Supplementary Information

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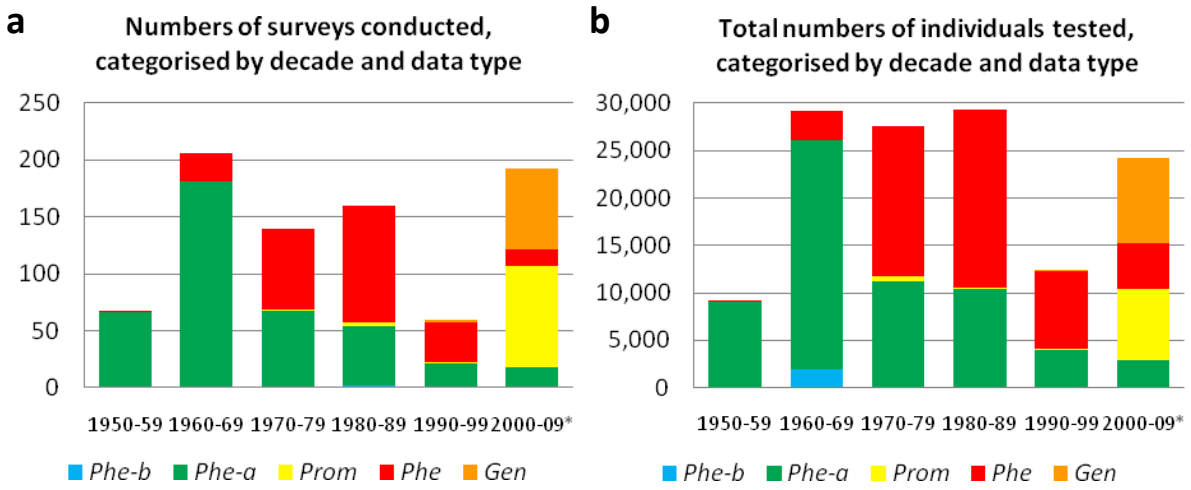
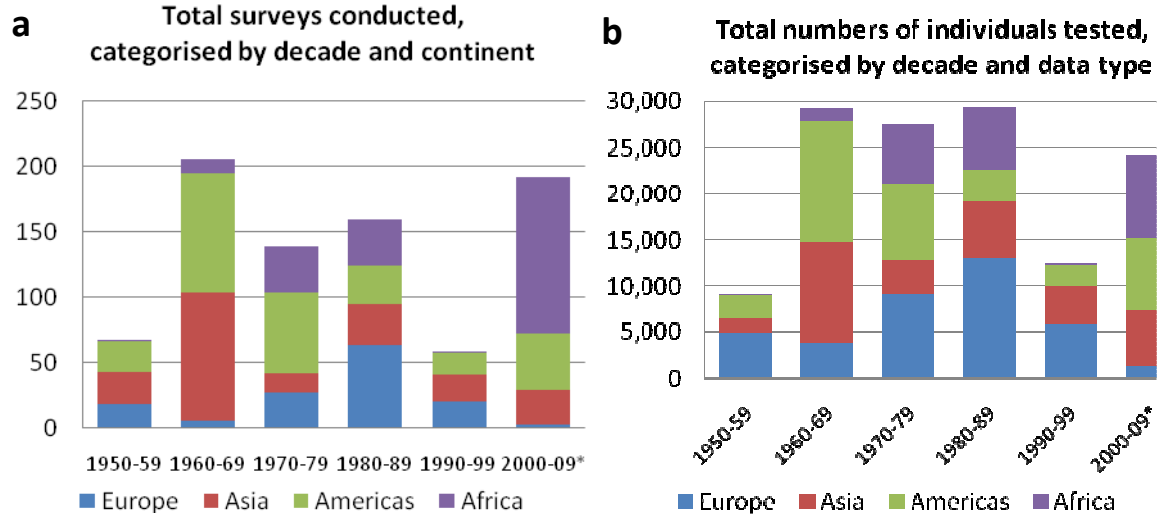
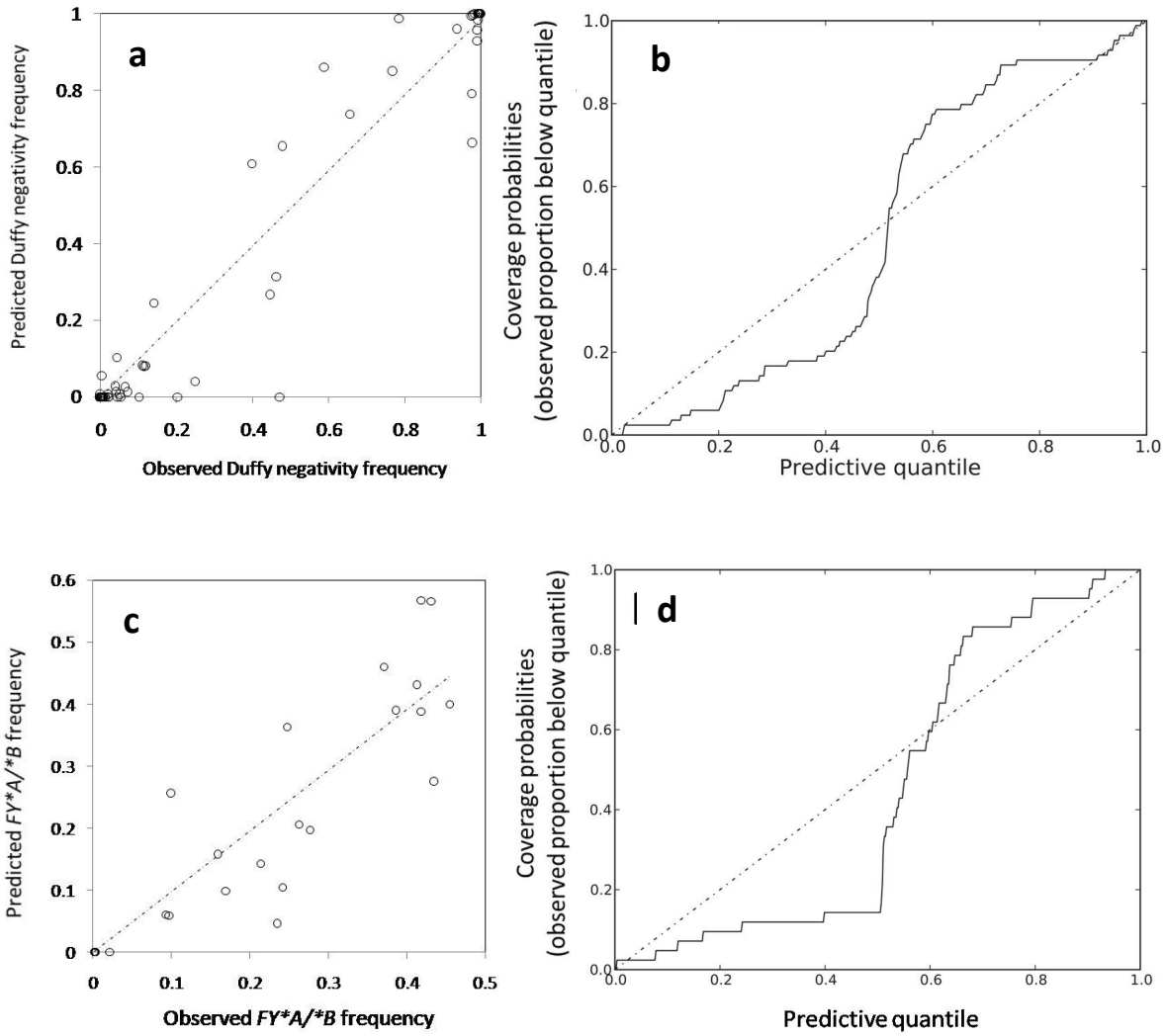
Supplementary Figures



