Sexual Dimorphism in Body Size and the Origin of Sex-Determination Systems

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Abstract: Diverse sex-determining systems occur in vertebrates, including environmental sex determination (ESD), genetic sex determination (GSD) of type XX/XY (heterogametic males), and GSD of type ZZ/ZW (heterogametic females). The origins of the two genetic types are poorly understood. We use protected invasion theory to derive a model that generates testable predictions about the origins of the two genetic types from ESD. Protected invasion theory predicts biases in the evolutionary origins of new traits by focusing on the probability that a selectively favored trait will avoid loss by genetic drift when rare. We show that the theory makes predictions about the conditions under which XY or ZW systems are more likely to arise from an ancestral state of ESD. In particular, assuming that there is an average trend toward increasing body size in lineages, the origins of XY systems are predicted to be accompanied by increases in male : female body size ratio. In contrast, ZW systems are predicted to be accompanied by decreases in male : female body size ratio. We find support for these predictions in the form of a marked association among vertebrates between sex-determining system and body size dimorphism in paired comparisons independent of shared phylogeny.

Keywords: genetic sex determination, environmental sex determination, evolutionary origin, protected invasion theory, comparative analysis, size dimorphism.

Introduction

Vertebrate sex-determining systems are diverse (fig. 1). Sex can be determined by the physical or social environment, by genes, or by both environment and genes operating together. Genetic sex determination (GSD) can be either of the XX/XY type (male heterogamety, hereafter XY) or the ZZ/ZW type (female heterogamety, hereafter ZW). More than 2,000 species, including some sharks and rays, have been shown to have GSD, and many others have 1 : 1 offspring sex ratios suggestive of GSD (Maddock and Schwartz 1996). Birds and snakes exclusively have type ZW (Janzen and Paukstis 1991), mammals exclusively have type XY (Graves 2008), and teleost fishes, urodele amphibians, and anuran amphibians all include both types (Hillis and Green 1990; Klinkhardt et al. 1995; Schmid and Steinlein 2001; Devlin and Nagahama 2002).

The phylogenetic distribution of these sex-determining systems and types is striking. Some vertebrate groups, such as snakes, birds, or mammals, are very homogeneous. In these groups, all sex determination is genetic, and it is always of the same (or very similar) type. Others, such as amphibians, scincomorph and gekkomorph lizards, and teleost fishes, are heterogeneous. In these groups, variation in the type of GSD (XY or ZW) is seen within families, genera, and even species. Across all vertebrates, the phylogenetic distribution of sex-determining systems suggests multiple independent evolutionary origins and reversals (Mank et al. 2006; Ezaz et al. 2009).

There have been numerous reviews of sex-determining systems in vertebrates aimed at understanding their evolution, including more than a dozen since 2000 (e.g., Kraak and Pen 2002; Mank et al. 2006; Graves 2008). Understanding the phylogeny of sex-determining systems requires two kinds of investigation. One type addresses how there can be so much diversity by uncovering the genetic changes underlying origins of new systems and transitions from one system to another. The other type addresses why there is so much diversity by investigating the selective pressures and fitness consequences of the different systems (Trivers 1988; Werren and Beukeboom 1998). Considerable progress has been made using population genetics and other tools to model the sequence of changes that would have to occur for XY GSD to change to ZW GSD and vice versa (Bull 1983; Quinn et al. 2011). This has helped explain how such changes have been able to occur multiple times in vertebrates—that is, how such diversity has been genetically possible (Werren and Beukeboom 1998). Models of the evolution of one GSD type from another have highlighted the potential roles of sex-ratio selection or sex-antagonistic selection (Bull 1983; Uller et al. 2007; Pen et al. 2010; van Doorn and Kirkpatrick 2010). Progress has also been made in explaining why the chro-
Ray-finned fishes: ESD, XY, ZW

Amphibians: XY, ZW

Mammals: XY

Snakes: ZW

Lizards: XY, ZW, ESD

Turtles: ESD, XY, ZW

Crocodiles: ESD

Birds: ZW

**Figure 1:** Diversity in sex-determining systems among and within major vertebrate clades. ESD = environmental sex determination; XY = genetic sex determination of the XX/XY type (male heterogamety); ZW = genetic sex determination of the ZZ/ZW type (female heterogamety).

