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The Malaria Gordon Conference is organized every two years. The previous conference (2005) focused on fundamental research, the current on applied research in clinical malaria, epidemiology and vector control. The maximum number of attendants was limited to about 200 (50% from endemic countries) and there were plenary sessions from 9 am til 9.30 pm. The small number of attendants, the intensive programme sessions and the fact that most speakers presented non-published material created a very good atmosphere with a lot of discussion with junior and senior scientists. The location, the beautiful Magdalen College in Oxford, certainly added to the academic atmosphere.

Kevin Marsh chaired the meeting and there were sessions on severe malaria (chair: Charles Newton), pathogenesis of severe malaria (Terrie Taylor), antimalarial drugs (Nick White), malaria in pregnancy (Ogobara Doumbo), novel interventions (Feiko ter Kuile), malaria vectors (Janet Hemmingway), implementation of tools (Malcolm Molyneux) and malaria vaccines (Graham Brown). There was a long discussion about the reason why malaria transmission intensity appears to go down in most fieldsites. Is it the wide-scale use of long lasting nets? Is artemisinin-based combination therapy reducing malaria transmission on a population level? Importantly, now that we have tools that seem to work, how should we make sure they are available to the general public and that sufficient funding is available for the coming decades? Some senior researchers suggested that we can again aim for malaria eradication, at least in many (low) endemic areas. On every day, there was also a poster session. The limited number of posters (total of 100) and an elaborate poster walk by Kevin Marsh, ensured that all posters were discussed in detail. Five posters (including one of the two I presented) were selected for an oral presentation.

Poster and oral presentation: **Increased *Plasmodium falciparum* gametocyte production in mixed infections with *P. malariae*.** Teun Bousema, Chris Drakeley, Petra Mens, Theo Arens, Rein Houben, Sabah Omar, Louis Gouagna, Henk Schallig, Robert Sauerwein

Plasmodium falciparum and *P. malariae* occur endemically in many parts of Africa. Observations from malariatherapy patients suggest that co-infection with *P. malariae* may boost *P. falciparum* gametocyte production. We determined *P. falciparum* gametocyte prevalence and density by quantitative nucleic acid sequence-based amplification (QT-NASBA) following antimalarial treatment of Kenyan children with either *P. falciparum* mono-infection or *P. falciparum* and *P. malariae* mixed infection. In addition, we analysed the relationship between mixed species infections and microscopic *P. falciparum* gametocyte prevalence in three datasets from previously published studies.

In Kenyan children, QT-NASBA gametocyte density was elevated in mixed species infections ($p = 0.03$). We also observed higher microscopic prevalences of *P. falciparum* gametocytes in mixed species infections in studies from Tanzania and Kenya (OR 2.15 (0.99-4.65) and 2.39 (1.58-3.63)) but not in a study from Nigeria. These data suggest that co-infection with *P. malariae* is correlated with increased *P. falciparum* gametocytaemia.