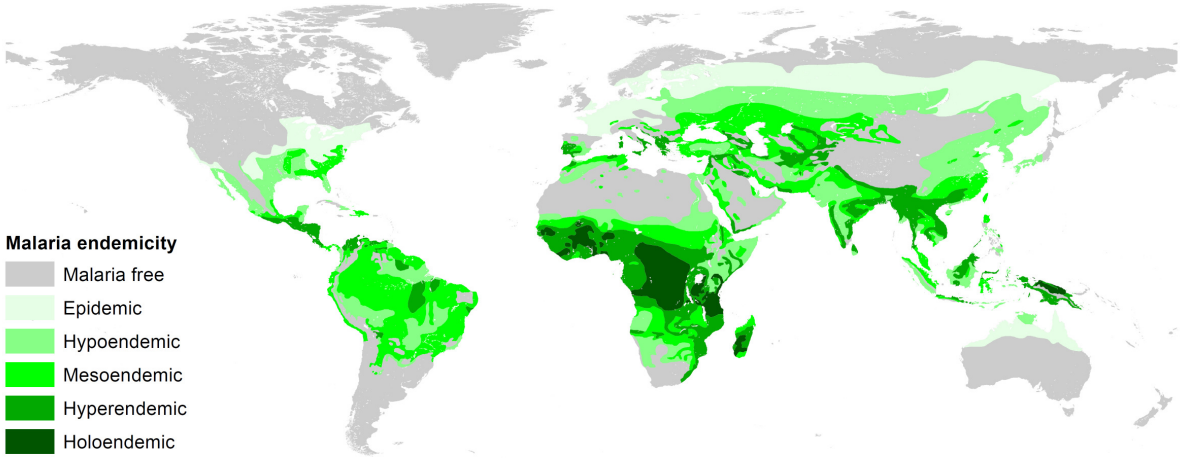
Supplementary Figure S1. Historical patterns of data types in the input data set. (a) Patterns according to survey numbers with colour-coded according to data points in Figure 2; (b) Patterns according to total individuals sampled. (*includes data from unpublished sources acquired in 2010).



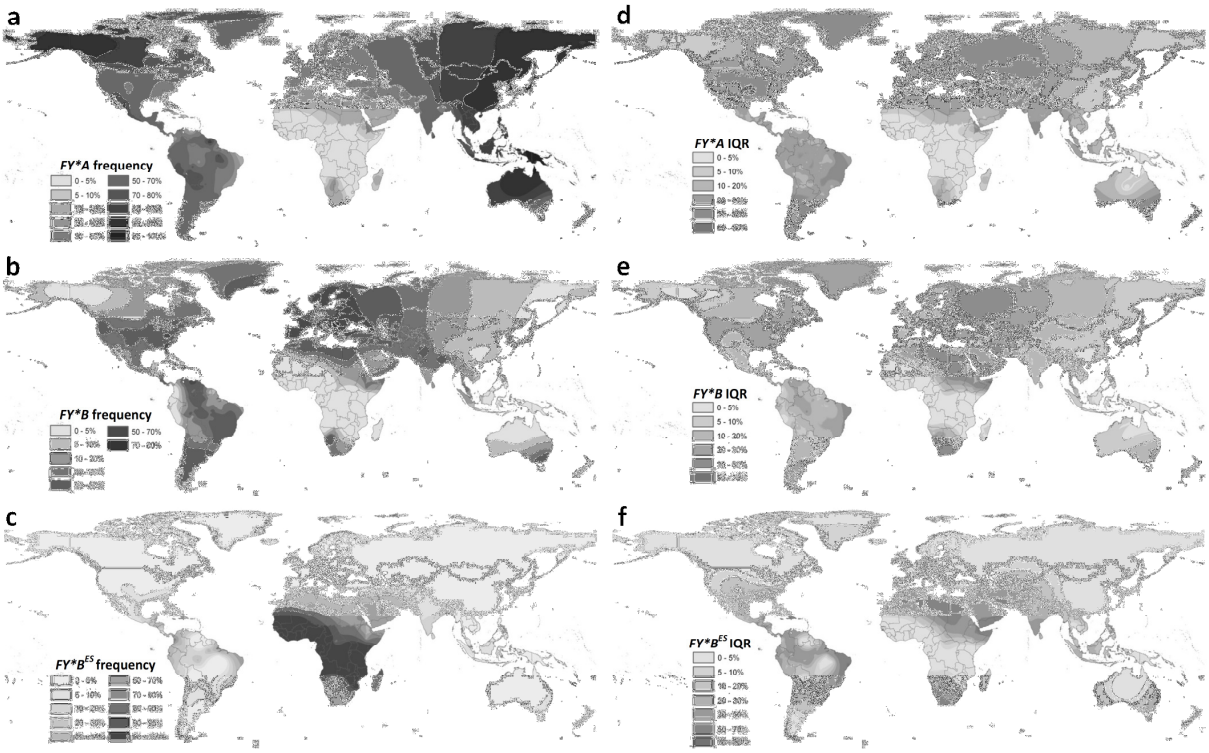
Supplementary Figure S2. Historical patterns of survey locations by continent in the input data set. (a) Patterns according to survey numbers; (b) Patterns according to total individuals sampled. (*includes data from unpublished sources acquired in 2010)



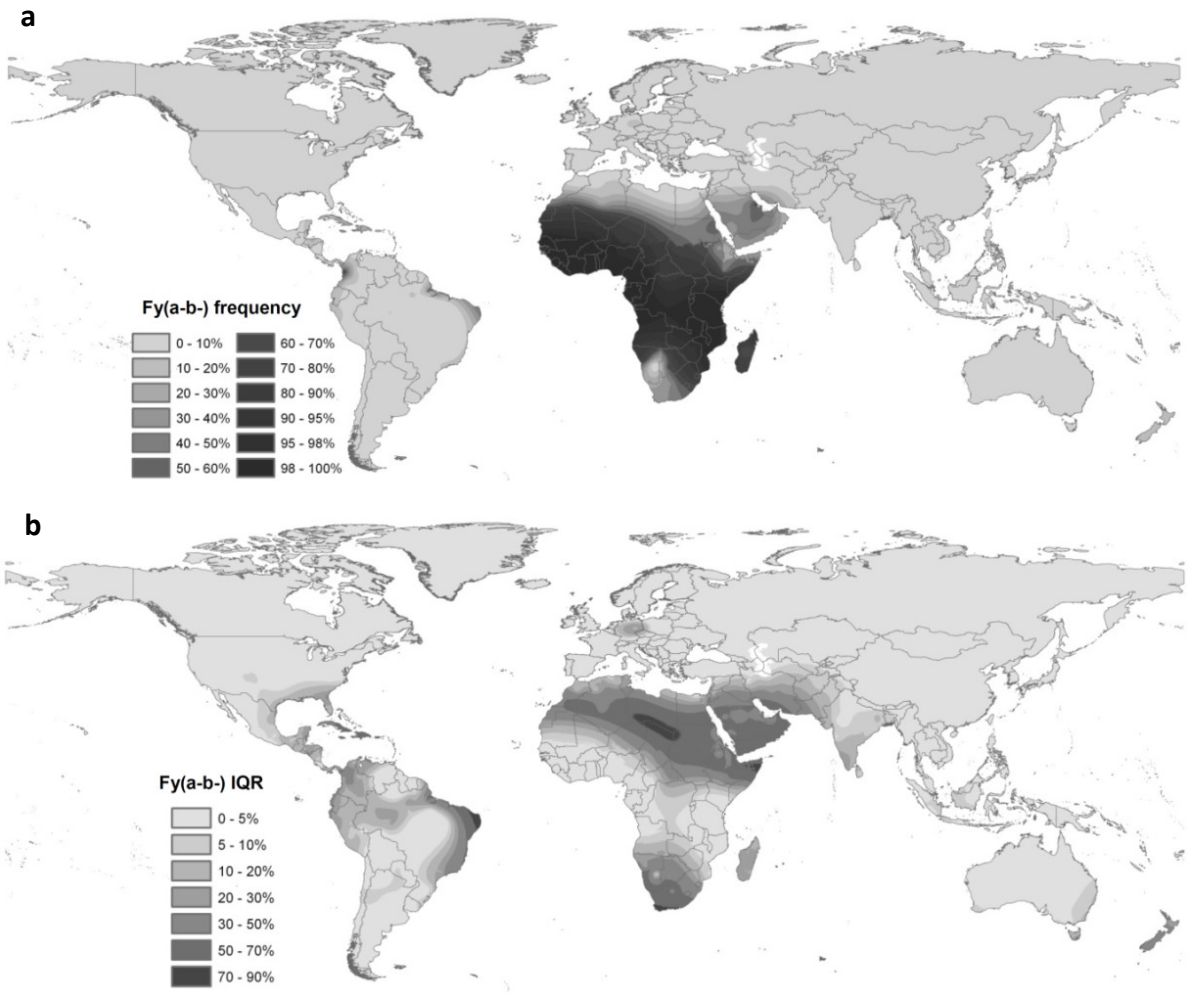
Supplementary Figure S3. Model validation plots. (a) Scatter plot of actual versus predicted point-values of Duffy negativity prevalence. (b) Probability-probability plot comparing predicted probability thresholds with the actual proportion of true values below quantile for Duffy negativity; (c) and (d) show equivalent plots for *FY*A/*B* heterozygosity validation. In all plots the 1:1 line is also shown (dashed line) for reference.