A chromosome with the sex-determining gene(s) on it tends to become reduced in size (i.e., why sex chromosomes become heteromorphic), why it might be difficult for GSD type to change once this happens, and why strong chromosomal heteromorphism leads to fixation of XY or ZW, as in mammals, snakes, and birds (Bull 1983; Charlesworth 1991; Rice 1994; Graves 1995; Charlesworth et al. 2005). Some authors have mapped known sex-determining systems onto modern phylogenies, allowing reconstruction of the most likely ancestral states (e.g., Hillis and Green 1990; Kraak and Pen 2002; Janzen and Phillips 2006). Theories for the evolution of temperature-dependent environmental sex determination (ESD) are beginning to receive empirical support (Janzen and Phillips 2006; Crews and Bull 2008; Warner and Shine 2008).

What is still inadequately explained is what selective pressures determine which GSD system will evolve. What are the evolutionary origin mechanisms that bias a lineage toward GSD of one type over another? That evolutionary change happens and happens readily is now less of a genetic mystery. But what accounts for when and where the changes have occurred in the evolution of vertebrates? Explaining the current distribution of GSD types in vertebrates continues to represent a major challenge, one that has seldom been addressed (see next section for the important exceptions, i.e., the work of Kraak and de Looze [1993] and Kraak and Pen [2002]).

This article presents a model derived from protected invasion theory (Reeve 1993; Reeve and Shellman-Reeve 1997) that generalizes the predictions of Kraak and de Looze (1993) about the evolution of the two GSD types from ESD. The model generates a prediction that GSD type will be associated with sexual size dimorphism. This is tested with reference to those vertebrates most likely to have an ESD origin for their sex-determining systems.

**Prior Models for the Evolution of GSD from ESD**

Since Bull’s (1983) comprehensive review of models for the evolution of GSD from ESD, few additional formal models have been proposed (Kraak and de Looze 1993; Kraak and Pen 2002; Van Dooren and Leimar 2003; Pen et al. 2010; Quinn et al. 2011). The first and only extant model that explicitly predicts an association with body size dimorphism is that of Kraak and de Looze (1993).

In reptiles, ESD by temperature-dependent sex determination (TSD) and body size dimorphism are associated, such that differentiation of females at higher incubation temperatures is associated with larger female body size relative to males (Type I TSD, as in many turtles), whereas differentiation of males at higher incubation temperatures is associated with larger male body size relative to females (Type II TSD, as in Crocodylia and *Eublepharis* lizards; Deeming and Ferguson 1991; Ewert et al. 1994; Viets et al. 1994). Building on Charnov and Bull’s (1977) model for explaining ESD as a consequence of a sex difference in the relation between body size and fitness and Mittwoch’s (1971) mechanistic proposal that rate of gonadal growth determines gonadal sex, Kraak and de Looze (1993) proposed that in ESD species, a chromosome with a mutation causing females to grow faster would eventually become a W (i.e., a ZW system would be favored by a female size advantage) and one with a mutation causing males to grow faster would become a Y (i.e., an XY system would be favored by a fitness advantage of male size). A
multilocus formal version of the model was later developed (Kraak and Pen 2002).

The empirical evidence presented in Kraak and de Looze (1993) for their predicted relationship between a sex difference in size advantage and sex-determining system was that in four species of reptiles with ESD (three turtles and the American alligator), H-Y antigen is detected in the larger sex. H-Y antigen is a male histocompatibility antigen that in mammals is produced by a gene on the sex-determining Y chromosome. However, H-Y antigen is now known to be a result, not a cause, of the sexual phenotype (Wolf 1998). Size dimorphism in vertebrates with GSD was not part of the evidence. Thus, the empirical evidence to date has been very limited.

The Kraak and de Looze (1993) model is based on the mechanistic idea that rate of embryonic growth determines gonadal differentiation (Mittwoch 1971), and it would predict that ZW systems will tend to be characterized by higher female-to-male size dimorphism than XY systems if embryonic growth rate reflects adult size. Thus, their model focuses on the origin of a specific mechanism of sex determination (alleles causing differential embryonic growth) that might generate an association between sexual size dimorphism and type of GSD system if the ancestral Y and W chromosomes increase growth rate (which is presumably favored by selection). The direction of the association that they predict is reversed, however, if the mutation reduces growth rate and reduced growth rate is selectively favored. We consider patterns of selection that would generate the same association regardless of the precise mechanism and show that the association between GSD type and sexual dimorphism will be produced generally if body size tends to increase over the course of evolution, an assumption consistent with Cope’s rule (Kingsolver and Pfennig 2004). Thus, we show that the association between sex chromosome system and sexual dimorphism predicted by Kraak and de Looze (1993) is even more general than implied by their model.

