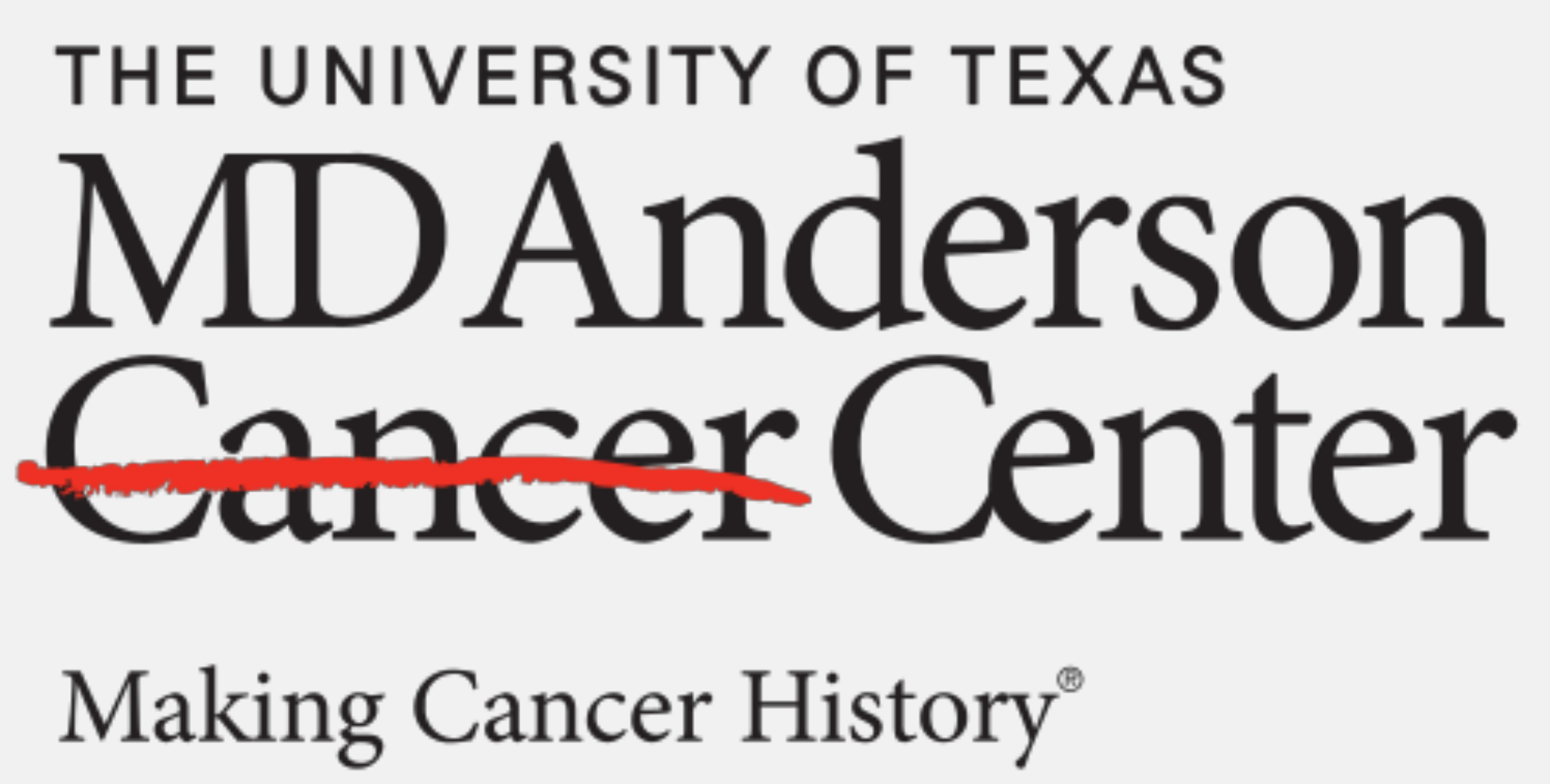


EPIDEMIOLOGIC ASSESSMENT OF RISK FACTORS FOR DUAL DIAGNOSIS OF B-CELL AND T-CELL LYMPHOMAS

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INTRODUCTION

- Epidemiologic studies demonstrate a nearly fivefold increased bidirectional risk between B-cell lymphomas (BCL) and T-cell lymphomas (TCL)
- The biologic mechanisms and shared risk factors underlying dual BCL/TCL diagnoses remain incompletely understood
- Comprehensive characterization of patients with dual diagnoses may provide insight into common mechanisms of lymphomagenesis

METHOD

- Study design: Retrospective single-center study at MD Anderson Cancer Center
- Study population: 116 adults with dual BCL and TCL diagnosed from separate biopsy specimen (January 2013 – May 2026)
- Exclusion criteria: Composite BCL and TCL in the same biopsy specimen
- Data collection: Demographics, lymphoma subtype, temporal sequence, non-lymphoid malignancies (NLMs), autoimmune comorbidities, family history of hematologic malignancies and viral serologies

CONCLUSIONS

- Patient cases with dual BCL/TCL diagnoses are frequently associated with additional malignancies, autoimmune comorbidities, EBV positivity of tumors and family history of malignancy
- These findings support a shared underlying immune dysregulation as predisposing factor in addition to environmental and genetic susceptibilities
- Future genomic and molecular studies are required to explore the underlying mechanisms of dual B-cell and T-cell lymphomagenesis

CONTACT INFORMATION

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RESULTS

Characteristics	Value
Sex	
Male	72 (62.1%)
Female	44 (37.9%)
Age at diagnosis	
First lymphoma	63.5 (18 – 86)
Second Lymphoma	67 (19 – 88)
Race	
White/ Caucasian	86 (74.1%)
Back/ African American	13 (11.2%)
Asian	8 (6.9%)
Other	9 (7.8%)
Viral serostatus	
HIV	0/96 (0%)
HCV antibody	1/93 (1.1%)
HBV core antibody	10/91 (11%)
CMV	35/61 (57.4%)

Tbl. 1: Demographic characteristics and viral serostatus in the study cohort. Categorical variables are presented as n (%), and age as median (range). Viral serostatus is reported as the proportion of positive cases among patients with available data.

