

The metaICA yielded 52 components, all of which were used to estimate their reproducibility, and the proportion of spatial map inside/outside grey matter. Details are reported in the section of the main text and of the supplementary materials describing the procedure for components selection. In this picture, we show the 33 components which were excluded from FNC analysis.
Out of the 52 spatial components (IC) estimated by the metaICA, we first qualitatively identified 16 ICs (red circles) on the basis of their anatomical plausibility, and spatial similarity with previously reported ICA results (see Methods). All but one (IC7) out of these 16 ICs clustered in high values of both reproducibility and proportion of spatial extent inside/outside GM. The region delimited with the green axis highlights the components which were considered for inclusion in further analyses. Of the previously excluded ICs (blue circles), 7 lay inside the region of the initially selected components; 4 of these (IC9, IC10, IC15, IC30) were included in the further FNC analysis, while three (IC11, IC14, IC45) were discarded as artifactual due to their anatomical implausibility (See Fig. eFigure 3). The final 19 ICs selected for FNC are marked with a green outline, and their spatial extent is shown in Figure 1 in the main text. In most cases, the selected 19 ICs bear a high spatial correlation with available previous results from the Smith70 (available at http://www.fmrib.ox.ac.uk/analysis/brainmap+rsns/) and Biswal20 (courtesy of Maarten Mennes) (See eFigure 7), however this was not used as a strict discriminant for selecting components, due to the different composition of the our sample, including also ASD participants besides controls, and to the different model order (52 ICs in our case), which can lead to some of our components to represent union or subdivision of previous studies using respectively either higher (70 ICs in Smith SM et al. (2009)23) or lower dimensionality (20 ICs in Biswal BB et al. (2010)5).
In functional connectivity analysis, it is customary to transform the Pearson correlation coefficients into Z scores using the Fisher r to Z transformation (see for instance http://en.wikipedia.org/wiki/Fisher_transformation) to improve the normality of the distribution of the correlation coefficient, in provision of using a parametric test. In our case, we carried out inference using nonparametric permutation testing on the correlation coefficients. However, we also tested whether the r-to-Z transform would have affected the results. The matrix in the middle shows that the results obtained on the r or on the Z scores are almost identical, although the final FDR corrected threshold was in this case p<.00125 with respect to p<.001 obtained with correlation coefficients (these results if p<.001 is used as final threshold).

We also performed our analysis adding the previously discarded components (IC7, IC11, IC14, IC45) bearing similar values of reproducibility and gray matter proportion with respect to the finally selected 19 components. For details about the procedure and the reasons that motivated the choice, see the eMaterial section on components selection, as well as the figure representing all the eventually discarded components (eFigure 3). No
significant group difference was found in 7/8 discarded components. The remaining component (IC7), located in the dorsal medial and lateral part of the occipital lobe, showed increased FNC with the RSN encompassing basal ganglia and thalamus, in line with our main results. While also in this case the final FDR threshold changed to \( p < 0.0008 \), the same group differences are found by using the \( p < 0.001 \) threshold determined by using FDR on the analysis carried out on the correlation coefficients.