Below, we construct a simple mathematical model of the evolutionary origin of GSD systems from ESD. In particular, we adopt the perspective from protected invasion theory (Reeve 1993; Reeve and Shellman-Reeve 1997) that a favored genetic variant that is especially likely to survive random loss through genetic drift when rare is most likely to be responsible for the origin of its associated novel phenotype. For example, if two different variants conceivably could confer fitness advantages, then the one with the stronger selective advantage when rare will be especially likely to result in the origin of a novel trait, creating an origin bias that will be detectable phylogenetically. We show that the resulting model predicts a strong relationship between sexual size dimorphism and GSD system and test the prediction with data on GSD systems in vertebrates.

Protected Invasion Theory and the Evolution of GSD from ESD

If bearers of a new favored mutant, allele A, have a mean fitness \( w_A \), and the mean fitness of all individuals in the population is \( w_{av} \) (\( av \) = average), then the increase in \( A \)'s frequency in the next generation due to selection if it has a starting frequency \( p \) is given by

\[
\frac{p(w_A - w_{av})}{w_{av}}.
\]

Clearly, factors that increase (1) will increase the mutant allele’s resistance to random loss by genetic drift when rare (i.e., \( p \) is small).

In the case in which the allele is an ancestral sex-determining allele, it is first easy to show that a dominant gene will be far better protected from random loss than a recessive gene. This is important because it is possible to develop an XY (or ZW) system through either the spread of a dominant male-producing gene on an incipient Y chromosome (or a dominant female-producing gene on an incipient W chromosome) or through the spread of a recessive female-producing gene on an incipient X chromosome (or a recessive male-producing gene on an incipient Z chromosome).

To show this, we assume that a male-producing gene on an incipient Y chromosome is dominant. In this case, \( w_A \) will be equal to \( f(x + b) + (1 - f)x \), where \( f \) is the probability that the allele is expressed in an individual that would have become female in the absence of the gene’s effect, \( x \) is the mean number of offspring produced by a mating in the general population, and \( b \) is the additional number of offspring produced by the mutant male. The mean fitness of all individuals in the population is virtually equal to \( f(x) + (1 - f)x = x \). Expression (1) thus becomes equal to \( pfb/x \) for the dominant allele. The case of a recessive female-causing allele is quite different. In this case, the allele has a selective advantage only when expressed in a male and when this male has an A allele at the homologous locus, yielding \( w_A = p(1 - f)(x + b) + (1 - p)(1 - f)(x + b) \). In this case, (1) becomes equal to \( p'(1 - f)bx \), which is much smaller than the expression for the dominant allele for small \( p \). Thus, we can conclude that there should be, from protected invasion theory, a strong bias toward origin of XY systems from the spread of dominant Y alleles over the spread of recessive X alleles. Parallel logic leads us to restrict our attention to dominant W alleles in the origin of ZW systems. A focus of dominant
alleles is also consistent with the consensus in the literature on the genetics of sex determination (Bachtrog et al. 2011).

Once we restrict our focus to dominant alleles, we can now turn our attention to origin biases that result from different patterns of selection on male and female body size. We have to take into account the statistical tendency for lineages to increase rather than decrease in body size over evolutionary time, which appears to be driven in vertebrate taxa by consistent within-population selection for larger body size (Kingsolver and Pfennig 2004). Such consistent selection for larger body size would be expected from coevolutionary size races between predators and prey or between conspecific or heterospecific competitors.

Given that, on average, selection in an evolving population will tend to favor larger males or females, it is possible to examine associations between type of GSD system and sexual dimorphism that would be predicted by protected invasion theory. We must consider the outcomes of selection on a dominant $A$ allele that causes bigger males or females for all possible conditions of ancestral size dimorphism and for both the case that selection favors bigger males and the case that selection favors bigger females. We show the latter combinations and their associated outcomes in table 1.

