Contrastive AI reveals the structure of individual variation in Autism Spectrum Disorder

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Autism Spectrum Disorder is highly heterogeneous. Identifying systematic individual differences in neuroanatomy could inform diagnosis and personalized interventions. The challenge is that these differences are entangled with variation due to other causes: individual differences unrelated to ASD and measurement artifacts. We used contrastive deep networks to disentangle ASD-specific neuroanatomical variation from individual variation in common with typically developing participants. ASD-specific variation correlated with individual differences in symptoms. The structure of this ASD-specific variation also addresses a long-standing debate about the nature of ASD: individuals do not cluster into subtypes; instead, they are organized along at least two continuous dimensions – one associated with concurrently enlarged temporoparietal junction and somatosensory cortex and compressed superior longitudinal fasciculus, the other associated with changes in the thalamus.

Psychiatric disorders affect hundreds of millions of people worldwide [32]. Heterogeneity is a key obstacle to understanding them: different individuals diagnosed with the same type of disorder often present with unique profiles of behavioral symptoms and with different genetic

variants [66]. We investigated heterogeneity within autism spectrum disorder (ASD): a highly prevalent neurodevelopmental condition [38; 50] characterized by impaired social interactions, restricted patterns of behavior, and communication deficits [70]. Individuals with ASD differ substantially in the severity of social and behavioral symptoms [54; 59]. Moreover, ASD's genetic underpinnings are highly variable, with more than 200 associated copy number variations (CNVs), distributed differently across individuals and affecting multiple distinct pathways [34; 41; 48]. Understanding neuroanatomical heterogeneity within ASD could be pivotal to improving quality of life in affected individuals [26], by leading to more specific diagnoses and targeted behavioral interventions [39].

Recent studies attempted to investigate individual differences in neuroanatomy within the ASD population. However, researchers have struggled to identify systematic neural variation that correlates with symptoms and that replicates across different groups of participants [49; 56; 60; 65]. Different studies found different effects [13; 37; 56] or none at all [5].

We hypothesized that ASD-specific variation is being obscured by the many other factors that lead brains to vary. Brains differ from one to another due to numerous genetic and environmental causes unrelated to ASD [36]. Neuroanatomical data from different individuals, in addition, vary also because of methodological artifacts, such as systematic differences due to the scanners at different scanning sites [30; 43; 45]. ASD-specific variation may be difficult to identify within this mass of irrelevant variation. Researchers have developed several methods for addressing this problem, but they remain unsatisfactory. For instance, case-matching ASD and TD participants works in theory, but it assumes we know exactly what factors we need to match. However, brain anatomy is shaped by a multitude of genetic and environmental factors [36], some of which are unknown. This makes it very difficult (if not impossible) to match them all in practice.

In order to better characterize ASD-specific neuroanatomical variation, we employed an

alternative that disentangles it from variation in common with the general population. Specifically, we modeled structural MRI data using contrastive variational autoencoders (CVAEs, [55]). CVAEs take as inputs samples from two distinct populations, and can be trained to isolate features that capture variation specific to one population from features that are common to both (see Figure S1). We used CVAEs to disentangle features that capture anatomical variation within the ASD group ('ASD-specific') from 'shared' features that capture anatomical variation common to both ASD and TD participants (See Figure 1A).

First, we validated these neuroanatomical features by testing whether the ASD-specific features were differentially related to clinical symptoms while the shared features were differentially related to scanner type and non-clinical individual characteristics. We then replicated the results with a zero-free-parameter generalization to an independent dataset. Next, we explored the structure of the ASD-specific features, applying cluster analysis to determine whether there are distinct subtypes of ASD neuroanatomy. Finally, in order to identify anatomical loci of individual variation, we leveraged the properties of the CVAE to generate counterfactual TD brains closely matched to the brains of the ASD participants ("synthetic twins"). Comparing the anatomy of ASD participants to their synthetic twins allowed us to detect specific brain regions whose alterations vary within the ASD population.