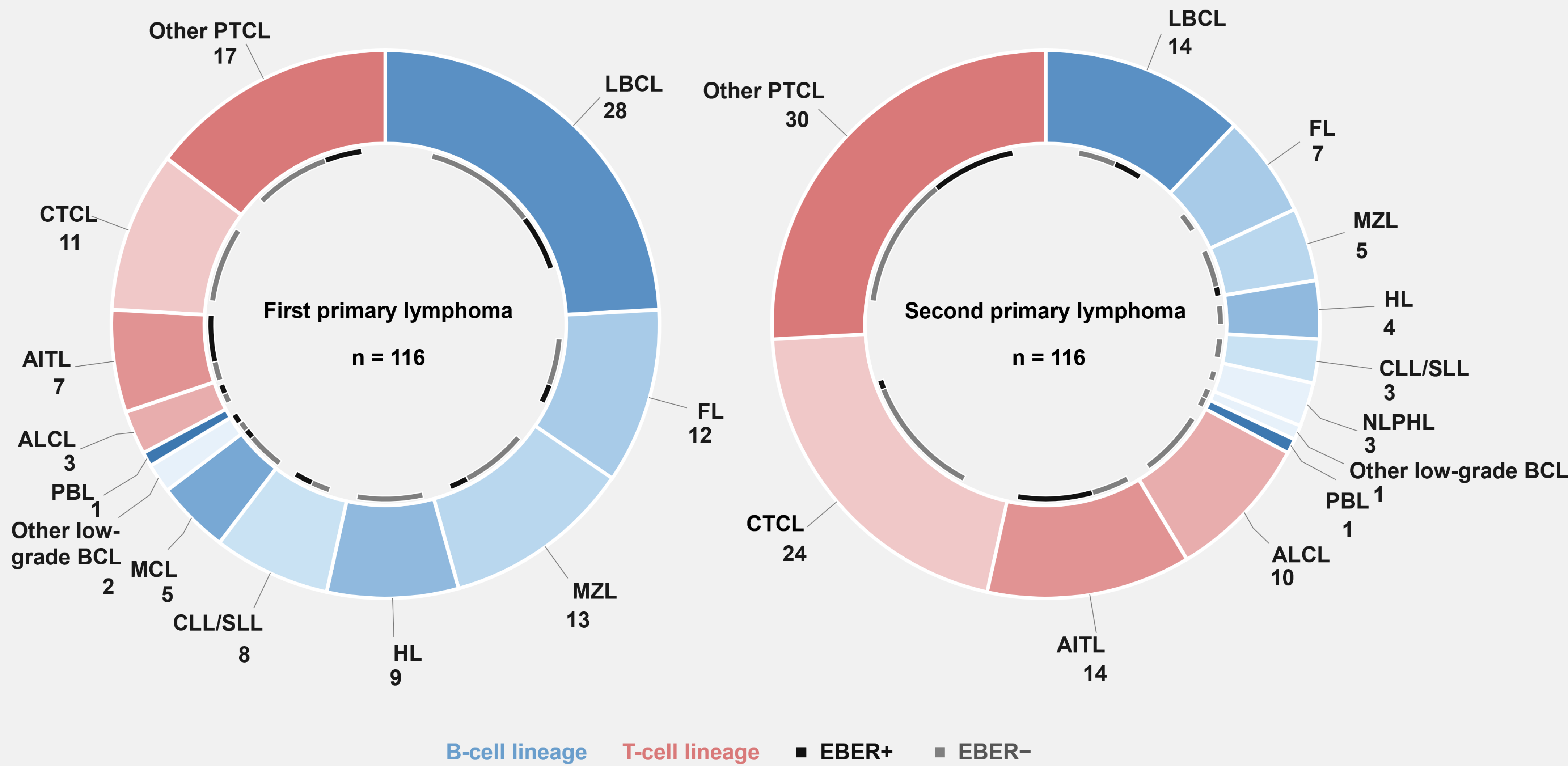


Fig. 1: Distribution of BCL and TCL subtypes at first and second primary lymphoma diagnosis (outer ring, n=116). The inner ring indicates tumor Epstein-Barr virus-encoded RNA (EBER). Blank portions represent cases with unreported status. LBCL: large BCL, FL: follicular lymphoma, MZL: marginal zone lymphoma, HL: Hodgkin lymphoma, CLL/SLL: chronic lymphocytic leukemia/small lymphocytic lymphoma, MCL: mantle cell lymphoma, NLPHL: nodular lymphocyte-predominant HL, PBL: plasmablastic lymphoma, PTCL: peripheral TCL, CTCL: cutaneous TCL, AITL: angioimmunoblastic TCL, ALCL: anaplastic large cell lymphoma.