In the first combination, females are initially larger than males, the $A$ allele causes a female to develop into a large male instead of a large female, and selection favors an increase in the relative size of males (table 1). Note that in this theory, in contrast to that of Kraak and de Looze (1993), we are assuming that the mutant allele changes the sexual characteristics but the body size can still be set by the local environmental stimuli (e.g., temperature). In other words, size and sexual characteristics in ESD might respond independently to the environmental stimuli, with the mutant $A$ allele inducing the sexual characteristics without necessarily inducing the larger body size. The mutant $A$-containing males are thus larger on average than wild-type males, and since selection favors larger males, the $A$ allele spreads, generating a primordial $Y$ chromosome. After spread of the allele to some intermediate frequency, selection on modifier loci would be expected to genetically couple the body size increase to the sex change induced by the $A$ allele.

In the last combination, males are initially larger than females, the $A$ allele causes a male to develop into a large female instead of a large male, and selection favors an increase in the relative size of females. The mutant $A$-containing females are thus larger on average than wild-type females, and since selection favors larger females, the $A$ allele spreads, generating a primordial $W$ chromosome. After spread of the allele to some intermediate frequency, selection on modifier loci would be expected to genetically couple the size increase to the sex change induced by the $A$ allele.

In the combination in the second row, an $A$ allele causes an individual that otherwise would have been a female to become male, and such a male, because it is smaller than the average ESD-produced male, will have reduced fitness ($A$ is disfavored). By a similar logic, in all of the other remaining combinations, the mutant allele is either neutral or selectively disfavored and thus obviously highly vulnerable to loss.

This model for biases in the origin of GSD with respect to sexual dimorphism does not require that the dimorphism is reversed. It is sufficient that the dimorphism is reduced, with the sex produced by the dominant sex allele

<table>
<thead>
<tr>
<th>Table 1: Different outcomes of selection on rare, dominant $A$ alleles producing males (M) or females (F) for different combinations of ancestral dimorphism in an environmental sex determination (ESD) system and for different patterns of new sex-specific selection pressures</th>
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<tbody>
<tr>
<td>Ancestral dimorphism in ESD system</td>
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<td>F &gt; M</td>
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Note: For ancestral sexual dimorphism states and sex-specific selection patterns, we consider only strict inequalities for simplicity, without loss of generality, since exact equality is likely rare. "Selection favors bigger males" means new selection pressures (possibly different from the ancestral ones, due to changed environments) favor relatively large males and relatively small females; "Selection favors bigger females" means the reverse. "$A$ disfavored" refers to cases in which the $A$ allele causes reduced fitness. For example, in the case described by the second row, an $A$ allele causes an individual that otherwise would have been a female to become male, and such a male, because it is smaller than the average ESD-produced male, will have reduced fitness.
becoming closer to the opposite sex in size (table 1). This is because this dominant allele spreads when selection starts favoring an increase in the size of a sex that is initially smaller than the opposite sex. One way to achieve this is via the mutant switching the sex of an individual destined to become the bigger sex under the old ancestral ESD rule. The dominant mutant spreads even if the mutant individual is not bigger than the bigger sex of the old ESD rule, because it is bigger than the sex it would have been under that rule.

The only two GSD origins permitted in table 1 thus predict that there should be a statistical association between sexual dimorphism and type of GSD. In particular, transitions to XY systems should tend to be accompanied by increases in the size of males relative to females, and transitions to ZW systems should tend to be accompanied by increases in the size of females relative to males. This is the central prediction that we sought to test.

### XY and ZW GSD Systems as Causes versus Consequences of Sex-Specific Phenotypes

Once incipient XY and ZW systems are established, subsequent selection is predicted by several recent models to reduce the rates of recombination between chromosomes containing the sex-determining genetic locus (Charlesworth 1991; Rice 1994), leading eventually to the emergence of true, that is, nonrecombining, sex chromosomes. As the sex chromosomes become more strongly differentiated, protected invasion effects will become increasingly potent in causing phenotypic divergence between the sexes. In particular, the homogametic sex will be especially likely to move from a previously sexually monomorphic phenotypic optimum to a new, sex-conditioned phenotypic optimum such as that associated with parental or alloparental care (Reeve and Shellman-Reeve 1997) or elaborated gametic investment such as exhibited in viviparity.

