A Life History Approach to the Evolution of Small Adult Body Size in *Homo Floresiensis*

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INTRODUCTION

In their original paper presenting LB1, the *H. floresisnsis* type specimen, Brown *et al*. [1] suggested her small stature was caused by insular dwarfism [1]. This conclusion was not without critics and alternative explanations for LB1 are that she 1) was, or was closely descended from, an *Australopithecine*, 2) was microcephalic [1, 2], or 3) was a pygmoid *H. erectus* or *H. sapiens*.

The physical, geographic, and temporal evidence, however, do not support the notion that *H. floresiensis* was an *Australopithecine* [3], and the microcephalic and pygmoid hypotheses become less plausible as skeletal evidence builds, supporting a new species designation [1, 3-7]. The insular dwarfism hypothesis, as further developed by Bromham and Cardillo [8], deserves further attention.

The "island rule," is the tendency for small animals to become larger and large animals to become smaller in isolated ecological niches like islands [4, 9]. Recent research suggests specific orders react to islands differently [9, 10], and that the island rule should be applied on a case by case basis, not as a sweeping generality [11]. With this in mind, there are several examples of mammalian species which have undergone changes in body size upon reaching islands [12]. These include the Wrangel Island mammoth (*Mammuthus primigenius*), which experienced a 65% size reduction in just 5,000 years [10]; the Bali tiger (*Panthera tigris balica*); the island fox (*Urocyon littoralis*); the extinct, elephant-like, *Stegadon* that once inhabited Flores [1, 13, 14]; and several primate species [8].

Limited food availability, widened diet breadth, changes in interspecific competition and predation, and increased mortality may lead to insular dwarfism [10, 15, 16]. No single cause, however, seems to explain all instances of the phenomenon, which furthers the notion that the "island rule" is less generalizable than once thought. As to not fall victim to such overgeneralization, this paper focuses on one genus, primates, and one cause of insular dwarfism, a change in life history.

Palkovacs [15] suggests that insular dwarfism might be explained by the effect of increased mortality on body size. This connection is formally modeled for primates by Charnov and Berrigan [17], who show that a population-wide change in the timing or rate of mortality affects how long individuals have to mature and reproduce (detailed derivations and descriptions of this model appear in Charnov [18-20] and Charnov and Berrigan [17, 21]). The essence of the relationship is the following: where mortality is high, growth and reproduction must happen quickly; where it is low, more time and energy can be spent on somatic growth and reproduction of larger, ostensibly higher quality, offspring. Instantaneous adult mortality rate, therefore, influences both offspring production and adult body size [17-22]. *H. floresiensis* was about half the size of *H. erectus*, suggesting a mortality difference between the two species.

RESULTS

Substituting the weights of *H. erectus* and *H. floresiensis* into Equation 1 gives instantaneous adult mortality rates for each. McHenry and Coffing [23] estimate an adult female *H. erectus* weighed 56 kg. There is not yet a consensus on *H. floresiensis* body weight, so I averaged two estimates for the type specimen, LB1. The first put her at 16 - 28.7 kg based on height, while the second put her at 36 kg based on femoral cross-section measurements [1]. These average to 29.175 kg. Substituting W = 56 kg for *H. erectus* gives $M = 0.053$, and for *H. floresiensis*, $W = 29.175$ kg gives $M = 0.068$. The difference between these estimates is 0.015, which, divided by the original *H. erectus* mortality rate, produces a mortality difference of 0.281, or about 28%.

What of the error in the model? The maximum estimates of M for *H. erectus* and *H. floresiensis* are 0.122 and 0.135, respectively. Dividing their difference of 0.013 into the maximum *H. erectus* mortality estimate, 0.122, produces a mortality difference of 0.107, or about 11%. Using the same method with minimum M estimates gives a mortality difference of -106%, insinuating a *decrease* in mortality could have led *H. erectus* to shrink; however, this requires using a negative mortality rate, -0.106, for *H. erectus*, which is impossible in nature since the lower limits of both W and M are greater than zero. Furthermore, the suggestion that decreasing mortality increases body size disagrees with both accepted theory and the empirical data [8].

Even if I arbitrarily assign a very low value of $M = 0.001$ for *H. erectus* and use the minimum estimate given in the model for *H. floresiensis*, $M = 0.001$, the predicted mortality difference is 0%. If I pair the maximum mortality estimate for *H. erectus*, $M = 0.122$, with the minimum for *H. floresiensis*, 0.001, and divide back into the *H. erectus* maximum, a 99.2% mortality difference emerges. These error figures are broad, but reasonable. At maximum mortality levels for both species, an 11% increase in mortality accounts for the size difference; at minimum levels, a negligible, even non-existent, change does; and at the maximum difference, a 99% increase explains the disparity.

