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How supplemental food may induce abnormality in wild Japanese macaque populations

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ABSTRACT

Food has been supplied to many wild Japanese macaque (*Macaca fuscata*) populations in Japan since the 1950s. Since then, the monkeys have often exhibited various physical abnormalities such as anomalies and malformation of the limbs and other body parts. No conclusive explanation exists for these anomalies. Here we propose a new population-level hypothesis: feeding relaxes selection intensity such that abnormal babies tend to be born and survive to adulthood. We build a discrete population dynamic model and use it to simulate successfully the macaque population of Mt. Takasaki. Contrary to our usual intuition that a bad effect is caused by a bad factor, in some situations frequent abnormalities could be caused by the shift to a good environment.

Keywords: abnormalities, feedings, Japanese macaque, selection intensity.

INTRODUCTION

Since the early 1950s, food provisioning has been occurring for many wild Japanese macaque (*Macaca fuscata*) populations across Japan. Feedings to the macaques of Saki-shima Island began in summer 1952 and in that fall to those of Mt. Takasaki (Itani, 1954, 1975).

We could find no record of abnormalities in the wild macaques before food provisioning. However, a few years after the feedings started (*c.* the mid-1950s), physical abnormalities began to be reported. For instance, monkeys might be missing a foot or a hand, or have cleft hands and feet [Itani and Mizuhara, 1957; Furuya, 1966; Iwamoto, 1967; Kawamura, 1974; Japanese Monkey Abnormality Problem Research Group (JMAP), 1979; Homma, 1980].

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Abnormalities have been documented in detail in the Mt. Takasaki population (Itani and Mizuhara, 1957; Furuya, 1966; Iwamoto, 1967; JMAP, 1979). Data have recently been collected at the Mt. Takasaki Nature Park, Oita City. In Mt. Takasaki, the first abnormal monkey was observed in 1955 (Itani, 1954, 1975; Itani and Mizuhara, 1957). For the next 20–30 years, the number of abnormal monkeys increased quite rapidly. Then, for no apparent reason, it decreased to a low level. Recently, few monkeys have been abnormal, but some are still seen in most years. This pattern is quite similar to that in all other monkey populations in Japan (Kawamura, 1974; JMAP, 1979; Homma, 1980).

Domestic and zoo animal populations also exhibit abnormalities, but less frequently. Such abnormalities pose a serious problem for the management of both domestic and wild animal populations.

Many hypotheses have been proposed and evaluated to account for the high abnormality rate in Japanese macaques: environmental pollution, food contamination (by agricultural chemicals such as pesticides) and genetic problems (such as incompatibility between mates, inbreeding depression and incest mating) (Nishida, 1966; Iwamoto, 1967; Iwamoto and Hirai, 1970; Iwamoto *et al.*, 1975; JMAP, 1979; Nakamichi *et al.*, 1983). However, no conclusive evidence has been found even after their detailed evaluation (JMAP, 1979). The reason for frequent abnormalities in Japanese macaques remains unclear.

Here we propose an alternate hypothesis: a natural cause. We suggest that the reduction in selection intensity due to feedings enables many abnormal babies to be born and survive in wild monkey populations. Using a discrete population model, we show that high abnormality rates can be induced by a rapid population increase. Then we compare the model output to the historical records of wild monkey populations in Mt. Takasaki (Itani, 1954).

ASSOCIATION OF MORTALITY AND ABNORMALITY

Abnormalities are rarely observed in newborns in wild animal populations. In natural conditions where food is scarce, the pregnancy of abnormal individuals is easily terminated/aborted due to poor maternal condition. Furthermore, babies born with an abnormality usually die during early infancy due to their poor ability to gather food or escape predation. Therefore, we expect that the proportion of abnormal individuals in a wild population should be extremely low in a rigorous environment with high death rates. In contrast, if death rates fall, abnormalities might increase.

We observed this correlation in Mt. Takasaki. A high rate of abnormalities appears only when mortality is low; it is almost zero when mortality is high (Fig. 1). Therefore, our model assumes that the abnormality rate in a population decreases with increasing death rates (Fig. 2).