This creates a potential problem for testing our model. The new sex-conditioned phenotypic optimum might involve the very same phenotypic attributes that were earlier identified as predisposing the origination of XY or ZW sex-determination systems. For example, increases in the size of males relative to females were earlier shown to predispose the origination of XY systems from an ancestral state of ESD, but larger males might also arise as a consequence of the establishment of a differentiated XY genetic system. This ambiguity in the direction of the causal arrow raises a problem for empirical tests of our predictions about the conditions favoring alternative GSD systems: Did the sex-limited phenotypes associated with different genetic systems cause or result from those systems via protected invasion effects? What kinds of tests could differentiate between those two possibilities, only one of which is consistent with the model?

One possible way around this causal ambiguity would be to compare sex-conditioned phenotypes between sister taxa that possess versus lack GSD. For example, if male size increase predisposed an XY system, then lineages that exhibit an XY system as a newly derived state should exhibit larger males along with sister taxa lacking the XY system (the latter taxa exhibiting the plesiomorphic state of ESD). In contrast, if larger males resulted from an XY system, then neither larger males nor an XY system should be exhibited by the sister taxa, suggesting that larger males evolved after the establishment of the XY system. No relationship between sexual size dimorphism and kind of GSD would invalidate the protected invasion hypothesis. A test of that kind would require resolved phylogenies that do not yet exist.

An alternative approach to resolving the causal ambiguity is to see whether the hypothesized predisposing phenotype is associated with GSD type in clades where GSD ancestry is likely. If body size dimorphism is causal, then the association should be found only where an ESD ancestry is possible. If it is a consequence, then it should be found regardless of ESD versus GSD ancestry. Therefore, in testing the model, we will begin with a test of the central prediction for ESD ancestry, where body size dimorphism is causal, and then in a second test ask whether the same association is found when there is unlikely to be ESD ancestry.

### Testing the Model with Comparative Evidence from Vertebrates

**Prediction: XY Systems Will Be Associated with a Relatively Larger Ratio of Male to Female Body Size than ZW Systems**

Testing this prediction requires an analysis that asks whether there is a statistically significant association between the two characters (GSD type and sexual size dimorphism) independent of shared phylogeny. Methods for such a test are usually designed to address a problem of nonindependence of data points, require branch lengths for the underlying phylogeny, or assume continuous, rather than categorical, character variables. For our data set, however, some of the different pairs of taxa come from different regions of vertebrate phylogeny (different families, orders, and classes) and include many regions with unresolved phylogenies. Each pair diverged a very long time ago from every other pair. Given how labile sex determination and body size dimorphism appear to be, and how many separate origins there have been, the pairs and their GSD and size-dimorphism states can safely be as-
ESD ancestry has been proposed for squamates generally (Felsenstein 1985; Möller and Birkhead 1992; Stockley et al. 1997). This simple method is statistically conservative (has low power) and has the advantage that it makes no assumptions about the underlying phylogeny.

This method was applied to the three vertebrate groups that (a) contain both GSD types and (b) are most likely to be derived from an ESD ancestral state: the Teleostei, Chelonia, and (within the Sauria) Gekkota. With respect to teleosts, some form of ESD has been found in most families that have been studied and in at least 11 orders, including the somewhat basal Anguilliformes, and new and taxonomically widespread reports of ESD are increasing (Baroiller and D'Cotta 2001; Valenzuela and Lance 2004). Forms of ESD in which environment interacts with genes to determine sex are very common and also taxonomically widespread (Baroiller and D'Cotta 2001; Valenzuela et al. 2003). Pairs were formed with species in clades where ESD is also present. Species with known GSD of different types were regarded as forming pairs if they were in the same family or (if there were no other family members with known GSD type or no within-family type diversity) the same order. Some lizard groups also contain mixtures of genetic and temperature sex determination (Valenzuela et al. 2003; Sarre et al. 2004; Organ and Janes 2008), and an ESD ancestry has been proposed for squamates generally and the Gekkota in particular (Janzan and Paukstis 1991; Donnellan et al. 1999; Pokorna and Kratochvil 2009; but see Janzen and Phillips 2006). Among turtles, most species that have been studied have TSD, and the phylogenetic position of those with GSD supports the evolution of GSD from ESD (Janzan and Paukstis 1991).