(to correctly visualize colors in this picture, please print it in PDF and visualize it using Adobe® Reader®)
As described in the section on component selection, the final selection of RSN was based on the consistency with previously published ICA analyses, as well as one the two features of reproducibility and gray matter proportion. As an additional procedure, we ran Classic Multidimensional Scaling on feature vectors quantifying for each of the initial 52 components their GM proportion, reproducibility, dynamic range of the spectrum and fALFF. The results of this more complex approach converged with the one considering only proportion of GM and reproducibility (See eFigure 4). This led us to use the simpler approach using only GM proportion and reproducibility to aid the selection of components to be considered for FNC analysis. The axis report the values determined by the employed procedure for Classic Multidimensional Scaling in Matlab.
This picture reports the spatial distribution of the RSN selected for FNC, after thresholding with the significance values obtained from the spatial mixture modelling implemented in Melodic ICA, in the same way as reported in the Fig. 1 in the main text. Abbreviations are described in Table 2. The values underneath the name/abbreviations for each IC report the value of spatial correlation with the results obtained by Biswal BB et al. (2010) and Smith SM et al. (2009). For instance, for the IC1 (V1), R(B1)=0.69 means that the spatial map for our component IC1 has a spatial correlation of 0.69 with the component IC1 found by Biswal BB et al. (2010); analogously, R(S1)=0.7 means that the spatial map for our component IC 1 has a spatial correlation of 0.7 with the component IC 1 found by Smith SM et al. (2009). For each of our ICs, we report the value of spatial correlation for the component in Biswal BB et al. (2010) and Smith SM et al. (2009) which had the highest correlation.
Given the concerns for motion-induced artifacts in resting-state functional connectivity, we repeated the analysis of group differences in FNC using two additional steps during preprocessing. 1) Middle matrix: data were despiked using AFNI 3dDespike, as a first preprocessing step. By leaving all the subsequent steps unchanged, we replicated our results on the non-despiked data (presented in the top matrix). 2) Bottom matrix: in this case, the selection of participants used stricter criteria for motion: we included only participants with mean framewise displacement (FD) of at most 0.227mm, corresponding to one standard deviation above the mean FD of ASD+TD participants – as opposed to 0.34, corresponding to two standard deviations. Also, additional participants were excluded from each group – TD with high relatively low (below 20th percentile) mean FD and ASD with relatively...
high (above 80th percentile) mean FD – to reach a stricter matching for motion between groups ($p=.36$). In this case 329 participants were included (151 ASD and 178 TD). The exact values are reported in eTable 4.

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To assess the likelihood of finding the 8 FNC differences reported for the whole sample of 166 ASD+193 TD participants in smaller sample sizes, we estimate group differences in 200 randomly chosen symmetric subsets of varying size from 10 ASD+10 TD to 166 ASD+166 TD participants, followed by 2 asymmetric subsets of 166 ASD and 175 and 193 TD, respectively. Hits denote a group difference in FNC in the whole group \((q(FDR) = 0.05; p < 0.001)\) and also in the subsample \((q(FDR) = 0.05)\). A False alarm is a difference in FNC which is far from significance in the whole sample \((p > 0.3)\) but significant \((q(FDR) = 0.05)\) in the subsample. The tightly dotted horizontal line indicates a false alarm rate of 5%. The average amount of False Alarms is always \(\leq 5\%\) and basically overlaps onto the \(X = 0\) axis. Note that ‘Hits’ and ‘False alarms’ are used here in relation to our larger sample (i.e. in a relative sense), rather than to the true (unknown) underlying difference (i.e. in an absolute sense).
eFigure 10 - Correlation between FNC and SRS scores.

These scatterplots show the relation between SRS, after group-wise demeaning, and FNC in the whole sample (ASD in red + TD in blue) for the 8 pairs of networks where a significant difference was found when comparing the mean FNC between groups. Before computing the correlation coefficients we regressed out IQ, Site of acquisition, Eyes open/closed at scan, and mean FD. The correlation coefficient reported on top of each scatterplot refer to the correlation analysis on the whole sample. After correction for multiple comparisons, correlation coefficients are significant at p<.007 (q(FDR)=.05). The lines in the scatterplot represent the linear fit within each group: red for ASD, blue for TD. The corresponding correlation coefficients and significance values, as well as group differences in correlation coefficients, can be found in eTable 2 and in the Results section of the main text.

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eFigure 11 - Correlation between FNC and age.

These scatterplots show the relation between Age and FNC in the whole sample (ASD in red + TD in blue) for the 8 pairs of networks where a significant difference was found when comparing the mean FNC between groups. Before computing the correlation coefficients (reported in eTable 3) we regressed out IQ, Site of acquisition, Eyes open/closed at scan, and mean FD. The correlation coefficient reported on top of each scatterplot refer to the correlation analysis on the whole sample. After correction for multiple comparisons, correlation coefficients are significant at p<.012 (q(FDR)=.05). The lines in the scatterplot represent the linear fit within each group: red for ASD, blue for TD. The corresponding correlation coefficients and significance values, as well as group differences in correlation coefficients, can be found in eTable 3 in the main text.