Results

We used the ABIDE dataset – one of the largest available structural MRI datasets including both ASD participants (N = 470) and TD controls (N = 512) [31; 46] – to train a CVAE and a non-contrastive VAE matched in the number of parameters that uses a single latent space but has the same number of latent features as the CVAE. The non-contrastive VAE allows us to test whether associations between neuroanatomy and ASD symptoms can be identified using variational autoencoding alone (effectively, a powerful method for factor analysis) without explicitly

disentangling ASD-specific and shared variation.

Thus, to establish a baseline, we first report the non-contrastive VAE results. We used Representational Similarity Analysis (RSA; [11]) to test whether the VAE's neuroanatomical feature space correlates with individual variation within the ASD participants in key clinical and non-clinical individual characteristics, such as ADOS scores (a numerical measure of ASD symptom severity), Vineland adaptive behavior scores, scanner type, and age. More specifically, we separately calculated the pairwise dissimilarity between participants with respect to the VAE neuroanatomical feature space and with respect to a set of clinical and non-clinical individual characteristics, obtaining a dissimilarity matrix for the VAE feature space and one for each characteristic (Figure 1B). The VAE feature space showed Kendall τ correlations with some of the non-clinical characteristics, such as scanner type (τ = 0.04, t(9) = 16.29, p $<$ .001), age (τ = 0.03, t(9) = 8.27, p $<$ .001) and gender (τ = 0.03, t(9) = 4.71, p = 0.001). While there was some relationship between neuroanatomical feature similarity extracted by VAE and DSM-IV behavioral subtypes (τ = 0.03, t(9) = 4.77, p = 0.001), there was no relationship with autism severity (ADOS total; τ = 0.00, t(9) = -1.08, p = 0.310) or Vineland adaptive behavior scores (τ = 0.00, t(9) = -0.29, p = 0.780). This is consistent with the idea presented above, namely that entangled measures of neuroanatomy (such as VAE features) fail to capture more subtle variations in symptom severity.

We then assessed whether explicitly disentangling ASD-specific and shared neuroanatomical variation with a CVAE would allow us to identify clinically-relevant individual variation. As described above, the CVAE segregates its internal representations into ASD-specific and shared latent features (Figure 1A, Figure S2). The CVAE was trained on the same ABIDE data. Critically, while the CVAE training implicitly makes a binary distinction between ASD and TD subjects, the model is not provided with any of the clinical and non-clinical individual characteristics of interest, such as continuous measures of symptomatology like ADOS. We used RSA

to compare the CVAE's ASD-specific and shared neuroanatomical spaces to each of the clinical and non-clinical individual characteristics. We expected to find that shared features correlate with non-clinical variation common to both ASD and TD participants, while ASD-specific features correlate with clinical ASD variation (Figure 1B).

Scanner type was strongly associated with subject similarity in the shared feature space (τ = 0.11, t(9) = 253.01, p $<$ .001) but not the ASD-specific space (τ = -0.01, t(9) = -14.16, p $<$ .001; shared vs ASD-specific: $\Delta\tau$ = 0.12, t(9) = 124.83, p $<$ .001). Thus, the CVAE was able to factor out a common source of "nuisance" variation in multi-site data [43]. In contrast, measures of ASD clinical symptoms were more associated with the ASD-specific space but generally not the shared space. These include DSM IV behavioral subtypes (ASD-specific: τ = 0.06, t(9) = 30.83, p $<$ .001; shared: τ = 0.02, t(9) = 29.02, p $<$ .001; comparison: $\Delta\tau$ = 0.04, t(9) = 20.04, p $<$ .001), ADOS total score (ASD-specific: τ = 0.01, t(9) = 16.85, p $<$ .001; shared: τ = 0.00, t(9) = -1.50, p = 0.167; comparison: $\Delta\tau$ = 0.01, t(9) = 11.59, p $<$ .001), and Vineland adaptive behavior questionnaire (ASD-specific: τ = 0.05, t(9) = 12.33, p $<$ .001; shared: τ = 0.00, t(9) = 1.17, p = 0.270; comparison: $\Delta\tau$ = 0.05, t(9) = 10.46, p $<$ .001) (see also Figure S4, SOM). Thus, the CVAE was not only able to disentangle neuroanatomical variation specific to ASD from general-population variation, but this neuroanatomical variation was differentially associated with ASD-specific and general-population phenotypic and demographic characteristics.