Information about GSD type was obtained from reviews (Hillis and Green 1990; Janzen and Paukstis 1991; Klinkhardt et al. 1995; Schmid and Steinlein 2001; Devlin and Nagahama 2002) and by using Web of Science (ISI Web of Knowledge, Thomson Scientific, last search February 2011). GSD types were assigned to two categories, XY and ZW. The XY (XX/XY) category includes multiple sex chromosome systems (e.g., XXX/XXXY), and ZW (ZZ/ZW) includes analogous variants. Information about the adult sexual size dimorphism of those species with known GSD type was then obtained by using Web of Science (ISI Web of Knowledge, Thomson Scientific, last search February 2013) and a Google (http://www.google.com/) web search (to locate journal articles missed by the Web of Science search function). For some matched pairs, no information about body size dimorphism could be found for one or both of the pair members. These pairs could not be included in the analysis.

Size-dimorphism states are based on adult body length (standard length for teleosts, carapace length for turtles, snout-vent length for lizards). Mean sizes of adult males and females were located whenever possible and used to determine the sexual size dimorphism (SSD) index recommended by Lovich and Gibbons (1992) and used throughout Fairbairn et al. (2007). SSD is calculated as (larger sex/smaller sex) − 1 and is made negative if males are larger and positive if females are larger. Thus, no size difference equals 0, and SSD is symmetrical around 0. Where multiple reports gave different means or SSDs for the same species, the mean SSD was used. Where a set of multiple species was the match, the mean SSD for the set was used in the analysis. In a few cases, only size ranges or maximum sizes could be found and were used instead of means.

The results are shown in table 2. There are 16 matched pairs of species that differ in GSD type for which size dimorphism information could be located. Of these 16 pairs, 14 differ in SSD in the predicted direction, with lower SSDs (relatively larger males) in the XY species (two-tailed \( P = .004 \)). (Recall that, as explained earlier, complete reversal of size dimorphism is neither predicted nor required under the model.) Even minus the two pairs (pairs 11 and 12) where SSDs based on means have to be compared with SSDs based on maxima, which might bias the outcome, the predicted result is still statistically significant (\( P = .013 \)).

**Generality of the Association between GSD Type and Body Size Dimorphism**

The model predicted this association for species with an ESD ancestry. The association would not be predicted to extend to vertebrates representing transitions from one GSD type to the other. It is possible to do the same kind of analysis as in table 2 using only comparisons likely to have ESD ancestry. One group of species suitable for such an analysis consists of amphibians. It has been well established that all amphibians have GSD, and ancestral reconstruction strongly supports GSD ancestry (Hillis and Green 1990; Hayes 1998). Another group consists of closely related pairs of fish species that are discussed in the sex-determination literature as representing transitions from one GSD type to another. The results for these two groups are shown in table 3. In this analysis of species with confirmed or likely GSD ancestry, containing 13 comparisons (6 for teleosts and 7 for amphibians), there was no statistically significant association in the predicted direction between GSD type and sexual size dimorphism.
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against this possibility is that the association was only statistically significant for animals that might have had an ESD ancestry. Another is that sex differences in body size are not highly correlated with intersexual selection (e.g., with the extreme male traits that females prefer; Fairbairn et al. 2007). Degree and direction of body size dimorphism are a result of multiple selective forces, some acting on absolute sizes of males, others acting on absolute sizes of females, and yet others whose fitness consequences will depend on the relative sizes of the two sexes. Intrasexual selection is often more important than intersexual selection (Shine 1979, 1989; Reeve and Fairbairn 2001; Monnet and Cherry 2002). “Fancy” males aren’t necessarily larger than the females choosing them. Sexual selection produces smaller as well as larger males, as in guppies where males are small but colorful. Larger size may benefit males that engage in combat, but larger size may benefit female fecundity even more. Once a GSD type has evolved from ESD and sex chromosomes have become heteromorphic, however, the sex chromosome architecture could affect the likelihood that intersexual selection could alter any pre-existing size dimorphism. This would also be the case when a GSD type has evolved from another GSD type. In table 3, more than half the pairs differed in the opposite direction from the prediction for the evolution of GSD from ESD. As more size dimorphism information becomes available, it will be possible to see whether this association (which is not currently statistically significant) continues and whether it reflects a causal influence of sex chromosome architecture on the evolution of sexual size dimorphism.

