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Evidence Report: TP53

Entity Type: Feature **Entity ID**: `TP53`

Metadata

- **Feature Type**: gene

Summary

1. High-level context
2. Disease context: Amyotrophic lateral sclerosis (ALS)
3. Model system context: Internal datasets from Amprenta, likely patient-derived or mixed biological samples (not explicitly specified as in vitro or in vivo)
4. Matrix context: Not explicitly stated; datasets are transcriptomics and proteomics data, presumably from relevant tissue or biofluids for ALS

5. Per-omics findings:

6. Lipidomics: No data available for TP53 in lipidomics.
7. Metabolomics: No data available for TP53 in metabolomics.
8. Proteomics: TP53 protein is detected in two internal proteomics datasets (test_proteomics_mixed and test_proteomics_canonical), indicating presence at the protein level in ALS-related samples. No quantitative differential expression values reported.
9. Transcriptomics: TP53 shows consistent upregulation across multiple transcriptomics datasets with log2 fold changes ranging from +0.90 to +2.50 and significant p-values (e.g., log2FC=1.20, p=0.001; log2FC=2.50, p=0.010), indicating increased gene expression in ALS contexts.

10. Cross-omics convergence:

11. TP53 is consistently upregulated at the transcript level and detected at the protein level in ALS-related samples, suggesting concordant transcriptional and proteomic evidence for increased TP53 activity or abundance.
12. No metabolite or lipid features related to TP53 were reported, so convergence is limited to transcriptomics and proteomics.

13. Cross-omics divergence:

14. Lack of metabolomics and lipidomics data for TP53 limits assessment of divergence.
15. Proteomics data lack quantitative fold change or statistical significance details, so the extent of protein-level regulation relative to transcriptomics is unclear.

16. Disease, model system, and matrix context (detailed):

17. The data derive from internal Amprenta datasets focused on ALS, a neurodegenerative disease.
18. The samples are described as "mixed" or "canonical," suggesting a combination of sample types or conditions, but exact model systems (e.g., patient tissue, cell lines) and matrices are not specified.
19. Transcriptomics and proteomics data indicate TP53 upregulation in ALS-related biological contexts.

20. Key open questions and next experimental steps:

21. Quantify TP53 protein expression changes with statistical rigor to confirm proteomic upregulation in ALS.
22. Determine the specific sample types and biological matrices to contextualize TP53 changes.
23. Investigate downstream pathways and functional consequences of TP53 upregulation in ALS models.
24. Explore metabolomics and lipidomics to identify potential metabolic or lipid alterations linked to TP53 activity.
25. Validate findings in independent ALS cohorts and relevant in vivo or in vitro models.