Results for age, gender, and full-scale IQ (FIQ) were of particular interest, since these are known to differently relate to neuroanatomy in the TD and ASD populations [33; 62]. Thus, it is not surprising that each of these was significantly related to both the ASD-specific space (age: τ = 0.05, t(9) = 48.60, p $<$ .001; gender: τ = 0.02, t(9) = 8.13, p $<$ .001, FIQ: τ = 0.02, t(9) = 20.22, p $<$ .001) and the shared space (age: τ = 0.08, t(9) = 89.29, p $<$ .001, gender: τ = 0.05, t(9) = 35.34, p $<$ .001, FIQ: τ = 0.01, t(9) = 15.57, p $<$ .001), indicating that the CVAE was able to disentangle general effects of age, gender, and FIQ from those that specifically

interact with ASD. As expected, shared features captured greater variation in age and gender than ASD-specific features (age: $\Delta\tau$ = 0.03, t(9) = 24.11, p $<$ .001; gender: $\Delta\tau$ = 0.03, t(9) = 11.90, p $<$ .001). Conversely, variation in full-scale IQ was more related to ASD-specific than shared features ($\Delta\tau$ = 0.01, t(9) = 12.86, p $<$ .001).

In sum, the CVAE was not only able to disentangle individual neuroanatomical variation that is specific to ASD from variation that characterizes the population as a whole, but these patterns of variation were differentially associated with clinical and non-clinical participant characteristics. This contrasts with the control VAE model, whose unitary neuroanatomical space showed weaker correlations with individual characteristics. Indeed, there was no individual characteristic that was most strongly associated with the control VAE space (Figure S4, SOM).

Generalization to an independent dataset.

Generalization to a new dataset that was not used for training is a very stringent test of a model's performance. In the context of diagnosis, generalization across datasets is especially important, because a model trained on one group of participants may need to be used to inform the diagnosis of novel participants that were not included in the training dataset. To test generalization to a new dataset, we applied the ABIDE-trained CVAE to the anatomical scans of participants from the SFARI VIP dataset (N = 121) [22] using a parameter-free fit. SFARI VIP is a particularly interesting test case because it includes information about ASD-relevant CNVs, allowing us to study whether ASD-specific neuroanatomical features correlate with genotype.

We expected that if CVAE features were robust, shared features should have high correlations with properties of scanning site, age and gender, and ASD-features should correlate with ASD-related individual differences such as DSM-IV behavioral subtypes. This is exactly what we found. Compared to ASD-specific features, shared features correlated better with scanner-type ($\Delta\tau$ = 0.09, t(9) = 12.81, p $<$ .001), age ($\Delta\tau$ = 0.06, t(9) = 15.09, p $<$ .001) and gender

($\Delta\tau$ = 0.01, t(9) = 3.17, p = 0.011). In contrast, ASD-specific features, compared to shared features, had higher Kendall tau values for DSM-IV behavioral subtypes ($\Delta\tau$ = 0.01, t(9) = 2.34, p = 0.044) suggesting that CVAE identified population-wide patterns of neuroanatomy, some of which are shared between ASD and non-ASD participants, and some of which are only present in those with ASD. Additionally, the SFARI VIP dataset allowed us to ask whether neuroanatomical differences observed in 16p11.2 deletion and duplication carriers are consistent with patterns of variation in the typically developing population, or whether they match patterns of variation within ASD. Similarity between deletion and duplication CNVs was better reflected in ASD-specific than shared features ($\Delta\tau$ = 0.05, t(9) = 14.54, p $<$ .001).

