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SUPPLEMENTAL MATERIALS AND METHODS

Study Participants

NEPTUNE Cohort

The composition of the Nephrotic Syndrome Study Network (NEPTUNE) has been described previously.[1-3] The study is registered at clinicaltrials.gov (NCT01209000) and each participant (or their parent/guardian) provided written, informed consent prior to participation. Participants were enrolled from 24 clinical sites in North America with local IRB approval at each institution. Briefly, adults and children with nephrotic-range proteinuria (≥500 mg/d on a 24 h urine sample or urinary protein-to-creatinine (UP:C) ratio ≥0.5 g/g on spot urine) were eligible for enrollment at the time of a first clinically indicated kidney biopsy. Because kidney biopsy at presentation is not the standard of care in pediatrics, children (≤18 years) were also eligible for enrollment based upon the above proteinuria criteria alone. Exclusion criteria included life expectancy <6 months, prior solid organ transplantation, and kidney manifestations of systemic disease. For the purposes of the present study, participants with a recorded prescription for anticoagulant or antiplatelet medications were excluded. Participants with a pre-enrollment history of VTE were also excluded from this study to decrease potential confounding from non-NS hypercoagulable conditions. Participants' phenotypic data was collected from the NEPTUNE Data Coordinating Center database (tranSMART). The NEPTUNE cohort consisted of plasma samples from 150 unique subjects randomly selected to represent the available proteinuria spectrum within the NEPTUNE biorepository as of October 2014 (including patients with active disease as well as those who had achieved partial or complete remission). Three (2%) samples had undetectable thrombin generation indicating that the plasma had clotted during the pre-analytic phase; the remaining 147 samples were thus included in this study.[4]

PNRC Cohort

Children with incident NS were recruited from Pediatric Nephrology Research Consortium (PNRC) participating centers. As described previously, the study protocols and consent documents were approved by the Nationwide Children's Hospital Institutional Review Board (IRB05-00544, IRB07-004, & IRB12-00039) and at each participating PNRC center.[5] Participants were enrolled between 2008-2014 and their samples were banked at

Nationwide Children's Hospital until analyzed. Glucocorticoid (GC) therapy naïve children 1-18 years of age presenting with edema and proteinuria ≥3+ by dipstick were eligible for enrollment.

Columbus Cohort

Participants with incident nephrotic syndrome were subject to the same eligibility criteria as the NEPTUNE participants, including exclusion for pre-enrollment VTE history, anticoagulant, or antiplatelet medications.[4] Sample were collected at diagnosis prior to initiation of any nephrotic syndrome-directed therapy. Participants were enrolled between 2013-2015 and their samples were banked at Nationwide Children's Hospital until analyzed. This portion of the study was approved by the Nationwide Children's Hospital Institutional Review Board (IRB12-00290) in reciprocity with The Ohio State University Wexner Medical Center IRB. Written informed consent was obtained from each participant or parent/guardian.

Plasma Samples

Aliquots (≤200 µL) of plasma isolated from blood collected into 0.105 M buffered sodium citrate tubes (0.32% final concentration citrate) were provided by the NEPTUNE biorepository. Blood from the PNRC cohort was collected into 0.1 M sodium citrate cell preparation tubes containing a cell separator system (BD Vacutainer CPT (REF 362761) Becton, Dickinson and Company, Franklin Lakes, NJ) at an 8:1 blood-to-citrate ratio (~0.33% final concentration NaCitrate). For the Columbus Cohort, blood was collected into final concentration 0.32% sodium citrate / 1.45 µM corn trypsin inhibitor (Haematologic Technologies Inc., Essex Junction, VT, USA) and platelet poor plasma (PPP) was prepared as previously described, aliquoted, and frozen at ¯80°C until further analysis.[6, 7]

