weights of killed prey of up to 4.5 kg (ref. 3). It is not known how close to the nest these kills were made, as none was made within sight of the observer. But many studies of large African raptors include subjective assessments of the weights of animals collected and carried by birds over apparently long distances as being greater than the 1.7 kg suggested by Hedenström[1,3,4–8]. There are also accounts of eagles lifting animals presumably greater than 6 kg over short distances, and remains of animals found below nests suggest that this is not an uncommon occurrence[1,3,5,8,9].

Finally, as we have previously noted[1], we cannot discount the possibility that an extinct bird of prey, larger in body size than either the extant crowned or black eagle, was responsible for the fossil accumulations at Taung.

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Flower lifespan and disease risk

SIR — Ashman and Schoen[1,2] have proposed that plant fitness is an increasing function of pollination success, and a decreasing function of floral maintenance and production costs. They concluded that different pollination regimes, and different relative costs of flower construction and maintenance, determine optimal floral longevity for a species or population[1]. Here we suggest another factor relevant to the evolution of flower lifespans.

The pollinator-borne plant pathogen, the anther-smut *Microbotryum violaceum* (Pers.) Deml and Oberw, affects members of the pink family (Caryophyllaceae[3]) and is mainly transmitted by pollinators[4]. Successful infection requires several time-consuming processes. After a diploid teliospore, the dispersal unit of this basidiomycete fungus is carried to an uninfected plant by a pollinator, it undergoes meiosis, and two haploid sporidia of opposite mating type conjugate to produce the infectious dikaryon that invades the plant[5].

Flowers that remain open or on the plant longer are at higher risk of contracting this disease than are shorter-lived flowers, for at least two reasons. Longer-lived flowers have an increased chance of receiving infectious spores due to increased numbers of insect visits, as shown by gradual pollen accrual over time[6], and long-lived flowers offer the fungus a greater opportunity to colonize the plant. This is true both of flowers that are maintained open and those that have wilted but retain physiological connections with the plant. Ripening fruits of female and perfect flowers remain physiologically dependent on the plant. Male plants, on the other hand, can shed their flowers completely, losing nothing once their pollen is removed.

We measured floral longevity in full sib crosses of two dioecious species of the family Caryophyllaceae — the white and red campion, *Silene latifolia* and *S. dioica*. Both species harbour this disease, which may have been instrumental in the evolution of floral traits[7]. Male flowers remained on the plant for one or two days only (*S. latifolia*, 1.5 ± 0.05 days; *S. dioica*, 1.1 ± 0.05 days; mean ± s.e.); female flowers wilted soon after ovule fertilization, but retained their physiological connections for a much longer time (*S. latifolia*, 8.9 ± 0.27 days; *S. dioica*, 9.6 ± 0.40 days).

Female plants seem to be more prone to anther-smut in natural populations. Field surveys throughout Switzerland showed that females of these dioecious species had significantly higher infection rates than did males. In six naturally infected populations of *S. latifolia*, on average 7.4% of females, but only 4.2% of males, were infected (analysis of deviance, $F_{(1,4)}=36.03$; $P<0.005$). Similarly, in three populations of *S. dioica*, on average 3.6% of the female plants were infected compared with only 1% of the males ($F_{(1,2)}=24.88$; $P<0.05$). Higher infection of females in natural populations may have other explanations, such as higher mortality rates of infected male than infected female plants (A. Biere, personal communication), but females may be more susceptible to this fungal pathogen, simply because their flowers remain longer on the plants, while it may have been a selective force shaping the rapid phenology of male flowers.

A logical extension of this hypothesis is that populations or species with a higher risk of infection should have shorter optimal floral lifespans. Infection is known almost exclusively from perennial members of the Caryophyllaceae[3], so we predict that annual species in this family should have longer floral longevity, as will any populations that have evolved in the absence of disease. We also suggest that anther-smut disease may facilitate the evolution of dioecy in this plant family, by providing an additional advantage to individuals that lose their female function if they concomitantly acquire the ability to shed flowers early and hence reduce their risk of contracting this disease.

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SIR — A recent article[1] proposed a model of flower longevity based on the number of flowers per plant, the cost to produce and maintain a flower, and the male and female fitness accrual rates. I contend that potential problems in the authors' field study of 11 plant species have been underestimated. First, data were collected only during one season and at one location, and so may not adequately represent the typical responses of the species studied. Second, the longevity data were obtained without controlling pollinator access. I would challenge the proposition that floral longevity measured in the field is a good measure of maximum floral longevity in the absence of pollinators. Many flowers become unattractive to pollinators shortly after pollination[8,9], so, in the absence of any control on pollinator access, flower longevity may simply be a measure of how long it took the flower to become pollinated.

The percentage variance in reported data accounted for by the model (r^2) was only 0.40 (see figure), but when the flower longevity data are plotted against the time to 80% emptying of the anthers (from Fig. 2 of ref. 1), a much better fit is found ($r^2=0.94$; see figure). The authors appear to have determined, at least partly, pollinator activity in flowers whose life is pollination-dependent.

The effect of pollinators on floral longevity is mediated by a complex internal signalling system, in which ethene (ethylene) is a key molecule[9,10]. Exogenous ethene usually mimics the effects of pollination on flower petals[10,11]. Regulation of flower life by ethene has been found to be similar within plant families; for example, ethene can hasten perianth abscission or wilting[10]. Some families, by contrast, show flower wilting that is independent of ethene[10], and no examples are apparently known of flower life shortened by pollina-

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