

## ONLINE SUPPORTING INFORMATION

### BET-HEDGING AS A MECHANISM FOR THE EVOLUTION OF POLYANDRY, REVISITED

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#### 1. **Supplementary material and methods**

##### METAPOPULATION STRUCTURE AND DISPERSAL AMONG PATCHES

Each subpopulation received immigrants from 2 neighboring subpopulations in every generation (Fig.S1), and four (1, 5, 10, or 15%) migration rates were simulated for the female offspring produced in each subpopulation. After dispersal, and for each subpopulation, all offspring (containing immigrants plus the resident polyandrous and monandrous genotypes) compete (with equal chances of being successful) for the limiting carrying capacity ( $K$ ), which ranged between  $K=5$  to  $K=100$  females for the patches (metapopulation contains 100~500 individuals in total).

In addition, a proportion ( $D$ ) of settled individuals are assumed to die (at the same rate for both genotypes) in order to make room for immigrants in each subpopulation.

The effect of the reduction coefficient  $D$  is relatively larger in small populations ( $D = r(1-K)/(K+200)$ , with  $r$  being a uniform random number from 0 to 1). This reduction supposes random genetic drift or stochastic environmental catastrophes (e.g. the destruction of insect host plants by large herbivorous mammals). Consequently, natural selection on mating frequency works only through differential reproductive success in a patch rather than through dispersal success.

## MALE QUALITY VARIATION

### *Setting the continuous distributions of intrinsic male quality variation*

In addition to the binomial male quality settings shown in the main text, we performed the simulations based on continuous male quality distributions. We generated a truncated normal distribution shown in Fig. S2A using normally distributed random numbers (mean=0.5 and SD=0.167) and setting values  $>1$  or  $<0$  to 1 and 0, respectively. Other distributions were modified from this distribution, by subtracting 0.37 (Fig.S2B), setting values  $<0.37$  as 0 (Fig.S2C), or adding 0.25 (Fig.S2D). Based on Garcia-Gonzalez's (2004) review about the extent of infertile matings in natural populations (see main text), the bimodal distribution (Fig.S2C), in which the frequency of unsuitable males is 22%, was the distribution most extensively simulated. In the simulations, male intrinsic quality determines offspring survival (if females mate with a male with 0.1 quality, only 10% eggs are viable). For simplicity and heuristic reasons we assume that male quality is determined environmentally implying that it is not heritable.

### *Setting the simulation of genetic incompatibility*

The difficulties of simulating appropriate mechanisms underlying genetic incompatibility in simulation studies that require a large number of generations is often overlooked. The main problems include the exhaustion of male quality variance and the incorporation of fully-incompatible matings that are often observed in empirical studies (e.g. Zeh 1997, Newcomer et al. 1999). As an example, incompatibility caused by cytoplasmic endosymbionts such as *Wolbachia* disappears after the endosymbionts spread all over the population (Werren 1997). Incompatibility by *Wolbachia* occurs only in the mating between uninfected females and infected males (Werren 1997). Because of the one-way nature of the incompatibility (i.e. uninfected females cannot produce any offspring when mating with infected males while infected females can reproduce with both uninfected and infected males), *Wolbachia* quickly infest all individuals in the population (Werren 1997). In the case of incompatibility based on heterozygote

superiority (overdominance), often claimed to underlie inbreeding-avoidance polyandry (Stockley et al. 1993, Tregenza and Wedell 1998), if homozygous offspring (genotypes AA and BB) are inviable, only heterozygote individuals (genotype AB) exist in population, implying that genotypic variation is depleted. Therefore, under both mechanisms of genetic incompatibility (endosymbiont model and overdominance model) the variance in male quality is exhausted.