Figure 2 shows the assumed rates of abnormality in a population as a function of death rate (m) (Fig. 2A). The instantaneous abnormality density is $g(m)$. Areas A1, A2, A3 and A4 represent the proportions of dead normal, dead abnormal, live normal and live abnormal individuals, respectively. The rate of abnormality r is given by

$$r(m) = [1/(1 - m)] \int_m^1 g(m)\mathrm{d}m = (\mathrm{A4})/(\mathrm{A3} + \mathrm{A4})$$

For numerical calculations, the instantaneous abnormal density $g(m)$ is arbitrarily set to

$$g(m) = (0.9\ \mathrm{e}^{-20m})/[1 - (1 - (0.9/0.93)\ \mathrm{e}^{-20m})]$$

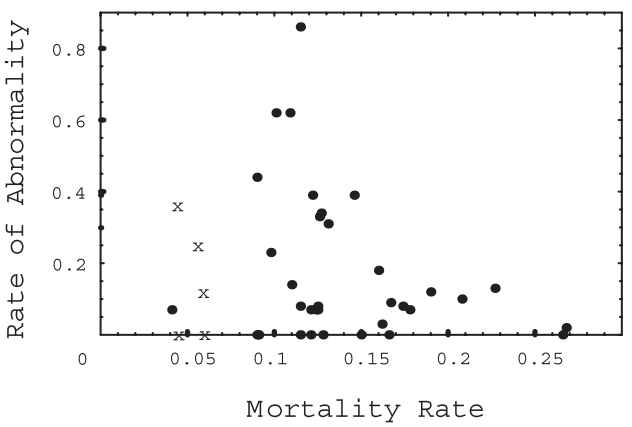

Fig. 1. The relationship between rate of abnormality and estimated mortality in wild Japanese macaques in Mt. Takasaki, 1953–99 (dots: 1965–2000; crosses: 1953–57). Mortality is estimated from population sizes and the number of births, assuming no migrations. Data points (years) with negative mortality (due to immigration) were eliminated from the analyses. Data for 1950–52 are taken from JMAP (1979) and those for 1953–58 and 1965–2000 were collected from Mt. Takasaki Nature Park, Oita, and JMAP (1979). (Abnormality data are missing for 1959–64.)

Figure 2B shows that a reduction in the death rate induces a drastic increase in the rate of abnormalities. At the death rate $m(t)$, the rate of abnormality is close to zero, while at $m(s)$, it increases considerably. Figure 2C plots the rate of abnormality $r = r(m)$ as a function of mortality (m). If m is above 0.3, r is almost zero.

A MODEL OF POPULATION DYNAMICS

We built a discrete population dynamics model to illustrate the emergence of abnormalities (Fig. 3A). In Mt. Takasaki, the monkey population has increased almost ten-fold since feedings began. We simulated this population increase with the model and calculated the mortality rates for such an increase, such that $r = r(m)$. The number of abnormal individuals is the product of population size (Fig. 3A) and the abnormality rate (Fig. 2C).

The model expresses population dynamics as a discrete Ricker model:

$$N(t+1) = N(t)\,[e^{B(t)-M(t)}]$$

where $N(t)$, $B(t)$ and $M(t)$ are the population size, birth rate and death rate, respectively, at year t. Here $B(t)$ and $M(t)$ are defined as

$$B(t) = b_k + (b_0 - b_k)(1 - N(t)/K)$$

and

$$M(t) = m_0 + (m_k - m_0)\,N(t)/K$$

where b_0, b_k, m_0, m_k and K are the intrinsic birth rate (at $N \cong 0$), the equilibrium birth rate (at $N = K$), the intrinsic death rate, the equilibrium death rate and the carrying capacity, respectively (Yoshimura and Clark, 1994). Note that, by definition of equilibrium, $b_k = m_k$. In each generation, we set $m = M(t)$ to calculate the expected rate of abnormality, r (Fig. 2). Then the estimated number of abnormal individuals is given by the product $M(t)N(t)$.

