Developed IFIs. However, several IFIs were reported with real-life use of IBR. In randomized clinical trials, <1% of IBR-treated CLL patients received a diagnosis of invasive fungal infection (IFI). Of the 821 CLL IBR-treated patients, 24 developed probable or proven IFI (2.9%). Of these infections, 21 occurred within 30 days (d) of last IBR dose, while 3 IFIs occurred at 94, 135 and 221 d post IBR, respectively. The majority of patients with IFI were male (83%) with a median age of 66 years at IFI diagnosis. The median prior duration of therapy for Richter's transformation at the time of IFI diagnosis was 2 years, while 2 patients had prior stem cell transplant. The average time from start of IBR to diagnosis of IFI was 338 d, with only 7 cases of IFI within the first 3 months of IBR. The majority of IFIs were proven/probable aspergillosis (63%), including 9 cases of Aspergillus fumigatus. The remaining infections consisted of Cryptococcus neoformans (21%), Fusarium spp. (8%), with one case each of candidiasis, histoplasmosis, mucormycosis, and Pneumocystis jiroveci pneumonia. Three patients had evidence of poly-fungal IFI. The sites of infection were pulmonary (88%), blood (13%), CNS (13%), and sinus (8%). Five patients were diagnosed with disseminated IFI, including Cryptococcus spp. (2 cases), Rhizopus spp., Aspergillus spp., and Candida spp. The 42-day mortality rate post IFI diagnosis was 25%.

Conclusion. We report the largest single-center cohort of CLL patients on IBR treated with IFIs (71%) were diagnosed > 3 months after starting IBR. We are currently conducting a case–control comparison of IBR–treated CLL patients with no infection to uncover risk factors associated with IFIs in these patients.

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1720. Isolation and Characterization of Candida auris From an Active Surveillance System in Texas

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Background. Candida auris is an emerging new multi-drug-resistant fungal pathogen spreading globally. C. auris is associated with outbreaks due to the bloodstream, ear, and wound infections with a high mortality rate (30 to 60%). As part of our multi-pathogen surveillance system, we began screening for C. auris to understand the ecology, sources, and epidemiology of this important pathogen from leftover stool samples collected from hospitalized patients.

Methods. Four hundred and seventeen stool samples were collected, enriched in brain heart infusion broth for 2–3 d at 37°C, and sub-cultured onto selective Candida agar plates. Agar plates were incubated at 37°C for another 2–3 d and suspect colonies were streaked onto blood and MacConkey agar. C. auris colonies were identified by PCR (0.7%; 3/417) of which 3 were confirmed by PCR. PCR amplicons were sequenced to confirm the identification of C. auris. Enrichment samples were also screened by PCR to directly detect C. auris. Minimum inhibitory concentration (MIC) of various anti-fungal drugs was determined by the micro-dilution method using a commercial MIC plate (Sensititre YeastOne™).

Results. Three C. auris samples were identified by PCR (0.7%; 3/417) of which one was able to be cultured. The isolated strain was resistant to fluconazole, itraconazole, and voriconazole, posaconazole, and caspofungin. WGS data analysis demonstrates our isolate has high identity with the Pakistan strains.

Conclusion. We have detected C. auris from stool samples of hospitalized patients in Texas for the first time. WGS data indicate our isolate has high similarity with South Asian patient isolates. Long-term surveillance of C. auris is essential to understand the infection or colonization sources and epidemiology of this newly emerging fungal pathogen.

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1721. A Transcriptional Signature of Acute Aspergillus Infection Offers High Diagnostic Accuracy In Vitro Despite the Presence of Immunosuppression

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Background. Aspergillosis (IA) is a major cause of critical illness in immunocompromised (IC) patients. However, current fungal testing methods have significant limitations and there is a clear need for new diagnostic options. Disease-specific gene expression patterns in circulating host cells show promise as novel diagnostic markers, but it is unknown whether a "signature" exists. We hypothesized,
there is a need for better understanding of the effect of iatrogenic immunosuppression (present in most cases of IA) on such host response-driven biomarkers.

**Methods.** Male BALB/c mice were separated into an *Aspergillus fumigatus* inhalational exposure group and a placebo group. These two groups were each subdivided into three additional sets based on immunocompromised status (no immunosuppression, cyclophosphamide, and corticosteroids) for a total of six experimental groups. Mice were sacrificed 4 days post-infection. Whole blood was assayed for transcriptional responses via microarray. Bayesian techniques were utilized to develop classifiers of IA and leave one out cross-validation was used to estimate predictive probabilities.