The nature of variation

Researchers have long debated whether individual differences in ASD are better understood as distinct subtypes or as variation along continuous dimensions [24; 49; 57; 65]. Having identified a space of ASD-specific features makes it possible to test these hypotheses directly. We used Gaussian mixture modelling [2] to identify clusters of subjects in each of the feature spaces, and we used the Bayesian Information Criterion (BIC [1]) to select the optimal number of clusters (Figure 1D).

The distribution of subjects in the VAE feature space, which conflates ASD-specific and shared variation, was consistent with a single cluster (p $<$ .01): one cluster was the optimal solution for 100/100 samples of the latent space (Figure 1D). The CVAE results were more nuanced. The shared features showed evidence of subtypes, revealing more than one cluster (p $<$ .01): the one cluster solution was optimal in 0/100 samples. Instead, the three cluster solution provided the best fit (selected in 88/100 samples), followed by the two cluster solution (12/100 samples) (binomial two-tailed test p $<$ .001). However, the subject distribution in the space of ASD-specific features again suggested continuous variation: the optimal solution assigned all

participants to a single cluster in 100/100 samples ($p < .01$). Thus, the results of cluster analysis show that once disentangled from typical variation, ASD-related neuroanatomical variation is better captured by continuous dimensions, rather than by discrete categories.

Neuroanatomical interpretation

Even though variation in the space of ASD-specific features is better described by continuous dimensions than by distinct categories, different dimensions might nonetheless be associated with different anatomical changes. Whether ASD-specific individual differences in neuroanatomy are focal (affecting one or few regions), or distributed (affecting many regions) - remains an open question. To address it, we tested whether different ASD-specific features are associated with neuroanatomical changes in localized brain regions. We identified loci of anatomical variation between ASD subjects following a three step process. First, for each ASD participant, we generated a simulated brain of a counterfactual "synthetic TD twin" by setting ASD-specific feature values to zero and using the CVAE decoder to reconstruct their brain only using the inferred shared features (see: Methods in Supplementary Materials (SM)). This "synthetic twin" is effectively a case-control for each ASD participant's brain, matched on shared variation features (including scan site artifacts as well as neuroanatomical variation due to age, gender, and other dimensions discovered from the data by the CVAE). In the second step, we estimated a nonrigid transformation morphing the counterfactual TD brain to match the corresponding ASD participant's brain. This produced a vector field describing the differences between the ASD brain and the corresponding TD brain (see: Methods in SM). Finally, we calculated the Jacobian determinant of the vector field. This measure captures the local volumetric compression/expansion needed to morph the simulated TD brain into the corresponding ASD brain. Repeating this procedure for multiple participants with different values of ASD-specific features, we computed interpretable gray and white matter alterations that vary across the ASD

participant population.

To organize the search of interpretable neuroanatomical features, we projected the 16-dimensional space of ASD-specific neuroanatomical features on a two-dimensional subspace (using UMAP, [53]). We then measured systematic variation in the compression/expansion of different brain regions along each of the two principal dimensions by comparing the Jacobian determinant maps of subjects ranking high (N = 50) and low (N = 50) on each dimension (Figure 2). By focusing on the two largest, most informative UMAP dimensions, we simplify interpretation and reduce the number of comparisons. Using more dimensions might reveal additional anatomical loci, but it would diminish power due to the need to correct for multiple comparisons.

Comparing extremes of the x dimension (negative vs. positive), we observed enlarged parietal cortex and compressed superior longitudinal fasciculus (SLF). The affected portion of parietal cortex (734 voxels in size) is centered in secondary somatosensory cortex (SII, MNI x = 62 y = -22 z = 24) and extends posteriorly into the temporoparietal junction (TPJ). The SLF cluster (466 voxels in size) is centered at MNI coordinates x = 32 y = -22 z = 30, and extends anteriorly terminating in the inferior frontal gyrus (BA44/BA45 MNI x = 42 y = 22 z = 30). Anatomical locations of SLF and SII were confirmed using the Juelich Histological Atlas [61]. The TPJ location was confirmed using Neurosynth's [19] meta-analytic activation maps generated using the "theory of mind" search term (meta analysis of 181 studies).