In this study, in order to avoid these problems, we adopt a single-locus gametophytic incompatibility system (McCubbin and Kao 2000). A compatibility controlling locus has several alleles (e.g. A-D in Table S1), and homozygotes (e.g. AA, BB, CC....) for this locus are lethal due to incompatibility. Thus, only heterozygotes exist in the population and each heterozygote (e.g. AB, AC, BC....) has equal genotypic frequency in evolutionary equilibrium because homozygote mortality produces strong negative frequency-dependent selection on this locus. By assuming that this stabilizing selection is far stronger than selection on female mating frequency via bet-hedging, we assume that the compatibility locus rapidly reaches the genetic equilibrium and that the evolution of mating frequency occurs on this stable genetic background. The compatibility phenotype of the sperm is determined by its own gametophytic haploid genotype, while that of the egg is determined by the diploid genotype of the female producing it. For example, sperm of haplotype A produced by AB males are incompatible with eggs produced by females of genotypes AB and AC, while B sperm in the same ejaculate are compatible with AC eggs (Table S1). This corresponds to the plant system that haplotype  $S_1$  pollen cannot elongate the pollen tube into stigmas of genotype  $S_1S_2$  and  $S_1S_3$  (McCubbin and Kao 2000). If the compatibility phenotype of the eggs was determined by their own haploid genotype, the fully-incompatible mating combination would not be present in Table S1 (e.g. ABxAB mating would produce AB viable offspring). Thus, the gametophytic incompatibility system is an appropriate genetic mechanism for our analyses. Table S1 shows the fitness consequence of offspring from all possible mating combinations in the cases in which the compatibility locus has either three- or four-alleles. In general, when the number of alleles (A, B, C....) is  $n$ , the number of genotypes (AB, AC, AD....) is  ${}_nC_2$  (the number of 2-combinations from a given set of  $n$  elements =  $n!/(2!(n-2)!)$ ). All genotypes have the same frequency in population and this frequency distribution is constant over generations because of random mating and homozygote mortality. Females of a given genotype (e.g. AB) randomly sample mates from these  ${}_nC_2$  different male genotypes (e.g. AB, AC and BC males if  $n = 3$  and AB, AC, AD, BC, BD and CD males if  $n = 4$ ) with equal probability (based on random mating due to unreliable mate quality discrimination). Out of these mating

combinations, a proportion  $p = 1/nC_2$  of matings are fully incompatible (i.e. all offspring are inviable), a proportion  $q = (2n-4)/nC_2$  are partially incompatible (i.e. 50% of offspring are inviable) and the remaining proportion  $(1-p-q)$  is fully-compatible (i.e. all offspring are viable) (Table S1). As the number of alleles (and genotypes) in the compatibility locus increases, the probability of incompatible matings (that is, the population genetic load due to incompatibility) decreases (Fig.S3).

## 2. Supplementary results

### INTRINSIC MALE QUALITY SCENARIO WITH CONTINUOUS QUALITY DISTRIBUTIONS

Results were qualitatively similar to the results for the binomial male quality case shown in the main text: the frequency distribution of unsuitable males in the population (Fig.S2) emerges as the primary factor determining the maintenance of polyandrous behavior. When the distribution of male quality has a mean equal to or greater than 0.5, polyandry has no selective advantage over monandry along the parameter space simulated, except for the cases where polyandry bears no costs (Fig. S4). However, if the male quality distribution shifts to a lower average, or shows a bimodal pattern with frequent occurrence of total reproductive failure, polyandry achieves higher fixation probabilities than monandry, even if it entails 10% or higher fitness costs (Fig. S4). Consequently, we performed simulations mainly based on the use of a bimodal male quality distribution with mean = 0.44. Both in the symmetrical competition (Fig.S5) and the invasibility analysis (Fig.S6), the results mirror those obtained under the binomial male quality case (Fig.3~5). This implies that only the existence and abundance of completely unsuitable males (rather than the shape of male quality distribution) are important for the evolution of polyandry by bet-hedging. Therefore, the risk of total reproductive failure would mainly promote females to mate multiply.

