tides for nutrition or of templates for DNA repair), as long as most of the available DNA is from the same species and there is occasional recombination between incoming DNA and the chromosome. Indeed, if acquisition of nucleotides is the function of transformation then any DNA will serve, but the feedback between sequence abundance and receptor specificity will only occur if some of the incoming DNA recombines with the chromosome. The model has not, however, been rigorously examined, and is only one possible explanation for uptake specificity.

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SIR — Kirkpatrick and Ryan suggest[1] that there is growing support for the hypothesis that mating preferences evolve because of direct rather than indirect effects on female fitness. I believe that this conclusion is premature and stems, in part, from the way that tests have been attempted of one of the indirect effects, the Hamilton–Zuk[2] (or parasite) hypothesis.

Kirkpatrick and Ryan point out that many of the across-species comparative studies investigating the parasite hypothesis have found no relationship between the extremity of male display traits and the level of parasitism. They regard this as evidence that parasites are not important in the evolution of male displays. Indeed, they suggest that such comparative tests may be "the most fruitful way" to investigate the parasite hypothesis.

Previous authors[3,4] have pointed out problems with various aspects of these tests, such as the difficulties associated with objectively measuring brightness and in obtaining holistic measures of the levels of parasitism. There seems, however, to be a more fundamental problem with the tests, stemming from the basic assumption that the parasite hypothesis predicts that the absolute level of parasitism in a species should be correlated with the extremity of the displays in that species. The crux of the parasite hypothesis is that the exaggerated male traits allow females to choose among males within their population and mate with a more resistant male. Thus, there is no reason to assume that there will be an across-species relationship between the level of parasitism and the degree of exaggeration of the trait.

Imagine, for example, a population of birds that has a very high level of parasitism but a very low variance in the level of parasitism or very low heritability in host resistance to the parasite. In this population, the parasite hypothesis would predict the absence of exaggerated male display traits as females have very little to choose between males and, if heritability is low, nothing to gain by making a choice. Conversely, a population with low overall levels of parasitism but with high variance in parasite levels within the population and high heritability for resistance should have extreme display traits as females have much indirect fitness to gain by discriminating among males. So a more valuable across-species comparison would be to compare the extremity of the male trait with differences in the heritability and variance of resistance to parasites.

The relative importance of direct and indirect fitness effects in the evolution of exaggerated male traits can only be measured once these different factors have been more thoroughly examined in wild populations.

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Simplified gene nomenclature

SIR — Vertebrate genes encoding several distinct voltage-gated potassium channels have recently been identified in many laboratories, but the names assigned to them are frequently confusing. We propose a simplified nomenclature for one major family of these genes, based on sequence relatedness. This family comprises at least four subfamilies that represent vertebrate homologues of the four potassium channel genes in *Drosophila* (*Shaker*, *Shab*, *Shaw* and *Shal*). In the table a name is proposed for each gene. For example, the proposed name for MBK1/RCK1/RBK1 in our nomenclature is Kv1.1; that is, a K-channel gene (K) which is voltage dependent (v), and is the first identified member of the *Shaker*-related subfamily 1 (1.1).

The tissue origin of these genes will not be included in the name as many of these genes can be expressed in several tissues. The species origin of a gene (such as Kv1.1) will not be included in the name, but could be described, for example, as rat Kv1.1 or human Kv1.1. The list of genes will be updated periodically; genes published in the interim will have a 'w' prefix to signify 'nomenclature being worked out'. Genes encoding channels whose voltage-dependence has not been experimentally confirmed can be indicated with an empty parenthesis, the 'v' to be added when functional data are available. As additional subfamilies are discovered, they will be numbered based on their date of discovery.

Other voltage-dependent potassium channel genes that are structurally distinct from the *Shaker*/*Shaw*/*Shab*/*Shal* proteins (for example the Isk channels) could be added to the current list of genes as a separate family (for example, Ks1.1 or Kvs1.1). The proposed nomenclature could be used for other types of K^+ channels as well. Ligand-gated K^+ channels could be similarly named, the particular ligand (Ca, ATP) being substituted for v.

A list of references and the affiliations of the signatories will be provided upon request from K.G.C..

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A SIMPLIFIED NOMENCLATURE FOR A FAMILY OF VERTEBRATE VOLTAGE-DEPENDENT K^+ CHANNEL GENES

Property	*Xenopus*	Mouse	Rat	Human
Shaker-related subfamily 1				
Kv1.1	–	MBK1 MK1	RCK1 RBK1	HK1
Kv1.2	XSha2	MK2	RBK2 RCK5 NGK1	HK4
Kv1.3	–	MK3	RCK3 RGK5 KV3	HPCN3
Kv1.4	–	–	RCK4 RHK1	HK2 HPCN2 HK1
Kv1.5	–	–	KV1	HPCN1 HK2
Kv1.6	–	–	KV2 RCK2	HBK2
K()1.7	–	MK6 MK4	RK6	HaK6
Shab-related subfamily 2:				
Kv2.1	–	Mshab	DRK1	–
Shaw-related subfamily 3:				
Kv3.1	–	NGK2 Mshaw22	Kv4*	–
Kv3.2	–	MShaw12	RKShIIIA Rshaw12	–
K()3.3	–	Kv3.3 Mshaw19	–	–
Kv3.4	–	Kv3.4	Raw3	–
Shal-related subfamily 4:				
Kv4.1	–	Mshal1	–	–
Kv4.2	–	–	RK5	–

*Kv4 is an alternatively spliced version of NGK2.