**Results.** *Aspergillus* infection triggers a powerful response in non-IC hosts, with 2896 genes differentially expressed between IA and controls. We generated a 146-gene expression classifier able to discriminate between non-IC mice with IA and uninfected non-IC mice with 100% accuracy. However, the presence of immunosuppressive drugs exhibited a strong confounding effect on the transcriptional classifier that was derived in the absence of immunosuppression. After controlling for the genomic effects of immunosuppressive drugs, we were able to generate a 187-gene classifier with a sensitivity of 100% and specificity of 97% across all IC states.

**Conclusion.** The host transcriptional response to IA is robust and highly conserved. Pharmacologic perturbation of the host immune response unsurprisingly has powerful effects on gene expression-based classifier performance and must be taken into account when developing novel diagnostics. When appropriately designed, host-derived peripheral blood transcriptional responses to IA demonstrate the ability to accurately diagnose *Aspergillus* infection, even in the presence of immunosuppression.

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1722. Histoplasmosis Acquired in Alberta, Canada, 2011–2018
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**Background.** Histoplasmosis is a serious fungal infection caused by the geographically restricted, dimorphic fungus *Histoplasma capsulatum*. In Canada, the geographic range of *H. capsulatum* is classically thought to be restricted to southern parts of Ontario and Quebec. Over the past decade, histoplasmosis has occasionally been diagnosed in patients in Alberta without travel to areas of known geographic risk (Figure 1). We studied the epidemiology and geographic distribution of histoplasmosis in Alberta to assess evidence for locally-acquired infections.

**Methods.** We retrospectively reviewed all laboratory-confirmed (culture, antigen and/or immunodiffusion positive) cases of histoplasmosis diagnosed from January 1, 2011 to June 30, 2018. Data collected by public health and clinical charts were reviewed for clinical presentation, exposure and travel histories, and geographic distribution of cases. Cases of histoplasmosis in patients who had not left Alberta or associated with a local point source were classified as definite local acquisition; cases in patients with remote travel but with local exposures and appropriate timing of disease onset were deemed "probable" cases of local infection. University of Alberta's Research Ethics Board approved this study.

**Results.** We identified 45 laboratory-confirmed cases of histoplasmosis, including 17 cases that were locally acquired. Among these, there were 12 cases of definite local acquisition, including 8 patients from 3 point-source outbreaks—all involving exposure to bats and/or their droppings in chimneys or attics of private dwellings or churches—and 4 sporadic cases in patients who had never traveled. Of the other 5 cases probably acquired in Alberta, patients had previously traveled (n = 4) or travel history was incomplete (n = 1) but local exposures preceding infection were considered compelling. The mean incidence rate of locally acquired infection was 0.062/100,000 population with an incidence increasing since 2014. Table 1 shows features of locally acquired cases.

**Conclusion.** This study, for the first time, establishes Alberta as a region of geographic risk for histoplasmosis. The diagnosis should be considered in patients with compatible symptoms and exposure history, even in the absence of travel.

1723. Human Serum Albumin Regulates the Growth of *Candida auris* in vitro
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**Background.** *Candida auris* is commonly detected in human ear secretions. However, *C. auris* occasionally causes bloodstream infections even in immunocompetent patients resulting in poor prognosis. It was speculated that *C. auris* growth within the blood might be regulated by proteins in the bloodstream. Thus, in this study, the potential role of blood proteins in the regulation of *C. auris* growth was investigated.

**Methods.** Five Candida species (*C. albicans*, *C. auris*, *C. glabrata*, *C. parapsilosis*, and *C. tropicalis*) were incubated overnight. Colony suspensions for each species were prepared and adjusted to OD 1.0 at absorbance 0.1. Then, human serum albumin (HSA) and bovine serum albumin (BSA) were diluted (2.5 g/dL–0.002 g/dL) and mixed with the suspensions. Mixed samples were adjusted to 100 μL and incubated on MHA plates at 35°C for 2 days. Then, 50 μL of the combined sample was extracted and streaked onto Yeast extract-Pepote-Dextrose (YPD) agar. The remaining 50 μL sample was analyzed using an XTT assay. Further testing was then conducted on the effects of a specific blood protein albumin on *Candida*. Thereby, *C. albicans* and *C. auris* were cultured following the procedure above and stained with Annexin V and PI.

**Results.** The growth of *C. auris* mixed with a high albumin concentration (2.5–0.15 g/dL) was regulated compared with that of other Candida species (P = 0.001) (Figures 1 and 2); however, the growth of *C. auris* mixed with a lower albumin concentration was similar to that of other species. The wash-out study showed that *C. auris* growth and survival in the high albumin concentration was not different than that of other species.

**Conclusion.** HSA and BSA regulated *C. auris* growth which led to increased necrosis of *C. auris*. Conversely, growth of the other Candida species was not regulated. Therefore, albumin might be involved in the growth and necrosis of *C. auris*. As the highest concentration at which albumin regulated *C. auris* growth was similar to that found in human serum, it is possible that serum albumin might help prevent *C. auris* from entering the bloodstream via the ear or skin.