Comparing extremes of the y dimension (negative vs. positive), we observed deformations corresponding to the thalamus bilaterally - more prominently expressed on the left hemisphere. The deformation cluster spanned 537 voxels and was centered at MNI x = 6.35, y = -12.31 z = .53, $p < .048$ (cluster-level FWE corrected, two-tailed test). We used the Oxford thalamic connectivity probability atlas [4] to determine the anatomical location more precisely. This procedure confirmed that the cluster is centered on a subdivision of the thalamus with predom-

inant connections to prefrontal cortex previously associated with individual differences in ASD symptom severity, such as ADOS scores [42; 68].

Discussion

Behavioral heterogeneity is a defining characteristic of ASD; to understand its biological basis we need to identify systematic neural heterogeneity. Furthermore, individuals with similar behavioral symptoms might have different anatomical alterations that could help clinicians to select distinct, tailored interventions.

Unfortunately, identifying systematic neuroanatomical variation between individuals with ASD has proven challenging [44; 65], due to variation caused by ASD-unrelated factors. Using a contrastive variational autoencoder (CVAE), we revealed a space of neuroanatomical features that captures ASD-specific variation, disentangled from neuroanatomical variation common to both ASD and TD participants. We then found that individual differences in ASD-specific neuroanatomical features are associated with individual differences in diagnosis and ASD-related genotype. Additional analyses identified two distinguishable components of this neural variation, linked to localized anatomical changes. One dimension of neuroanatomical variability is right lateralized and affects the secondary somatosensory cortex (SII), the temporoparietal junction (TPJ), and the superior longitudinal fasciculus (SLF); the other affects the thalamus bilaterally. These findings demonstrate that different variants of ASD can have a concerted impact on multiple anatomical structures, affecting in conjunction cortical areas and white matter tracts.

Analyses further demonstrated that ASD-specific neuroanatomical differences are best characterized by continuous variation along at least two dimensions (Figure 2), rather than by distinct subtypes as suggested by prior work [49; 57; 65]. This evidence indicates that – at least at the level of neuroanatomy – dimensional approaches (such as the Research Domain Crite-