## 3. References

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#### 4. Supplementary Table S1

Table S1 Offspring genotypes produced from all possible matings under gametophytic incompatibility, and their viability\*

##### a. Three alleles in the compatibility locus

	AB father	AC father	BC father
AB mother	AA, 2AB, BB	AA, AB, <u>AC</u> , <u>BC</u>	AB, <u>AC</u> , BB, <u>BC</u>
AC mother	AA, <u>AB</u> , AC, <u>BC</u>	AA, 2AC, CC	<u>AB</u> , AC, <u>BC</u> , CC
BC mother	<u>AB</u> , <u>AC</u> , BB, BC	<u>AB</u> , <u>AC</u> , BC, CC	BB, 2BC, CC

Fully-compatible mating
Partially-compatible mating
Fully-incompatible mating

##### b. Four alleles in the compatibility locus

	AB father	AC father	AD father	BC father	BD father	CD father
AB mother	AA, 2AB, BB	AA, AB, <u>AC</u> , <u>BC</u>	AA, AB, <u>AD</u> , <u>BD</u>	AB, <u>AC</u> , BB, <u>BC</u>	AB, <u>AD</u> , BB, <u>BD</u>	<u>AC</u> , <u>AD</u> , <u>BC</u> , <u>BD</u>
AC mother	AA, <u>AB</u> , AC, <u>BC</u>	AA, 2AC, CC	AA, AC, <u>AD</u> , <u>CD</u>	<u>AB</u> , AC, <u>BC</u> , CC	<u>AB</u> , <u>AD</u> , <u>BC</u> , <u>CD</u>	AC, <u>AD</u> , CC, <u>CD</u>
AD mother	AA, <u>AB</u> , AD, <u>BD</u>	AA, <u>AC</u> , AD, <u>CD</u>	AA, 2AD, DD	<u>AB</u> , <u>AC</u> , <u>BD</u> , <u>CD</u>	AB, AD, <u>BD</u> , DD	AC, AD, <u>CD</u> , DD
BC mother	<u>AB</u> , <u>AC</u> , BB, BC	<u>AB</u> , <u>AC</u> , BC, CC	<u>AB</u> , <u>AC</u> , <u>BD</u> , <u>CD</u>	BB, 2BC, CC	BB, BC, <u>BD</u> , <u>CD</u>	BC, <u>BD</u> , CC, <u>CD</u>
BD mother	<u>AB</u> , <u>AD</u> , BB, BD	<u>AB</u> , <u>AD</u> , <u>BC</u> , <u>CD</u>	<u>AB</u> , <u>AD</u> , BD, DD	BB, <u>BC</u> , BD, <u>CD</u>	BB, 2BD, DD	<u>BC</u> , BD, <u>CD</u> , DD
CD mother	<u>AC</u> , <u>AD</u> , <u>BC</u> , <u>BD</u>	<u>AC</u> , <u>AD</u> , CC, CD	<u>AC</u> , <u>AD</u> , CD, DD	<u>BC</u> , <u>BD</u> , CC, CD	<u>BC</u> , <u>BD</u> , CD, DD	CC, 2CD, DD

\* Viable offspring is underlined.

## 5. Supplementary Figures S1~S6

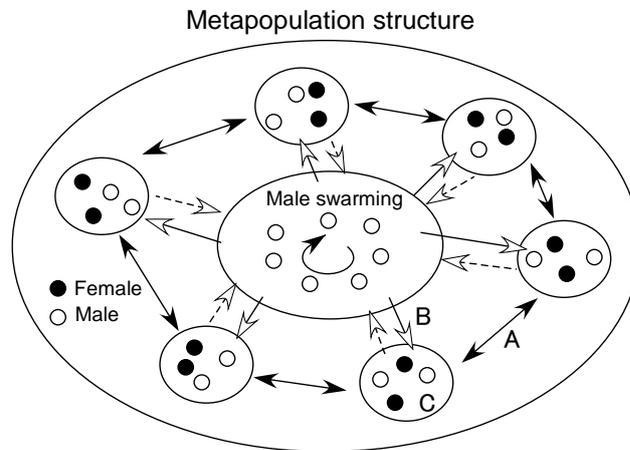


Figure S1: Diagram of metapopulation dynamics. (A) Females disperse to 2 neighboring patches. (B) Males disperse randomly metapopulation-wide after swarming. (C) After dispersal, females compete against each other and survivors mate with males within each patch.