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eFigure 12 – FNC group differences at p<0.05 uncorrected

T values for FNC group differences significant at p<.05 uncorrected

T scores corresponding to significant (p<.05) FNC group differences before correction for multiple comparison. Negative values (light to dark blue) indicate higher FNC in the TD group; positive values (yellow to dark red) indicate higher FNC in the ASD group.

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eFigure 13A – FNC values in the ASD group for network pairs showing a significant group difference at p<.05 uncorrected

(to correctly visualize colors in this picture, please print it in PDF and visualize it using Adobe® Reader®)
eFigure 13B – FNC values in the TD group for network pairs showing a significant group difference at p<.05 uncorrected

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Values of difference in FNC between groups. Positive values (dark) indicate higher FNC in the ASD group. Negative values (bright) indicate higher FNC in the TD group.

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eTable 1 - Site-specific inclusion criteria for participants with Autism Spectrum Disorders (ASD) for each Site included in the presented analyses.

<table>
<thead>
<tr>
<th>Site</th>
<th>Inclusion criteria for ASD participants (see <a href="http://fcon_1000.projects.nitrc.org/indi/abide/">http://fcon_1000.projects.nitrc.org/indi/abide/</a> for full details)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leuven: Sample 1 (N&lt;sub&gt;ASD&lt;/sub&gt;=10; N&lt;sub&gt;TD&lt;/sub&gt;=12) SRS=22</td>
<td>(1) Diagnosis of autistic disorder according to the DSM-IV-TR; (2) SRS (Adult Version, parent-reported) raw scores &gt; 60</td>
</tr>
<tr>
<td>NYU (N&lt;sub&gt;ASD&lt;/sub&gt;=56; N&lt;sub&gt;TD&lt;/sub&gt;=69) ADOS=56; ADI-R=53 ; SRS=125</td>
<td>(1) A Clinician’s DSM-IV-TR diagnosis of Autistic Disorder, Asperger’s Disorder, or Pervasive Developmental Disorder Not-Otherwise-Specified, which was supported by review of available records; (2) ADOS; (3) ADI-R when possible</td>
</tr>
<tr>
<td>OLIN (N&lt;sub&gt;ASD&lt;/sub&gt;=14; N&lt;sub&gt;TD&lt;/sub&gt;=13) ADOS=14</td>
<td>(1) ADOS and (2) either ADI-R or SCQ Lifetime administration to confirm ASD diagnosis from clinicians outside the Institute</td>
</tr>
<tr>
<td>Pittsburgh (N&lt;sub&gt;ASD&lt;/sub&gt;=19; N&lt;sub&gt;TD&lt;/sub&gt;=18) ADOS=16; ADI-R=16</td>
<td>(1) Participants from 7 to 35 years of age with a well-characterized Autistic Disorder; (2) ADI-R; (3) ADOS; (4) expert clinical opinion; (5) exclusion of participants with PDD-NOS or Asperger’s syndrome; (6) exclusion of participants with FIQ &lt; 80; (7) exclusion of participants known to have an associated disorder such as tuberous sclerosis or Fragile-X syndrome</td>
</tr>
<tr>
<td>SDSU (N&lt;sub&gt;ASD&lt;/sub&gt;=7; N&lt;sub&gt;TD&lt;/sub&gt;=15) ADOS=7; ADI-R=6</td>
<td>Clinical diagnoses confirmed using (1) ADI-R; (2) ADOS; (3) expert clinical judgment according to the DSM-IV-TR criteria; (4) exclusion of participants with ASD-related medical conditions (e.g. Fragile-X syndrome, tuberous sclerosis) and other neurological conditions (e.g. epilepsy, Tourette’s Syndrome)</td>
</tr>
<tr>
<td>Stanford (N&lt;sub&gt;ASD&lt;/sub&gt;=13; N&lt;sub&gt;TD&lt;/sub&gt;=14) ADOS=12; ADI-R=13</td>
<td>(1) ADI-R and/or (2) ADOS, administered by research reliable clinician; (3) exclusion of participants with known genetic, psychiatric, or neurological disorders (e.g., Fragile X syndrome or Tourette’s syndrome); (4) no currently prescribed antipsychotic medication.</td>
</tr>
<tr>
<td>USM (N&lt;sub&gt;ASD&lt;/sub&gt;=40; N&lt;sub&gt;TD&lt;/sub&gt;=33) ADOS=40; SRS=72</td>
<td>Inclusion as an individual with ASD required meeting full (1) ADOS and (2) DSM-IV-TR criteria for Autism; (3) Parents were interviewed with ADI-R; (3) exclusion of participants with known medical conditions such as tuberous sclerosis, Fragile X, neonatal ischemic/hypoxia; (4) exclusion of participants with blindness or deafness, history of seizures, severe head injury or severe medical problems.</td>
</tr>
<tr>
<td>Yale (N&lt;sub&gt;ASD&lt;/sub&gt;=7; N&lt;sub&gt;TD&lt;/sub&gt;=19) ADOS=1; ADI-R=6 ; SRS=24</td>
<td>Cutoff scores for ASD on the (1) ADOS (for those who received it) and (2) ADI-R, administered by a research-reliable clinician; (3) DSM-IV-TR diagnosis of ASD confirmed by one of three expert clinicians at the Yale Child Study Center</td>
</tr>
</tbody>
</table>