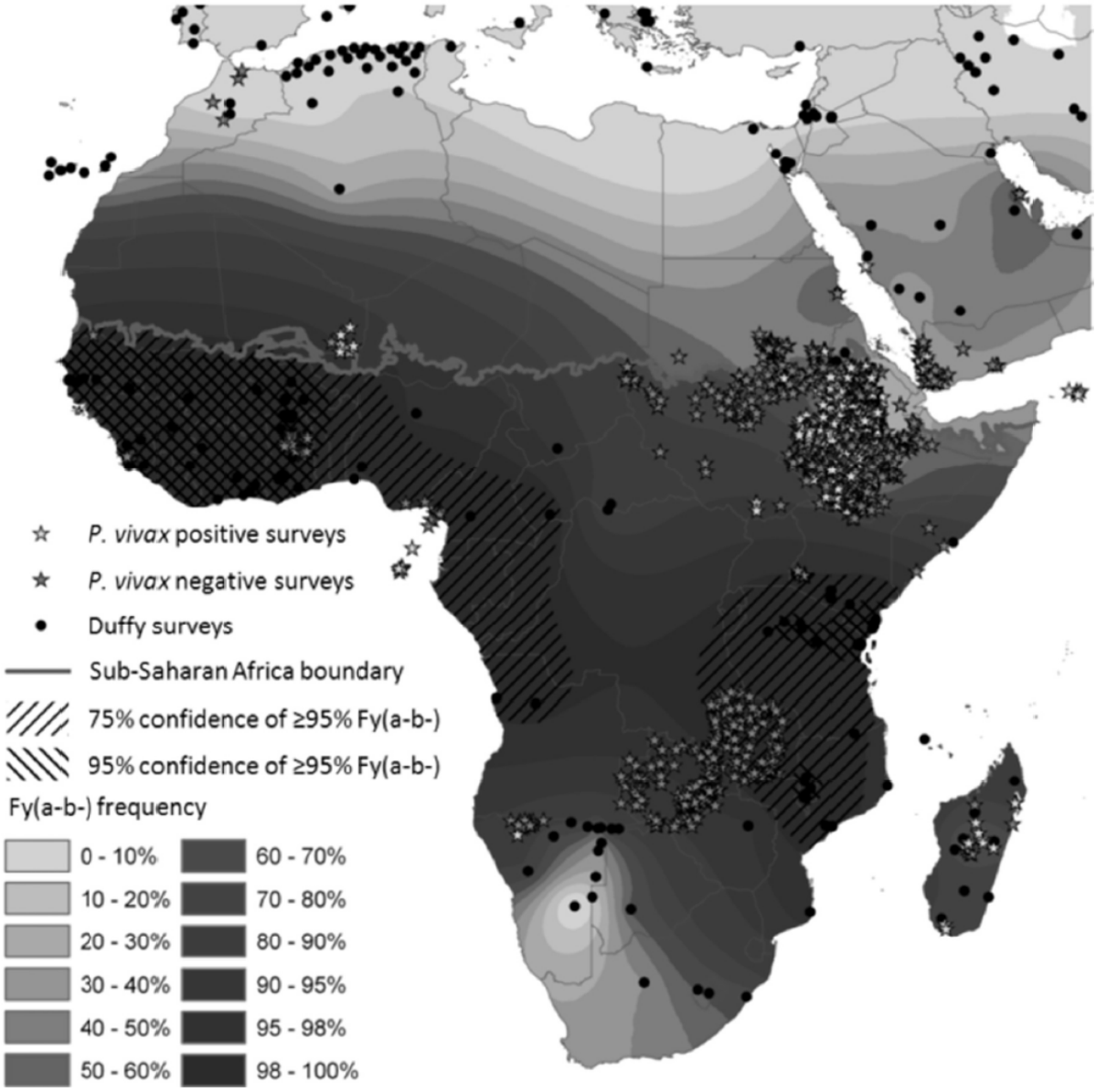
Supplementary Figure S4. Historical malaria endemicity map, originally generated by Lysenko and Semashko[52], recently republished and fully described by Piel *et al*[53].The classes are defined by parasite rates (PR_{2-10}, the proportion of 2 to 10 years olds with parasites in their peripheral blood): malaria free, $PR_{2-10} = 0$; epidemic, $PR_{2-10} \approx 0$; hypoendemic, $PR_{2-10} < 0.10$; mesoendemic, $PR_{2-10} \geq 0.10$ and < 0.50; hyperendemic, $PR_{2-10} \geq 0.50$ and < 0.75; holoendemic $PR_{0-1} \geq 0.75$ (this class was measured in 0 to 1 year olds)[53].