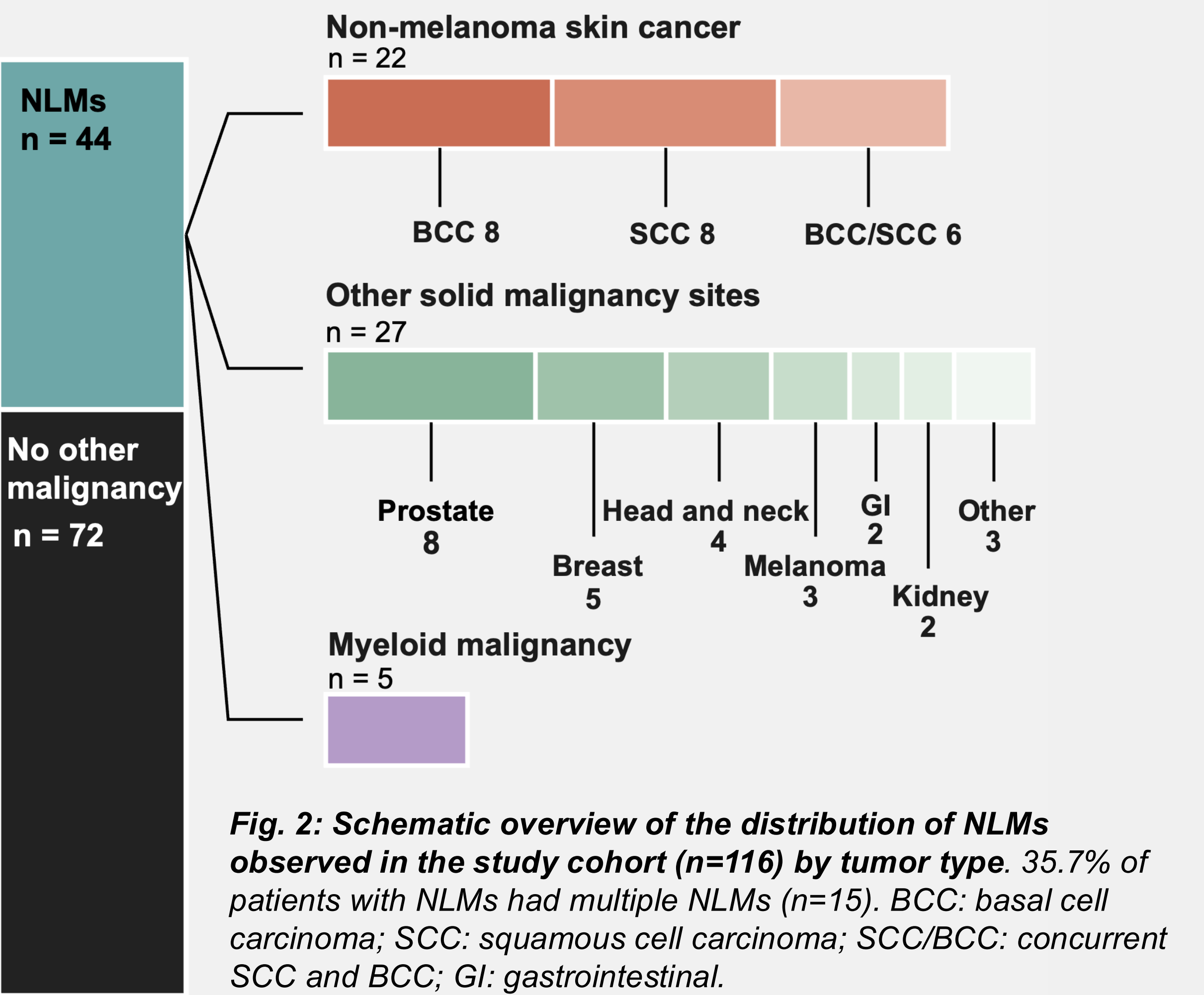
