Prefixes

- **schizo-**: split 分裂
  - schizophrenia 精神分裂症，schizocarp 双悬果， schizogenesis 裂殖
- **auto-**: self 自我
  - autism 自闭症
- **cortex-**: outer layer
  - cortical 皮层的，cortex皮层
- **homo-**: same
  - homologous同源的，homogeneous 均匀的，homogeneity 同质性
- **hetero-**: different
  - heterologous，heterogeneous，heterogeneity
- **commensal-**: sharing
  - commensal 共生的, commensalism 共生现象
- **opt-**: eye, vision
  - optic optical视觉的 眼睛的 光学的，optogenetic光遗传学的

Suffixes

- **-iatric**: relating to medical treatment
  - neuropsychiatric 神经心理的， pediatric 儿科的
- **spectrum**: range
  - spectrum 光谱, broad-spectrum 广谱的, autism spectrum 自闭症谱系
- **-ome, -omic, -omics**: 组，组学的，组学
  - genome, microbiome, connectome, transcriptome
- **-itis**: inflammation
  - hepatitis 肝炎
- **-oid**: like, resembling, similar
  - steroid 类固醇 （cholesterol 胆固醇，chole 胆，sterol固醇，甾醇），corticoid = corticosteroid 皮质类固醇

[1] Neuropsychiatric genome-wide association studies (GWASs), including those for autism spectrum disorder and schizophrenia, show strong enrichment for regulatory elements in the developing brain. **[2]** However, prioritizing risk genes and mechanisms is challenging without a unified regulatory atlas. Across 672 diverse developing human brains, we identified 15,752 genes harboring gene, isoform, and/or splicing quantitative trait loci, mapping 3739 to cellular contexts. **[3]** Gene expression heritability drops during development, likely reflecting both increasing cellular heterogeneity and the intrinsic properties of neuronal maturation. Isoform-level regulation, particularly in the second trimester, mediated the largest proportion of GWAS heritability. **[4]** Through colocalization, we prioritized mechanisms for about 60% of GWAS loci across five disorders, exceeding adult brain findings. Finally, we contextualized results within gene and isoform coexpression networks, revealing the comprehensive landscape of transcriptome regulation in development and disease.

[1] The complexity and heterogeneity of schizophrenia have hindered mechanistic elucidation and the development of more effective therapies. Here, we performed single-cell dissection of schizophrenia-associated transcriptomic changes in the human prefrontal cortex across 140 individuals in two independent cohorts. [2] Excitatory neurons were the most affected cell group, with transcriptional changes converging on neurodevelopment and synapse-related molecular pathways. [3] Transcriptional alterations included known genetic risk factors, suggesting convergence of rare and common genomic variants on neuronal population-specific alterations in schizophrenia. [4] Based on the magnitude of schizophrenia-associated transcriptional change, we identified two populations of individuals with schizophrenia marked by expression of specific excitatory and inhibitory neuronal cell states. [5] This single-cell atlas links transcriptomic changes to etiological genetic risk factors, contextualizing established knowledge within the human cortical cytoarchitecture and facilitating mechanistic understanding of schizophrenia pathophysiology and heterogeneity.

[1] Recent studies suggest that human-associated bacteria interact with host-produced steroids, but the mechanisms and physiological impact of such interactions remain unclear. **[2]** Here, we show that the human gut bacteria *Gordonibacter pamelaeae* and *Eggerthella lenta* convert abundant biliary corticoids into progestins through 21-dehydroxylation, thereby transforming a class of immuno- and metabo-regulatory steroids into a class of sex hormones and neurosteroids. **[3]** Using comparative genomics, homologous expression, and heterologous expression, we identify a bacterial gene cluster that performs 21-dehydroxylation. **[4]** We also uncover an unexpected role for hydrogen gas production by gut commensals in promoting 21-dehydroxylation, suggesting that hydrogen modulates secondary metabolism in the gut. **[5]** Levels of certain bacterial progestins, including allopregnanolone, better known as brexanolone, an FDA-approved drug for postpartum depression, are substantially increased in feces from pregnant humans. **[6]** Thus, bacterial conversion of corticoids into progestins may affect host physiology, particularly in the context of pregnancy and women's health.

[1] Flies groom in response to competing mechanosensory cues in an anterior-to-posterior order using specific legs. From behavior screens, we identified a pair of cholinergic command-like neurons, Mago-no-Te (MGT), whose optogenetic activation elicits thoracic grooming by the back legs. **[2]** Thoracic grooming is typically composed of body sweeps and leg rubs in alternation, but clonal analysis coupled with amputation experiments revealed that MGT activation only commands the body sweeps: initiation of leg rubbing requires contact between the leg and thorax. **[3]** With new electron microscopy (EM) connectome data for the ventral nerve cord (VNC), we uncovered a circuit-based explanation for why stimulation of posterior thoracic mechanosensory bristles initiates cleaning by the back legs. **[4]** Our previous work showed that flies weigh mechanosensory inputs across the body to select which part to groom, but we did not know why the thorax was always cleaned last.

[5] Here, the connectome for the VNC enabled us to identify a pair of GABAergic inhibitory neurons, UMGT1, that receives diverse sensory inputs and synapses onto both MGT and components of its downstream circuits. **[6]** Optogenetic activation of UMGT1 suppresses thoracic cleaning, representing a mechanism by which mechanosensory stimuli on other body parts could take precedence in the grooming hierarchy. **[7]** We also anatomically mapped the pre-motor circuit downstream of MGT, including inhibitory feedback connections that may enable rhythmicity and coordination of limb movement during thoracic grooming. **[8]** The combination of behavioral screens and connectome analysis allowed us to identify a neural circuit connecting sensory-to-motor neurons that contributes to thoracic grooming.