**Objective:** Crohn’s disease is a clinically heterogeneous disease, with multiple approved treatments that target different arms of the host immune system. Although treatment efficacy may very well vary by subgroups commonly documented in clinical trials, our understanding of IBD precision medicine remains in its infancy and current treatment selection largely ignores these factors. This is in part because individual studies are insufficiently powered to assess these groups and a methodologically-proper subgroup analysis requires access to individual participant level-data (IPD) from the original trials. We sought to estimate the treatment efficacy of all FDA-approved treatments for moderate-to-severe Crohn’s disease in each of these clinically-distinct groups.

**Methods Used:** We performed a search of all phase 2/3, placebo-controlled randomized trials of all drugs for moderate-to-severe Crohn’s disease that studied their FDA-approved dose in adults. We requested and obtained access to 27 trials (~13,303 patients) of IPD, distributed across 3 separate virtual machines.

We began a process of data harmonization and quality control followed by fitting non-linear mixed effects model that treats the outcome (Crohn’s Disease Activity Index, CDAI) as an asymptotic function of time and treatment assignment. We sought to sequentially assess a series of other covariates by evaluating model convergence and goodness-of-fit. The fixed covariates included age, gender, height, weight, smoking status, race, lower GI disease location, upper GI disease location, presence of a perianal fistula, use of immunomodulators, and the use of glucocorticoids. Random effects included patient identifier, trial identifier, and drug target. These models were fit using the lme4 package within the R statistical computing environment.

Outcome measures included the asymptotic CDAI score, CDAI half-life, and estimates of CDAI at week 8 and week 26 by post-hoc model estimation. We planned to compute bootstrap confidence intervals and export summary estimates from each platform to facilitate a frequentist network meta-analysis. Summary estimates taken from trials that only follow induction-phase responders into the maintenance phase were to be corrected by using estimates derived from other trials that followed all induction participants to the maintenance timepoint. The bias of the overall analytic procedure was to be estimated by simulation.

**Results:**

In the process of quality control, we identified some minor differences between the calculated CDAIs and the site investigator-reported CDAI scores. We successfully performed harmonization of the data corresponding to the included trials from Certolizumab, Infliximab, and Ustekinumab. We successfully fit the planned mixed-effect model for a preliminary model incorporating CDAI as a function of visit and assignment to placebo vs non-placebo (Infliximab and Ustekinumab combined) with random effects terms for the subject-specific baseline and asymptotic CDAI score. This process culminated in interpretable parameters and a baseline goodness-of-fit. Additional planned work and analysis are still ongoing.

**Conclusions:** N/A