with ST-3672 (n=22) predominant. At the bivariate level, infection with ST-3672 was associated with younger age (62% of those infected were 15–19 years old, p=0.002), and chlamydia co-infection (67% vs 37%, p=0.012). In multivariable analysis, age group remained significant, while an interaction between inner-core residency and chlamydia co-infection was detected. Case-contact networks were highly-fragmented, consisting mainly of dyads and triads. Of 85 components, the largest component included 6 nodes, while 61% were dyads. CUG testing indicated in-degree centralization was lower than expected (p<0.05). Genotyping combined with case-contact data increased the potential size and geographic reach of each component. Of potential components found after incorporating subtypes, 32% (10/33) were dyadic, with the largest component consisting of 45 nodes.

Conclusion Molecular data revealed connections that were not apparent from case-contact investigations alone, leading to more cases potentially linked together, and over a wider geographic area. A handful of subtypes were responsible for the majority of infections. Early identification of dominant subtypes may potentially curtail transmission of NG.

Disclosure No significant relationships.

P689 TURNING GONORRHEA AGAINST HIV: LATENT HIV ‘SHOCK-AND-KILL’ USING A GONOCOCCAL-DERIVED METABOLITE

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Background Clinical studies have long indicated that a pathological synergy exists between Neisseria gonorrhoeae and HIV, with gonococcal infection increasing HIV transmission between HIV serodiscordant sexual partners. In trying to understand this association, we discovered that N. gonorrhoeae liberate a small molecule that stimulates HIV replication from latently infected CD4+ T cells. This led to our discovery that heptose phosphate (HP)-containing metabolites, 7-carbon phospho-sugars not produced by animals, serve as a molecular cue that bacteria are present in the tissues and elicit an NF-kB-dependent transcriptional response. Based upon these observations, this study aims to test the hypothesis that HP can function both to (i) drive the virus from latency and (ii) stimulate the antiviral response to work in synergy with available highly active antiretroviral therapies to cure HIV infection.

Methods We have used a combination of cell line and primary human leukocyte-based models to test the effect of natural and synthetic analogues of HP to stimulate HIV from latency, both alone and in combination with potential latency reversing agents, and to understand their effect of HPs on different leukocytic populations that have potential to either promote or inhibit HIV infection.

Results We show that HP has a superior combination of HIV latency reversal without toxicity often evident with conventional LRAs, and HP activity synergizes with other LRAs such that these can be administered at lower concentrations. Finally, we observed that HP stimulates primary human leukocytic responses with anti-viral potential.

Conclusion Our findings suggest that HP-based agonists are a novel LRA capable of both driving HIV from latency and stimulating immune responses so as to help control the infection. By virtue of its synergy with other LRAs and clinically available anti-retroviral agents, this represents an enticing new avenue in ongoing efforts to develop a cure for established HIV infection.

Disclosure No significant relationships.