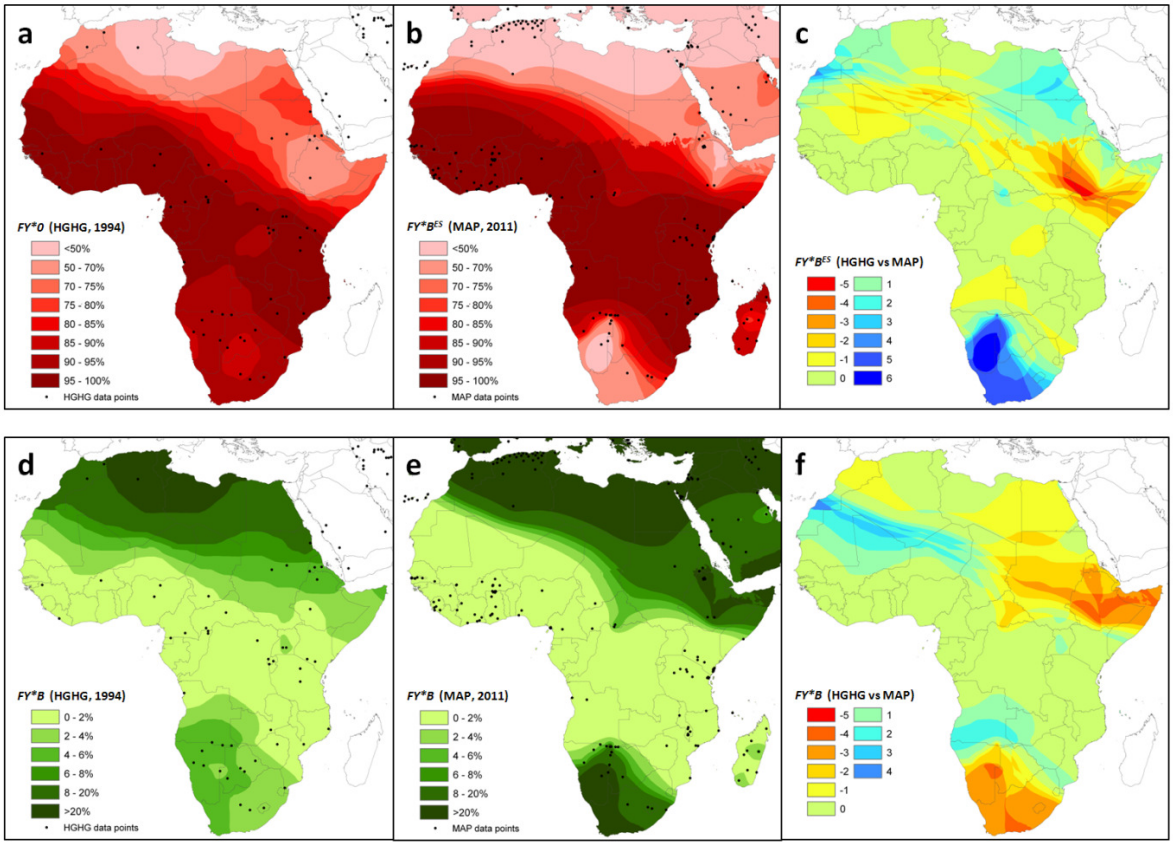
Supplementary Figure S5. Greyscale image of Figure 3: Global Duffy blood group allele frequencies and uncertainty maps. (a), (b) and (c) correspond to *FY*A*, *FY*B* and *FY*B*^{ES} allele frequency maps, respectively; (d), (e) and (f) show the respective inter-quartile ranges (IQR) of each allele frequency map (25% to 75% interval). Predictions are made on a 5 x 5 km grid in Africa and 10 x 10 km grid elsewhere.



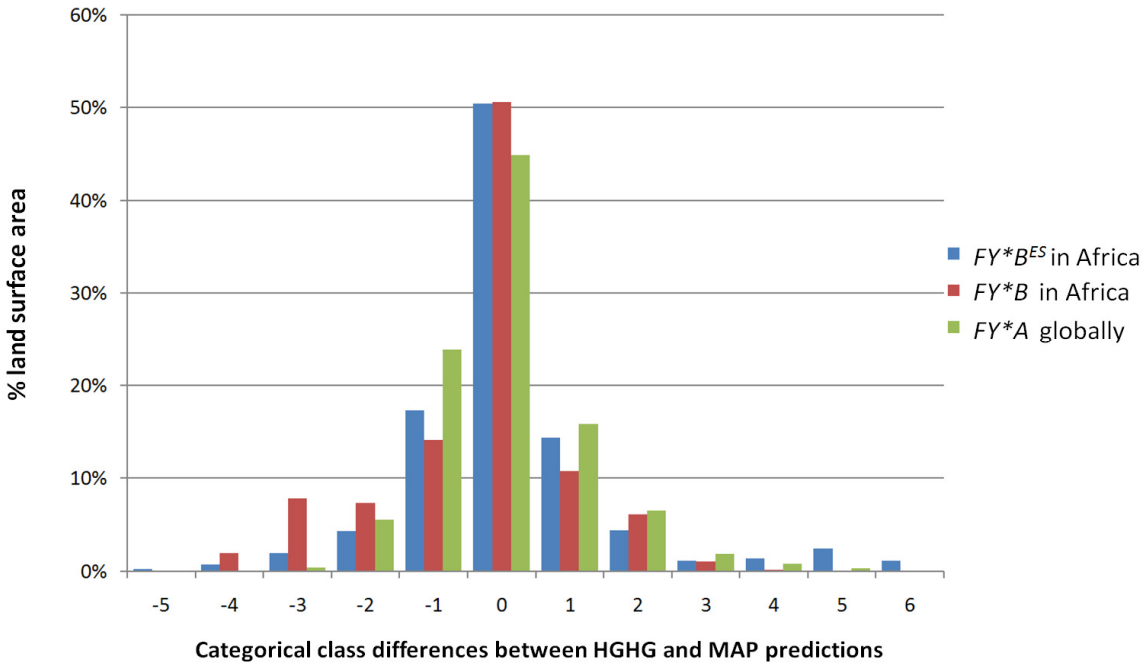
Supplementary Figure S6. Greyscale image of Figure 4: Global distribution of the Duffy negativity phenotype. (a) Global prevalence of Fy(a-b-); (b) associated uncertainty map. Uncertainty is represented by the interval between the 25% and 75% quartiles of the posterior distribution (IQR).



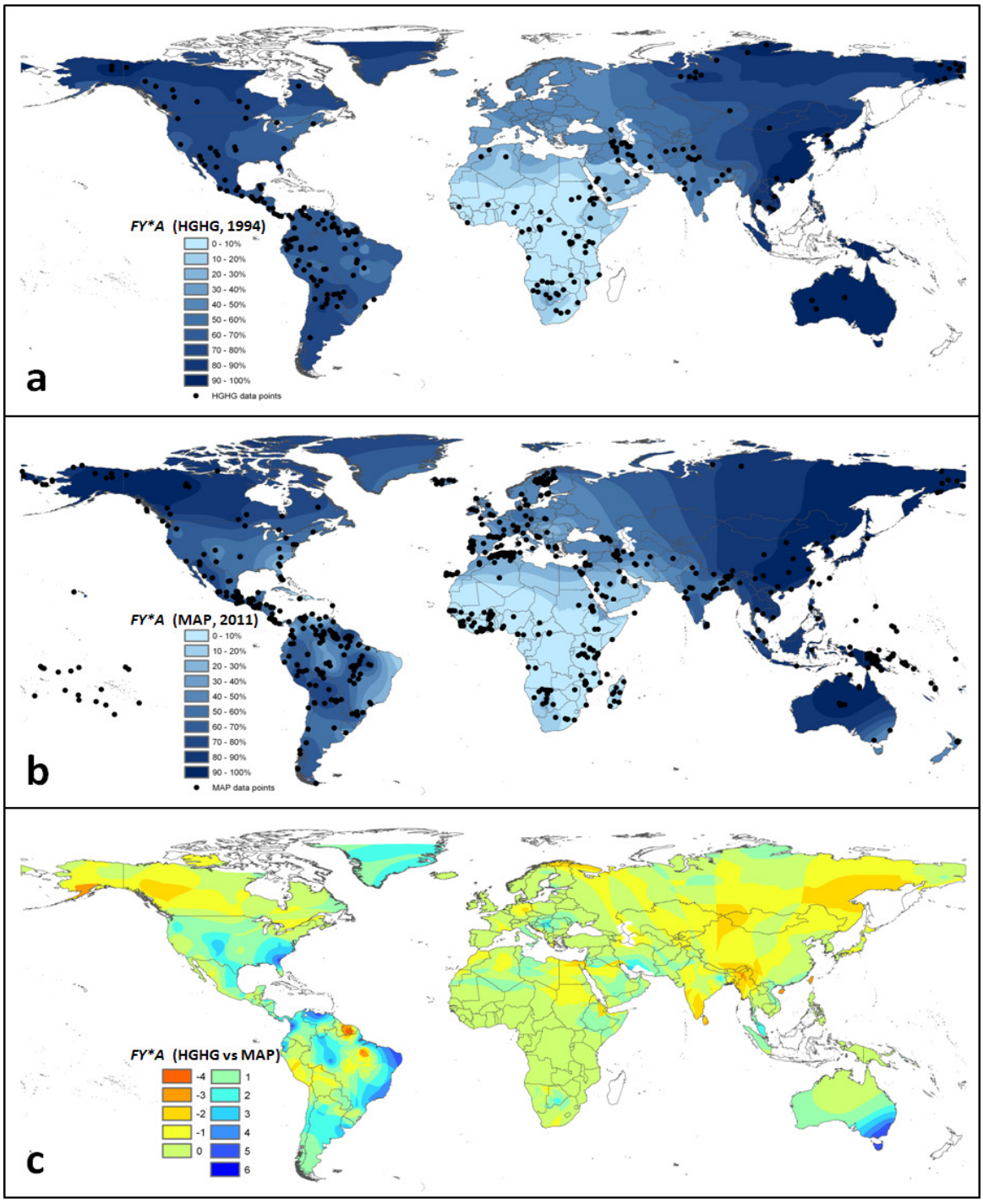
Supplementary Figure S7. Greyscale image of Figure 5: Characteristics of the Duffy negativity phenotype in Africa. This figure shows the covariate line (in pale grey) which separates sub-Saharan African populations from the rest of the continent; hatched areas indicate areas of confidence in the distribution of ≥95% Duffy negativity frequency: with 75% and 95% confidence. Black data points correspond to the input Duffy data points (n=821). Pale grey stars indicate locations of *P. vivax* positive community surveys (n=354), and darker grey stars *P. vivax* negative surveys (n=1405) [data assembled by the Malaria Atlas Project].