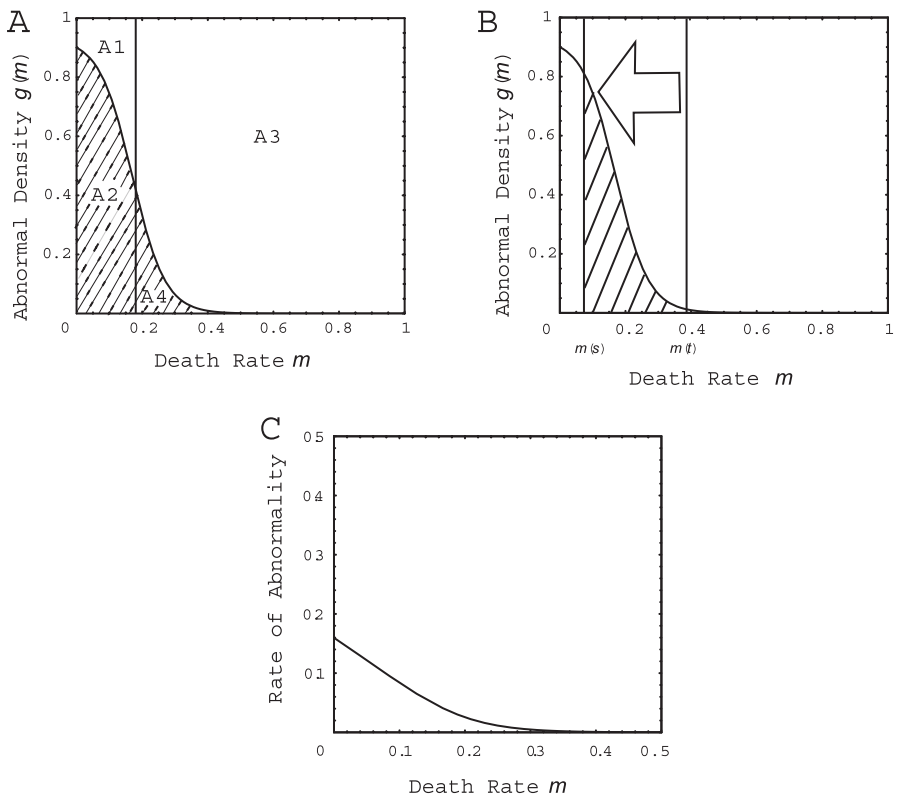

Fig. 2. The supposed rates of abnormal individuals in a population in relation to death rate. (A) The instantaneous abnormality density function $g(m)$ for death rates m. The vertical line indicates the current death rate. Areas A1, A2, A3 and A4 represent the proportions of dead normal, dead abnormal, live normal and live abnormal individuals, respectively. (B) The drastic increase in abnormality rate caused by the reduction in death rate. At death rate $m(t)$, the abnormality rate is close to zero (no area under the curve), while at $m(s)$, the rate increases considerably (large area under the curve). (C) The rate of abnormality $r = r(m)$ as a function of mortality m.

RESULTS

The monkey troop consisted of 166 individuals in 1950 when the survey began (Itani, 1954). In the late 1980s, the population had reached about 2000. Afterwards, it fluctuated around that level.

We set the simulation parameters to match the observed data of monkeys at Mt. Takasaki (Fig. 3). We set the carrying capacity before initial feedings to $K_{1950} = N(1950) = 166$, and that from 1952 to $K_{1952} = 2000$ (Fig. 3A). We also set $b_0 = 0.27$, $b_k = m_k = 0.25$ and $m_0 = 0.05$ to achieve visual curve matching (population dynamics) as a result of reducing the death rates.

The simulated abnormality rate is plotted with the observed data in Fig. 3B and the number of abnormals in Fig. 3C. Note that the high abnormality rates appeared around the 1970s (JMAP, 1979).

