Thioridazine is a piperadine phenothiazine derivative classified as a ‘typical’ antipsychotic for its potent inhibition of D2 receptors. Thioridazine displays activity at D1-5 receptors, H1/2 histamine receptors, M1-5 muscarinic acetylcholine receptors (mAChRs), α1/2-adrenergic receptors, and 5-HT1/2/5/6/7 receptors; thioridazine also modulates activity of the norepinephrine transporter (NET). In addition to its well-established antipsychotic and sedative activities, thioridazine also exhibits antibacterial, anti-angiogenic, and anticancer properties. In vitro, thioridazine enhances β-lactam antibacterial capabilities; this compound inhibits peptidoglycan synthesis by interfering with the formation of pentaglycine branches and inducing amino acid shortages. Thioridazine inhibits phosphorylation of Akt, PDK-1, mTOR, and p70S6K, inhibiting migration, invasion, and capillary-like tube formation of cells. In cervical and endometrial cancer cells, thioridazine downregulates expression of cyclins D1 and A as well as cyclin-dependent kinase 4 (CDK4) and upregulates expression of p21 and p27, inducing apoptosis. In vivo, this compound decreases colony-forming units of *Mycobacterium tuberculosis*, inducing expression of the sigma6 regulon and Rv3160c-Rv3161c operon. Thioridazine, like other antipsychotics, also inhibits hERG K+ channels, potentially inducing QT prolongation and acts as a functional inhibitor of acid sphingomyelinase (FIASMA).

**References**