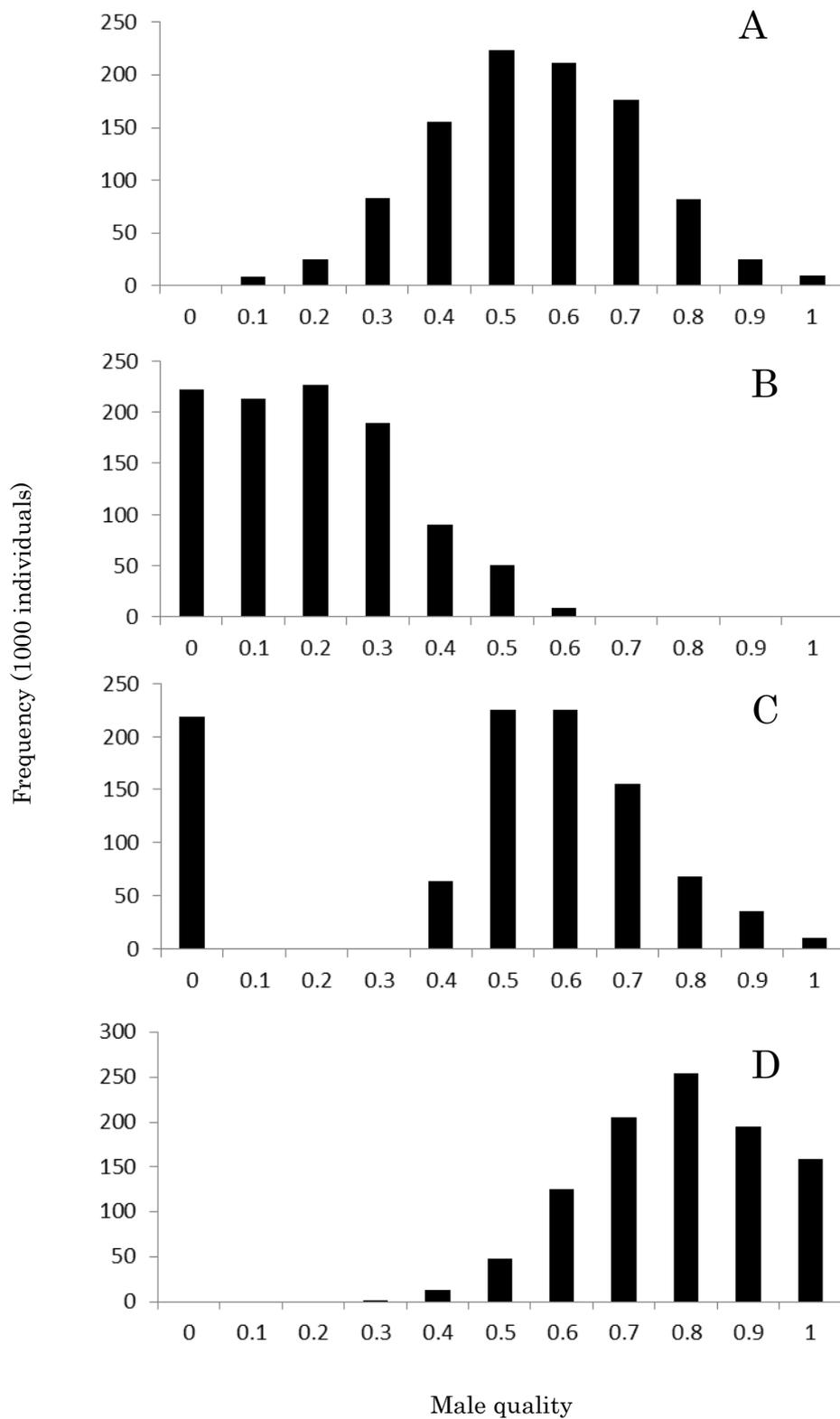


Figure S2. Four examples of male quality (ability of males to produce viable offspring measured as a proportion ranging from 0 to 1) distributions under the intrinsic male quality variation scenario. One thousand individuals were derived from a truncated normal distribution with constant SD (0.1667). (A) Truncated normal distribution with mean = 0.5, (B) Skewed distribution with mean = 0.15, (C) Bimodal distribution with mean = 0.44, (D) Skewed distribution with mean = 0.74. Note ca. 22% of males are totally infertile in (B) and (C).

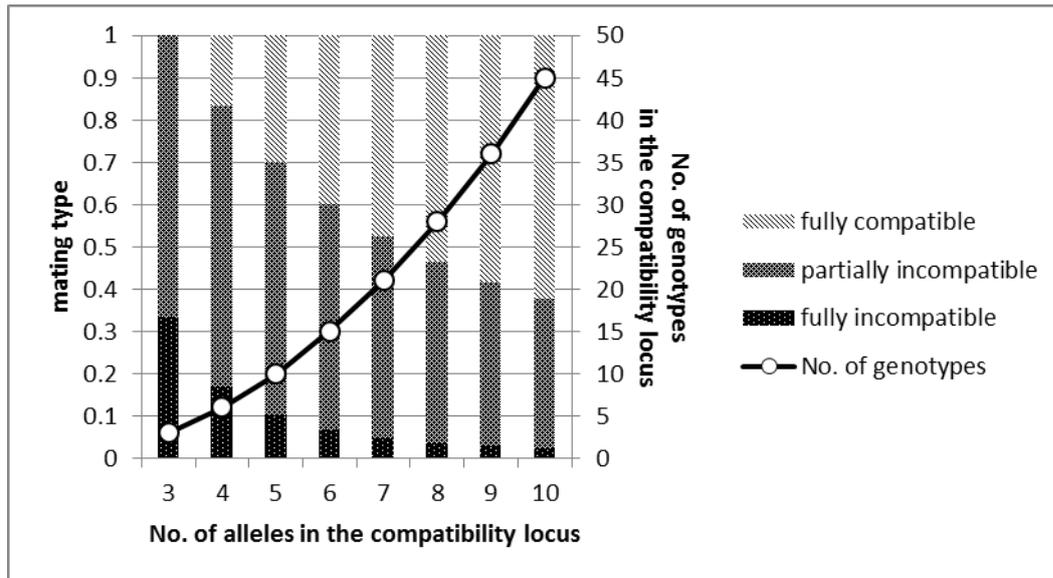


Figure S3. Probability of compatible or incompatible matings in relation to the number of alleles in the compatibility-controlling locus.