Supplementary Figure S8. Comparative display of the HGHG and MAP allele frequency maps for *FY*B*^{ES} and *FY*B* in Africa. Supplementary Figures S8a-c represent the silent *FY*B*^{ES} allele (denoted *FY*0* in the HGHG), and S8d-f the *FY*B* allele frequencies. Differences between the HGHG maps (S8a and S8d) and the new maps (S8b and S8e) are shown in panels S8c and S8f, respectively. Discrepancies are represented by the difference in number of classes between the two versions, based on the categorical classes defined by the HGHG maps. Negative differences (yellow to red) indicate areas where the MAP version predicted higher frequencies than the HGHG version, positive differences (in blue) are where the HGHG version predicted frequencies higher than in the new version. Same class predictions in the HGHG and MAP versions appear in pale green. Black datapoints represent input data points (HGHG: n=41; MAP: n=203).



Supplementary Figure S9. Histogram of differences between frequency categories of the HGHG and MAP predictions for *FY*B*^{ES} and *FY*B* allele frequencies in Africa (HGHG – MAP prediction). Negative differences indicate higher frequency predictions in the MAP version than in the HGHG version; positive differences mean the HGHG version predicted frequencies higher than in the new version. The differences are quantified by proportions of land surface area.



Supplementary Figure S10. Comparative display of the HGHG and MAP allele frequency maps of *FY*A* globally. Differences between the HGHG map (S10a) and the new map (S10b) are shown in panel S9c. Discrepancies are represented by the difference in number of classes between the two versions, based on the categorical classes defined by the HGHG maps. Negative differences (yellow to orange) indicate areas where the MAP version predicted higher frequencies than the HGHG version; positive differences (in blue) are shown where the HGHG version predicted frequencies higher than in the new version. Pale green indicates same class predictions in both versions. Blanks in the HGHG and difference maps indicate areas where no prediction was made, including many islands, most prominently Madagascar and large parts of Indonesia. Black datapoints represent input data points (HGHG: n=751; MAP: n=821). [*Although it was only possible to digitise 241 of the HGHG datapoints from their published map – many appear to be spatial duplicates, and the European datapoints were missing from their global* FY*A *map*].

Supplementary Tables

Parameter	Model term p_{ab}	Parameter	Model term p_0	Parameter	Model term p_1
ϕ_{ab} mean	3.57	ϕ_0 mean	7.02	p_1 mean	0.0119
ϕ_{ab} median	3.36	ϕ_0 median	6.26	p_1 median	0.0119
ϕ_{ab} std	1.24	ϕ_0 std	2.70	p_1 std	0.000921
ϕ_{ab} IQR	1.64	ϕ_0 IQR	3.36	p_1 IQR	0.00130
ϕ_{ab} 95 BCI	4.91	ϕ_0 95 BCI	10.5	p_1 95 BCI	0.00368
θ_{ab} mean	4.99	θ_0 mean	3.05		
θ_{ab} median	4.83	θ_0 median	2.23		
θ_{ab} std	3.04	θ_0 std	2.74		
θ_{ab} IQR	4.02	θ_0 IQR	3.11		
θ_{ab} 95 BCI	11.9	θ_0 95 BCI	10.7		
ν_{ab} mean	0.377	ν_0 mean	0.422		
ν_{ab} median	0.380	ν_0 median	0.427		
ν_{ab} std	0.0412	ν_0 std	0.0473		
ν_{ab} IQR	0.0640	ν_0 IQR	0.0769		
ν_{ab} 95 BCI	0.156	ν_0 95 BCI	0.152		
V_{ab} mean	0.152	V_0 mean	0.345		
V_{ab} median	0.149	V_0 median	0.323		
V_{ab} std	0.0322	V_0 std	0.123		
V_{ab} IQR	0.0492	V_0 IQR	0.163		
V_{ab} 95 BCI	0.114	V_0 95 BCI	0.478		

Supplementary Table S1. MCMC output parameter values at the three modelled loci. The first columns refer to the locus differentiating *FY*A* from *FY*B*. The association of the *FY*B* variant with the silencing Duffy negative mutation (-33C; i.e. the *FY*B*^{ES} allele) is considered in the middle columns. The third term, the *p1* variant, represents the constant modelling the frequency of association between the *FY*A* variant and the silencing promoter mutation (i.e. the *FY*A*^{ES} allele). Spatially variable parameters reported include amplitude (ϕ_0 and ϕ_{ab}), scale (θ_0 and θ_{ab}), degree of differentiability (ν_0 and ν_{ab}) and nugget variances (V_0 and V_{ab}). Summary statistics of the MCMC output include mean and median values, standard deviation ('std'), 50% interquartile range ('IQR') and 95% Bayesian credible intervals ('95 BCI'). Scale is measured in units of earth radii; other parameters are unitless. Values are presented to three significant figures.

Statistic	Sub-Saharan Africa covariate effect
Median	0.832
Mean	0.829
STD	0.310
IQR	0.420
95% BCI	1.214

Supplementary Table S2. Summary statistics for the sub-Saharan Africa (β_{africa}) covariate effect. The posterior is concentrated in positive values, indicating that the covariate increases likelihood of association between the silencing mutation and the *FY*B* locus in the sub-Saharan Africa region. This is reflected by increased frequencies of $FY*B^{ES}$ south of the covariate boundary. (Full model details are given in the Supplementary Methods 1, pages S2-4).

