phosphorus regeneration in a given population. For example: if we take a hypothetical zooplankton population composed of 10 percent by weight of ciliates weighing 1×10^{-2} μg each and 90 percent crustaceans the size of *Calanus finmarchicus*, weighing about 0.3 mg each (*12*), the ciliates would contribute over 70 percent of the total dissolved phosphorus released by the population. The substitution of phagotrophic flagellates for ciliates would shift the balance further in favor of the domination of nannozooplankton in nutrient release processes, while the substitution of smaller crustaceans, such as *Acartia clausii*, for *Calanus finmarchicus* would increase the dissolved phosphorus contribution of the net zooplankton.

Thus the contribution of microzooplankton to phosphorus regeneration is much greater than their contribution to total zooplankton biomass. Their participation in other ecologically important processes (for example, regeneration of other nutrients, food consumption) must likewise exceed the importance of their biomass.

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Genetic Regulatory Mechanisms at the Population Level in Man

Abstract. *The distribution of the Rhesus and ABO blood groups, thalassemia, and glucose-6-phosphate dehydrogenase deficiency in Europe and in Jewish populations argues for a regulatory mechanism at the population level which adjusts gene frequencies so that the overall genetic load is not excessive, since gene frequencies giving a high genetic load at one locus are often associated with those giving low genetic loads at other loci.*

Genetic polymorphisms are common in man. The occurrence of polymorphism implies a balanced genetic load (*1*) made up of components derived from the segregation of genotypes less fit than the optimum, and incompatibility. If each polymorphic locus is completely independent of every other, the genetic load would perhaps be so great that it could not be tolerated, especially in a species such as man with a low reproductive potential. However, the development of fitness interactions between genes at different loci may lead to the total load over all loci becoming less than directly cumulative (*2*). An example in man is the ABO-Rh incompatibility interaction (*3*).

A second, but not entirely distinct, method of compensation may be by the control of gene frequencies such that, if there is a high load associated with one polymorphic locus, the gene frequencies at other loci might be adjusted to give relatively low genetic loads. There may, therefore, be a regulatory mechanism at the population level controlling gene frequencies.

In Sardinia, there is a high frequency of thalassemia (*4*) and glucose-6-phosphate dehydrogenase (G6PD) deficiency (*5*). Genes for these conditions may be maintained at a high frequency in the population as a consequence of malaria. Thalassemia, in particular, implies a high genetic load as homozygotes are relatively inviable. Genes for both these conditions are also frequent in the Ferrara region of the Po Valley, and in regions of southern Italy. A map by Morganti (*6*) shows that the frequency of Rh-negative individuals is extremely low in Sardinia, and, furthermore, Ceppellini (*4*) has presented evidence suggesting that within Sardinia the frequency of Rh-negative individuals is low in the malarial regions where the gene frequencies for thalassemia and G6PD deficiency are high, whereas in the mountainous nonmalarial districts, where the gene frequencies for thalassemia and G6PD deficiency are lower, the frequency of Rh-negative individuals is higher. Similarly, Ferrara is a region of low Rh-negative gene frequency, and on the whole there are fewer Rh-negative individuals in southern than in northern Italy. Thus low Rh-negative frequencies, implying a low Rh-incompatibility load, are associated with a high load due to thalassemia and presumably G6PD deficiency.

Morganti (*6*) gives maps of the A, B, O, and Rh gene frequencies for 90 Italian provinces. Let the gene frequencies of the A, B, and O alleles be p, q, and r, respectively, such that $p + q + r = 1$, and let d_A and d_B be the probabilities of death due to incompatibility based on A and B sperm, respectively. The ABO incompatibility load is then (*7*)

$$d_A p(1 - p)^2 + d_B q(1 - q)^2.$$

As q does not vary much in Italy, variations in the ABO incompatibility load are due mainly to variations in p. Maximum A load occurs at $p = 1/3$. The lowest values of p (0.15 to 0.25) occur in Sardinia (*6*). In Ferrara, p is lower than in the neighboring districts, and in southern Italy p is lower than in northern Italy (*6*), but nowhere is p greater than 1/3. Thus low ABO and Rh incompatibility loads occur in regions where the genetic load due to thalassemia and G6PD deficiency is high.