Supplemental Table 1: Key elements of studies included in meta-analyses

First Author	Publication Year	Study Design	Patients (*N*)	Age Range (Years)	Antithrombin Assay(s)	Nephrotic Features
Adult Studies (Majority of Patients >18 years)						
Thomsom et al[8]	1974	CCSC	16	14-65	Activity	Proteinuria (>5 g/d); hypoproteinemia
Gomperts et al[9]	1977	CCSC	5	19-38	Both Activity and Antigen	Proteinuria (>5 g/d); hypoalbuminemia (<3.5 g/dl)
Thaler et al[10]	1978	CCSC	14	9-70	Both Activity and Antigen	Moderate to severe proteinuria (with histological diagnosis)
McGinley et al[11]	1983	CCSC	21	39.4±3.6	Activity	Proteinuria (>3 g/d); hypoalbuminemia (<3.5 g/dl)
Panicucci et al[12]	1983	CCSC	20	"Adults"	Both Activity and Antigen	Moderate to severe proteinuria; hypoproteinemia
Vaziri et al[13]	1984	CCSC	20	16-60	Both Activity and Antigen	Proteinuria (>3 g/d); hypoalbuminemia (<3.5 g/dl)
Mannucci et al[14]	1986	CCSC	24	15-70	Both Activity and Antigen	Proteinuria (>3 g/d); hypoalbuminemia
Rydzewski et al[15]	1986	CCSC	17	21-61	Antigen	Proteinuria (>3.5 g/d); hypoalbuminemia (<3.5 g/dl)
Hannedouche et al[16]	1987	CCSC	15	42±18	Activity	Proteinuria (5.7±4.2 g/d); hypalbuminemia (2.19±0.72 g/dl)
Grandrille et al[17]	1988	CCSC	10	16-82	Activity	Proteinuria (>2.5 g/d); hypoalbuminemia (<3.5 g/dl)
Grau et al[18]	1988	CCSC	68	12-72	Both Activity and Antigen	Proteinuria (>2.5 g/d)
Mori et al [19]	1988	CCSC	9	46-76	Both Activity and Antigen	Proteinuria (>4 g/d); hypoalbuminemia (<3.5 g/dl)
Toulon et al [20]	1992	CCSC	33	16-82	Both Activity and Antigen	Proteinuria (>3 g/d); hypoalbuminemia (<4 g/dl)
Joven et al[21]	1997	CCSC	22	25-69	Antigen	Proteinuria (≥3.5 g/d); hypoalbuminemia (<3.1 g/dl)
Pediatric Studies (Majority of Patients ≤18 years)						
Boneu et al[22]	1981	CCSC	27	3-13	Both Activity and Antigen	Nephrotic features not specified

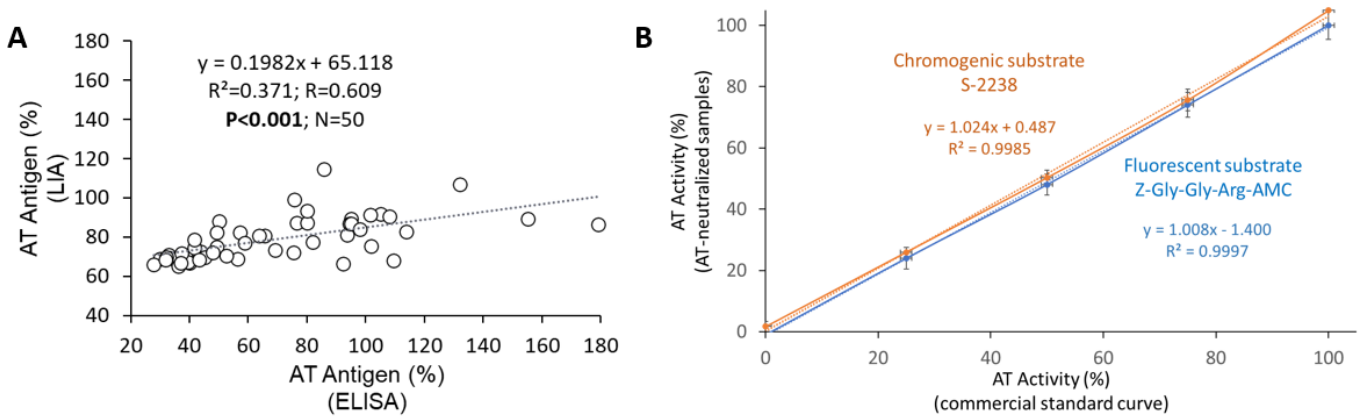
Elidrissy et al[23]	1985	CCSC	25	3-11	Activity	Proteinuria (≥3+ by dipstick or >40 mg/h/m^2); hypoalbuminemia (<2.5 g/dl)
Sie et al[24]	1988	CCSC	33	2-6	Activity	Proteinuria; hypoalbuminemia (<3.7 g/dl)
Fukui et al[25]	1989	CCSC	18	“Children”	Both Activity and Antigen	Nephrotic features not specified
Ueda et al[26]	1990	CCSC	23	3-16	Antigen	Proteinuria (3.4±2.2 g/d); hypoalbuminemia (2.3±0.5 g/dl)
Elidrissy et al[27]	1991	CCSC	39	2-14	Activity	Proteinuria (≥3+ by dipstick or >40 mg/h/m^2); hypoalbuminemia (<2.5 g/dl)
Andre et al[28]	1994	CCSC	29	1-14	Activity	Proteinuria; hypoproteinemia (<5.5 g/dl)
Yalcinkaya et al[29]	1995	CCSC	15	2.5-13	Activity	Proteinuria (>40 mg/h/m^2); hypoalbuminemia (<3 g/dl)
Al-Mugeiren[30]	1996	CCSC	41	2-14	Activity	Proteinuria (>40 mg/h/m^2); hypoalbuminemia (<2.5 g/dl)
Citak et al[31]	2000	CCSC	49	1-16	Antigen	Proteinuria (>1 g/m^2/d); hypoalbuminemia (<2.5 g/dl)
Prandota et al[32]	2001	PCC	19	1.5-18	Activity	Proteinuria (151±65 mg/kg/d); hypoalbuminemia (1.56±0.5 g/dl)
Ozkayin et al[33]	2004	CCSC	26	2-15	Both Activity and Antigen	Proteinuria (>40 mg/m^2/hour); hypoalbuminemia
Mortazavi et al[34]	2008	CCSC	30	1.4-11	Antigen	Proteinuria (>1 g/m^2/d); hypoalbuminemia (<2.5 g/dl)

