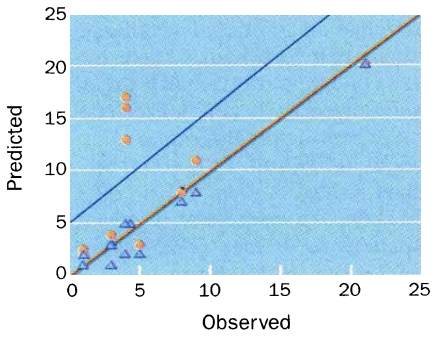

Relationship between predicted and observed flower longevity in 11 species. The red line is the ideal relationship. Longevity as observed in the field is plotted against predicted values; linear regression is shown as a blue or green line. Values predicted based on ref. 1 (circles, $r^2 = 0.40$; blue line), or based on 80% of emptying of the anthers (triangles, $r^2 = 0.94$; green line).

tion, in these families (for example, ref. 11). Ethene, furthermore, causes the loss of other cues for pollinators, such as colour[11] and permanent flower closure[12]. It is likely that the principal function of the regulation of flower longevity by ethene is to direct pollinator activity; avoidance of pollinated flowers increases the time spent by pollinators on virgin flowers[8].

Petal wilting or abscission strongly reduce visual 'advertising' by flowers, and are usually considered to conclude floral life. Permanent flower closure often precedes wilting or abscission, and also reduces the visual presence of flowers. It is unclear whether the authors also considered permanent closure to end flower life, but in the present context, I define floral life as the time from (first) opening to petal wilting or abscission.

Among the species investigated by Ashman and Schoen[1], at least five belong to families in which floral longevity is shortened by ethene[10]; one is from the Boraginaceae, a family known to show petal abscission[13] (hence their flower life seems ethene-regulated) and in which the flower life of *Borago* sp. is shortened by pollination[14], and another, *Oenothera flava*, is from a genus known to contain at least one species whose flower life is shorter after pollination[15].

The male and female fitness accrual rates, as used by the authors, are also a function of pollinator activity, as male fitness accrual rate was defined as "the rate at which pollen is disseminated and enters the pool of pollen that competes to fertilize ovules", and female fitness accrual as "the rate at which pollen is received to fertilize ovules".

More than a century ago, Kerner von Marilaun[16] suggested that flower life was related to plant and flower morphology, and to the chance of reproductive success. He noted that flowers with many anthers and much pollen tend to have a short life, whereas those with one anther are long-lived, particularly when their pollen is all in one package. Flower life in species with many flowers per individual also tends to be short, especially when these open sequentially over a long period. In contrast, flowers are relatively long-lived on plants that produce only one or a few blooms per individual. A long flower life was also predicted for species that experience few pollinator visits due to pollinator specialization, and for species with obligate outcrossing. These hypotheses have, however, never been properly tested.

An insight into these relationships will require flower longevity to be monitored at several sites and over several seasons. In addition, cessation of pollinator cues, such as colour (including the ultraviolet range) and permanent flower closure, along with wilting or abscission of the perianth, should be taken into consideration. In these studies, access by pollinators must be regulated to enable intrinsic flower longevity to be distinguished from the shorter flower life that may occur in the presence of pollinators.

Wouter G. van Doorn
Agrotechnological Research Institute (ATO-DLO), PO Box 17, 6700 AA Wageningen, The Netherlands

ASHMAN AND SCHOEN REPLY — Shykoff *et al.* suggest that because vulnerability to receipt of the anther-smut pathogen (as well as subsequent infection) increases as flowers remain open and physiologically connected to the plant, natural selection should operate to reduce floral longevity, and should do so more in males than in females, where longer-duration floral connections are required for completion of fertilization. We agree that these plant–parasite interactions could influence floral longevity in the species of *Silene* studied. The essential elements of interaction could, in fact, be easily accommodated by extending our optimal floral longevity model[1] to consider the rate at which the infectious spores arrive at flowers during the pollination process, and the extent to which disease infection initiated at one point in time diminishes future reproductive success. Moreover, because male and female reproductive functions are separated in dioecious species, there would be no constraint on male floral longevity (in the case of plants with bisexual flowers) brought about through possibly slower rates of fitness accrual of female fitness. Thus, factors that select for reduced floral longevity, such as increasing disease susceptibility with increasing floral lifespan, would be free to lead to divergence in optimal male and female flower longevities.

With regard to the comment by van Doorn, we have acknowledged elsewhere that post-pollination responses, such as ethylene production and floral-senescence response, can contribute to variation in floral longevity among and within species[17]. There is, however, a close correlation between floral longevity measured in flowers protected from pollination (MFL) and floral longevity in flowers of the same species that have been exposed to pollinators (RFL) (MFL = 0.88(RFL) + 1.72; $P < 0.0001$; $n = 23$ plant species), suggesting that using either measure of longevity would give results that are compatible with our model's predictions .

van Doorn's suggestion that variation in floral longevity can be accounted for primarily by floral senescence in response to ethylene production is at odds with the fact that flowers of different species differ in durability (and therefore longevity). For example, the flowers of morning glory (and other species), whether pollinated or not, senesce on the same day they are produced. It has been suggested that 'pollination-induced' senescence is mediated by production of ethylene by the stigma[10]. If such a mechanism is the main determinant of floral longevity, we would expect a strict correlation between completion of female function and floral longevity. van Doorn re-analyses our data and reports that the correlation between observed longevity and "time to 80% emptying of the anthers" is better than with floral longevities predicted by our model. However, when one uses the more appropriate measure of completion of female function, which we provided in our original data (that is, time to 80% receipt of pollen required for full seed set), the correlation is far less supportive of his hypothesis ($r^2 = 0.16$; $P > 0.2$).

We agree that it would be valuable to collect more data on floral longevity and its relationship to male and female fitness accrual from additional populations and species. We have, in fact, analysed floral longevity and correlated male and female fitness accrual in additional species, and the results support the predictions of our model[4].

Finally, there is a deeper issue. van Doorn argues for the primacy of a proximate explanation of floral longevity, rather than for an ultimate (evolutionary) explanation. This view that floral longevity variation is simply a physiological problem misses the issue of why plants have evolved mechanisms to control floral longevity. We believe that this ability came about from past selection to optimize floral longevity in the face of varying floral maintenance costs and varying rates of contributions of flowers to fitness. It would not be surprising or at odds with our model that cues such as ethylene production could be used by plants to realize such optimal floral longevities.

Tia-Lynn Ashman
Department of Biological Sciences, University of Pittsburgh, Pittsburgh, Pennsylvania 15260, USA
Daniel Schoen
Department of Biology, McGill University, Montreal, Quebec H3A 1B1, Canada