family members). These novel, emergenic, phenomena may arise in the individual from interacting co-occurring traits or the interaction of underlying genetic and biological factors, and are exemplified by seizures and intellectual disability. Consideration of the role of emergence in autistic neurobiology and behavior complements a reductionist approach and may help illuminate the components and complexities of autism.

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We-D-15

**Recent cognitive and FMRI data bridge cellular to behavioral alterations in autism, and support the enhanced perceptual functioning model**

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We now dispose of multiple, replicated behavioral (visual search, block-design, embedded figure task) and fMRI (superior activation of expertise regions during pattern processing) results demonstrating enhanced mid-level perception in the visual modality in autism. There also some findings in the same direction in low-level visual perception (symmetry detection, luminance discrimination). In the auditory modality, this pattern is reversed, with strongly replicated findings demonstrating enhanced low level perception (pitch discrimination) and some results in the same direction in mid-level perception (enhanced local processing of melodies). These perceptual atypicalities have been accounted for by a common enhanced perceptual functioning model (Mottron et al., 2006). However, we ignore if these enhanced performances result from a predisposing alteration of perceptual neural networks, common to both perceptual modalities, or from a secondary and compensatory overtraining favouring different processes and modality for each subject. We now present the first series of data demonstrating a common alteration in low-level auditory and visual perceptual processes in autism. These data represent a strong argument in favour of an alteration of neural networks mediating temporal and spatial frequency processing, which may yield cascade effects in most, if not all, cognitive operations performed by the autistic brain.

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We-D-16

**Nonsyndromic autism: On the waiting list of syndromic autism?**

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Advances in clinical and molecular genetics have increased the current state of knowledge on genetic disorders associated with autism. First, a review of various genetic disorders associated with autism will be presented and discussed with regard to possible common underlying mechanisms leading to a similar autistic behavioral syndrome. In particular, the role of epigenetic mechanisms will be emphasized. Finally, the pertinence of distinguishing nonsyndromic autism (isolated autistic disorder) from syndromic autism (autistic disorder related to known genetic disorders) will be reconsidered, given that nonsyndromic autism could be viewed as related to currently unknown disorders on the waiting list of syndromic autism. It highlights the need to conduct systematically a clinical genetic examination searching for underlying genetic disorders in all individuals with autistic behavioral syndrome.

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