For each site, we report in brackets the number of included ASD and TD participants. Additionally, we report the number of participants for which an ADOS, ADI-R or SRS score was available among the included participants. For the final sample of 359 participants, ADOS was available in 88% of included ASD, ADI-R in 57% of included ASD, SRS in 61% of all participants. Abbreviations: ADI-R: Autism Diagnostic Interview-Revised; ADOS: Autism Diagnosis Observation Schedule; FIQ: Full-IQ; PDD-NOS: Pervasive Developmental Disorder - Not Otherwise Specified; SCQ: Social Communication Questionnaire; SRS: Social Responsiveness Score.

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eTable 2 – Confirmation of the result using data despiking and additional control for subject motion (2)

<table>
<thead>
<tr>
<th>Brain region</th>
<th>FNC values presented in the main text</th>
<th>mean ASD</th>
<th>mean TD</th>
<th>ASD - TD</th>
<th>T</th>
<th>p</th>
<th>Cohen d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bg+Th ~ dSI/MI/mPMC</td>
<td>0.17</td>
<td>-0.02</td>
<td>0.19</td>
<td>4.60</td>
<td>1.1E-04</td>
<td>d=0.49</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ pfus + V1</td>
<td>0.09</td>
<td>-0.02</td>
<td>0.11</td>
<td>3.23</td>
<td>4.1E-04</td>
<td>d=0.34</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ TE+pSTG+PARop</td>
<td>0.12</td>
<td>-0.02</td>
<td>0.14</td>
<td>3.56</td>
<td>2.0E-04</td>
<td>d=0.38</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ vSI+vMI+plC</td>
<td>0.14</td>
<td>0.01</td>
<td>0.13</td>
<td>3.51</td>
<td>4.6E-04</td>
<td>d=0.37</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ TS+LH IFG</td>
<td>0.16</td>
<td>0.02</td>
<td>0.13</td>
<td>3.13</td>
<td>1.0E-03</td>
<td>d=0.33</td>
<td></td>
</tr>
<tr>
<td>TE+pSTG+PARop ~ vSI+vMI+plC</td>
<td>0.44</td>
<td>0.55</td>
<td>-0.10</td>
<td>-4.06</td>
<td>1.1E-04</td>
<td>d=0.43</td>
<td></td>
</tr>
<tr>
<td>CRB anterior ~ STS+LH IFG</td>
<td>-0.03</td>
<td>-0.16</td>
<td>0.13</td>
<td>3.20</td>
<td>1.0E-03</td>
<td>d=0.34</td>
<td></td>
</tr>
<tr>
<td>CRB anterior ~ dSI/MI/mPMC</td>
<td>0.06</td>
<td>-0.07</td>
<td>0.13</td>
<td>3.35</td>
<td>3.0E-04</td>
<td>d=0.35</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Brain region</th>
<th>FNC values using data preprocessed with 3Ddespike</th>
<th>mean ASD</th>
<th>mean TD</th>
<th>ASD - TD</th>
<th>T</th>
<th>p</th>
<th>Cohen d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bg+Th ~ dSI/MI/mPMC</td>
<td>0.15</td>
<td>-0.02</td>
<td>0.17</td>
<td>4.32</td>
<td>1.5E-04</td>
<td>d=0.46</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ pfus + V1</td>
