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ENIGMA ADHD Working Group

ENIGMA ASD Working Group

ENIGMA OCD Working Group

Supplemental Information 1:

Image Exclusion Criteria, Key-MRI feature selection, Age-distribution plots

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Disclosures and Acknowledgments

ENIGMA ADHD Working Group

Disclosures

David Coghill served in an advisory or consultancy role for Lilly, Medice, Novartis, Oxford outcomes, Shire and Viforpharma. He received conference support or speaker's fee by Janssen McNeil, Lilly, Medice, Novartis, Shire and Sunovian. He is/has been involved in clinical trials conducted by Eli Lilly and Shire. The present work is unrelated to the above grants and relationships. *Jonna Kuntsi* has given talks at educational events sponsored by Medice; all funds are received by King's College London and used for studies of ADHD. *Anders Dale* is a Founder of CorTechs Labs, Inc. He serves on the Scientific Advisory Boards of CorTechs Labs and Human Longevity, Inc., and receives research funding through a Research Agreement with General Electric Healhcare. *Paulo Mattos* was on the speakers' bureau and/or acted as consultant for Janssen-Cilag, Novartis, and Shire in the previous 5 years; he also received travel awards to participate in scientific meetings from those companies. The ADHD outpatient program (Grupo de Estudos do Déficit de Atenção/Institute of Psychiatry) chaired by Dr. Mattos has also received research support from Novartis and Shire. The funding sources had no role in the design and conduct of the study; collection, management, analysis, or interpretation of the data; or preparation, review, or approval of the manuscript. *Tobias Banaschewski* served in an advisory or consultancy role for Lundbeck, Medice, Neurim Pharmaceuticals, Oberberg GmbH, Shire, and Infectopharm. He received conference support or speaker's fee by Lilly, Medice and Shire. He received royalities from Hogrefe, Kohlhammer, CIP Medien, Oxford University Press. The

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ENIGMA ASD Working Group

Disclosures

Dr. Anagnostou has served as a consultant or advisory board member for Roche and Takeda; she has received funding from the Alva Foundation, Autism Speaks, Brain Canada, the Canadian Institutes of Health Research, the Department of Defense, the National Centers of Excellence, NIH, the Ontario Brain Institute, the Physicians' Services Incorporated (PSI) Foundation, Sanofi-Aventis, and SynapDx, as well as in-kind research support from AMO Pharma; she receives royalties from American Psychiatric Press and Springer and an editorial honorarium from Wiley. *Dr. Arango* has served as a consultant for or received honoraria or grants from Acadia, Abbott, Amgen, CIBERSAM, Fundación Alicia Koplowitz, Instituto de Salud Carlos III, Janssen-Cilag, Lundbeck, Merck, Instituto de Salud Carlos III (co-financed by the European Regional Development Fund "A way of making Europe," CIBERSAM, the Madrid Regional Government [S2010/BMD-2422 AGES], the European Union Structural Funds, and the European Union Seventh Framework Programme under grant agreements FP7-HEALTH-2009-2.2.1-2-241909, FP7-HEALTH-2009-2.2.1-3-242114, FP7-HEALTH-2013-2.2.1-2-603196, and FP7-HEALTH-2013-2.2.1-2-602478), Otsuka, Pfizer, Roche, Servier, Shire, Takeda, and Schering-Plough. *Dr. Freitag* has served as a consultant for Desitin regarding issues on ASD. *Dr. De Martino* is a coauthor of the Italian version of the Social Responsiveness Scale, for which she may receive royalties. *Dr. Rubia* has received speaking honoraria from Eli Lilly, Medice, and Shire. *Dr.*

Buitelaar has served as a consultant, advisory board member, or speaker for Eli Lilly, Janssen-Cilag, Lundbeck, Medice, Novartis, Servier, Shire, and Roche, and he has received research support from Roche and Vifor. *Dr. Gallagher* received funding from are the Meath Foundation and the National Childrens Research Centre in Ireland. The other authors report no financial relationships with commercial interests.

