Pirin

- Nuclear Pirin promotes HCC by acting as a key inflammation-facilitating factor
- Effects of Pirin-like proteins on PAT biosynthesis and stress resilience in postharvest apple pathogen Penicillium expansum
- Corrigendum to "Pirin is a nuclear redox-sensitive modulator of autophagy-dependent ferroptosis" [Biochem. Biophys. Res. Commun. 536 (2021) 100-106]
- The m6A hypermethylation-induced PIR overexpression regulates H3K4me3 and promotes tumorigenesis of uveal melanoma
- Inhibition of mast cell activation by Jaranol-targeted Pirin ameliorates allergic responses in mouse allergic rhinitis
- Genome-Wide Identification and Characterization of the Pirin Gene Family in Nicotiana benthamiana
- Mechanistic insights into Rho/MRTF inhibition-induced apoptotic events and prevention of drug resistance in melanoma: implications for the involvement of pirin
- A plasmid-chromosome crosstalk in multidrug resistant enterobacteria

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Experimental studies

In a **Experimental study** using in vitro cell lines and in vivo mouse models, supplemented with transcriptomics, western blotting, qRT-PCR, immunofluorescence, and immunohistochemistry. **Ma et al.** from the Xiamen University, Xiamen, China. published in the *Gut Journal*, online ahead of print: **June 27, 2025**. to investigate **Pirin (PIR)** as a redox-sensitive transcriptional regulator and pro-inflammatory factor in hepatocellular carcinoma (HCC) pathogenesis [1)].

Key Results

1. PIR undergoes **redox-mediated nuclear translocation**, enhancing inflammatory transcription via RELA. 2. This creates a **positive feedback loop** that increases proinflammatory cytokines and promotes HCC progression. 3. PIR inhibition (genetic/pharmacological) or treatment with **N-acetyl cysteine** attenuates tumor growth and inflammation in mouse models. 4. Liver parenchymal cells demonstrate autocrine cytokine production under PIR influence, directly contributing to malignancy.

Critical Review

- **Strengths**:

1. Mechanistically rich; integrates redox biology, transcriptional regulation, and tumor immunopathology.
2. Robust experimental design across molecular, cellular, and organismal levels.
3. Data support PIR as a **central inflammatory amplifier** with translational potential.

- **Limitations**:

1. The study remains **preclinical** — lacking validation in human tissue cohorts or prospective clinical samples.
2. While inflammation is implicated, **direct links to immune cell behavior or microenvironmental shifts** remain underexplored.
3. Therapeutic interventions like NAC, although promising, need careful **pharmacokinetic validation** for clinical extrapolation.
4. The oncogenic specificity of PIR needs contrast with other tumor models—i.e., is this HCC-specific or broadly pro-tumorigenic?

Final Verdict

- **Score**: 8/10 — a conceptually innovative, well-executed mechanistic study with significant translational implications, pending clinical correlation.

Takeaway for Neurosurgeons

While not directly neurosurgical, this study reinforces the **broader principle** that redox-sensitive transcriptional regulators (like PIR) may be **pro-inflammatory oncogenic drivers**—a paradigm potentially relevant in gliomas or other CNS tumors influenced by inflammation.

Bottom Line

Pirin’s nuclear activity under oxidative stress orchestrates a self-sustaining inflammatory loop that drives HCC, suggesting a novel antioxidant-modifiable target for tumor suppression.

Citation

Nuclear Pirin promotes HCC by acting as a key inflammation-facilitating factor. Ma H, et al. *Gut.* Published online June 27, 2025. doi:10.1136/gutjnl-2024-334087. Corresponding authors: Qinxi Li (liqinxi@xmu.edu.cn), Weiling He (wlhe@xah.xmu.edu.cn)

[1)]
Ma H, Cao T, Zhang F, Sun D, Chen L, Lin Y, Lai S, Jiang B, Zhou Y, You J, Liu X, Wang Y, Lin F, Liu Y, Wang J, He W, Li Q. Nuclear Pirin promotes HCC by acting as a key inflammation-facilitating factor. Gut. 2025 Jun 27:gutjnl-2024-334087. doi: 10.1136/gutjnl-2024-334087. Epub ahead of print. PMID: 40579121.

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