PA-018 **SEASONAL MALARIA CHEMOPREVENTION WITH SULPHADOXINE-PYRIMETHAMINE AND AMODIAQUINE SELECTS DHFR-DHPS QUINTUPLE MUTANT GENOTYPE IN MALI**

Hamma Maiga,¹ Estrella Lasry,² Modibo Diarra,¹ Issaka Sagara,¹ Amadou Bamadio,¹ Aliou Traore,¹ Samba Coumare,¹ Bahanan Soma,² Yeyia Dicko,¹ Nouhoum Diallo,¹ Boubou Sangare,¹ Aly Tembely,¹ Djibril Traore,¹ Hamidou Niangaly,¹ François Dao,¹ Aboubecrin Haidara,¹ Alassane Dicko,¹ Ogobara Doumbo,¹ Abdoulaye Djimde¹. ¹MRTC, University of Bamako, Mali; ²Médecins Sans Frontières, United States of America

10.1136/bmjgh-2016-000260.55

Background Seasonal malaria chemoprevention (SMC) with sulfadoxine-pyrimethamine (SP)+amodiaquine (AQ) is being scaled up in countries of the Sahel in West Africa. However, the potential development of *Plasmodium falciparum* resistance to the respective component drugs is a major concern.

Methods Two cross-sectional surveys were conducted before (August 2012) and after (June 2014) a pilot implementation of SMC in Koutiala, Mali. Children aged 3–59 months received 7 rounds of curative doses of SP+AQ over two malaria seasons. Genotypes of *P. falciparum* dhfr codons 51, 59 and 108; dhps codons 437 and 540, pfcr1 codon 76 and pfmdr1 codon 86 were analysed by PCR on DNA from samples collected before and after SMC, and in non-SMC controls.

Results In the SMC population 191/662 (28.9%) and 85/670 (13.7%) of children were *P. falciparum*-positive by microscopy and were included in the molecular analysis before (2012) and after SMC implementation (2014), respectively. In the control population 220/310 (71%) were successfully PCR analysed. In the SMC children the prevalence of all molecular markers of SP resistance increased significantly after SMC including the dhfr-dhps quintuple mutant genotype, which was 1.6% before but 7.1% after SMC ($p=0.02$). The prevalence of Pfmdr1-86Y significantly decreased from 26.7% to 15.3% ($p=0.04$) while no significant change was seen for pfcr1 K76T. In 2014, prevalence of all molecular markers of SP resistance were significantly higher among SMC children compared to the non-SMC control population ($p<0.01$). No dhfr – 164 mutation was found neither at baseline nor post SMC.

Conclusions SMC increased the prevalence of molecular markers of *P. falciparum* resistance to SP in the treated children. However, there was no significant flow of these resistance genes into the general parasite population after 2 years and 7 rounds of SMC.