CCSC: Cross Sectional Case-Control; PCC: Prospective Case-Control

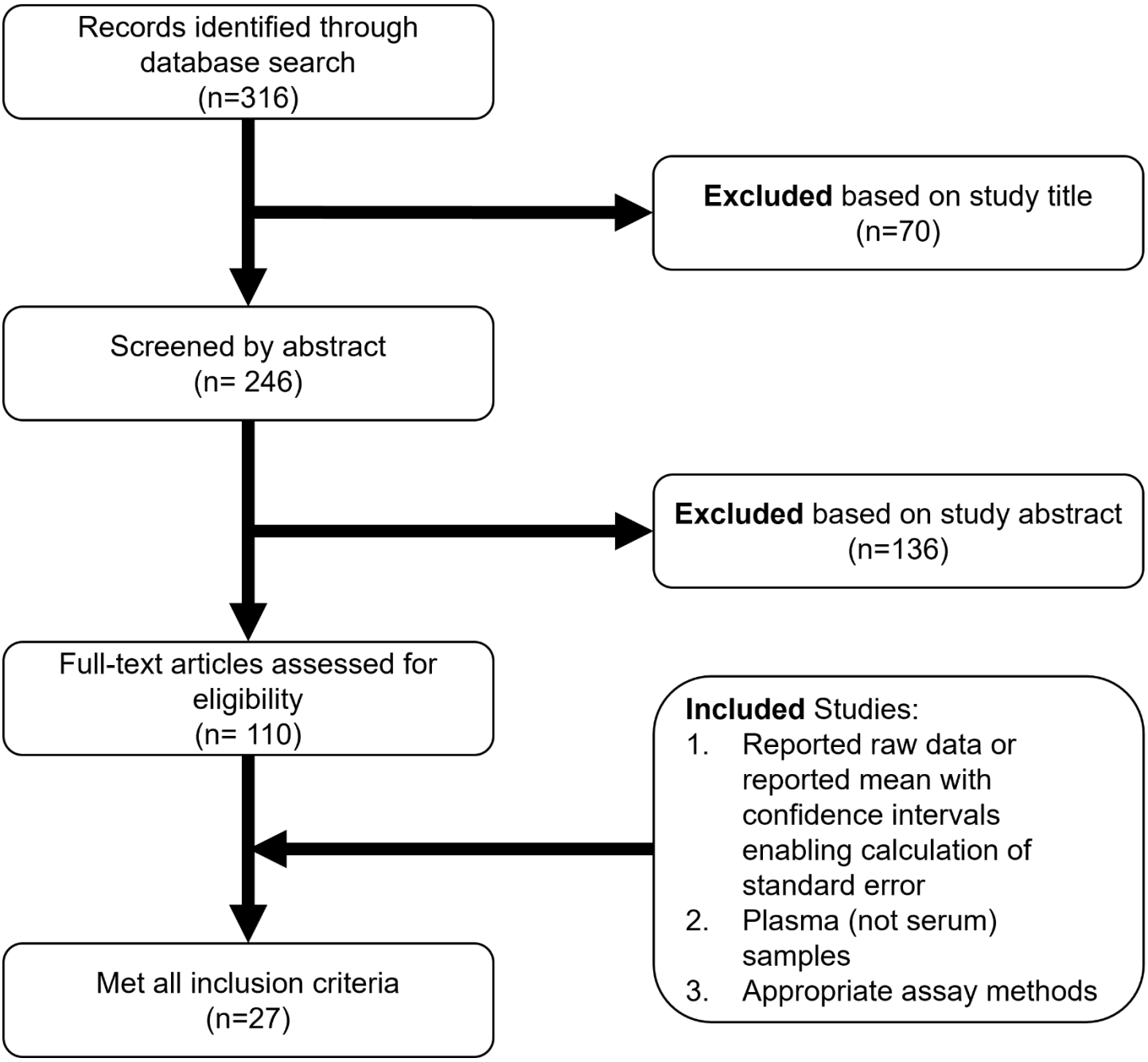
Supplemental Table 2: Newcastle-Ottawa scale risk-of-bias assessment for meta-analyses studies

