

## 19.103

**Pneumocystis jiroveci pneumonia the who and the where identifying the population at risk**L.A. McCorry<sup>a,\*</sup>, A. O'grady<sup>b</sup><sup>a</sup> Belfast Trust, Infection, Belfast, ANTRIM/UK<sup>b</sup> Royal Victoria Hospital, Medical, Belfast/UK

**Purpose:** *Pneumocystis jiroveci* is an opportunistic pathogen which can lead to life threatening respiratory failure. It has a documented mortality of between 5–20%. Historically it was almost exclusive to HIV patients however; an increase in immunosuppressive therapies has led to a reciprocal increase in the prevalence of *Pneumocystis pneumonia* (PJP) in this non-HIV population.

Despite clear guidelines for PJP prophylaxis in HIV, there is a haphazard approach in other immunosuppressed populations.

A growing body of evidence suggests that immunosuppressed patients are at an increased risk of PJP, but to what extent, or to whom that risk is greatest is not certain.

Given this uncertainty we felt it prudent to review regional rates of PJP to develop a clearer understanding of the potential at risk population.

**Methods & Materials:** We audited a random cohort of 103 patients with *Pneumocystis pneumonia* over a 5 year period in a region in the United Kingdom. We collated information on potential risk factors, morbidity and mortality.

**Results:** The highest proportion of patients were cancer patients, however the most at risk population were rheumatology patients, with a 73% mortality rate in those who tested positive for PCP.

Admission to ICU was 38% and mortality was 32%. Mortality was highest when prescribed three modalities of immunosuppression in combination; however prednisolone alone carries a mortality rate of 62%.

**Conclusion:** We feel that prophylaxis guidelines should be considered in these identified high risk groups, but much more study is required on the absolute risk, and on how prophylaxis should be approached.

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## 19.105

**Reducing the impact of emerging infections through data sharing**L. Merson, A. Vessiere<sup>\*</sup>

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**Purpose:** Data sharing has the potential to shift the paradigm of outbreak response in the public health, clinical and scientific communities. Increasing access to more data can address some of the key challenges of the limited, fragmented, poor quality data, which characterise emerging infections. To achieve this, we must design well-governed, equitable, collaborative data sharing models driven by the questions relevant to those directly affected, and ensure that the benefits of sharing are returned to them. We must also implement methodologies that maximize the usability of data to generate evidence.

**Methods & Materials:** A secure, flexible, technical architecture was developed based on lessons learned from other industries. Careful, centralised curation of varied types and sources of data has been applied to minimize overlapping resource investment and expedite implementation of evidence-generating analysis. We

continue to work towards the establishment of appropriate governance models and benefits sharing for each disease community.

**Results:** Working with the research community addressing the emergence of antimalarial resistance, we have built a collaborative model of sharing data that has successfully pooled and analysed more than 80% of the clinical trials on critical antimalarials to deliver policy-changing evidence. This has served as the prototypic model for the development of new platforms for Ebola and Zika, with the aim of enabling sharing within a useful timeframe to affect the outbreak response. The varied technical and governance frameworks of the platforms assert that different types of data, and different communities of data generators, have different needs.

**Conclusion:** Failure to share data on emerging infections impedes outbreak response. In order to address this, investment must be made in sustainable infrastructure and governance for data sharing in advance of the outbreak. Data sharing has the potential to improve knowledge on many diseases, but resource limitations mean that efforts should be focused where they can have the greatest impact. The global threat of emerging infections puts these high on the priority list.

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## 19.108

**Environmental persistence of Human Rotavirus causing a large outbreak in Western Mindanao, Philippines, 2016**J.M. Bonifacio<sup>a,\*</sup>, M.A. Igoy<sup>b</sup>, S. Lupisan<sup>c</sup>, A. Tandoc<sup>a</sup><sup>a</sup> Research Institute for Tropical Medicine, Department of Virology, Muntinlupa City/PH<sup>b</sup> Research Institute for Tropical Medicine, Molecular Biology, Muntinlupa City/PH<sup>c</sup> Research Institute for Tropical Medicine, Muntinlupa City/PH

**Purpose:** Enteric viruses are a leading cause of acute diarrhea outbreaks in the Philippines. In March 2016, a large diarrheal outbreak occurred in Zamboanga del Sur, affecting 1,538 cases with 6 reported deaths. Human specimens from the outbreak showed insignificant numbers of bacterial and parasitic pathogens were isolated in. To develop effective preventive measures, epidemiological information of viral agents must be determined. This study aims to investigate the presence of three enteric viruses in stools and water samples collected during the Zamboanga outbreak.

**Methods & Materials:** A total of 106 stool and 10 water samples (approximately 1-L each) from water refilling stations, household water and deep wells were collected to determine the presence of three enteric viruses: group A rotavirus, norovirus and adenovirus by real-time polymerase chain reaction. Water samples were concentrated by precipitation using polyethyleneglycol hydroextraction technique. Nucleotide sequencing and analysis using MEGA 6 software were performed to determine the prevailing genotypes on samples detected with rotavirus, norovirus and adenovirus.

**Results:** Enteric viruses were detected in 80 (75.5%) of the 106 cases of acute diarrhea. In 106 samples, rotavirus A was the most frequent virus identified in 65 (61.3%) cases, followed by norovirus in 5 (4.7%) cases, and adenovirus in 1 (0.9%) case. 9 (8.5%) out of the total specimens also showed mixed infection. Only rotavirus A was identified in 5 of the 10 water samples collected. G9P[8] and G3P[8] rotavirus genotypes were identified in both water and human samples.