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PUBLICATIONS OF RELEVANCE TO PAPUA NEW GUINEA AND MELANESIA

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- 1 **Allen SJ, O'Donnell A, Alexander NDE, Alpers MP, Peto TEA, Clegg JB, Weatherall DJ.** Alpha+-thalassemia protects children against disease caused by other infections as well as malaria.
Proc Natl Acad Sci USA 1997 Dec 23;94(26):14736-14741
In the South-West Pacific region, the striking geographical correlation between the frequency of alpha+-thalassemia and the endemicity of *Plasmodium falciparum* suggests that this hemoglobinopathy provides a selective advantage against malaria. In Vanuatu, paradoxically, alpha+-thalassemia increases the incidence of contracting mild malaria in the first 2 years of life, but severe disease was too uncommon to assess adequately. Therefore, we undertook a prospective case-control study of children with severe malaria on the north coast of Papua New Guinea, where malaria transmission is intense and alpha+-thalassemia affects more than 90% of the population. Compared with normal children, the risk of having severe malaria was 0.40 (95% confidence interval 0.22-0.74) in alpha+-thalassemia homozygotes and 0.66 (0.37-1.20) in heterozygotes. Unexpectedly, the risk of hospital admission with infections other than malaria also was reduced to a similar degree in homozygous (0.36; 95% confidence interval 0.22-0.60) and heterozygous (0.63; 0.38-1.07) children. This clinical study demonstrates that a malaria resistance gene protects against disease caused by infections other than malaria. The mechanism of the remarkable protective effect of alpha+-thalassemia against severe childhood disease remains unclear but must encompass the clear interaction between this hemoglobinopathy and both malarial and nonmalarial infections.
- 2 **Al-Yaman F, Genton B, Taraika J, Anders R, Alpers MP.** Association between cellular response (IL-4) to RESA/Pf155 and protection from clinical malaria among Papua New Guinean children living in a malaria-endemic area.
Parasite Immunol 1997 Jun;19(6):249-254.
A prospective study in 207 children aged 0.5-15 years was carried out to examine the relationship between cellular responses to *Plasmodium falciparum* ring-infected erythrocyte surface antigen (RESA) and malaria infection and morbidity. The prevalence of lymphoproliferative response to RESA was 13%, IFN-gamma prevalence was 40% and IL-4 prevalence was 22%. Only the IFN-gamma response to RESA increased significantly with age. When proliferation or stimulation of either cytokine was used to assess T-cell activation the overall frequency of responders increased to 55%. The proliferative and IFN-gamma response to RESA were positively associated. Although there was no association between any of the CMI responses to RESA and concurrent morbidity the prevalence of IL-4 response to RESA was significantly lower in children who experienced clinical malaria in the following year. These results coupled with our earlier data showing a negative relationship between humoral responses to RESA and malaria morbidity support the inclusion of RESA in a subunit vaccine against malaria.
- 3 **Al-Yaman F, Genton B, Reeder JC, Anders RF, Smith T, Alpers MP.** Reduced risk of clinical malaria in children infected with multiple clones of *Plasmodium falciparum* in a highly endemic area: a prospective community study.
Trans R Soc Trop Med Hyg 1997 Sep-Oct;91(5):602-605.
A prospective community study in a highly malaria-endemic area of Papua New Guinea found that infection with multiple *Plasmodium falciparum* genotypes was an indicator of lowered risk of subsequent clinical attack. The results suggest that concurrent or very recent infections provide protection from superinfecting parasites. The finding of an association between reduced risk of clinical malaria and infection with parasites of merozoite surface protein 1 (MSP-1) type RO33 or MSP-2 type 3D7 further suggests that the concomitant immunity is, at least in part, a consequence of a response to these major merozoite surface proteins.
- 4 **Barat LM, Bloland PB.** Drug resistance among malaria and other parasites.
Infect Dis Clin North Am 1997 Dec;11(4):969-987.
Recent decades have witnessed the emergence and spread of parasites resistant to standard drug therapies, particularly malaria. Chloroquine-resistant *Plasmodium falciparum* has now spread to most malarial areas, and resistance to other antimalarial drugs, including mefloquine and sulfadoxine-pyrimethamine, have become significant problems in some parts of Southeast Asia and South America. Chloroquine-resistant *P. vivax* is well established in Papua New Guinea and Indonesia and has been reported in other areas. *Trichomonas* and *Giardia* infections resistant to metronidazole have also been documented. This article reviews the current status of drug resistance among parasites, particularly malaria, and offers strategies for managing patients with these infections.