First Author	Publication Year	Selection (/4)	Comparability (/2)	Exposure (/3)*	Total (/6)*
Adult Studies (Majority of Patients >18 years)					
Thomsom et al[8]	1974	★★★	0	N/A	3
Gomperts et al[9]	1977	★★★★	★	N/A	5
Thaler et al[10]	1978	★★★	0	N/A	3
McGinley et al[11]	1983	★★★	★★	N/A	5
Panicucci et al[12]	1983	★★★★	0	N/A	4
Vaziri et al[13]	1984	★★★★	★★	N/A	6
Mannucci et al[14]	1986	★★★★	★	N/A	5
Rydzewski et al[15]	1986	★★★★	★	N/A	5
Hannedouche et al[16]	1987	★★★★	★	N/A	5
Grandrille et al[17]	1988	★★★★	0	N/A	4
Grau et al[18]	1988	★★★★	0	N/A	4
Mori et al [19]	1988	★★★★	★	N/A	5
Toulon et al [20]	1992	★★★★	★	N/A	5
Joven et al[21]	1997	★★★★	★	N/A	5
Pediatric Studies (Majority of Patients ≤18 years)					
Boneu et al[22]	1981	★★★★	★	N/A	5
Elidrissy et al[23]	1985	★★★★	★	N/A	5
Sie et al[24]	1988	★★★★	★	N/A	5
Fukui et al[25]	1989	★★★★	★	N/A	5
Ueda et al[26]	1990	★★★★	★	N/A	5
Elidrissy et al[27]	1991	★★★★	★★	N/A	6
Andre et al[28]	1994	★★★	★	N/A	4
Yalcinkaya et al[29]	1995	★★★	★	N/A	4
Al-Mugeiren[30]	1996	★★★	★	N/A	4
Citak et al[31]	2000	★★★	★	N/A	4
Prandota et al[32]	2001	★★★	★	N/A	4
Ozkayin et al[33]	2004	★★★	★	N/A	4
Mortazavi et al[34]	2008	★★★	★	N/A	4

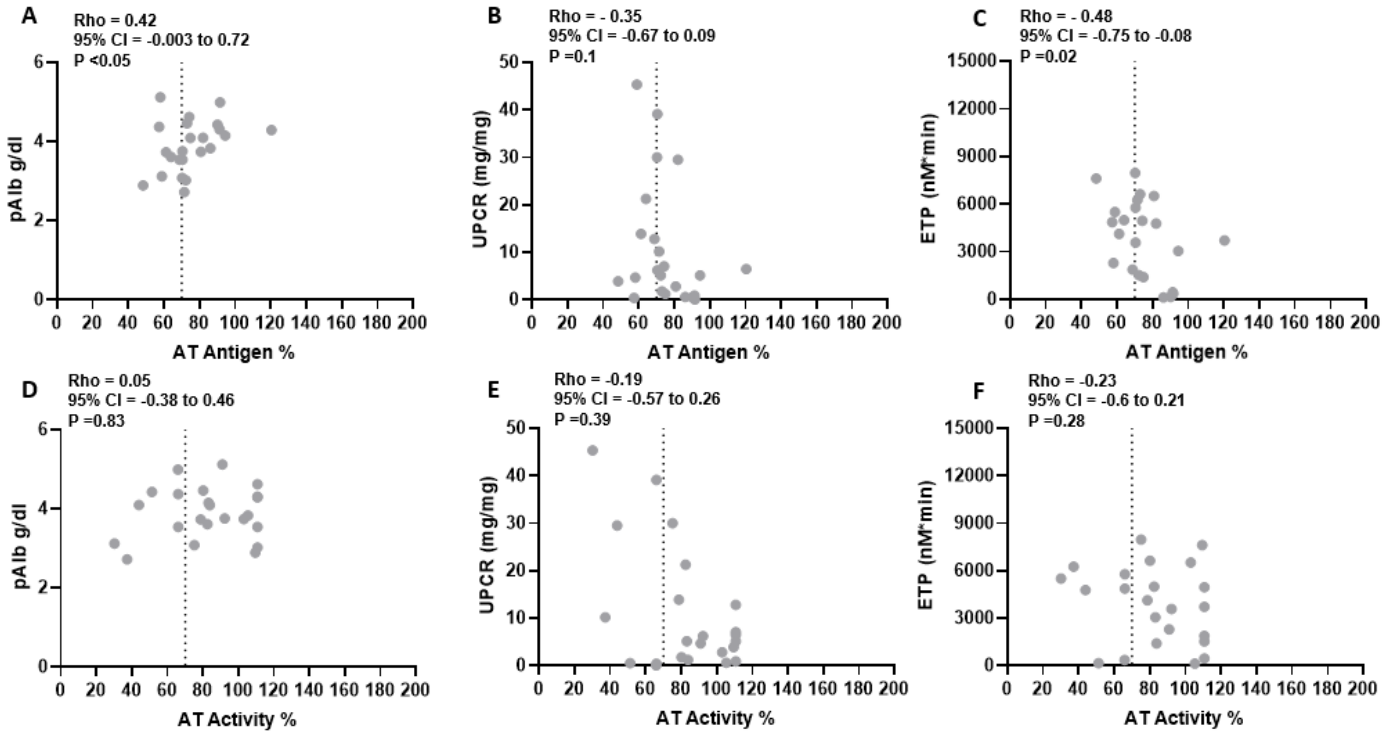
*The Newcastle-Ottawa scale defines exposure as an intervention. The studies included in these analyses were non-interventional.