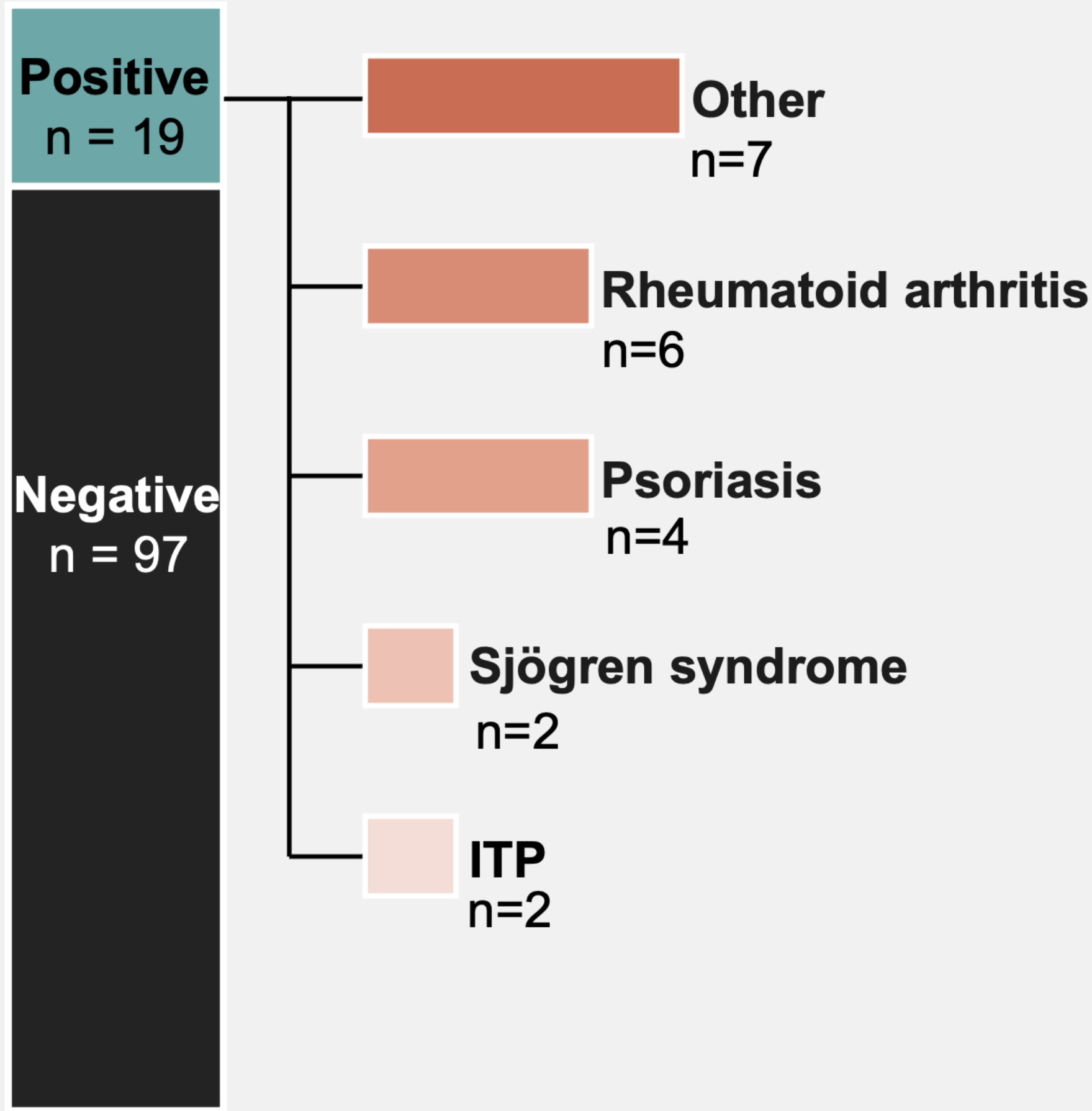