- 5 **Barclay L.**
Midwifery in Australia and surrounding regions.
Midwifery 1997 Sep;13(3):111-114.

- 6 **Bindon JR, Baker PT.**
Bergmann's rule and the thrifty genotype.
Am J Phys Anthropol 1997 Oct;104(2):201-210.

One of Roberts' key contributions was his work demonstrating the applicability of several ecological rules to human populations (Roberts [1953] *Am. J. Phys. Anthropol.* 11:533-558; [1978] *Climate and Human Variability*, 2nd ed., Menlo Park, CA: Cummings). His finding that average body weight systematically covaries with mean annual temperature was widely taken as confirmation of Bergmann's rule for humans. More recently his findings on weight and temperature have been extended and confirmed (Ruff [1994] *Yrbk. Phys. Anthropol.* 37:65-407; Katzmarzyk and Leonard [1995] *Hum Biol Council Program Abstracts* 132) although the strength of the association may be decreasing when considering more recent surveys (Katzmarzyk and Leonard [1995]). Roberts noted in 1953 that Oceanic populations may be somewhat of an exception to Bergmann's rule, and we propose that Neel's ([1962] *Am. J. Hum. Genet.* 14:353-362) thrifty genotype model may account for some of the deviation from predicted weights among these populations. We provide an updated version of the thrifty genotype model, suggesting that selection for energetic efficiency may have occurred for some Oceanic populations during the voyaging to and settlement of their island homes. Under conditions of modernization the thrifty genotype may be manifesting as high rates of obesity and NIDDM among Polynesians and Micronesians. First, using measurements of adult male weight from 19 Oceanic populations, we demonstrate the extreme nature of their deviation from predicted weight based on Roberts' regression of weight on mean annual temperature. Next, we regress the deviations from predicted weight on NIDDM prevalence for these 19 populations, producing a highly significant regression ($r^2 = 0.46$; $p < 0.001$), consistent with expectations if the thrifty genotype is responsible for the high weights.

- 7 **Bockarie MJ, Alexander NDE, Hyun P, Dimber Z, Bockarie F, Ibam E, Alpers MP, Kazura JW.**
Randomised community-based trial of annual single-dose diethylcarbamazine with or without ivermectin against *Wuchereria bancrofti* infection in human beings and mosquitoes.
Lancet 1998 Jan 17;351(9097):162-168.

BACKGROUND: WHO has targeted lymphatic filariasis for elimination. Studies of vector-parasite relations of *Wuchereria bancrofti* suggest that a reduction in the microfilarial reservoir by mass chemotherapy may interrupt transmission and thereby eliminate infection. However, no field data exist on the impact of chemotherapy alone on vector efficiency and transmission intensity of *W. bancrofti*. We compared the impact of an annual community-wide single-dose treatment with diethylcarbamazine alone or with ivermectin on rate and intensity of microfilaraemia, and