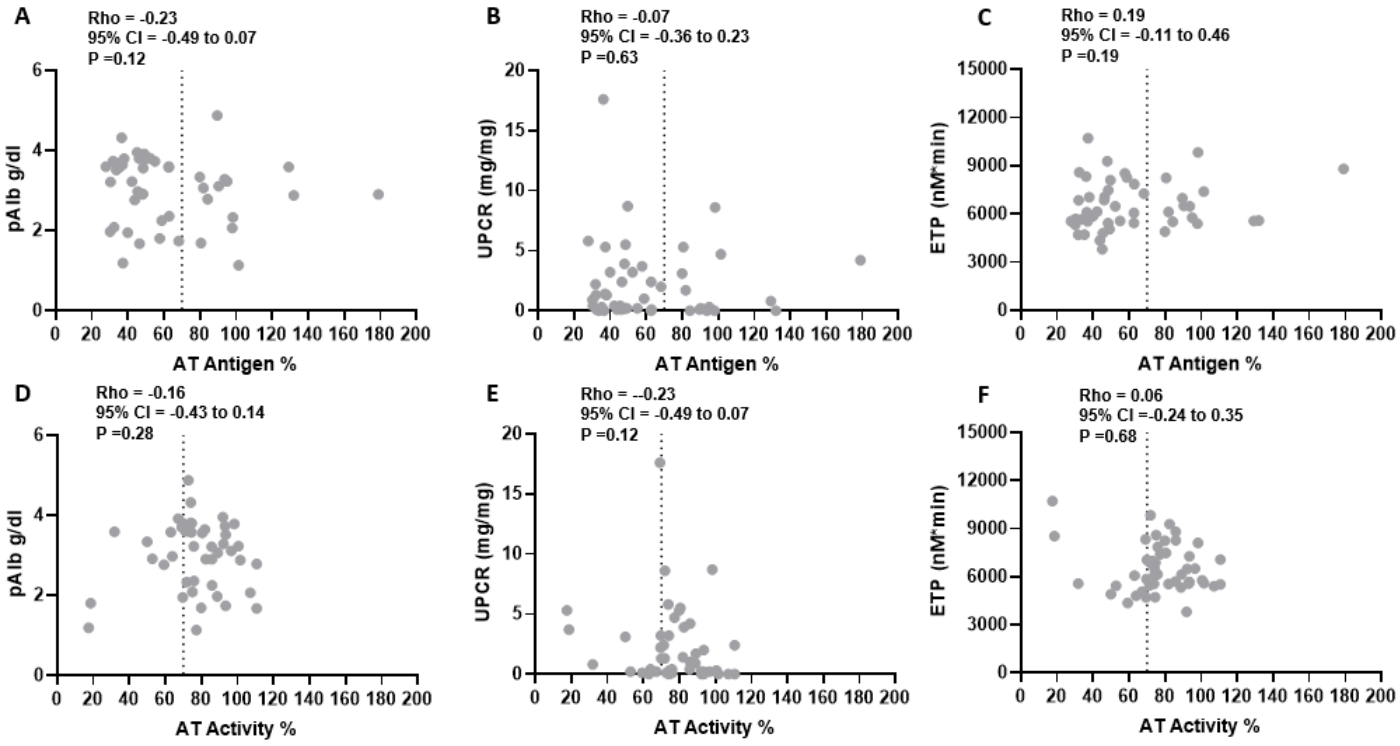
Supplemental Figure 1: Antithrombin Assay Validation. Antithrombin (AT) antigen quantification by latex immunoassay (LIA) and enzyme-linked immunosorbent assay (ELISA) were significantly correlated (*n*=50 NEPTUNE samples; **A**). Similarly, AT activity quantified using amidolytic assays with either chromogenic or fluorogenic reporters provided similar results (**B**). Two types of standard curve were utilized in **B**: On the x-axis standards were created with proportionate mixing of AT immunodepleted plasma and pooled normal plasma whereas the y-axis standards were generated with a set of healthy control plasmas treated with varying concentrations of AT neutralizing antibody (*n*=3-4 samples per point on the standard curve).