	N sites	N samples (all sites)	Variants		% Duffy positive†
Genotype	22	272	$FY*B^{ES}/FY*B^{ES}$	272	0%
Phenotype	11	3,738	Fy(a+b+) Fy(a+b-) Fy(a-b+) Fy(a-b-)	1 13 12 3,712	0.70%
Promoter	83	7290	Fy-pos Fy-neg	24 7,266	0.33%
Phenotype-a	5	508	Fy(a+) Fy(a-)	7 501	1.38% (excludes any Fy(a-b+))
Phenotype-b	2	148	Fy(b+) Fy(b-)	1 147	0.68% (excludes any Fy(a+b-))

Supplementary Table S3. Duffy positive samples in the predicted 98-100% Duffy Negative region. Total surveys conducted in this region: n=123.

† Of the 11,956 individuals surveyed across the predicted 98-100% Duffy negativity region, 58 Duffy positive individuals were identified at 22 sites across nine countries: Angola, Cameroon, Côte d'Ivoire, The Gambia, Kenya, Malawi, Mozambique, Nigeria and the United Republic of Tanzania.

Supplementary Discussion

Comparison with existing maps.

The only previously published maps of the Duffy alleles are those of Cavalli-Sforza *et al*, in their History and Geography of Human Genes (HGHG[54]). The following discussion centres on (i) the general and (ii) the specific cartographic differences between these two mapping efforts.

In general terms, both efforts employed a similar conceptual framework: an evidence-base of Duffy blood group surveys which informs a statistical model and generates a prediction map. The main methodological differences between the HGHG and the current mapping efforts can, therefore, be examined as (i) the evidence-base, and (ii) the statistical modelling method employed. The third aspect of this discussion will examine the specific differences between the resulting maps.

1. Database

The most obvious advantage that the MAP database has over the HGHG evidence base is twenty years of additional data collection, including all the genotyped data (the most informative data type). The data assembly effort, including reference gathering methods, criteria for survey inclusion and geopositioning protocols are documented in more detail in this effort than previously in the HGHG. In addition, from our review of their sources, it is apparent that non-representative samples of communities were included in the HGHG dataset, such as studies selecting specific ethnic groups from ethnically diverse communities, groups of related individuals and malaria patients, providing potentially biased allelic frequency estimates. These various limitations were addressed in the present study.

The methodological descriptions provided in this paper and the referenced sources for additional information are intended to provide sufficient detail to enable independent reproduction and thus objective evaluation. This addresses another significant limitation to the methods of the HGHG protocols, where only limited documentation is presented on the cartographic methodology employed. The full list of sources used here is given in the Supplementary References; the complete derived database will be made freely accessible online in mid-2011 (the model input extracted from the database is published here as Supplementary Data); the statistical code is freely available for download from the open-access github repository (https://github.com/malaria-atlas-project).

2. Statistical mapping model

The statistical mapping methods employed in the present effort benefit from two decades of development in the field of geostatistics, enabling better representation of small-scale variation in gene frequencies. The most significant development, however, relates to the Bayesian framework which allows the map surface to be interpreted according to its relative reliability by generating numerous iterations of predictions from which various summary indicators (e.g. mean or median) and uncertainty measures can be derived. An immediate advantage of this is enabling predictions for regions where data are absent, and quantifying the certainty in the predictions; in contrast, data

limitations, such as in Madagascar, prevented Cavalli-Sforza *et al* from making predictions in data-poor areas. Further, the importance of uncertainty metrics has recently been emphasised to the public health research community[55].

Furthermore, the HGHG model input was restricted to surveys specifically informing the frequency of each variant, with each variant being mapped in isolation of the others, in spite of their intricate associations. The multi-allelic framework employed here, adapted for the five data types previously described, allowed all data to inform the predictions of each allele simultaneously.

3. Spatial differences between maps

Comparisons of the predictions for all three alleles are discussed here. As Cavalli-Sforza *et al*[54] do not present global frequency maps for all alleles, predictions of *FY*B* and *FY*B*^{ES} frequencies focus on the African continent, while the *FY*A* maps are discussed on a global scale.

A limitation of the practical applicability of the HGHG maps is that their outputs include only gene frequency maps. We present here the first published Duffy negativity phenotype map, as we believe this output will be most informative to the anticipated end-user community. A further limitation of the HGHG maps derives from their presentation, specifically the categorical boundaries employed. The highest allele frequency band (95-100%) corresponds to a wide range of Duffy negative phenotype frequencies (90-100%), which could encompass a wide range of *P. vivax* epidemiological scenarios. Modelled as a continuous measure, the new version can be used as a continuous or categorical surface (available on request from the authors).

i. Null Duffy allele, *FY*B*^{ES}, and *FY*B* in Africa

Visual comparison of the *FY*B*^{ES} and *FY*B* series of maps for Africa (Supp. Figure S8) reveals roughly comparable unskewed outputs, an observation supported by quantitative summaries of the difference maps (Supp. Figure S9). The categorical difference maps (Supp. Figure S8c and S8f) reveal 50% and 51% concordance for *FY*B*^{ES} and *FY*B* frequency predictions, respectively, between the HGHG and MAP maps (Supp. Figure S9). A quarter (24%) of the *FY*B* prediction surface area differed by two classes or more (≥4% difference in allelic frequency), and 18% of the *FY*B*^{ES} predicted area differed by 20% or more (2 frequency classes).

Due to the additive nature of the variant frequencies, the location of the discrepancies overlaps for both alleles, namely the boundary zone around the region of highest Duffy negativity prevalence. The most striking area of discrepancy is the prediction for southern Africa, where the HGHG map predicts much higher frequencies of *FY*B*^{ES} and lower frequencies of *FY*B* than the new iteration does, a reflection of the new dataset which includes surveys reporting very low rates and even absence of Duffy negativity in the local population, an element not reflected in the HGHG map. At the eastern limit of the high frequency zone, the indent of lower *FY*B*^{ES} and higher *FY*B* frequencies into Sudan is less pronounced into the HGHG map than the current one. Similarly, predictions in Ethiopia for frequencies of both allele frequencies are generally lower in the HGHG map. The position of the northern boundary of high Duffy negativity differs by one or two classes between the maps, with higher frequencies of *FY*B*^{ES} stretching further north and correspondingly lower frequencies of *FY*B* predicted in the new maps.

The database updates yielded a five-fold increase in the input evidence-base, from n_{Africa}=41 in the HGHG version (though only 27 points could be digitised from their published maps) to n_{Africa}=203 in the current iteration (including 37 *Genotype* and 61 *Phenotype*), (Figure 2). The differences in the southern and eastern parts of the distributions are directly informed by the updated dataset. Notably, the highly informative genotype datapoints in southern Ethiopia enable the more spatially convoluted prediction in this area. However, being transition regions becoming increasingly heterozygous, both are also areas of high uncertainty (Figure 3e-f). The spectrum of frequencies across the western and central Sahara is informed by a much larger dataset in the current iteration than the HGHG one, both across the sub-Saharan countries (Nigeria and West Africa: 3 datapoints in the HGHG, 64 in the updated dataset) and the Maghreb region (west of Libya: 1 datapoint in the HGHG, 29 in the updated version). However, no surveys were identified within the Sahara region, likely a reflection of the low population densities in the area. The exact position of the frequency class boundaries therefore, as it corresponds to almost zero population levels, is of minor consequence. MAP predictions across the desert are moderated by increased uncertainty (Figure 3d-f), contrasting with the much more certain predictions across central Africa.

ii. *FY*A* global distribution

Only *FY*A* and *FY*B* are commonly found outside the Africa region. To avoid repetition, only the one set of maps are discussed here – *FY*A*, the only global Duffy allele frequency map presented in the HGHG.