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ENIGMA OCD Working Group

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Supplemental Information

Supplemental Information SI1

Image Exclusion Criteria:

Prior to data processing scans were visually inspected at each site locally and scans with gross brain pathology, artifacts or poor image quality hampering image segmentation were excluded. A neuroimaging researcher at each site individually examined each segmentation by three major steps following standardized ENIGMA protocols to harmonize quality control procedures across multiple sites.

First, outliers were determined by calculating the interquartile range for each of the volumes per cohort and per group (patients and healthy controls). For each subject that was marked as a statistical outlier, a re-inspection of the subject's segmentation was conducted in 3D to evaluate whether it was segmented properly, and excluded if necessary. Second, cortical segmentations were overlaid directly on a subject's T1-weighted scan and snapshots from internal slices of the brain were presented on a webpage for easy checking. Third, webpages were created with external views of the segmentations from different angles of each subject. For these latter two steps, we instructed the sites to perform extensive quality checking according to a standardized manual (available on http://enigma.ini.usc.edu/protocols/imaging-protocols/) encompassing the most common segmentation errors by FreeSurfer based on our own experience of several samples across different sites and scanners.

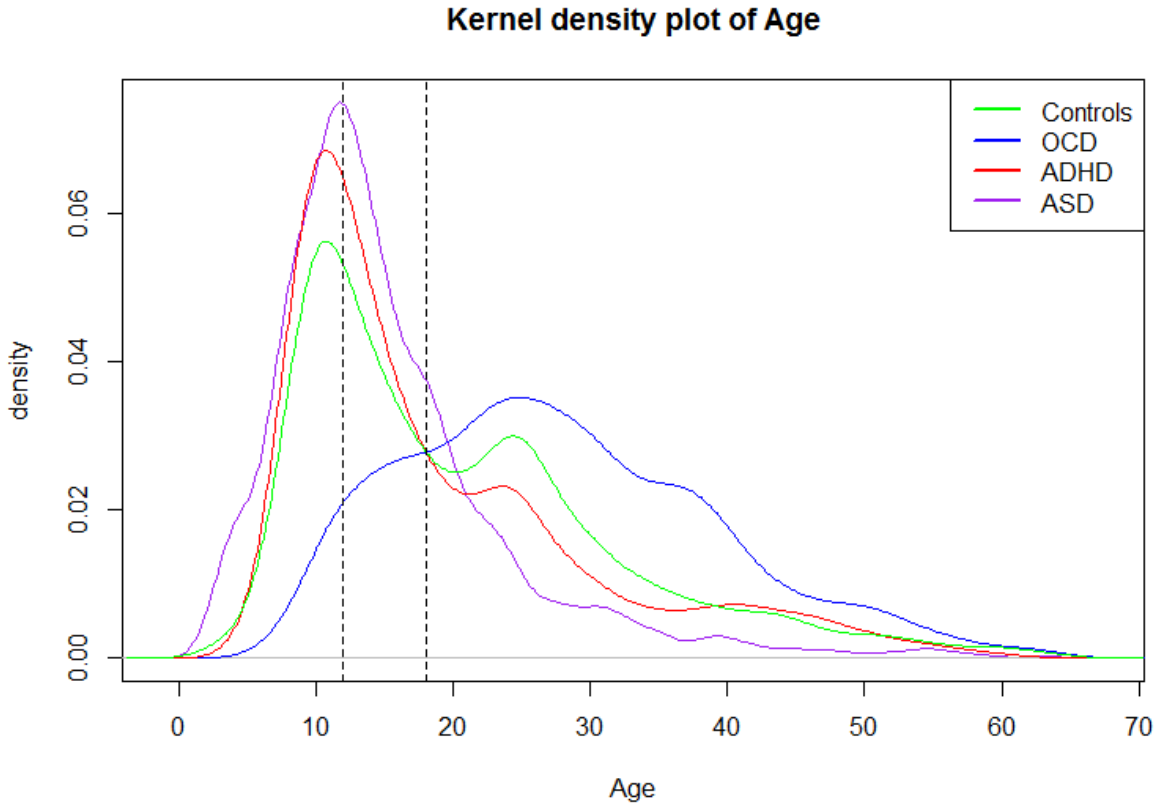
Further, we collected study-wide statistics (means and standard deviations) as well as histogram plots in order to identify non-normally distributed data and major outliers. Lastly, the coordinating PI site per disease working group checked the histograms and summary statistics of every site for possible irregularities that would indicate incorrect segmentations. Where needed, sites were asked to re-inspect suspicious irregularities.

