Table S1. RNAi of *acy-1*, *acy-2* or *acy-3* does not block fertility

RNAi treatment of wild-type hermaphrodites	% Fertility (*n*)
Control RNAi*	100% (20)
acy-1(RNAi)	100% (26)†
acy-2(RNAi)	100% (20)†
acy-3(RNAi)	100% (32)†
gsa-1(RNAi)‡	0% (20)

Control RNAi was L4440. RNAi was used for *acy-1* and *acy-2* because deletion mutations are lethal.

†No apparent defects in meiotic maturation or ovulation were observed by DIC microscopy. The identity of the clones was verified by DNA sequencing.

‡In our prior analysis, we concluded that it was unclear which of the four *acy* genes participates in the regulation of meiotic maturation (Govindan et al., 2006). We noted, however, that some *acy-1(ce2*gf*)* gain-of-function (gf) females exhibited slightly de-repressed meiotic maturation rates and thus incorporated an involvement of *acy-1* in our model (Govindan et al., 2006). The finding that *acy-4* is required for oocyte meiotic maturation and fertility is sufficient to explain the requirement for *gsa-1*. We cannot eliminate the possibility that *acy-1*, *acy-2* or *acy-3* might augment the essential function of *gsa-1* and *acy-4*. A weak involvement of *acy-1*, *acy-2* or *acy-3* might explain the weak suppression of *acy-4(lf)* infertility by phosphodiesterase inhibitors (see Fig. S2 in the supplementary material)