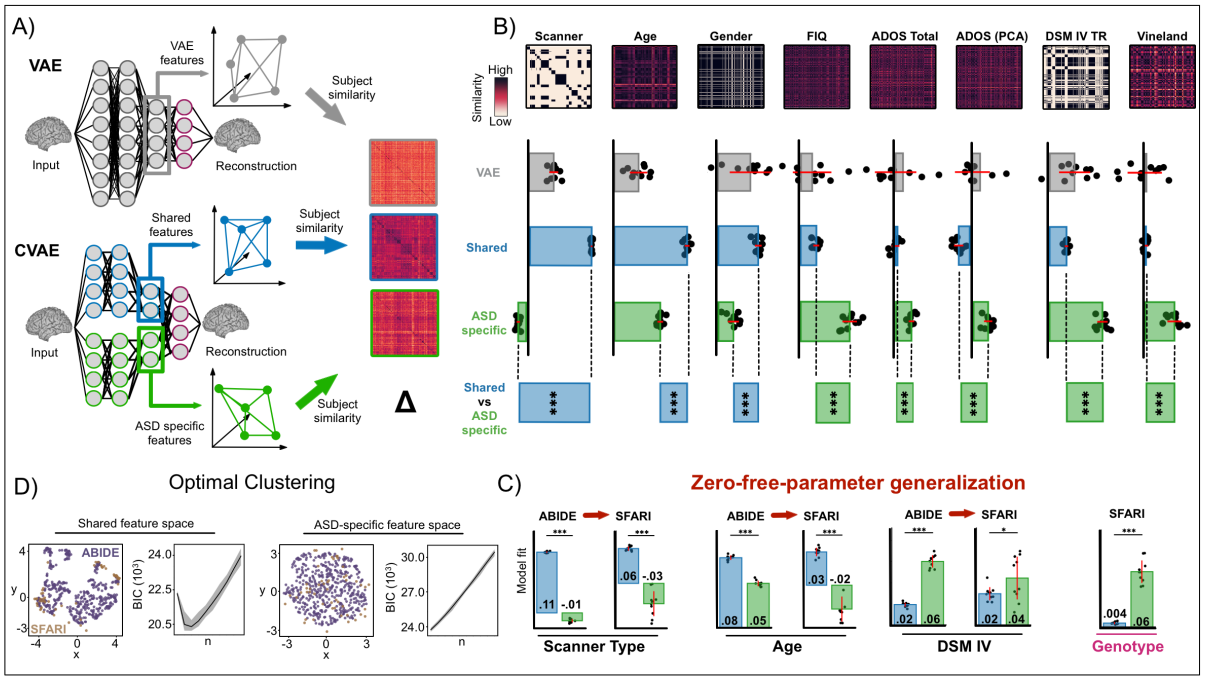

Figure 1: Patterns of individual variation captured in the learned latent spaces. **A)** Representations of the subjects' neuroanatomy in the three latent feature spaces (VAE: gray, CVAE shared: blue, and CVAE ASD-specific: green) are used to construct neuroanatomical similarity matrices. **B)** Neuroanatomical similarity matrices are compared to models of similarity based on different participant properties (e.g. demographic variables, clinical measurements). Significance values indicated by stars, $p < .05$ (*), $p < .0001$ (***). Variables common to TD and ASD participants are best captured by the shared latent neuroanatomical features. Variables associated with ASD-related variation are best captured by the ASD-specific latent neuroanatomical features. Model fit for control model (VAE) is noticeably poorer across all variables. **C)** *Zero-free-parameter generalization*. RSA analyses using SFARI data that was not used to train or tune the CVAE. In both ABIDE and SFARI datasets, the shared space shows higher Kendall τ values with variation in scanner-type and age, and ASD-specific space has higher Kendall τ values with variation associated with ASD severity (DSM IV behavioral subtypes). Participants sharing similar copy-number-variations associated with increased risk of ASD (16p11.2 deletion or duplication) are more similar in ASD-specific, but not shared neuroanatomical features. **D)** *Optimal clustering*. Scatter-plots show inter-subject similarity of ABIDE ASD (Purple dots, N = 492) and SFARI 16p11.2 CNV (Orange dots, deletion N = 26, duplication N = 25) subjects in shared and ASD-specific feature spaces after dimensionality reduction using UMAP. Line plots show Gaussian mixture model fit (Bayesian Information Criterion; BIC) for different numbers of clusters (1-10). Clustered structure can be seen in the shared feature space (optimal clustering n = 3) but not in the ASD-specific feature space (optimal clustering n = 1).

