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MEETING ABSTRACT

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Dopamine in schizophrenia: where does it stand in the cascade of pathological events?
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The dopamine (DA) dysfunction in schizophrenia has recently moved from being a hypothesis based on clinical observations, to a stage of refined and topographically precise sets of alterations documented across multiple labs with positron emission tomography (PET) imaging studies, that showed excess striatal dopamine synthesis, release, and D2 supersensitivity [1]. Recent data suggest also profound extrastriatal deficits in DA release [2]. The presence of opposing findings of striatal excess and extrastriatal deficit including midbrain deficit is puzzling as it suggests that striatal excess may not be a consequence of midbrain DA cells overactivity. A mechanistic understanding of the DA dysfunction is missing. Animal models may shed some light on the pathogenic mechanisms involved. In particular, the D2 overexpressing (D2-OE) [3] mouse has shown that cortical-dependent cognitive deficit and abnormal cortical DA signaling can be a consequence of developmental abnormalities in striatal D2 stimulation. Since many developmental factors, both genetic [4] and environmental, have been shown to be at play in schizophrenia, and shown to affect dopaminergic indices, these combined lines of evidence suggest that DA dysfunction may be an early event leading to additional consequences on the rest of the circuitry and behavior. We will review the evidence critically and develop a model as well as future directions to move this understanding further.

References

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