Overall the *FY*A* HGHG and MAP maps reveal good correspondence (Supp. Figure S10), with 85% of the prediction surfaces being within 10% of the other. Largest differences are in the Americas, where the population composition is known to be highly heterogeneous with populations of diverse origins. Predictions from south eastern Australia also differ by up to 50% (5 prediction classes), with the MAP prediction falling to frequencies of 40 to 50% in areas predicted to be 90 to 100% by the HGHG map. The MAP prediction in this region is informed by two relatively large surveys from the SE Australian coast (n=304[56] and n=788[57]) and three from northern New Zealand, contrasting with the uniform HGHG prediction informed only by data from central Australia. The prediction across much of Africa and Europe is largely concordant between maps. The exact position of the class boundaries across Asia varies slightly, but both show the same general trend of increasing *FY*A* frequencies eastwards across the continent.

***FY*X* variant: potential further elaboration to the model.**

A number of mutations additional to those encoding the common Duffy variants discussed here (*FY*A*, *FY*B*, $FY*B^{ES}$, $FY*A^{ES}$) have been described[58], most notably the *FY*X* allele, which reaches polymorphic frequencies among populations of European origin[59]. Despite being relatively common, insufficient data prevented its inclusion in the current mapping project. Additional data on the prevalence of this variant would have allowed extension of the model to include the C265T and G298A loci which encode the *FY*X* allele in association with the *FY*B* variant[58]. These mutations cause reduced expression levels of Fy^b antigen, characterised as the Fy(b+^{weak}) phenotype.

The lack of reliable data on this variant is largely due to the low sensitivity of agglutination diagnostics. The low levels of Fy^b antigen expressed by *FY*X* (10% wildtype levels[60]) are not consistently or reliability detected by agglutination assays, due to differences in antiserum reactivity and experimental procedures. This inconsistency means that the Fy(b+^{weak}) phenotype may occasionally have been misclassified as the Fy(b-) phenotype, overlooking the presence of the Fy^b antigen.

The *FY*B* map presented here is, therefore, defined only in terms of expression at two loci for which sufficient data existed for reliable modelling: nucleotide -33 in the promoter GATA-box region and at position 125 of exon 2. Where detected, expression of the *FY*X* allele is included in the *FY*B* map, due to its correspondence at the two loci considered. The availability of more multi-locus *Genotype* data would allow full refinement of the model.

Supplementary Methods

Mathematical description of the Bayesian geostatistical model and its implementation.

This supplement provides information on the geostatistical model used to map the allelic frequencies of the monogenic Duffy blood group variants. General principles of the geostatistical framework used have been previously described by Diggle and Ribeiro[61].

1. Random fields

To accommodate the five input data types previously detailed (Table 2), and to simultaneously model the distribution of both the positive alleles, *FY*A* and *FY*B*, and their corresponding negative variants, *FY*A*^{ES} and *FY*B*^{ES}, the model targets two spatially-varying allele frequencies: the frequency of the Fy^{b} variant (125A, which is present in both *FY*B* and *FY*B*^{ES}), and the frequency of the promoter region silencing variant (-33C) in association with the 125A coding region variant (thus the frequency of the *FY*B*^{ES} allele). These are denoted $p_{ab}(x)$ and $p_0(x)$ respectively, where x is a location. A third allele frequency, the frequency of the silencing variant occurring in association with the 125G variant (thus the *FY*A*^{ES} allele) is modelled as a small constant denoted p_1. This allele's highly restricted distribution, as reported in the assembled database, and its low frequencies where it was detected, prevent its distribution being modelled spatially as for the other alleles. The model for these allele frequencies is as follows:

$$
\begin{aligned}
m_{ab} &\sim \text{Normal}(0, 10000) \\
m_0 &\sim \text{Normal}(0, 10000) \\
\beta_{\texttt{africa}} &\sim \text{Normal}(0, 10000) \\
p_1 &\sim \text{Uniform}(0, .05)
\end{aligned}
$$

$$
\begin{aligned}
M_{ab}(x) &= \beta_{\text{africa}} 1_{x \text{ in africa}} + m_{ab} \\
M_0(x) &= m_0
\end{aligned}
$$

$$
\begin{aligned}
\phi_{ab} &\sim \text{Exponential}(.1) \\
\phi_0 &\sim \text{Exponential}(.1) \\
\theta_{ab} &\sim \text{Exponential}(.1) \\
\theta_0 &\sim \text{Exponential}(.1) \\
\nu_{ab} &\sim \text{Uniform}(0, 3) \\
\nu_0 &\sim \text{Uniform}(0, 3) \\
V_{ab} &\sim \text{Exponential}(.1) \\
V_0 &\sim \text{Exponential}(.1)
\end{aligned}
$$

$$
\begin{aligned}
C_{ab}(x, y) &= \phi_{ab} \text{Matèrn}(d(x, y)/\theta_{ab}; \nu_{ab}) + V_{ab} 1_{x=y} \\
C_0(x, y) &= \phi_0 \text{Matèrn}(d(x, y)/\theta_0; \nu_0) + V_0 1_{x=y}
\end{aligned}
$$

$$
\begin{aligned}
f_{ab} &\sim \text{GP}(M_{ab}, C_{ab}) \\
f_0 &\sim \text{GP}(M_0, C_0) \\
p_{ab}(x) &= \text{logit}^{-1}(f_{ab}(x)) \\
p_0(x) &= \text{logit}^{-1}(f_0(x))
\end{aligned}
$$

The mean function for f_0 simply returns a constant, m_0. The mean of f_{ab} takes presence in sub-Saharan Africa as a covariate with coefficient β_{africa}, and also a constant term m_{ab}.

Both fields use the Matèrn covariance function[62]. The range parameters of f_0 and f_{ab} are θ_0 and θ_{ab}, respectively; the corresponding amplitude parameters are ϕ_0 and ϕ_{ab}; the degree-of-differentiability parameters are denoted ν_0 and ν_{ab}; and the nugget variances are V_0 and V_{ab}. The distance function d gives the great-circle distance between its arguments.

The Gaussian random fields are converted to probabilities using the standard inverse logit link function.

2. Likelihood

The probability of the 125A variant being present (variant encoding Fy^b antigen) is $p_{ab}(x)$, and should be much higher for x in Africa. Given that $b = 1$, the promoter region "erythrocyte silent" -33C variant occurs with probability $p_0(x)$. Given that $b = 0$, the -33C variant happens with probability p_1, which is assumed to be a small, constant value (representing the *FY*A^{ES}* allele). Hardy-Weinberg assumptions apply to the genotype frequencies.

Input data falls into five categories: *gen, phe, prom, aphe, bphe* defined by the diagnostic method used (Table 1), and thus what phenotypic/genotypic information is discernable. The nomenclature

system used includes a prefix denoting the data category (**gen***, **phe***, **prom***, **aphe***, **bphe***) followed by a symbol for each variant:***a** for *FY*A* or Fy^a variant, ***b** for *FY*B* or Fy^b variant, allelic variant ***0** for $FY*B^{ES}$, allelic variant ***1** for $FY*A^{ES}$, phenotypic variant ***0** to denote absence. These are fully described as follows:

gen* : Genotype data. Allelic frequencies are:

$$\begin{aligned}
\mathbf{gena}\ & (1-p_{ab}(x))(1-p_1)\\
\mathbf{genb}\ & p_{ab}(x)(1-p_0(x))\\
\mathbf{gen0}\ & p_{ab}(x)p_0(x)\\
\mathbf{gen1}\ & (1-p_{ab}(x))p_1
\end{aligned}$$

The genotype frequencies (**genaa** (or *FY*A/FY*A*), **genab** (or *FY*A/FY*B*), etc.) can be obtained using the standard Hardy-Weinberg formula. For example, the frequency of **genab** is twice the product of the frequencies of **gena** and **genb**, which is $2(1-p_{ab}(x))(1-p_1)p_{ab}(x)(1-p_0(x))$[63,64].

phe* : Phenotype data. Studies where full phenotype resolution was provided.

pheab	This can only happen if the genotype is **genab**.
phea	This can only happen if the genotype is **gena0**, **gena1** or **genaa**.
pheb	This can only happen if the genotype is **genb0**, **genb1** or **genbb**.
phe0	This can only happen if the genotype is **gen00**, **gen01** or **gen11**.

prom* : Molecular data. Studies that considered only the promoter region variant (T-33C).

prom0	This can only happen if the genotype is **gen00**, **gen01** or **gen11**.
promab	This corresponds to the complement of **prom0**.

aphe* : Phenotype data. Only anti-Fya antibody was used in the diagnosis.

aphea	This can only happen if the genotype is **genaa**, **genab**, **gena1** or **gena0**.
aphe0	This corresponds to the complement of **aphea**.

bphe* : Phenotype data. Only anti-Fyb antibody was used in the diagnosis.

bpheb	This can only happen if the genotype is **genbb**, **genab**, **genb0** or **genb1**.
bphe0	This corresponds to the complement of **bpheb**.