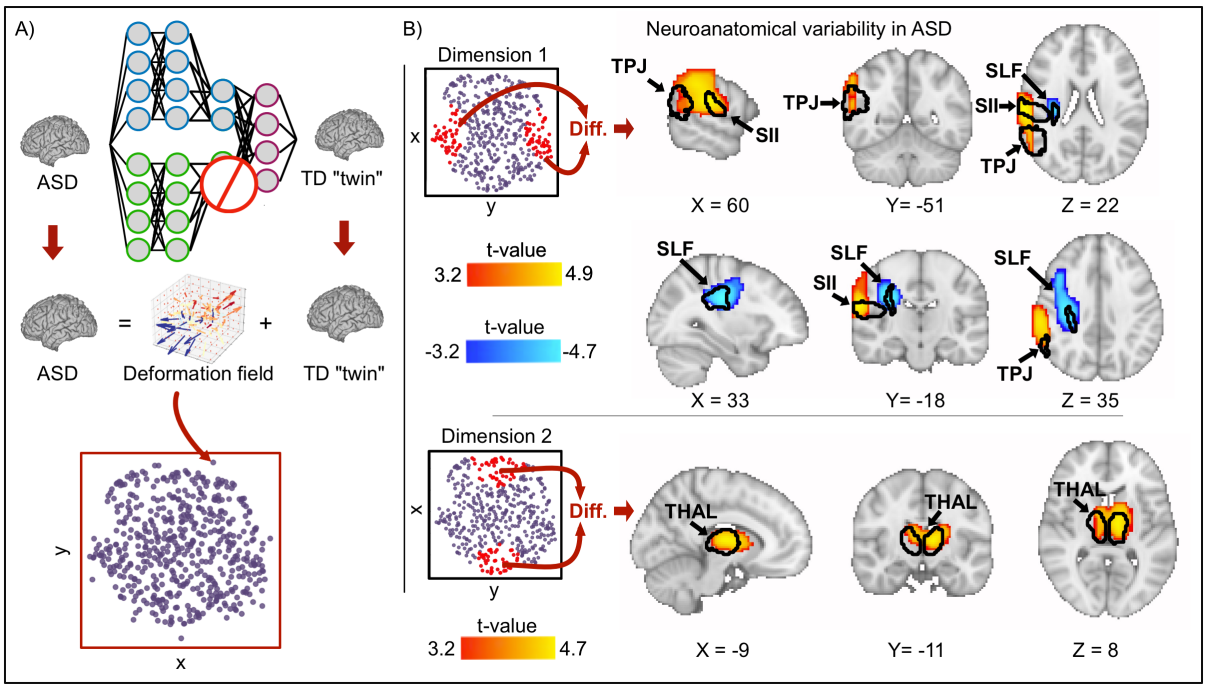

Figure 2: Anatomical loci of individual variation within the ASD group. **A)** For each ASD subject we calculated a synthetic "TD-twin" brain that is matched on ASD-unrelated neuroanatomical features, by setting to zero the features capturing ASD-specific variation. We then computed a nonrigid transformation required to morph the synthetic "TD-brain" into the corresponding ASD brain, obtaining a deformation field. **B)** Comparing the deformation fields along different dimensions in the space of ASD specific features (after UMAP dimensionality reduction) revealed individual variation in specific anatomical loci. Differences along the x-axis revealed an expansion of the parietal lobe, accompanied by a compression of the superior longitudinal fasciculus (SLF). The parietal effects were centered on the secondary somatosensory cortex (SII) and extended posteriorly into temporoparietal junction (TPJ). Along the y-axis we observed expansion of the thalamus (THAL), and specifically in subregions of thalamus connected with prefrontal cortex. Anatomical locations were confirmed using the Juelich histological atlas (SLF, SII), Oxford Thalamic Connectivity atlas (Thalamus) and Neurosynth meta-analytic activation map for theory-of-mind tasks (TPJ).

ria: RDoC, [71]) can provide a better account of individual variation than discrete diagnostic categories.

Individual differences in ADOS and Vineland scores were uniquely associated with ASD-specific neuroanatomical features, and differences introduced by the types of fMRI scanners used were uniquely associated with shared features. Of particular interest were properties that are shared by TD and ASD participants, but are linked to distinct development patterns in the ASD population: age, gender and FIQ [62]. These properties showed positive Kendall τ values for both shared and ASD-specific anatomical features, suggesting that they have an impact on both. Previous studies have documented how the brain matures differently in ASD, and that boys and girls with ASD have different ASD-related neuroanatomical differences [62]. Higher FIQ scores are associated with thicker cortex in TD participants, but with thinner cortex in ASD [62]. The significant correlations between the ASD-specific space and these properties indicate that its features are able to capture ASD-specific patterns of age, gender and FIQ related variability. More broadly, the ability of the CVAE to capture interactions between ASD and development makes it a promising approach to investigate other developmental disorders as well.