The number of abnormal individuals is expected to increase sharply right after feeding is initiated (1952), reach a peak and then decrease and converge to a new higher equilibrium

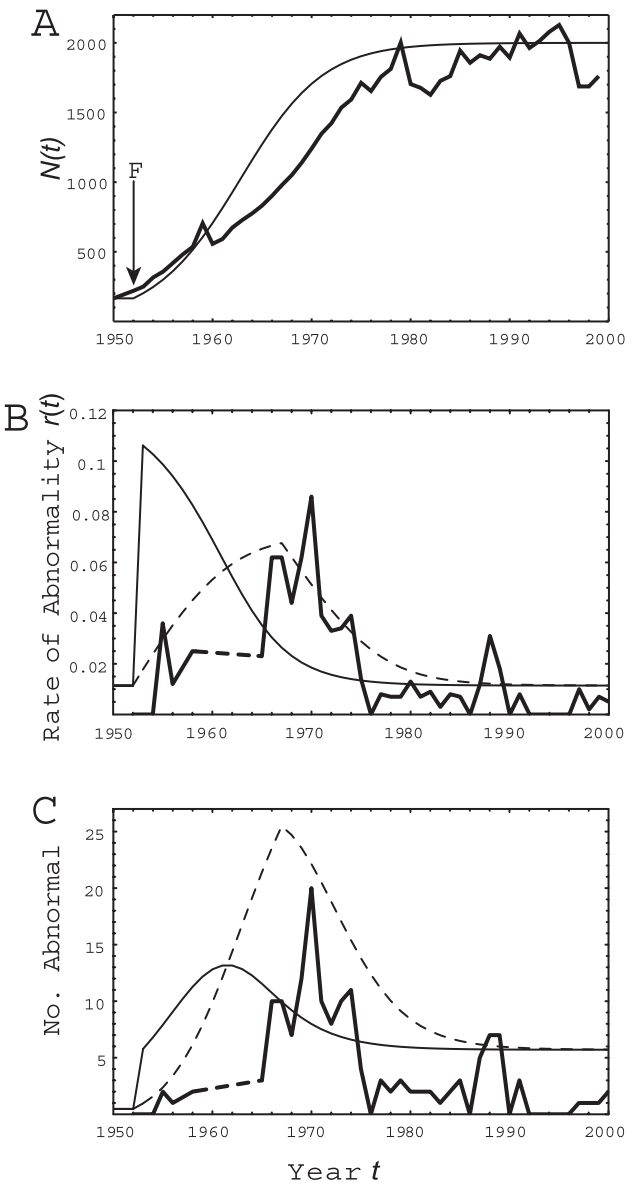

Fig. 3. Observed and simulated dynamics of population size and abnormality in Japanese macaques in Mt. Takasaki (1950–2000). Bold lines = observed values (dotted portions indicate missing data); thin lines = simulated values; dotted lines = simulated values with delay effects. (A) Population sizes $N(t)$. (The arrow and F indicate the start of feedings.) (B) Abnormality rates $r(t)$. (C) Number of abnormal individuals. Observed population sizes for 1950–52 are taken from JMAP (1979) and those for 1953–2000 from Mt. Takasaki Nature Park, Oita (unpublished data). Observed numbers of abnormal individuals for 1950–68 are taken from JMAP (1979) and those for 1969–2000 from Mt. Takasaki Nature Park, Oita (unpublished data). For this numerical example, the density of abnormal individuals in relation to death rate $g(m)$ is arbitrarily set as in Fig. 2.

value (Fig. 3, thin line). Indeed, the observed number of abnormal individuals was zero before feedings; it increased sharply to over 20 individuals around 1970 and then decreased to a few individuals (Fig. 3C, bold line). The observed data are qualitatively similar in temporal dynamics except for the absence of a sharp initial increase. We discuss this discrepancy below and attempt to account for it by introducing a simple lag effect to the model.

DISCUSSION

Our new hypothesis depends on population-level phenomena (DeWitt and Yoshimura, 1998). Thus it differs fundamentally from previous hypotheses, which are based on individual-level causes (JMAP, 1979). When a population is increasing, the number of abnormal monkeys increases sharply. When population size levels off, the number of abnormal monkeys decreases and converges to a new equilibrium. This new equilibrium exceeds the initial one because, although selection brought about by food shortage resumes, it is weaker.

Even though the current model is very simple and ignores many factors affecting monkey populations, the trend for abnormality is qualitatively similar to what is observed in Mt. Takasaki (Fig. 3). One other factor may further explain the pattern. This is the time lag due to the age structure of the monkey population. In the current population model, all individuals reflect the effects of abnormality instantaneously. But in natural populations, only the newborn cohort responds. It takes many years of reproduction before the abnormality rate in the population as a whole can catch up with the abnormality rate among newborns.

