

Identification of Sputum Biomarkers Predictive of Pulmonary Exacerbations in COPD

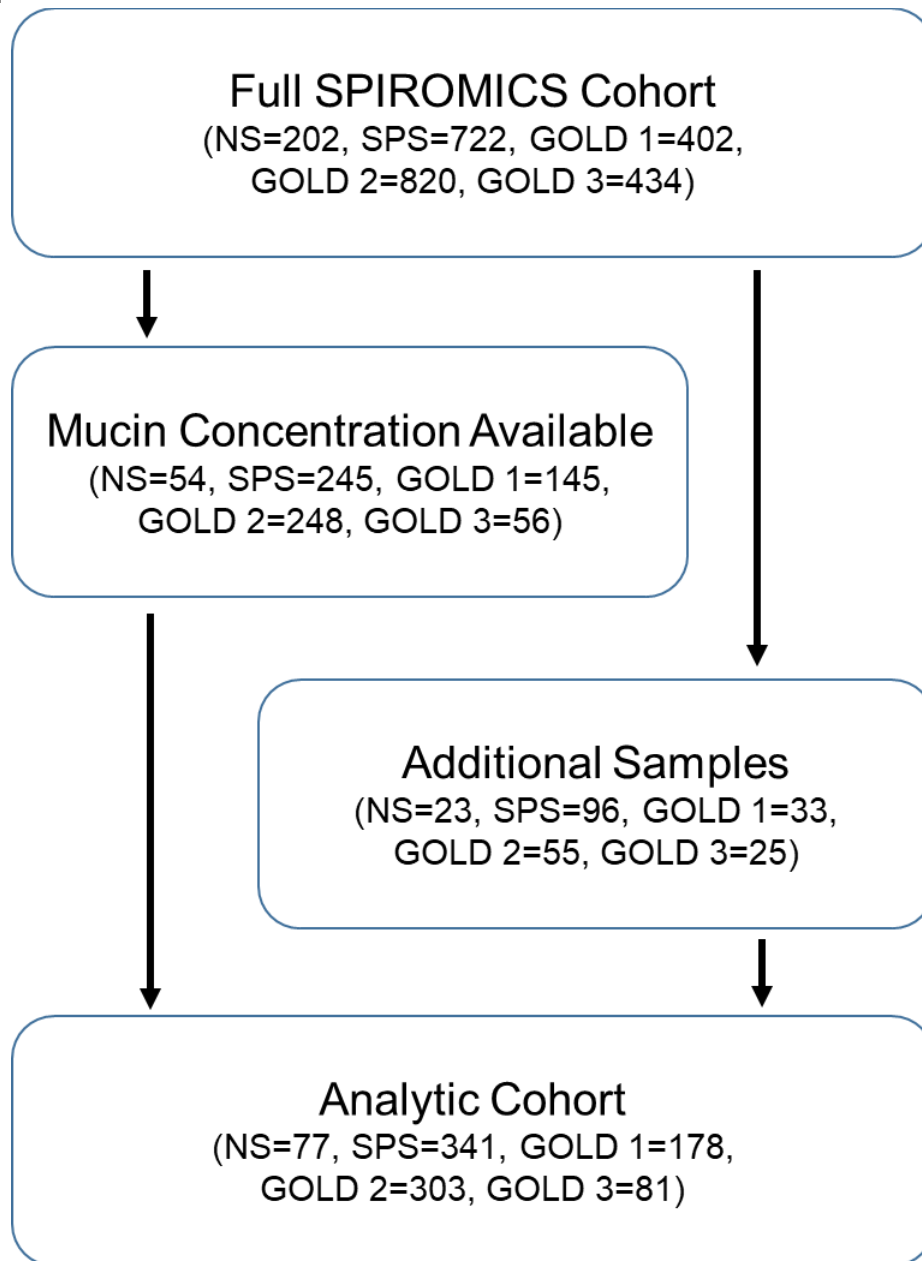