Our analyses suggested a key role for two dimensions of neuroanatomical variation. These two dimensions organize a variety of prior findings within a unified framework. One dimension of variability was characterized by a concurrent expansion of the parietal cortex, covering somatosensory and temporoparietal cortices, and compression of the SLF. Greater somatosensory cortex gray matter volume is associated with ASD-related tactile sensitivity [7; 35; 47]. Impaired anisotropy in the SLF, as revealed by diffusion tensor imaging, correlates with language delay in ASD [21] and successful behavioral intervention improving ASD-associated language outcomes proportionately improves the integrity of the SLF tract [58]. Our results showing that somatosensory cortex and SLF abnormalities covary within ASD help elucidate established be-

havioral observations that hypo-reactivity to sensory stimuli (including touch) is associated with impaired language development in children with ASD [9; 25].

The posterior part of the parietal cluster showed substantial overlap with a portion of right TPJ (rTPJ) within the Theory of Mind network: a key region for social cognition. Social impairments are a defining symptom of ASD [70]. Evidence of structural abnormalities is pivotal in establishing this region as a potential locus of social cognition related symptoms in ASD, but reports of altered cortical structure in rTPJ have been scarce [16; 23]. In addition, functional MRI studies have found largely similar amounts of activity in the rTPJ of ASD and TD participants [28]. However, recent work using multivariate analyses identified atypical development of rTPJ representations in ASD participants [64]. Our findings provide converging evidence of rTPJ alterations in ASD, and suggest that the observed differences in fMRI response patterns are likely due to abnormal gray matter development in this region (and not just to upstream alterations affecting the inputs received by rTPJ).

Another axis of variability was localized entirely in the thalamus, consistent with prior observation of substantial thalamic variability in ASD [6; 17; 20] such as above-average and below-average thalamic volumes in 16p11.2 duplication and deletion CNV [34]. While the relationship between thalamic variability and specific ASD symptom domains remains unclear [8; 51; 68], genetic studies have observed thalamic impairments to be common across multiple ASD associated mutations, particularly copy number deletions [52; 63; 67].

Considering ASD as a case study, the links between the present results and the previous literature provide an illustration of how computational psychiatry [40], and more specifically large-scale studies of individual differences that disentangle disorder-specific neural variation, can contribute to our understanding of complex disorders, enabling a synthesis of findings across multiple specialized studies focusing on particular facets.

To reach a more comprehensive understanding of ASD, several future steps are needed.

First, structural variation does not exhaust the ways in which brains can differ: results may differ for functional connectivity, diffusion tensor imaging, or evoked responses. Critically, the methods presented here can be straightforwardly applied to other data modalities. Second, the scope of our investigation of genotype and its relation to neuroanatomical variation was limited by the data available. Datasets with richer genetic information could reveal groups of patients with distinct genetic alterations affecting common pathways. Finally, finer grained measures of behavior will be needed to gain a deeper understanding of the interplay between genetics, anatomy and symptoms.

Our findings were enabled by a key methodological innovation: the use of CVAEs to parcel individual variation into shared variation and variation specific to ASD. As compared to classical case-control matching, this approach has the advantage that it can discover anatomical features that vary also among TD participants in a data-driven fashion. Importantly, this makes it possible (in theory) to control for confounds that are difficult to measure explicitly, such as ASD-unrelated genetic variation and effects of the environment [36]. The results in the present study show that the CVAE is effective in practice, and the comparison with a VAE matched in the number of parameters demonstrates that the contrastive architecture is essential. The features discovered by the CVAE generalize well to an independent dataset – this is especially important for the study of psychiatric disorders, because a model trained with a given dataset may need to be applied to data from new participants to inform diagnosis. Importantly, this novel approach for identifying disorder-specific variability in neuroimaging data can be applied to a variety of neurodevelopmental disorders, paving the way to the search for neurally-informed personalized interventions.

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