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Diagnostic Value of Seven Autoantibodies for Malignancy Risk Stratification in Pulmonary Nodules

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A 52-patient cohort compared benign and malignant nodules

Population and assays

Retrospective cohort: 52 pulmonary nodules

Benign: 9 (17.3%)

Malignant: 43 (82.7%)

Seven autoantibodies: p53, PGP9.5, SOX2, GAGE7,
GBU4-5, MAGEA1, CAGE

Five conventional markers: CEA, AFP, NSE,
CYFRA21-1, CA19-9

Statistical plan and data quality

Mann-Whitney U: benign vs malignant

Spearman: biomarker correlations

ROC: composite seven-antibody panel

Complete antibody data: all 52 patients

Conventional-marker missingness: AFP 17.3%, NSE
30.8%, CYFRA21-1 50.0%, CA19-9 61.5%

Interpretation: exploratory because of group
imbalance and incomplete conventional-marker data

No single antibody discriminated malignancy; panel AUC 0.693

Individual autoantibodies

0 of 7 reached $P < 0.05$

SOX2 $P=0.238$ **MAGEA1** $P=0.208$

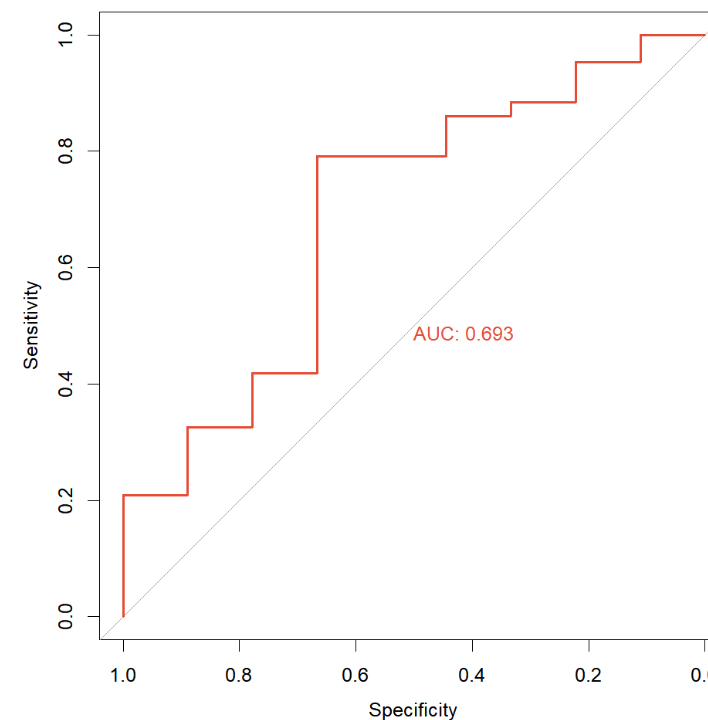
p53 $P=0.751$ **PGP9.5** $P=0.691$

GAGE7 $P=0.628$ **GBU4-5** $P=0.645$

CAGE $P=0.628$

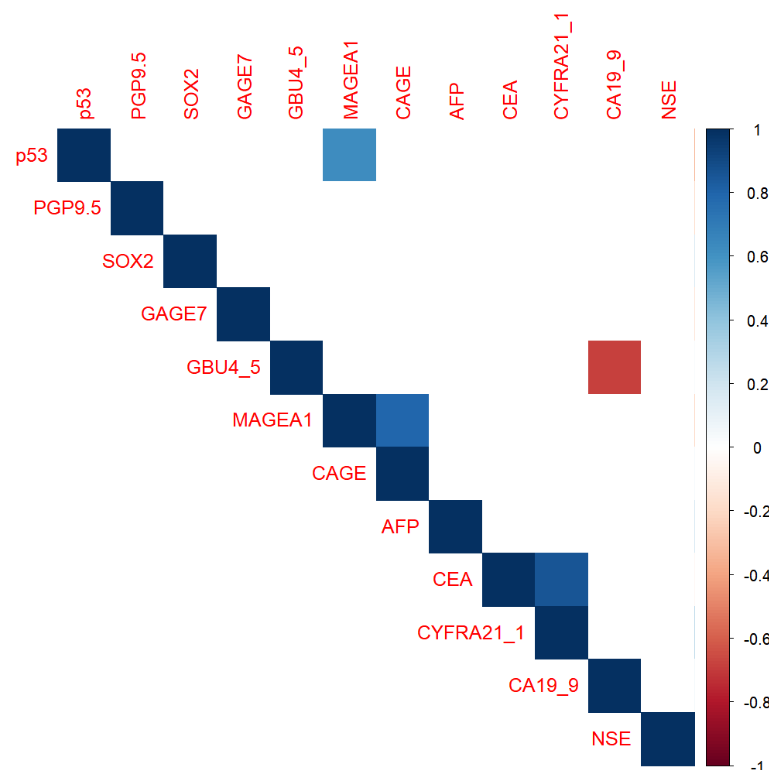
Result: single-marker concentrations showed substantial overlap between benign and malignant nodules.

Composite seven-antibody panel



Correlated antibody signals support panel-level analysis

Exploratory biomarker correlations



Clinical interpretation

MAGEA1-CAGE: $r=0.791$, $P=0.0037$

p53-MAGEA1: $r=0.629$, $P=0.038$

Meaning: coordinated antibody signals may carry more information than any single marker.

Diagnostic role: AUC 0.693 indicates low-to-moderate discrimination and does not support stand-alone use.

Limitations: only 9 benign cases, imbalanced groups, missing conventional-marker data, and no external validation.

Next step: validate in a larger cohort and examine associations with minimal residual disease.

Disclosure

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