<td>0.08</td>
<td>-0.03</td>
<td>0.11</td>
<td>3.46</td>
<td>3.0E-04</td>
<td>d=0.37</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ TE+pSTG+PARop</td>
<td>0.11</td>
<td>-0.03</td>
<td>0.14</td>
<td>3.53</td>
<td>3.5E-04</td>
<td>d=0.37</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ vSI+vMI+plC</td>
<td>0.12</td>
<td>-0.00</td>
<td>0.12</td>
<td>3.46</td>
<td>6.0E-04</td>
<td>d=0.37</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ TS+LH IFG</td>
<td>0.15</td>
<td>0.01</td>
<td>0.14</td>
<td>3.32</td>
<td>5.5E-04</td>
<td>d=0.35</td>
<td></td>
</tr>
<tr>
<td>TE+pSTG+PARop ~ vSI+vMI+plC</td>
<td>0.45</td>
<td>0.55</td>
<td>-0.10</td>
<td>-4.00</td>
<td>5.0E-05</td>
<td>d=0.42</td>
<td></td>
</tr>
<tr>
<td>CRB anterior ~ STS+LH IFG</td>
<td>-0.07</td>
<td>-0.22</td>
<td>0.15</td>
<td>3.85</td>
<td>1.0E-04</td>
<td>d=0.40</td>
<td></td>
</tr>
<tr>
<td>CRB anterior ~ dSI/MI/mPMC</td>
<td>0.06</td>
<td>-0.07</td>
<td>0.13</td>
<td>3.41</td>
<td>5.5E-04</td>
<td>d=0.36</td>
<td></td>
</tr>
<tr>
<td>CRB posterior ~ IFG+ppPC</td>
<td>-0.11</td>
<td>-0.00</td>
<td>-0.10</td>
<td>-3.02</td>
<td>8.5E-04</td>
<td>d=0.32</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Brain region</th>
<th>FNC values excluding subjects with mean FD &gt; 0.227 and matched for SRS at p=.36</th>
<th>mean ASD</th>
<th>mean TD</th>
<th>ASD - TD</th>
<th>T</th>
<th>p</th>
<th>Cohen d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bg+Th ~ dSI/MI/mPMC</td>
<td>0.17</td>
<td>-0.03</td>
<td>0.20</td>
<td>4.54</td>
<td>2.0E-04</td>
<td>d=0.48</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ pfus + V1</td>
<td>0.09</td>
<td>-0.03</td>
<td>0.12</td>
<td>3.66</td>
<td>5.0E-04</td>
<td>d=0.39</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ TE+pSTG+PARop</td>
<td>0.10</td>
<td>-0.04</td>
<td>0.13</td>
<td>3.30</td>
<td>9.0E-04</td>
<td>d=0.35</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ vSI+vMI+plC</td>
<td>0.14</td>
<td>0.00</td>
<td>0.14</td>
<td>3.60</td>
<td>4.0E-04</td>
<td>d=0.38</td>
<td></td>
</tr>
<tr>
<td>Bg+Th ~ TS+LH IFG</td>
<td>0.15</td>
<td>0.01</td>
<td>0.14</td>
<td>3.33</td>
<td>2.0E-04</td>
<td>d=0.35</td>
<td></td>
</tr>
<tr>
<td>TE+pSTG+PARop ~ vSI+vMI+plC</td>
<td>0.44</td>
<td>0.53</td>
<td>-0.09</td>
<td>-3.50</td>
<td>5.0E-04</td>
<td>d=0.37</td>
<td></td>
</tr>
<tr>
<td>CRB anterior ~ STS+LH IFG</td>
<td>-0.03</td>
<td>-0.17</td>
<td>0.14</td>
<td>3.59</td>
<td>2.0E-04</td>
<td>d=0.38</td>
<td></td>
</tr>
<tr>
<td>CRB anterior ~ dSI/MI/mPMC</td>
<td>0.07</td>
<td>-0.07</td>
<td>0.13</td>
<td>3.28</td>
<td>7.0E-04</td>
<td>d=0.35</td>
<td></td>
</tr>
</tbody>
</table>

