3.28.

Faria NR, Azevedo RD, Kraemer MU, Souza R, Cunha MS, Hill SC, Thézé J, Bonsall MB, Bowden TA, Rissanen I, Rocco IM, Nogueira JS, Maeda AY, Vasami FG, Macedo FL, Suzuki A, Rodrigues SG, Cruz AC, Nunes BT, Medeiros DB, Rodrigues DS, Nunes Queiroz AL, Silva EV, Henriques DF, Travassos da Rosa ES, de Oliveira CS, Martins LC, Vasconcelos HB, Casseb LM, Simith DB, Messina JP, Abade L, Lourenço J, Alcantara LC, Lima MM, Giovanetti M, Hay SI, de Oliveira RS, Lemos PD, Oliveira LF, de Lima CP, da Silva SP, Vasconcelos JM, Franco L, Cardoso JF, Vianez-Júnior JL, Mir D, Bello G, Delatorre E, Khan K, Creatore M, Coelho GE, de Oliveira WK, Tesh R, Pybus OG, Nunes MR, Vasconcelos PF. Zika virus in the Americas: Early epidemiological and genetic findings. Science. 2016 Mar 24.

Brazil has experienced an unprecedented epidemic of Zika virus (ZIKV), with ~30,000 cases reported to date. ZIKV was first detected in Brazil in May 2015 and cases of microcephaly potentially associated with ZIKV infection were identified in November 2015. Using next generation sequencing we generated seven Brazilian ZIKV genomes, sampled from four self-limited cases, one blood donor, one fatal adult case, and one newborn with microcephaly and congenital malformations. Phylogenetic and molecular clock analyses show a single introduction of ZIKV into the Americas, estimated to have occurred between May-Dec 2013, more than 12 months prior to the detection of ZIKV in Brazil. The estimated date of origin coincides with an increase in air passengers to Brazil from ZIKV endemic areas, and with reported outbreaks in Pacific Islands. ZIKV genomes from Brazil are phylogenetically interspersed with those from other South American and Caribbean countries. Mapping mutations onto existing structural models revealed the context of viral amino acid changes present in the outbreak lineage; however no shared amino acid changes were found among the three currently available virus genomes from microcephaly cases. Municipality-level incidence data indicate that reports of suspected microcephaly in Brazil best correlate with ZIKV incidence around week 17 of pregnancy, although this does not demonstrate causation. Our genetic description and analysis of ZIKV isolates in Brazil provide a baseline for future studies of the evolution and molecular epidemiology in the Americas of this emerging virus.T

Comment: Another major outbreak that underlines the importance of having trained microbiologists and a reactive collaborative network to implement the appropriate measures, including development of accurate diagnostic tools

3.29.

Cauchemez S, Besnard M, Bompard P, Dub T, Guillemette-Artur P, Eyrolle-Guignot D, Salje H, Van Kerkhove MD, Abadie V, Garel C, Fontanet A, Mallet HP. Association between Zika virus and microcephaly in French Polynesia, 2013-15: a retrospective study. Lancet. 2016 Mar 15.

BACKGROUND: The emergence of Zika virus in the Americas has coincided with increased reports of babies born with microcephaly. On Feb 1, 2016, WHO declared the suspected link between Zika virus and microcephaly to be a Public Health Emergency of International Concern. This association, however, has not been precisely quantified.

METHODS: We retrospectively analysed data from a Zika virus outbreak in French Polynesia, which was the largest documented outbreak before that in the Americas. We used serological and surveillance data to estimate the probability of infection with Zika virus for each week of the epidemic and searched medical records to identify all cases of microcephaly from September, 2013, to July, 2015. Simple models were used to assess periods of risk in pregnancy when Zika virus might increase the risk of microcephaly and estimate the associated risk.