DISCUSSION

According to the empirical model, a 28% increase in instantaneous mortality best accounts for the nearly 50% difference in body size between *H. erectus* and *H. floresiensis*. Even at its most conservative, the model shows a doubling of mortality explains the difference. Such an increase may seem high, but the model is static and says nothing of time; recall that the Wrangel Island mammoth underwent a 65% size reduction in just 5,000 years. *H. erectus* reached Flores about 840 kya [13] while *H. floresiensis* appeared there around 90,000 kya [14], leaving selection more than 700,000 years in which to act. Indeed, many primates have experienced significant changes in adult body mass since being isolated on islands following the last glacial maximum [8]

If *H. floresiensis* was the result of increased mortality on *H. erectus*, what caused it? Disease seems an obvious first candidate, but the population of *H. erectus* on Flores was probably not large enough to support communicable varieties at high rates. Predation is another possible cause, and others have noted that komodo dragons (*Varanus komodoensis*) lived alongside *H. floresiensis* [14, 24]; is it possible that the large lizard targeted hominins?

The komodo dragon reached Flores long before *H. erectus* or *H. floresiensis* [25]. The animals are fierce predators and the average adult is 2.5 meters long and weighs 70 kg [25]. They can run 20 km/hr, have excellent senses of smell and sight, and their saliva contains high loads of *Escherichia coli* bacteria [26]. Modern komodo dragons hunt Rousa deer weighing 90 kg, goats, and wild boar [25]. They even occasionally kill adult humans. Komodo dragons clearly could have taken prey the size of *H. erectus* or *H. floresiensis*. In addition, Liang Bua, the cave were LB1 was found, contains deposits of komodo dragon bones, suggesting interaction with *H. floresiensis* [14].

H. erectus may have spread throughout the Indonesian islands, encountering komodo dragons along the way. Although large, technologically sophisticated, and intelligent, *H. erectus* could have experienced high casualty rates at the hands of the dragons; after all, just being wounded could have caused fatal infections. In this scenario, increased mortality led to selection for earlier maturation and reproduction, and thus smaller size. As individuals became smaller, they became even better targets for predators until mortality rates and body size eventually stabilized in a new species, *H. floresiensis*.

Life history theory links many physiological attributes, including body size, to mortality rates. In this paper, I described the theoretical relationship between mortality rates and body size as shown in Charnov and Berrigan (1993). Then, I developed an empirical model of this relationship and used it to show that a 28% increase in mortality accounts for the difference in size between *H. erectus* and *H. floresiensis*. Last, I presented an evolutionary scenario suggesting komodo dragons were the cause of this increased mortality.

Insular dwarfism is a complex phenomenon and body size is likely regulated by several mechanisms which behave differently across clades [11]. Life history is just one mechanism by which insular dwarfism works, and the model presented here is an application of the theory. Although I have focused on primates, the same method should apply to other mammals.

MATERIAL AND METHODS

I use linear regression to show how a reasonable increase in adult instantaneous mortality rates (“survival for one more year” [19]) could have led a *H. erectus* population to “shrink” to the size of *H. floresiensis*. To do this, I quantified the mortality rates of fourteen extant primate species, regressed their lifespans on those rates, and then used the relationship to generate mortality estimates for *H. floresiensis* and *H. erectus*. The results show that a reasonable increase in mortality accounts for much of the small body size of *H. floresiensis*.

I generated the mortality levels in Table 1 using the method of dividing average lifespan into 1 as shown in Charnov and Berrigan (1993). Average lifespan and body size data came from Alvarez (2000). For example, the average lifespan of *Callitrichus jaccus* is 4.7 years. Divided into 1, this gives 0.213 as the mortality rate.

Next, I made a regression model using instantaneous mortality (M) as the dependent variable and body weight (W) as the independent. The relationship between the two is significant (n=14, p<.001, F=25.667, d.f.=1,12, R^2=0.682), and shown on logged axes in Figure 1. The linear equation for the fit line is below (Equation 1). It is arranged to generate easy to interpret mortality rates (M) using the natural log of W:

Equation 1: $M = .146 - .023 * \ln(W)$

The empirical data support Charnov and Berrigan’s theoretical argument that mortality and adult body weight are related.

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