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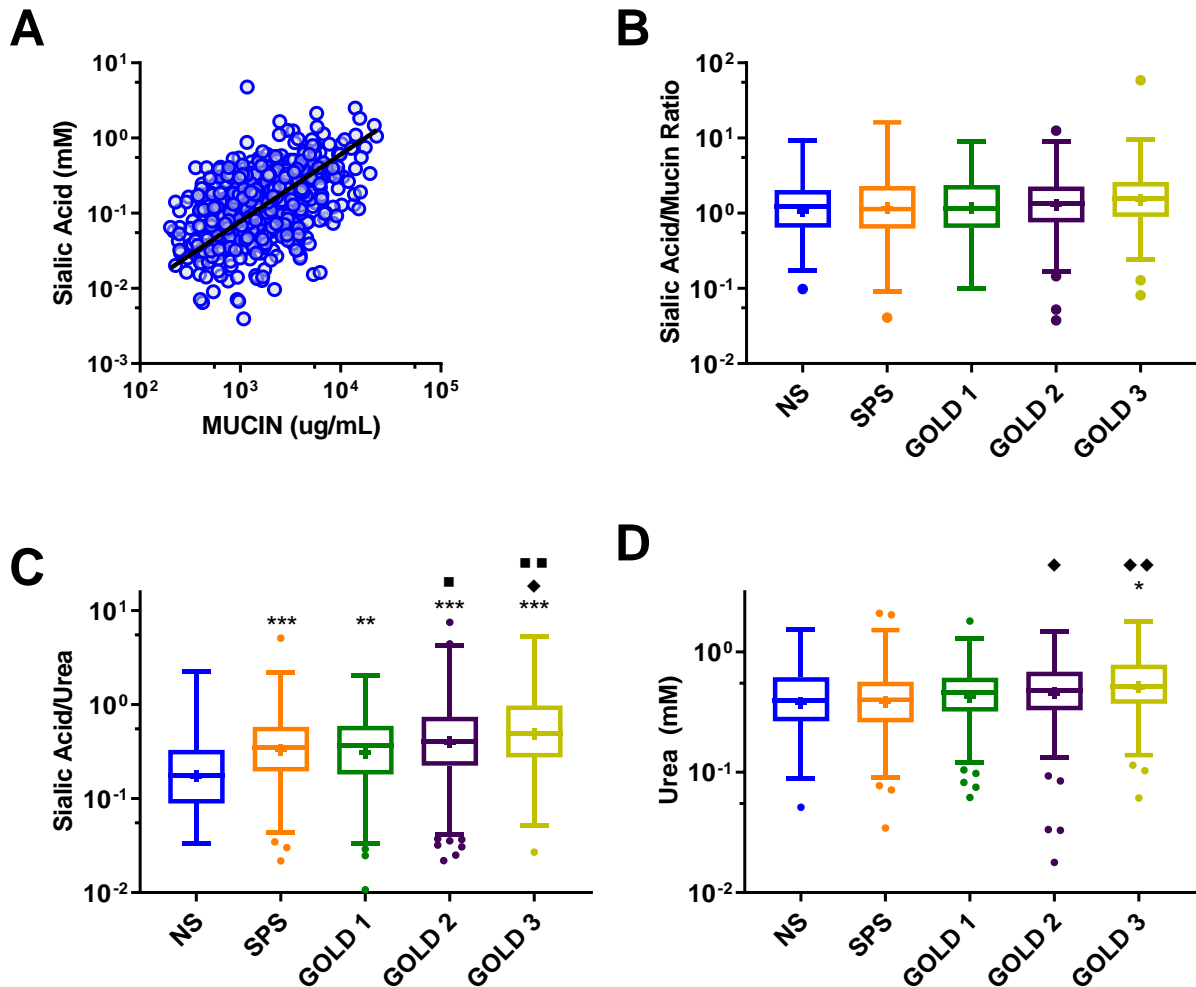
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e-Figure 1