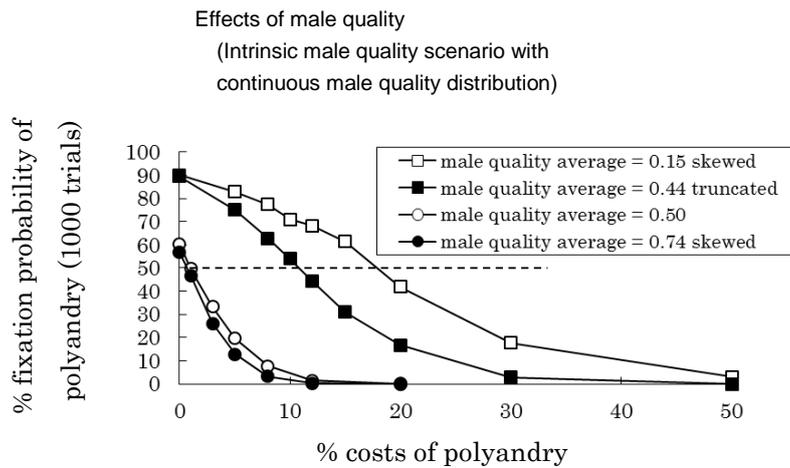


Figure S4: Fixation probabilities of polyandry obtained in the simulations under the intrinsic male quality scenario with various male quality distributions (Fig. S2). The frequency of mating genotypes in the initial generation is 1:1 (polyandry:monandry). Percent-costs of polyandry are equivalent to the decrease of mean egg production in the polyandry genotype (see Methods in the main text). General conditions: Polyandry mating frequency = 5; Number of patches = 10; Patch size = 10 females (i.e., Metapopulation size = 100); Dispersal rate = 1% per generation. The horizontal dotted line represents the threshold above which polyandry is advantageous compared to monandry.

### Intrinsic male quality scenario

(continuous male quality with average = 0.44 truncated)