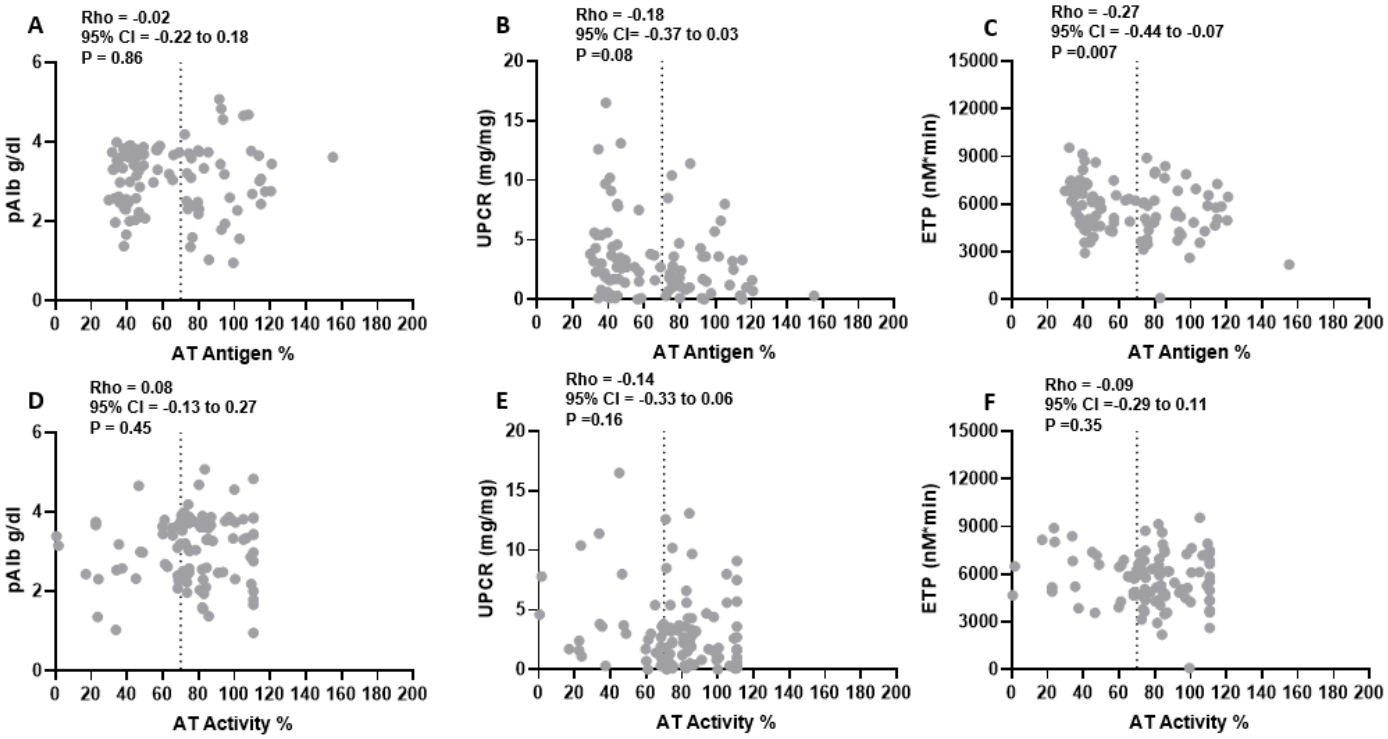
Supplemental Figure 2: Flow diagram illustrating exclusion and inclusion criteria of publications considered for the meta-analyses.