Key-MRI feature selection:

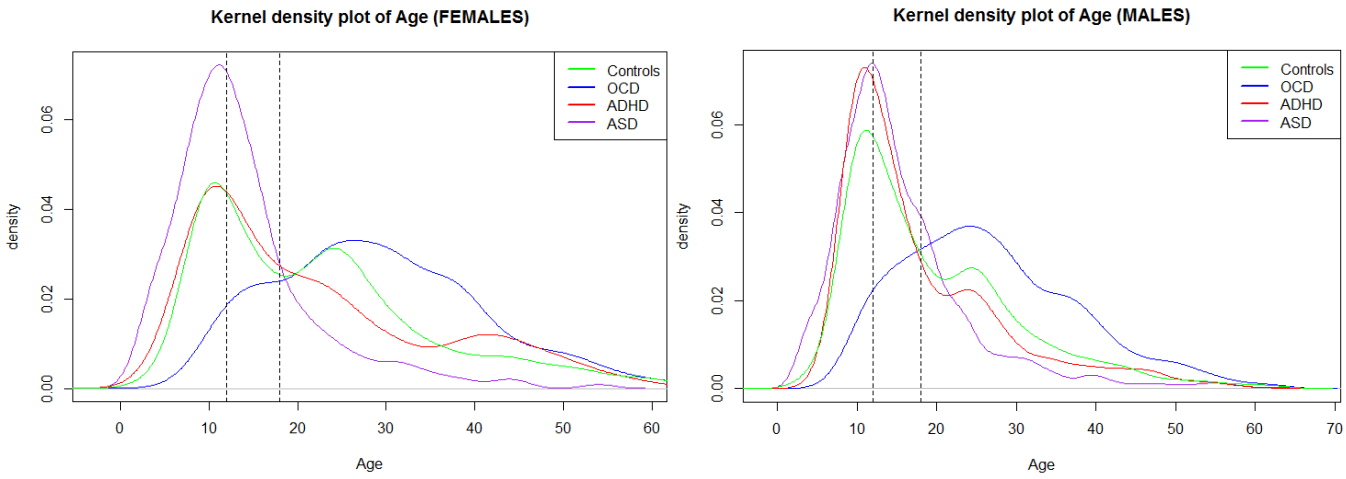
Several sensitivity analyses were performed to provide better insight into the main group effects presented in the main manuscript. However, given the large number of MRI features listed in the main analyses, we have opted to only display analyses on a subset of key MRI features. Specifically, a set of 10 cortical and subcortical MRI features was selected which demonstrate the strongest effect sizes, both between individual diagnostic groups and controls as well as between the different diagnoses. Therefore, these regions serve to best illustrate the size, direction and variance of our main group effects. These features are: Amygdala, Thalamus, Putamen, Hippocampus, ICV, total mean cortical thickness and full surface area, mean Orbitofrontal thickness, Mean Pars Triangularis thickness and Mean Posterior Cingulate thickness

Age-distribution plots:

Presented below are the kernel density plots by age for each diagnostic group, and additionally broken down by gender. These plots indicate that the ASD, ADHD and Control groups have a relatively similar distribution pattern, with the mean age centered around adolescence, and increasingly less coverage with increasing age. The OCD cohort is distributed differently, with a mostly adult population. Age distributions for males and females are almost identical.



SI Figure 1. Kernel density plot for the entire cohort, broken down by diagnostic group.



SI Figure 2. Kernel density plot for males and females separately, broken down by diagnostic group.