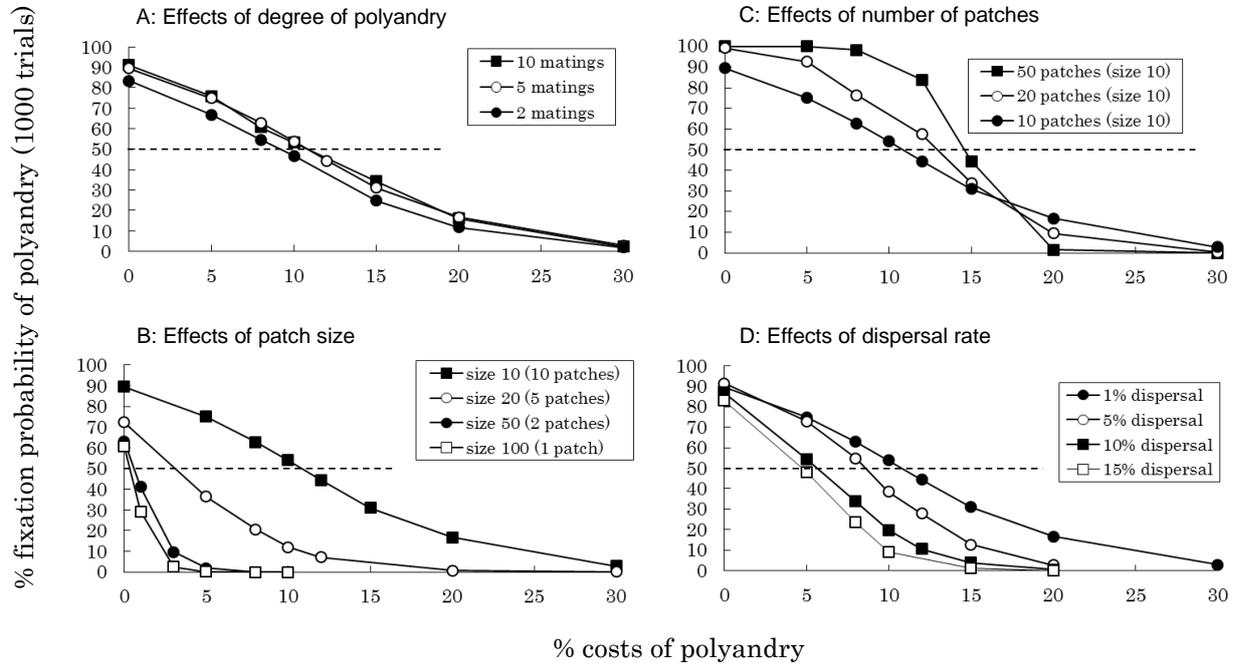


Figure S5: Fixation probabilities of polyandry obtained in the simulations under the intrinsic male quality scenario (truncated normal distribution with mean = 0.44 and SD = 0.1667; Fig. S2C). The frequency of mating genotypes in the initial generation is 1:1 (polyandry:monandry)(SC competition; see main text). (A) Effects of mating frequency for polyandrous females. Number of patches = 10; Patch size = 10 females (i.e., Metapopulation size = 100); Dispersal rate = 1% per generation. (B) Effects of divisioning pattern of metapopulation. Polyandry mating frequency = 5; Metapopulation size = 100 (i.e. patch size = 100, 50, 20 or 10 for number of patches = 1, 2, 5, or 10, respectively); Dispersal rate = 1% (except for the 1 population case). (C) Effects of number of