We changed the model to incorporate the time lag. We assumed 15 years of delay in reproduction. Then we introduced the effective abnormality rate r^*, which averages the previous 15 years of mortality to calculate the abnormality rate:

$$r^*(t) = (1/15) \sum_{i=t-14}^{t} r(m(t))$$

We based the 15-year figure on the maturation and reproduction of female monkeys (Itani, 1954, 1975). Expected abnormality in the time lag model better matches the observed trend (the thin dotted lines in Fig. 3B and C).

As our population-level mechanism shows, one does not need any external factors to explain abnormality rates. Influences such as food contamination, agricultural chemical contamination, genetic problems and diseases (Iwamoto and Hirai, 1970; Iwamoto *et al.*, 1975; JMAP, 1979) are not necessary. We suspect that the relaxation of selection intensity due to food provisioning is at least a major cause of high rates of abnormality in Japanese macaques in Mt. Takasaki and throughout Japan.

Ecologists often suspect that abnormalities are caused by bad environmental conditions. However, environmental improvement can also induce a higher rate of abnormalities. Nevertheless, the increase in abnormality rate due to environmental improvement is only temporary. The rate of abnormality declines to a very low rate once the monkey population nears its carrying capacity. Thus the increased abnormality is not actually caused by the good environment but by the relaxation of competition. Therefore, the good environment can be maintained without the rate of abnormality continuing to increase until all monkeys are missing a limb. In wildlife conservation and zoo animal management, a high level

of abnormality may not be a serious problem, but merely a temporary population phenomenon expressed only while the population has excess food.

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REFERENCES

DeWitt, T.J. and Yoshimura, J. 1998. The fitness threshold model: random environmental change alters adaptive landscapes. *Evol. Ecol.*, **12**: 615–626.

Furuya, Y. 1966. On the malformation occurred in the Gagyusan troop of wild Japanese monkeys. *Primates*, **7**: 488–492.

Homma, T. 1980. Sequential pattern of limb anomalies in Japanese monkeys on Awajishima Island. *Primates*, **21**: 20–30.

Itani, J. 1954. *Monkeys in Mt. Takasakiyama*. Tokyo: Kobunsha (in Japanese).

Itani, J. 1975. Twenty years with Mount Takasaki monkeys. In *Primate Utilization and Conservation* (G. Bermant and D.C. Lindburg, eds), pp. 101–125. New York: Wiley.

Itani, J. and Mizuhara, H. 1957. Notes on malformed individuals found in the wild group of Japanese macaques at Takasakiyama. *Bull. Exp. Anim.*, **4**: 105–107 (in Japanese).

Iwamoto, M. 1967. Morphological observations on the congenital malformation of limbs in the Japanese monkeys. *Primates*, **8**: 247–270.

Iwamoto, M. and Hirai, M. 1970. Case report on a Japanese monkey with congenital malformation of the limbs. *Primates*, **11**: 395–398.

Iwamoto, M., Shigehara, N. and Hakura, N. 1975. On the limb malformation in the Shiga A-troop in the Japanese monkeys. *Physiol. Ecol.*, **16**: 77–79 (in Japanese).

Japanese Monkey Abnormality Problem Research Group (JMAP). 1979. *Abnormal Monkeys*, revised edition. Tokyo: Shiobunsha (in Japanese).

Kawamura, S. 1974. The present situation of Japanese monkeys and consideration of their conservation. In *Proceedings of the Symposia of the Fifth Congress of the International Primatological Society* (S. Kondo, M. Kawai, A. Ehara and S. Kawamura, eds), pp. 501–507. Tokyo: Japan Science Press.

Nakamichi, M., Fujii, H. and Koyama, T. 1983. Behavioral development of a malformed infant in free-ranging group of Japanese monkeys. *Primates*, **24**: 52–66.

Nishida, T. 1966. A sociological study of solitary male monkeys. *Primates*, **7**: 141–204.

Yoshimura, J. and Clark, C.W. 1994. Population dynamics of sexual and resource competitions. *Theor. Pop. Biol.*, **45**: 121–131.