Fig. 2: Schematic overview of the distribution of NLMs observed in the study cohort (n=116) by tumor type. 35.7% of patients with NLMs had multiple NLMs (n=15). BCC: basal cell carcinoma; SCC: squamous cell carcinoma; SCC/BCC: concurrent SCC and BCC; GI: gastrointestinal.

A Autoimmune comorbidities



B Family history of hematologic malignancies

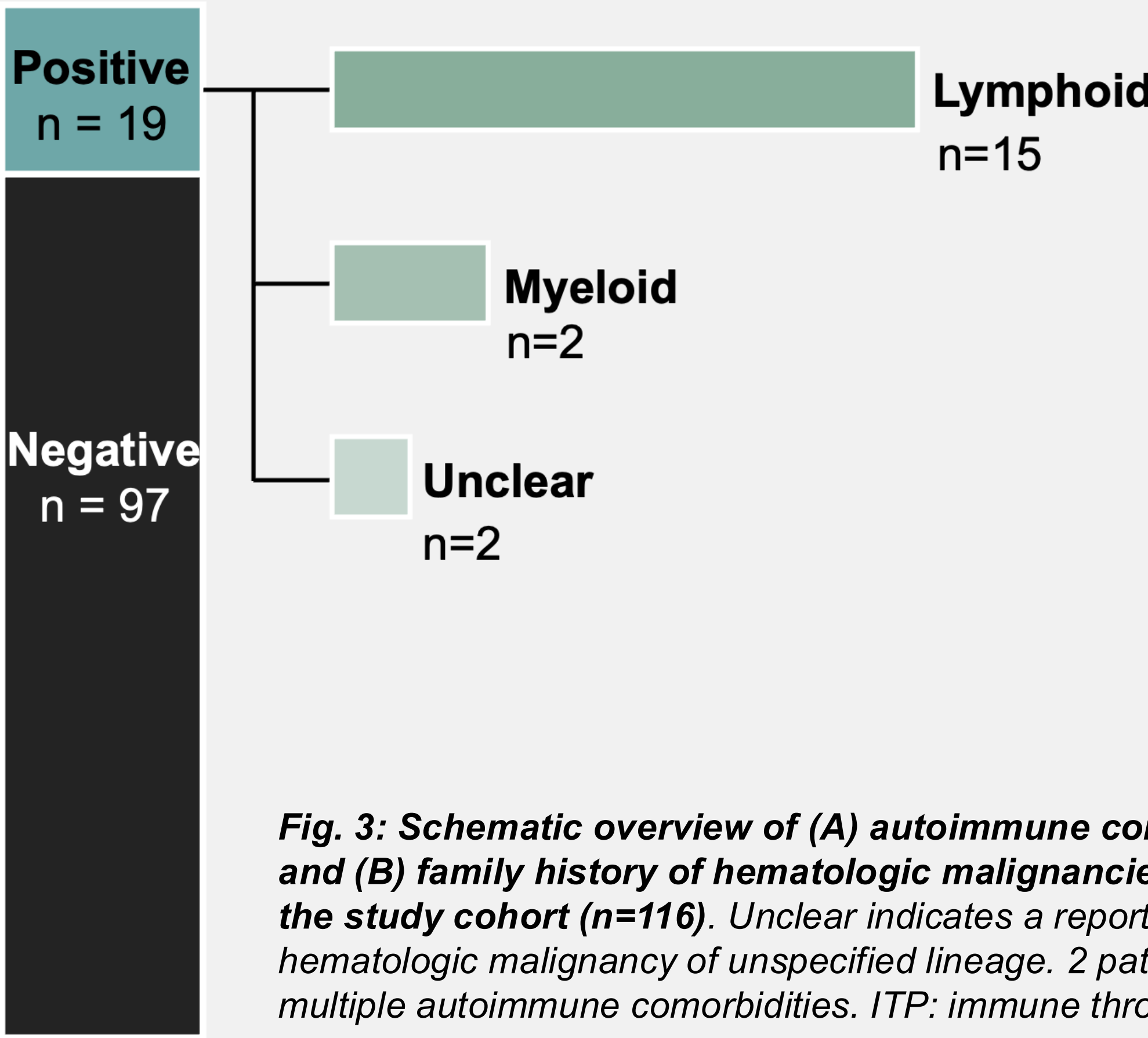


Fig. 3: Schematic overview of (A) autoimmune comorbidities and (B) family history of hematologic malignancies observed in the study cohort (n=116). Unclear indicates a reported hematologic malignancy of unspecified lineage. 2 patients had multiple autoimmune comorbidities. ITP: immune thrombocytopenia.