patches. Polyandry mating frequency = 5; Patch size = 10 (i.e., metapopulation size = 500, 200 or 100 for 50, 20 or 10 patch cases, respectively); Dispersal rate = 1%. (D) Effects of dispersal rate. Polyandry mating frequency = 5; Number of patches = 10; Patch size = 10 (metapopulation size = 100).

Intrinsic male quality scenario with continuous quality distribution

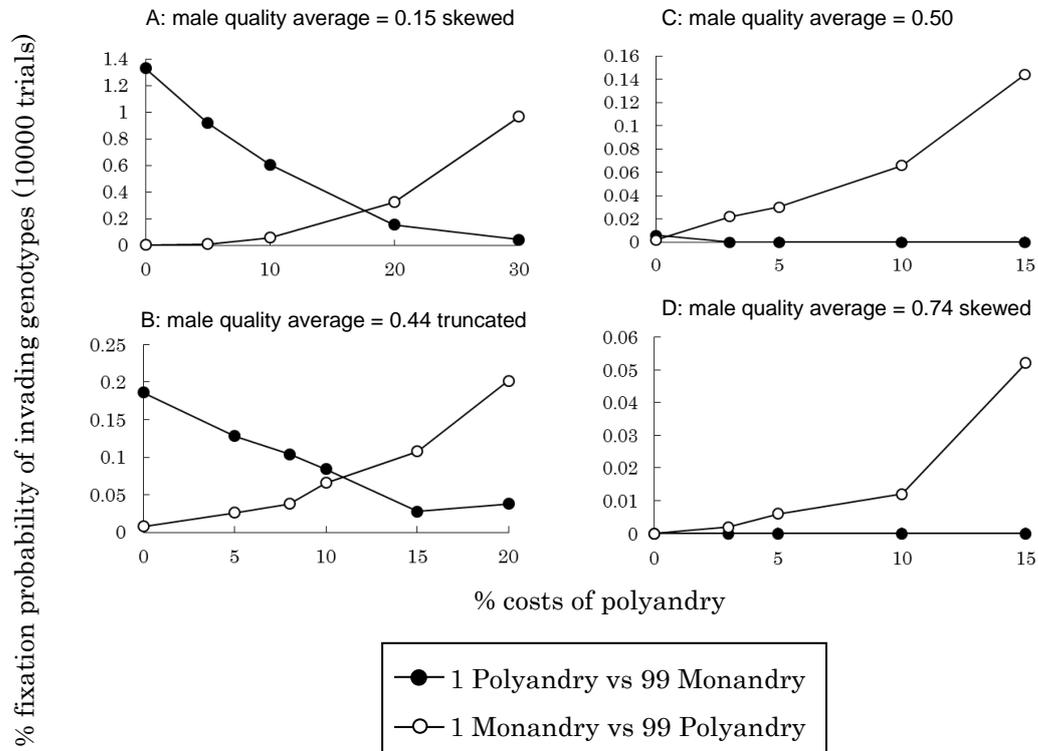


Figure S6: Probability (in %) of successful invasion by a mutant genotype (1 intruder genotype competing with 99 resident genotypes at generation 1) following 10000 trials under the intrinsic male quality scenario with various male quality distributions. Number of patches = 20; Patch size = 5 females (i.e. metapopulation size = 100); Mating frequency for polyandrous females = 5; Dispersal rate = 1% per generation.