The sampling distributions are assumed to be multinomial, conditional on the appropriate individual phenotype or genotype probabilities described above. This likelihood completes the Bayesian probability model.

3. Model implementation and output

As previously detailed[65], implementation of the modelling procedure was divided into two computational tasks: (i) the Bayesian inference stage which was implemented using the Markov Chain Monte Carlo (MCMC) algorithm[66] and was used to generate samples from the posterior distribution of the parameter set and the spatial random fields at the data locations; and (ii) a prediction stage in which samples were generated from the posterior distribution of allele frequencies at each prediction location on a global 10 x 10 km grid and 5 x 5 km grid across Africa.

Convergence of the MCMC tracefile was judged by visual inspection and verified using the Geweke convergence diagnostics[67]; 1.2 million MCMC iterations were run, with 10% recorded in the tracefile and the first 30,000 iterations excluded from the mapping stages. During the mapping process, the posterior distributions were thinned by 88, resulting in 1003 mapping iterations. MCMC dynamic traces[66] are available on request.

The model code was written in Python programming language (http://www.python.org), and is freely available from the MAP's code repository (http://github.com/malaria-atlas-project/duffy). MCMC algorithm[66], was used from the open-source Bayesian analysis package PyMC[68] (http://code.google.com/p/pymc). Maps were generated using Python and Fortran code, available from the MAP's code repository (http://github.com/malaria-atlas-project/generic-mbg).

Model validation procedure.

To assess the plausibility of the model's multiple outputs, two validation procedures were run to quantify the disparity between the model's predictions and hold-out subsets of the data[69]. First the frequency of the Duffy negativity phenotype was assessed, a measure determined directly by the *FY*B*^{ES} allele frequency map. Second, the frequency of heterozygosity was used to consider the reliability of all three allelic frequency predictions: *FY*A*, *FY*B*, *FY*B*^{ES}.

Selection of the validation sets:

1. Duffy negativity. Three of the five input data types (*Genotype*, *Phenotype*, *Promoter*) directly inform the frequency of the negative phenotype. A subset of these three data types corresponding to 10% of the overall dataset (n=84) were randomly selected as a hold-out dataset. The Bayesian geostatistical model was then implemented in full using the remaining 90% dataset.

2. Heterozygosity. Only molecularly diagnosed data, which assessed expression at both the promoter and coding-region loci can directly inform the frequency of allelic heterozygosity. This meant that only the *Genotype* data could be used in this hold-out dataset (n=73). Due to the small number of datapoints available, a smaller subset was held-back for validation (n=42), corresponding to 5% of the overall dataset.

Quantifying model performance

Simple statistical measures were used to quantify the model's ability to predict pixel values at unsampled locations by comparing model predictions with the held-out subset of observed values. The mean error (correlation coefficient between predicted and actual values) was used to assess

overall model bias in the predictions, and mean absolute error (summary of the model's general tendency to over/underestimate frequencies) quantified the overall prediction accuracy as the average magnitude of errors in the predictions[69].

Variability of Duffy typing diagnostic methods.

Uncertainty in relation to diagnostic methodology may arise due to a number of factors, each is discussed in turn.

First, diagnoses have historically been hampered by shortages of Fy^b anti-serum, thus leading authors to 'guess' the unknown phenotypes. Across sub-Saharan Africa Fy(a-) samples were assumed to be Duffy negative samples; outside this region, Fy(a-) individuals were assumed to be Fy(b+) (e.g. refs [70-72]). The obvious uncertainty associated with such assumptions was addressed directly through the model design. According to the diagnostic methodology employed, data were categorised into five data types (Table 2) and informed the model accordingly. Our modelling techniques therefore allow use of the complete dataset for each map without requiring any such tenuous assumptions to be made.

Uncertainty may arise, however, due to the inconsistently diagnosed Fy($b+^{weak}$) phenotype. This low copy-number variant may pass undetected by some antisera during agglutination assays[73]. Ideally, we would have included this additional locus as a spatially-variable term in the model. However, the allele's low frequencies, which are poorly and inconsistently reported, rendered it impossible to map this variant separately. For reasons discussed in Supplementary Discussion 2, the weakly agglutinating samples were treated as the Fy(b+) phenotype.

The second source of diagnostic uncertainty relates directly to the experimental procedures themselves. The main dichotomy between methods in terms of relative reliability is between serological methods (assessing phenotypes) and molecular methods (determining genotypes), corresponding to the greatest potential source of variation. Anticipated levels of variability between phenotypic and genotypic diagnoses can be ascertained from studies examining samples with multiple diagnostics. For example, this was recently done by Ménard *et al* on samples from Madagascar who found perfect concordance between a combination of phenotype-based methods (a microtyping kit and anti-sera, and flow cytometry) and molecular SNP analysis[74] (n = 661). Similar correspondence was reported from the range of diagnoses tested on a Brazilian sample: agglutination tests, flow cytometry analysis and PCR-RFLP DNA analysis and sequencing[75]; the same result was found among UK blood donors[76]. These diagnoses therefore support the commonly cited understanding of a tight phenotype-genotype association with Duffy blood types[76].

The third aspect of diagnostic uncertainty is introduced by experimental error. However, even poorly preserved blood samples need not necessarily be considered a major source a potential error, due to the remarkable stability of most blood antigens[70]. The ability of the antigens to maintain their integrity over time was demonstrated by the successful agglutination assay applied to six-month old samples from Papua New Guinea[77] and twelve-month old samples from the Maoris of New

Zealand[78]. However, when sample degradation does arise, there is evidence from the literature of such samples being excluded from analyses (e.g. due to poor refrigeration during transport[78]). Furthermore, results described by authors as likely to contain false-positives (e.g. Livingstone *et al* in West Africa[79]) were excluded from the present study to conform with the conservative approach defined in our data abstraction protocols (main manuscript pages 15-17).

We decided not to include diagnostic methodology as a covariate in the model as the model structure already allows for the differences between antigen and DNA-based methods. These were the only two diagnostic types encountered: serological anti-serum agglutination tests (n=659) and DNA-molecular methods (n=162). We therefore considered that little additional information would have been derived from this addition to the model. It is therefore not possible to quantitatively determine the level of variation introduced through synthesis of the different methods; however, we hope to have demonstrated that, within the modelling framework used here, we believe its influence to be very low.

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Sources from which data points were identified. (References 80 to 399)

Only sources reporting surveys included in the final model are listed. Population samples from these met the inclusion criteria for community representativeness and were geographically specific. References are ordered chronologically, and alphabetically by first author name within years; n=320.

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