Supplemental Information SI2

Trend results

Common subcortical and cortical alterations across clinical groups compared to healthy controls

Children with ADHD and those with ASD showed smaller amygdala volumes compared to control children (significant in children with ADHD *effect size*=-0.10; did not survive FDR-correction in children with ASD *effect size*=-0.09; Supplementary Table S5). *Cortical thickness* analysis revealed that both groups also had thinner cortices of the precentral gyrus (significant in children with ADHD effect size=-0.16, but did not survive FDR-correction in children with ASD *effect size*=-0.19), temporal pole (significant in children with ASD *effect size*=-0.24, but did not survive FDR-correction in children with ADHD effect size=-0.10) and fusiform gyrus (did not survive FDR-correction in both ADHD effect size=-0.14 and ASD effect size=-0.23) compared to control children (Supplementary Table S6). *Surface area* analysis in children with ADHD and those with ASD revealed smaller surface area of the caudal middle frontal gyrus (did not survive FDR-correction in both ADHD effect size=-0.10 and ASD effect size=-0.14; Supplementary Table S13). Adolescents with ADHD (effect size=-0.12) and those with ASD (effect size=-0.11) had a thinner cortex of the parahippocampal gyrus, however this finding did not held in both groups after FDR-correction. Adults with OCD and those with ASD had smaller hippocampal volumes compared to adult controls (significant in patients with OCD effect size=-0.09, but this finding did not survive FDR-correction in patients with ASD effect size=-0.11; Supplementary Table S10).

Disease-specific subcortical and cortical alterations

Children: Smaller hippocampal volumes were observed in children with ASD compared to those with OCD (*effect size*=-0.19), but did not survive FDR-correction. We also observed smaller putamen in children with ADHD compared to those with OCD (*effect size*=-0.23), however this also did not survive FDR-correction (Supplementary Table S14). The following *cortical thickness* results did not survive the FDR-correction, thinner temporal pole (effect size=-0.25) and entorhinal cortex (effect size=-0.29) in children with ASD compared to those with OCD, thinner entorhinal (effect size=-0.19), middle temporal (effect size=-0.22), transverse temporal cortices (effect size=-0.23), and thicker pericalcarine cortex in children with ASD compared to those with ADHD. The following cortical *surface area* results did not survive the FDR-correction, lower surface area of the banks of the superior temporal sulcus (effect size=-0.25) and larger surface area of the paracentral lobule (effect size=0.22) in children with ASD compared to those with OCD, larger surface area of the entorhinal cortex (effect size=0.15), isthmus of the cingulate cortex (effect size=0.18), paracentral lobule (effect size=0.18), and lower surface area of the banks of the superior temporal sulcus (effect size=-0.13) in children with ASD compared to those with ADHD.

Adolescents: Larger thalamus volume in adolescents with OCD compared to those with ASD (effect size=0.16) did not survive multiple comparison correction (Supplementary Table S17). The following *cortical* results did not survive FDR-correction, thinner lateral occipital (effect size=-0.21), thinner posterior cingulate (effect size=-0.20), thinner superior frontal cortices (effect size=-0.17), a lower

surface area of the paracentral lobule (effect size=-0.18) in adolescents with OCD compared to those with ADHD (Supplementary Table S18-S19), and a lower surface area in adolescents with OCD (effect size=-0.16) compared to adolescents with ASD (Supplementary Table S19).

Adults: The following cortical thickness results did not survive FDR correction: a thinner inferior parietal (effect size=-0.17) and lateral occipital cortex (effect size=-0.17) in adults with OCD compared to adults with ADHD and thicker cortices of the pars opercularis (effect size=0.14), pars orbitalis (effect size=0.16), rostral anterior cingulate (effect size=0.16), superior frontal gyrus (effect size=0.21), and frontal pole (effect size=0.15) in adults with ASD compared to those with ADHD (Supplementary Table S21). The following *surface area* results did not survive FDR-correction: lower surface area of the parahippocampal gyrus (effect size=-0.13) in adults with ADHD compared to those with OCD, lower surface area of the transverse temporal cortex (effect size=-0.17) in adults with OCD compared to those with ASD, and larger surface area of the pars triangularis in adults with ASD compared to those with OCD (effect size=0.14) and ADHD (effect size=0.16).