transmission intensity in an area of Papua New Guinea endemic for intense *W. bancrofti* transmission. **METHODS:** We carried out clinical and parasitological surveys in 14 communities in matched pairs. People aged 5 years or older in seven communities received randomly assigned diethylcarbamazine 6 mg/kg and people in the other seven communities received diethylcarbamazine 6 mg/kg plus ivermectin 400 micrograms/kg. We made physical examinations for hydroceles and leg oedema and investigated microfilarial densities by membrane filtration before and after treatment. We selected five communities for monthly entomological surveys between September, 1993, and September, 1995. Mosquitoes were collected in these communities by the all-night landing catch method and were individually dissected to identify rates of infection and infectiveness. **FINDINGS:** 2219 (87.6%) of 2534 eligible people received treatment. Microfilarial rate and density had decreased 1 year after treatment in all 14 communities; this decrease was significantly higher in communities given combined therapy than in those given diethylcarbamazine alone (mean decreases 57.5% and 30.6%, respectively; $p=0.0013$). Greater decreases were also seen in community-specific microfilarial intensity with combined therapy (mean reductions 91.1% and 69.8%, respectively; $p=0.0047$). The rate of leg oedema was not altered, but the frequency of advanced hydroceles decreased by 47% with combined therapy and 56% with diethylcarbamazine alone. 26,641 *Anopheles punctulatus* mosquitoes were caught during 499 person-nights of landing catches. Exposure to infective third-stage larvae decreased in all monitored five communities. Annual transmission potential decreased by between 75.7% and 98.8% in combined-therapy communities and between 75.6% and 79.4% in communities given diethylcarbamazine alone. Transmission was almost interrupted in two communities treated with combined therapy. **INTERPRETATION:** Annual single-dose community-wide treatment with diethylcarbamazine alone or with ivermectin is effective for the control of lymphatic filariasis in highly endemic areas, but combination therapy brings about greater decreases in rates and intensity of microfilaraemia.

- 8 **Brabin B, Piper C.**
Anaemia and malaria-attributable low birthweight in two populations in Papua New Guinea.
Ann Hum Biol 1997 Nov-Dec;24(6):547-555.

We studied 7300 singleton births in the highlands and 4881 in coastal Papua New Guinea in order to examine the separate contribution of anaemia or malaria to low birthweight. The highland sample was selected from a non-malarious area (Goroka) and the coastal sample from an area with perennial malaria transmission (Madang). There was an approximately three-fold increased risk of low birthweight (< 2500 g) in live-births in Madang compared to Goroka. The prevalence of anaemia in the two areas was strikingly different, with 29.2% of Goroka and 89.0% of Madang women anaemic. There was a trend towards increased low birthweight with

decreasing haemoglobin levels in both areas, but this was significant only for Madang. It was assumed that for a given haemoglobin level the increased low birthweight percentage in Madang compared to Goroka was due to malaria exposure, and on this basis relative risk values were estimated for the effect of malaria exposure on low birthweight. Using this approach separate estimates for anaemia and malaria population-attributable risk for low birthweight in Madang were calculated. These indicated that up to 40% of low birthweight babies born in malarious areas may be attributable to malaria and less than 10% attributable to severe anaemia (Hb < 7.0 g/dl). The magnitude of the malaria effect estimated in this analysis places a high priority on malaria control in pregnancy as a strategy for improving birthweight and child survival.

9 **Carman WF, Van Deursen FJ, Mimms LT, Hardie D, Coppola R, Decker R, Sanders R.**

The prevalence of surface antigen variants of hepatitis B virus in Papua New Guinea, South Africa and Sardinia.

Hepatology 1997 Dec;26(6):1658-1666.

Three assays, one based on monoclonal antibodies and the others on polyclonal antibodies, were employed to detect hepatitis B surface antigen (HBsAg)-reactive samples in both vaccinated and unvaccinated populations in areas of the world where hepatitis B virus (HBV) is endemic. Any discordant sera were tested by polymerase chain reaction (PCR) to confirm current infection, and sequence data were obtained from the DNA coding for the major hydrophilic region (MHR) of HBsAg of those samples positive for PCR. In all countries studied, samples that reacted in one HBsAg assay but not another were found. In the most extreme case, about 5% of viremic sera in Papua New Guinea were nonreactive in the monoclonal HBsAg assay; 9 of the 13 PCR-positive samples had novel or once-described variants, or a variant out of its usual genotype context. In South Africa, samples with sequences of subtype ayw2 reacted poorly, particularly in the polyclonal assay. Two had novel variants. In Sardinia, antibody to hepatitis B core antigen (anti-HBc) was analyzed as a marker of infection. A significant proportion of anti-HBc-positive, but monoclonal HBsAg-negative, vaccinees and unvaccinated persons were found to be PCR positive, as were some individuals without any markers of hepatitis B virus infection. Five more novel variants were found in these groups. There are implications for the design of HBsAg assays, which may have to be modified according to local sequence variability. Not all discordant samples were explained by variants, indicating that assay sensitivity is fundamental to diagnostic efficacy. Overall, this study defined 16 novel variants and 2 new potential epitope clusters.