The Rh incompatibility load (*2*) is

$$d_B l m^2,$$

where d_B represents the probability of death due to Rhesus incompatibility, and l and m are the frequencies of the R and r alleles respectively, such that $l + m = 1$. With the assumption that $d_A = d_B$, which is reasonable since

q is relatively fixed in Italy, the ABO incompatibility load was plotted against the Rh incompatibility load for the 90 provinces, giving a correlation coefficient -0.278 ($P < .01$). But omitting the three Sardinian provinces with low ABO and Rh loads, the correlation coefficient became -0.431 ($P < .001$). Thus on the whole, there is an inverse correlation between the ABO and Rh incompatibility loads, so that if the ABO load is high the Rh load is low, and vice versa, but in Sardinia, with high gene frequencies for thalassemia and G6PD deficiency, the ABO and Rh loads are both low. Thus it seems likely that complex regulatory mechanisms at the population level adjust gene frequencies to prevent the overall genetic load from becoming excessive.

Based on 33 points, a similar significant negative correlation coefficient ($P < .01$) was found from Mourant's (*8*) maps of the gene frequencies of blood groups in Europe (with Sardinia omitted). Two extreme populations deserve mention. First, the frequency of Rh-negative individuals in the Basque country is 30 to 40 percent, which is close to the frequency for the maximum possible Rhesus incompatibility load which occurs when the proportion of Rh-negative individuals is 45.45 percent. The ABO load, on the other hand, is very low in the Basque country, so a high Rh load is associated with a low ABO load. Secondly, in Lapland where $p \approx 0.5$ (Lapland being the only part of Europe where p is greater than 1/3), a relatively high ABO load is associated with a low Rh load. Thus these two populations do not disagree with the hypothesis. The most divergent population is, in fact, in Sardinia.

In ten Jewish populations, the lowest incidence of Rh-negative individuals is in the Kurdish Jews (*9*) which have both thalassemia and G6PD deficiency genes at a high frequency (*10*). The Jews also give a significant negative correlation coefficient ($P < .02$) between the ABO and Rh incompatibility loads (*11*). In many other parts of the world information is sparse, but in many of the regions where malaria is, or was, endemic the frequency of Rh negatives is extremely low or even zero (*8*).

Thus the evidence from Europe, in particular, is strongly suggestive of a regulatory mechanism at the population level adjusting gene frequencies so that the overall genetic load is not excessive. Such a mechanism would operate even apart from any specific fitness interactions between loci. Presumably the result of the regulatory systems will be the attainment of an optimum genetic load, since zero genetic load implies no evolution, and too great a genetic load would lead to extinction.

It is not suggested that these conclusions invalidate the uses of blood groups in physical anthropology. The broad blood group gene frequency variations are no doubt based on migration and differential sensitivities to disease, but within a specific region it can be argued that the regulatory mechanisms proposed will be important as shown by the evidence for the importance of malaria as an environmental control mechanism.

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Sporangium Discharge in Pilobolus: A Photographic Study

Abstract. *Stages in the discharge of sporangia by the fungus,* Pilobolus kleinii, *were photographed by means of a high-speed electronic flash triggered by a photocell. The photographs confirm that the sporangium is propelled by a jet of cell sap. The jet is deflected from the sporangium and attains a considerable length before it breaks into droplets.*

The forcible discharge of the mature sporangium of *Pilobolus* is a dramatic and characteristic feature of asexual reproduction in this genus of coprophilous Phycomycetes (*1*). The structure of the sporangiophore and the discharge of the sporangium have been described by Buller (*2*) and Ingold (*3*), but recent improvements in techniques of high-speed photography have made it possible to record and study stages in the process of discharge itself.

To photograph the discharge, *Pilobolus kleinii* van Tiegh. was grown in 60-mm petri dishes on a synthetic medium (*4*). Cultures were maintained at 24°C in darkness for 4 days; they were then subjected to a daily alternation of 12 hours of light and 12 hours of darkness to induce synchronous formation of sporangiophores. The fungus began to discharge sporangia about 3 hours after the beginning of the third light period. A group of sporangiophores was cut from a culture a few hours before the beginning of discharge and placed in a small glass chamber together with moist blotting paper to maintain a high relative humidity. This chamber, with the sporangiophores oriented horizontally, was placed on the stage of a compound microscope. Light from a small illuminator fitted with a red filter and a diaphragm to limit the diameter of the field was focused on a sporangium by the condenser of the microscope. The objective of the microscope was focused on the same sporangium with the aid of a beam-splitter and telescope from a Leitz Micca attachment. A cadmium selenide photocell (Clairex CL-3) was mounted at the focus of the ocular. This photocell was connected in series with a 22.5-v battery and the primary of a small transformer, the secondary of which was connected to an amplifier whose output was applied to the grid of a thyratron (*5*). The thyratron triggered the flash unit, a General Radio Strobotac (model 1531A) (*6*), which was used at the high intensity setting and gave a flash with a duration of 3 μsec. In some cases, a nerve stimulator (Grass SD-5) was interposed between the thyratron and the flash unit to permit a delay of 200 μsec between the time of discharge of