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# Editorial: New insights into investigating schizophrenia as a disorder of molecular pathways

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## KEYWORDS

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## Editorial on the Research Topic

### New insights into investigating schizophrenia as a disorder of molecular pathways

Schizophrenia (SZ) is a complex disorder with ~1% incidence world-wide involving multiple risk factors such as chemical imbalance (e.g., neurotransmitters), infection (e.g., Toxoplasmosis), genetic susceptibility (e.g., three-fold familial risk with a first-degree relative) and epigenetic factors (e.g., high discordance rates in monozygotic twins) (Saeima *et al.*, 2021). Many genetic and animal model studies have been carried out to date, but we are far from a complete understanding of the molecular/physiological basis for the onset and progression of SZ (e.g., Winship *et al.*, 2019; Trubetskov *et al.*, 2022). Considerable efforts were also directed for targeting molecules suspected to be involved in positive and negative symptoms to develop antipsychotic drugs [reviewed in Gorell (2020)]. However, these efforts have not yet provided completely curable or long-term solutions, mainly because of the lack of comprehensive understanding of the basic mechanisms, processes involving relapse, heterogeneity in the molecular abnormalities in patients, etc. (e.g., Farsi and Sheng, 2023).

Among the different lines of research involved in studying the basic mechanisms, the present article collection focuses on molecular pathways involved in SZ. In general, diverse sets of molecular pathways, broadly belong to four main categories of cellular signaling that are essential for various neurodevelopmental processes such as neuron cell survival, growth, death, neuron-to-neuron signaling, etc. (Table 1). Many of these pathways have been used to study the effects of some commonly used antipsychotic drugs, development of new generation of drugs and understand the basis of non-responsiveness in some cases. Nevertheless, as mentioned above, both basic and applied research are needed for better diagnosis and management of SZ.

Among the collection of articles under the Research Topic, one investigation involved the programmed cell death—associated genes dysregulated in SZ patients (Feng and Shen) who used transcriptome data from dorsolateral prefrontal cortex from the publicly available database containing 58 SZ patients and 175 controls as discovery group. The choice of cell death—related genes was also important because of the observations that the SZ patients showed accelerated aging effects with loss of gray and white matter (Cropley *et al.*, 2017). Out of the 2,684 differentially expressed genes (DEGs) identified, 263 were among the genes linked to programmed cell death. Following extensive bioinformatic

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