10 **Connelly M, King CL, Bucci K, Walters S, Genton B, Alpers MP, Hollingdale M, Kazura JW.**

T-cell immunity to peptide epitopes of liver stage antigen 1 in an area of Papua New Guinea in which malaria is holoendemic.

Infect Immun 1997 Dec;65(12):5082-5087.

Liver-stage antigen 1 (LSA1) is one of several pre-erythrocytic antigens considered for inclusion in a multiantigen, multistage subunit vaccine against falciparum malaria. We examined T-cell proliferation and cytokine responses to peptides corresponding to amino acids 84 to 107, 1813 to 1835, and 1888 to 1909 of LSA1 in asymptomatic adults living in an area of Papua New Guinea where malaria is holoendemic. Whereas T-cells from North Americans never exposed to malaria did not respond to any of the peptides, those from 52 of 55 adults from the area where malaria is endemic had vigorous proliferation responses to one or more of the LSA1 peptides (mean stimulation indices of 6.8 to 7.2). Gamma interferon (IFN-gamma) production driven by LSA1 peptides ranged from 34 to more than 3500 pg/2 x 10⁶ cells, was derived primarily from CD8+ cells, and was dissociated from T-cell proliferation. The frequencies of IFN-gamma response to the amino acid 1819 to 1835 and 1888 to 1909 peptides were significantly greater than that to the amino acid 84 to 107 peptide (87 and 88% versus 33% of subjects; P < 0.0001). In contrast to proliferation and IFN-gamma, interleukin 4 (IL-4) and/or IL-5 responses to LSA1 peptides were detected in only 18% of the subjects. These data show that T-cell immunity to epitopes in the N- and C-terminal regions of LSA1 are common in persons living in this area of Papua New Guinea where malaria is endemic. The dominance of type 1 CD8 cell IFN-gamma responses is consistent with a role for this T-cell population in immunity to liver-stage *Plasmodium falciparum* in humans.

11 **Gallagher EJ.**

The prion paradox.

J Emerg Med 1997 Sep-Oct;15(5):721-723.

12 **Maitland K, Williams TN, Peto TEA, Day KP, Clegg JB, Weatherall DJ, Bowden DK.**

Absence of malaria-specific mortality in children in an area of hyperendemic malaria.

Trans R Soc Trop Med Hyg 1997 Sep-Oct;91(5):562-566.

We conducted a prospective community-based malaria surveillance study on a cohort of children < 10 years old living in an area of hyperendemic malaria (spleen rates > 50% in children aged 2-9 years) in Vanuatu, Melanesia, supported by a concurrent prospective descriptive study of malaria admissions to the local hospital. The incidence of clinical malaria in children < 10 years old was 1.9 episodes/year. The annual incidence of severe malaria (severe malarial anaemia and cerebral malaria) was only 2/1000 in children aged < 5 years. The only manifestation of severe malaria seen in indigenous children was anaemia. No death could be attributed to malaria. While the incidence of uncomplicated clinical malaria in this population was comparable to that in many parts of Africa, the incidence of severe forms of the disease was significantly lower. This could not be attributed to differing rates of malaria transmission, chloroquine resistance, or to host protective or

behavioural factors. These findings suggest that studies which compare disease patterns in geographically disparate populations may be instrumental in developing a better understanding of the determinants of clinical outcome in *Plasmodium falciparum* malaria and that such regional differences must be considered when planning or interpreting the effects of malaria interventions.

13 **Morbidity and Mortality Weekly Report**

Progress toward poliomyelitis eradication in the Western Pacific Region, January 1, 1996 through September 27, 1997.
MMWR Morb Mortal Wkly Rep 1997 Nov 28;46(47):1113-1117.

In 1988, the World Health Assembly adopted the goal of global poliomyelitis eradication by 2000, which was endorsed in each of the six regions of the World Health Organization (WHO). In the Western Pacific Region (WPR), where the last known case of polio associated with isolation of wild poliovirus occurred in March 1997, the reported number of cases decreased from 5963 in 1990 to 197 in 1996. This report documents progress toward polio eradication in WPR from January 1, 1996, through September 27, 1997, in countries where polio is endemic (Cambodia, China, Laos, Papua New Guinea, Philippines and Vietnam) or recently was endemic (Malaysia and Mongolia) and describes the routine and supplemental vaccination activities necessary to interrupt wild poliovirus transmission in the region.