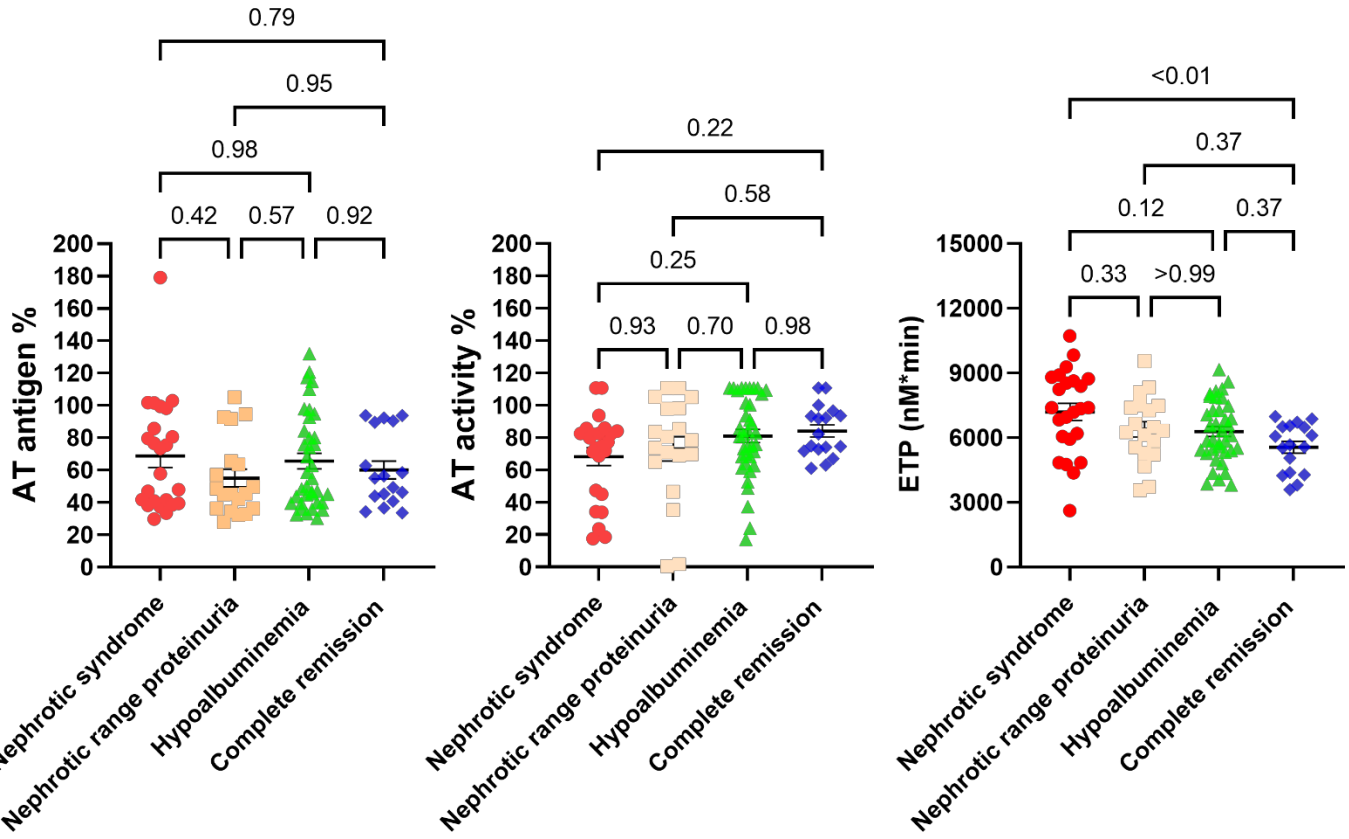
Supplemental Figure 3: Antithrombin relationships in the Columbus cohort. Antithrombin (AT) antigen was significantly correlated with plasma albumin (pAlb; **A**) and endogenous thrombin potential (ETP; **C**) but not with urinary protein-to-creatinine ratio (UPCR; **B**) in the Columbus cohort (*n*=23). AT activity was not significantly correlated with pAlb (**D**), UPCR (**E**), or ETP (**F**). The vertical dashed line in each panel represents 70% plasma AT, a commonly used threshold to define clinically relevant AT deficiency.