FINDINGS: The Zika virus outbreak began in October, 2013, and ended in April, 2014, and 66% (95% CI

62-70) of the general population were infected. Of the eight microcephaly cases identified during the 23-month study period, seven (88%) occurred in the 4-month period March 1 to July 10, 2014. The timing of these cases was best explained by a period of risk in the first trimester of pregnancy. In this model, the baseline prevalence of microcephaly was two cases (95% CI 0-8) per 10 000 neonates, and the risk of microcephaly associated with Zika virus infection was 95 cases (34-191) per 10 000 women infected in the first trimester. We could not rule out an increased risk of microcephaly from infection in other trimesters, but models that excluded the first trimester were not supported by the data

Comment: A work supporting a clear relationship between zikavirus and microcephaly when infection occurred during first trimester

3.30.

Cao-Lormeau VM, Blake A, Mons S, Lastère S, Roche C, Vanhomwegen J, Dub T, Baudouin L, Teissier A, Larre P, Vial AL, Decam C, Choumet V, Halstead SK, Willison HJ, Musset L, Manuguerra JC, Despres P, Fournier E, Mallet HP, Musso D, Fontanet A, Neil J, Ghawché F. Guillain-Barré Syndrome outbreak associated with Zika virus infection in French Polynesia: a case-control study. Lancet. 2016 Feb 29.

BACKGROUND: Between October, 2013, and April, 2014, French Polynesia experienced the largest Zika virus outbreak ever described at that time. During the same period, an increase in Guillain-Barré syndrome was reported, suggesting a possible association between Zika virus and Guillain-Barré syndrome. We aimed to assess the role of Zika virus and dengue virus infection in developing Guillain-Barré syndrome.

METHODS: In this case-control study, cases were patients with Guillain-Barré syndrome diagnosed at the Centre Hospitalier de Polynésie Française (Papeete, Tahiti, French Polynesia) during the outbreak period. Controls were age-matched, sex-matched, and residence-matched patients who presented at the hospital with a non-febrile illness (control group 1; n=98) and age-matched patients with acute Zika virus disease and no neurological symptoms (control group 2; n=70). Virological investigations included RT-PCR for Zika virus, and both microsphere immunofluorescent and seroneutralisation assays for Zika virus and dengue virus. Anti-glycolipid reactivity was studied in patients with Guillain-Barré syndrome using both ELISA and combinatorial microarrays.

FINDINGS: 42 patients were diagnosed with Guillain-Barré syndrome during the study period. 41 (98%) patients with Guillain-Barré syndrome had anti-Zika virus IgM or IgG, and all (100%) had neutralising antibodies against Zika virus compared with 54 (56%) of 98 in control group 1 (p<0·0001). 39 (93%) patients with Guillain-Barré syndrome had Zika virus IgM and 37 (88%) had experienced a transient illness in a median of 6 days (IQR 4-10) before the onset of neurological symptoms, suggesting recent Zika virus infection. Patients with Guillain-Barré syndrome had electrophysiological findings compatible with acute motor axonal neuropathy (AMAN) type, and had rapid evolution of disease (median duration of the installation and plateau phases was 6 [IQR 4-9] and 4 days [3-10], respectively). 12 (29%) patients required respiratory assistance. No patients died. Anti-glycolipid antibody activity was found in 13 (31%) patients, and notably against GA1 in eight (19%) patients, by ELISA and 19 (46%) of 41 by glycoarray at admission. The typical AMAN-associated anti-ganglioside antibodies were rarely present. Past dengue virus history did not differ significantly between patients with Guillain-Barré syndrome and those in the two control groups (95%, 89%, and 83%, respectively).

INTERPRETATION: This is the first study providing evidence for Zika virus infection causing Guillain-Barré syndrome. Because Zika virus is spreading rapidly across the Americas, at risk countries need to prepare for adequate intensive care beds capacity to manage patients with Guillain-Barré syndrome.

Comment: A major work providing evidences for a causative role of the Zika virus in Guillain-Barré syndrome