14 **Trott DJ, Combs BG, Mikosza AS, Oxberry SL, Robertson ID, Passey M, Taime J, Sehuko R, Alpers MP, Hampson DJ.**

The prevalence of *Serpulina pilosicoli* in humans and domestic animals in the Eastern Highlands of Papua New Guinea.

Epidemiol Infect 1997 Dec;119(3):369-379.

In a survey of five villages in the Eastern Highlands of Papua New Guinea, *Serpulina pilosicoli* was isolated from rectal swabs from 113 of 496 individuals (22.8%). Colonization rates ranged from 22.6 to 30.1% in four of the villages but was only 8.6% in the other village. In comparison colonization was demonstrated in only 5 of 54 indigenous people (9.3%) and none of 76 non-indigenous people living in an urban environment in the same region. Colonization did not relate to reported occurrence of diarrhoea, age, sex, or length of time resident in a village. A second set of 94 faecal specimens was collected from 1 village 6 weeks after the first set. *S. pilosicoli* was isolated from 27 of 29 individuals (93.1%) who were positive on the first sampling and from 7 of 65 individuals (10.8%) who previously were negative. In this case, isolates were significantly more common in watery stools than in normal stools. The annual incidence of infection in the village was calculated as 93.6%, with an average duration of infection of 117 days.

S. pilosicoli could not be isolated from any village pig (n = 126) despite its confirmed presence in 17 of 50 commercial pigs (34.0%) sampled at a local piggery. 4 of 76 village dogs (5.3%) and 1 of 2 village ducks were colonized with *S. pilosicoli*, suggesting the possibility of cross transmission between humans and animals.

15 **Ulijaszek SJ, Lourie JA.**

Anthropometry in health assessment: the importance of measurement error.
Coll Antropol 1997 Dec;21(2):429-438.

Anthropometry is the hallmark technique of biological anthropology, and has become increasingly important in health assessments across this century. Although the need for accurate anthropometric measurement has been repeatedly stressed, the ways in which measurement error can influence the characteristics of anthropometric data is poorly appreciated. In this article, guidelines for acceptable measurement error are examined critically, and in light of repeat measurements data collected by the two authors on adults in Papua New Guinea.

16 **Zietkiewicz E, Yotova V, Jarnik M, Korab Laskowska M, Kidd KK, Modiano D, Scozzari R, Stoneking M, Tishkoff S, Batzer M, Labuda D.**

Nuclear DNA diversity in worldwide distributed human populations.

Gene 1997 Dec 31;205(1-2):161-171.

Nucleotide variation was examined in an 8 kb intronic DNA bordering exon 44 of the human dystrophin gene on Xp21. Thirty-six polymorphisms (substitutions, small insertions/deletions and one (T)n microsatellite) were found using SSCP/heteroduplex analysis of DNA samples from mixed Europeans, Papua New Guineans as well as from six African, three Asian and two Amerindian populations. In this way the European bias in the nuclear polymorphism ascertainment has been avoided. In a maximum likelihood tree constructed from the frequency data, Africans clustered separately from the non-African populations. Fifteen polymorphisms were shared among most of the populations compared, whereas 13 sites were found to be endemic to Africans and four to non-Africans. The common sites contributed most to the average heterozygosity ($H_n=0.101\% \pm 0.023$), whereas the endemic ones, being rare, had little effect on this estimate. The F(ST) values were lower for Africans (0.072) than for non-Africans (0.158), suggesting a higher level of gene exchange within Africa, corroborating the observation of a greater number of segregating sites on this continent than elsewhere. The data suggest a recent common origin of the African and non-African populations, where a greater geographical isolation of the latter resulted in a smaller number of newly acquired polymorphisms.

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- 3 **Garner PA, Hill G.** Brainwashing in tuberculosis management. *PNG Med J* 1985;28:291-293.

- 4 **Cochrane RG.** A critical appraisal of the present position of leprosy. In: Lincicome DP, ed. *International Review of Tropical Medicine*. New York: Academic Press, 1961:1-42.

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