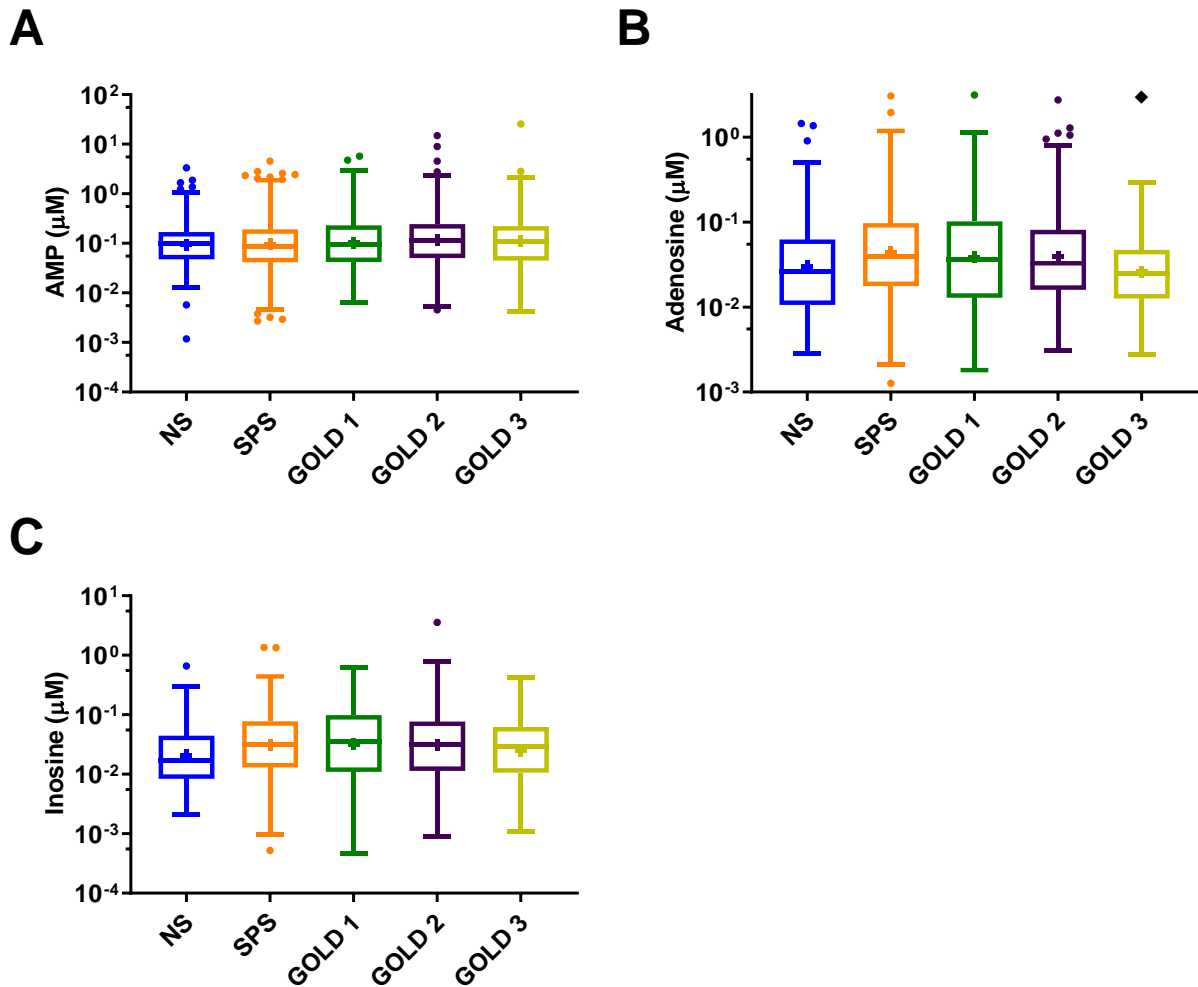
e-Figure 1. Study Analytic Cohort. From the full SPIROMICS cohort, sputum supernatanta samples were obtained from all subjects who had sputum mucin concentrations measured in a prior study and had sputum supernatant available for biomarker analysis (n=748). To reach the target analytic cohort of 980, samples from an additional 232 subjects were chosen at random from the full SPIROMICS cohort. Distribution of controls (NS = healthy non-smokers, SPS = smokers with preserved spirometry) and subjects with COPD stratified by GOLD status (GOLD 1, GOLD 2, and GOLD 3) are shown for each cohort.

e-Figure 2



e-Figure 2. Sputum sialic in COPD. A) Sputum sialic acid was positively correlated ($r=0.44$, $p<0.001$) with sputum mucins measured via physical methods in the 748 samples in which both measures were available. B) The ratio of sialic acid measured in this study to mucins measured independently via refractometry did not vary among cohorts. C) Sputum sialic acid/urea was increased in all GOLD cohorts relative to NS, and increased in GOLD 2 and GOLD 3 relative to S or GOLD 1 (** $=p<0.01$ vs NS, ◆ $=p<0.05$ vs S, ■ $=P<0.01$ vs GOLD 1, ▀ $=P<0.05$ vs GOLD 1). D) Sputum urea was increased in the most severe GOLD cohorts.

e-Figure 3



e-Figure 3. Sputum adenosine pathways. A) No differences among cohorts was observed for sputum AMP. B) In cohort analysis, sputum adenosine was lower in GOLD 3 than in S. C) No differences among cohorts was observed for sputum inosine.