These values are reported as color-coded matrices in eFigure 8.
eTable 3 - Test for non-zero group-level FNC within group in the 8 network pairs showing significant group differences in FNC

<table>
<thead>
<tr>
<th>RS-fmri</th>
<th>ASD</th>
<th></th>
<th>TD</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>T</td>
<td>p</td>
<td>mean</td>
</tr>
<tr>
<td>Bg+Th ~ dSI/MI/mPMC</td>
<td>0.17</td>
<td>5.72</td>
<td>0.00000</td>
<td>-0.02</td>
</tr>
<tr>
<td>Bg+Th ~ pfus + V1</td>
<td>0.09</td>
<td>3.49</td>
<td>0.0062</td>
<td>-0.02</td>
</tr>
<tr>
<td>Bg+Th ~ TE+pSTG+PARop</td>
<td>0.12</td>
<td>3.93</td>
<td>0.0012</td>
<td>-0.02</td>
</tr>
<tr>
<td>Bg+Th ~ vSI+vMI+pIC</td>
<td>0.14</td>
<td>4.90</td>
<td>0.00000</td>
<td>0.01</td>
</tr>
<tr>
<td>Bg+Th ~ STS+LH IFG</td>
<td>0.16</td>
<td>6.32</td>
<td>0.00000</td>
<td>0.02</td>
</tr>
<tr>
<td>TE+pSTG+PARop ~ vSI+vMI+pIC</td>
<td>0.44</td>
<td>18.95</td>
<td>0.00000</td>
<td>0.55</td>
</tr>
<tr>
<td>CRB anterior ~ STS+LH IFG</td>
<td>-0.03</td>
<td>-0.90</td>
<td>0.37034</td>
<td>-0.16</td>
</tr>
<tr>
<td>CRB anterior ~ dSI/MI/mPMC</td>
<td>0.06</td>
<td>2.03</td>
<td>0.04389</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

FNC values were tested against the null hypothesis of zero mean FNC at the group level using one sample t-test. FNC scores were to this aim previously transformed into Z scores to ensure the validity of the employed parametric test, however in the table the original mean FNC values are reported to facilitate the comparison with the values depicted in Figure 2 and shown in eTable 4 on the top. The threshold p value for significance was corrected for multiple comparison using q(FDR) =.05. Mean FNC which are significantly different from zero after FDR correction (in bold) are marked with a cross above the corresponding boxplot in Figure 2.

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**eTable 4 - Correlation between FNC and SRS scores (group-wise demeaned).**

<table>
<thead>
<tr>
<th>Network pair</th>
<th>ASD</th>
<th>TD</th>
<th>whole sample</th>
<th>group difference in slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bg+Th ~ dSl+MI+mPMC</td>
<td>R= 0.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>p&lt;0.0037</td>
<td>R= 0.01</td>
<td>NS</td>
</tr>
<tr>
<td>Bg+Th ~ pfus + V1</td>
<td>R= -0.04</td>
<td>NS</td>
<td>R= -0.04</td>
<td>NS</td>
</tr>
<tr>
<td>Bg+Th ~ TE+pSTG+PARop</td>
<td>R= 0.08</td>
<td>NS</td>
<td>R= 0.02</td>
<td>NS</td>
</tr>
<tr>
<td>Bg+Th ~ vSl+vMI+plC</td>
<td>R= 0.25&lt;sup&gt;a&lt;/sup&gt;</td>
<td>p&lt;0.0066</td>
<td>R= 0.04</td>
<td>NS</td>
</tr>
<tr>
<td>Bg+Th ~ STS+LH IFG</td>
<td>R= -0.03</td>
<td>NS</td>
<td>R= 0.04</td>
<td>p&lt;0.0452</td>
</tr>
<tr>
<td>TE+pSTG+PARop ~ vSl+vMI+plC</td>
<td>R= -0.04</td>
<td>NS</td>
<td>R= -0.11</td>
<td>p&lt;0.0006&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>CRB anterior ~ STS+LH IFG</td>
<td>R= 0.13</td>
<td>p&lt;0.0161</td>
<td>R= -0.07</td>
<td>NS</td>
</tr>
<tr>
<td>CRB anterior ~ dSl+MI+mPMC</td>
<td>R= 0.01</td>
<td>NS</td>
<td>R= -0.07</td>
<td>NS</td>
</tr>
</tbody>
</table>