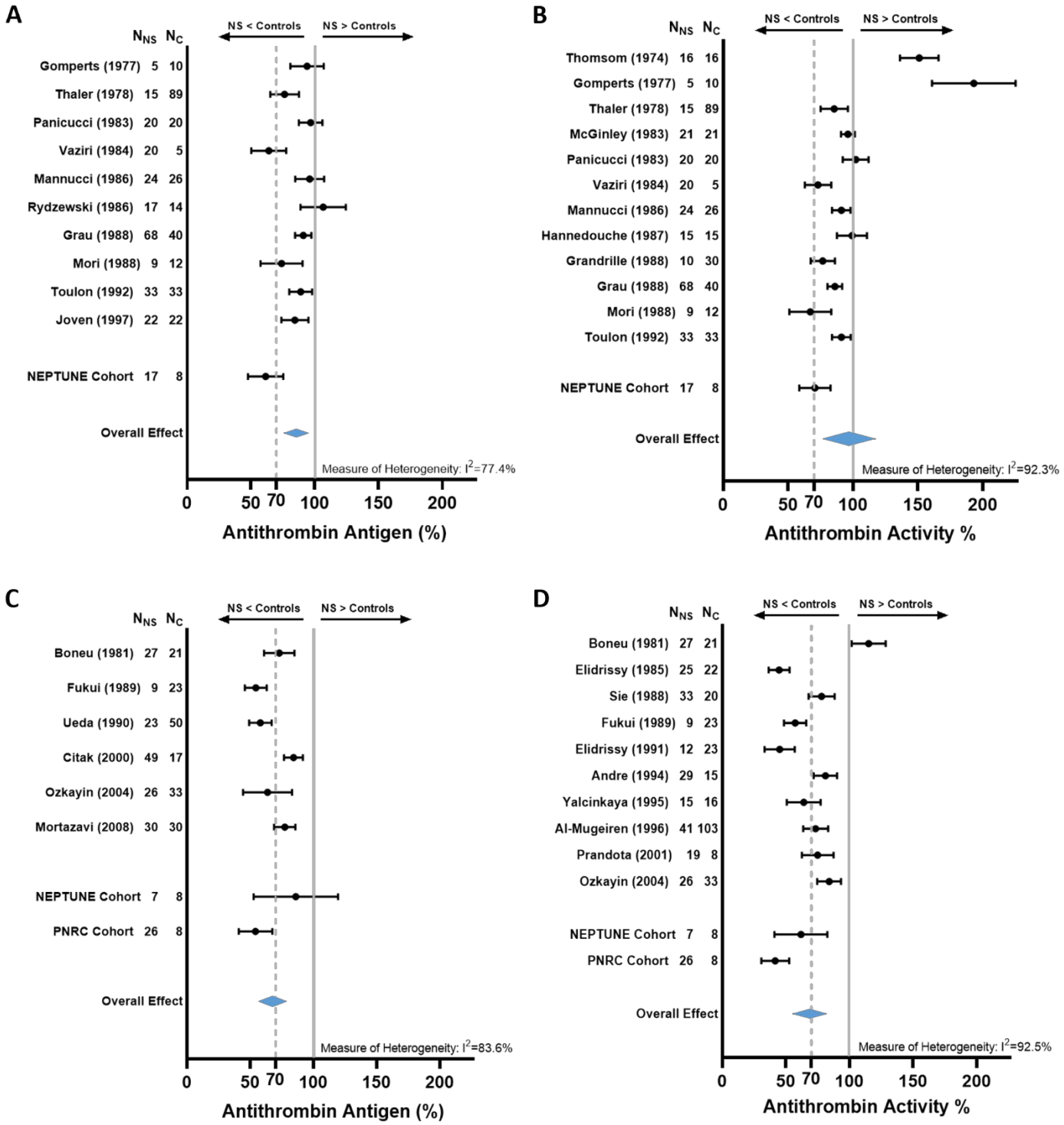
Supplemental Figure 4: Antithrombin was not correlated with hypercoagulopathy in the pediatric NEPTUNE subcohort. Neither antithrombin (AT) antigen (**A**, **B**, **C**) or activity (**D**, **E**, **F**) were correlated with plasma albumin (pAlb; **A**, **D**), urinary protein-to-creatinine ratio (UPCR; **B**, **E**), or endogenous thrombin potential (ETP; **C**, **F**) in the pediatric NEPTUNE subcohort (*n*=47). The vertical dashed line in each panel represents 70% plasma AT, a commonly used threshold to define clinically relevant AT deficiency.



Supplemental Figure 5: Antithrombin antigen was correlated with hypercoagulopathy in the adult NEPTUNE subcohort. Antithrombin (AT) antigen was correlated with endogenous thrombin potential (ETP; **C**) in the adult NEPTUNE subcohort (*n*=100). There was no significant correlation between AT activity and ETP (**F**) or between either AT antigen or activity with plasma albumin (pAlb; **A**, **D**) or urinary protein-to-creatinine ratio
(UPCR; **B**, **E**). The vertical dashed line in each panel represents 70% plasma AT, a commonly used threshold to define clinically relevant AT deficiency.



Supplemental Figure 6: ETP changes with nephrosis activity whereas antithrombin antigen and activity do not. NEPTUNE patients were divided into the following sub-groups: Nephrotic syndrome (UPCR ≥3.5 g/g and albumin ≤3.0 g/dl; n=24), Nephrotic range proteinuria (UPCR ≥3.5 g/g and albumin >3.0 g/dl; n=19), hypoalbuminemia (UPCR <3.5 g/g and albumin ≤3.0 g/dl; n=37), and for comparison, "complete remission" (UPCR ≤0.3 g/g and albumin >3.0 g/dl; n=17). (**A**) Antithrombin antigen was not significantly different amongst these groups. (**B**) Antithrombin activity was not significantly different amongst these groups. (**C**) Endogenous Thrombin Potential (ETP) was significantly decreased in the complete remission group compared to the nephrotic syndrome group.



Supplemental Figure 7: Antithrombin Deficiency was not a Uniform Feature of Nephrotic Syndrome. Based upon the overall effect estimates of these meta-analyses, clinically relevant antithrombin (AT) deficiency (<70%) was not expected in adults with active nephrotic syndrome (NS; **A**, **B**). AT deficiency was more likely to be observed in children with active NS but the confidence intervals for the overall effect estimates from these meta-analyses overlapped with normal AT values (≥70%; **C**, **D**). The vertical dashed line in each panel represents 70% plasma AT, a commonly used threshold to define clinically relevant AT deficiency.

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