<sup>a</sup>p<.0067 (corrected with q(FDR)=.053)

<sup>b</sup>p<.0006 (corrected with q(FDR)=.05)

<sup>c</sup>p<.007 (corrected with q(FDR)=.05)

NS indicates not significant at p<.05 uncorrected. NS values suggesting a trend to significance are reported with exact p value (uncorrected)

---

This boxplot shows the distribution of the SRS scores in the ASD and TD samples before the group-wise demeaning which was carried out in order to estimate unbiased correlation with FNC in the whole sample. The blue bar indicates the standard deviation of the SRS scores, the red bar the standard error of the mean.
eTable 5 - Correlation between FNC and Age.

<table>
<thead>
<tr>
<th>Network pair</th>
<th>ASD</th>
<th>p(ASD)</th>
<th>TD</th>
<th>p(TD)</th>
<th>whole sample</th>
<th>p(whole)</th>
<th>group difference in slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bg+Th ~ dSI/MI/mPMC</td>
<td>R= 0.02</td>
<td>NS</td>
<td>R= -0.09</td>
<td>p&lt;0.0433</td>
<td>R= -0.03</td>
<td>NS</td>
<td>Z= 1.02</td>
</tr>
<tr>
<td>Bg+Th ~ pfus + V1</td>
<td>R= -0.06</td>
<td>NS</td>
<td>R= -0.13b</td>
<td>p&lt;0.0089</td>
<td>R= -0.10d</td>
<td>p&lt;0.0073</td>
<td>Z= 0.69</td>
</tr>
<tr>
<td>Bg+Th ~ TE+pSTG+PARop</td>
<td>R= -0.06</td>
<td>NS</td>
<td>R= -0.13b</td>
<td>p&lt;0.0086</td>
<td>R= -0.09b</td>
<td>p&lt;0.012</td>
<td>Z= 0.67</td>
</tr>
<tr>
<td>Bg+Th ~ vSI+vMi+pIC</td>
<td>R= -0.04</td>
<td>NS</td>
<td>R= -0.18b</td>
<td>p&lt;0.0015</td>
<td>R= -0.11d</td>
<td>p&lt;0.0026</td>
<td>Z= 1.31</td>
</tr>
<tr>
<td>Bg+Th ~ STS+LH IFG</td>
<td>R= 0.04</td>
<td>NS</td>
<td>R= -0.08</td>
<td>p&lt;0.0267</td>
<td>R= -0.03</td>
<td>NS</td>
<td>Z= 1.11</td>
</tr>
<tr>
<td>TE+pSTG+PARop ~ vSI+vMi+pIC</td>
<td>R= -0.13</td>
<td>p&lt;0.0446</td>
<td>R= -0.03</td>
<td>NS</td>
<td>R= -0.09</td>
<td>NS</td>
<td>Z= -0.87</td>
</tr>
<tr>
<td>CRB anterior ~ STS+LH IFG</td>
<td>R= 0.18a</td>
<td>p&lt;0.0008</td>
<td>R= 0.08</td>
<td>NS</td>
<td>R= 0.14c</td>
<td>p&lt;0.0014</td>
<td>Z= 0.87</td>
</tr>
<tr>
<td>CRB anterior ~ dSI+MI+mPMC</td>
<td>R= 0.14</td>
<td>p&lt;0.0076</td>
<td>R= 0.11b</td>
<td>p&lt;0.0077</td>
<td>R= 0.12c</td>
<td>p&lt;0.0009</td>
<td>Z= 0.27</td>
</tr>
</tbody>
</table>

* p<.0008 (corrected with q(FDR)=.05)
* p<.009 (corrected with q(FDR)=.05)
* p<.012 (corrected with q(FDR)=.05)
NS indicates not significant at p<.05 uncorrected