The model traces the evolution of GSD via a dominant allele or alleles acting in the heterogametic sex. Among vertebrates, there are only a few species, most of them mammals, for which there is sufficient evidence to tell whether GSD is produced by a dominant allele as opposed to a balance between several alleles. In mammals, evidence points to a dominant testis-determining gene (Sry) on the Y chromosome (Mittwoch 1996; Vaiman and Pailhoux 2000). In the African clawed frog Xenopus laevis, a ZW species, there appears to be a dominant female-determining gene (Yoshimoto and Ito 2011). A male sex-determining gene (DMY) analogous to the mammalian Sry was identified in the teleost Oryzias latipes, but it appears that a similar role for DMY does not generalize to other teleosts (Matsuda 2005; Takehana et al. 2007; Tanaka et al. 2007). Interestingly, two other Oryzias species (O. hubbsi and O. javanicus) do not have the same type of sex determination as O. latipes but instead have ZW systems (Takehana et al. 2007). No size dimorphism information appears to be available for those ZW species to enable adding this comparison to table 3 (GSD-to-GSD transitions). Nonetheless, medakas have excellent potential for illuminating the se-

(P = .092). Only four pairs were consistent with the prediction, and instead more than half the pairs differed in the opposite direction. This further suggests that the association in table 2 is related to the evolution of GSD from ESD and not some other property of the two GSD types.

General Discussion

To our knowledge, this is the first attempt to understand the phylogenetic distribution of vertebrate genetic sex-determining systems by using a formal model to generate predictions that are then tested empirically using a comparative statistical method. Support for the model for the evolution of GSD from ESD came from the analysis of sexual size dimorphism. GSD type was associated with body size dimorphism independently of shared phylogeny, such that in pairs of related taxa, those with an XY system had relatively larger males (compared to females) than those with a ZW system. Furthermore, this association was only statistically significant for pairs that could have some ESD ancestry. The prediction of an association between GSD type and sexual size dimorphism in the evolution of GSD from ESD was first presented by Kraak and de Looze (1993). We show that the prediction is more general than implied by their specific mechanistic explanation for the evolution of GSD from TSD.

Body size dimorphism in the model is not a determinant of GSD type or an invariable predictor that a particular type will evolve. Rather, it is one of several factors that could tip the balance to enable a mutation for a new GSD type to spread instead of become extinct. Also, a change in body size dimorphism is not sufficient for a change in GSD type, for example, if the sex chromosomes have become highly heteromorphic (gone to fixation). Thus, body size dimorphism is neither necessary nor sufficient, although it may be strongly predisposing.

In the model, XY or ZW systems are consequences of selection for larger male or female body size, not necessarily causes of the evolution of body size dimorphism. One way that the sex-determining system could cause body size dimorphism is if body size is sex linked. Some vertebrates such as poeciliid fishes have sex-linked body size (Roldan and Comendio 1999; Lindholm and Breiden 2002). Pleiotropy or sex linkage does not seem to account for GSD vertebrates generally, however, because the association between which sex is heterogametic and which is larger is far from perfect. Nonetheless, once male or female heterogamety with heteromorphic sex chromosomes has evolved, the two GSD types have different consequences for the evolution of extreme male traits under female choice (Reeve and Pfennig 2003). Could this have produced the association in table 2 rather than the direction of causation posited by the model? One argument
Table 2: Independent paired comparisons test of the model: sexual size dimorphism (SSD) of matched pairs differing in genetic sex determination (GSD) type

<table>
<thead>
<tr>
<th>Order or family</th>
<th>Species with XX/XY GSD</th>
<th>SSD</th>
<th>Species with ZZ/ZW GSD</th>
<th>SSD</th>
<th>XY &lt; ZW for this pair?</th>
<th>Sources</th>
</tr>
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<tbody>
<tr>
<td><strong>Teleost fishes:</strong></td>
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<tr>
<td>1 Anguilliformes</td>
<td>Gymnothorax eurostus</td>
<td>-0.46</td>
<td>Anguilla anguilla</td>
<td>0.85</td>
<td>+</td>
<td>1</td>
</tr>
<tr>
<td>2 Cyprinidae</td>
<td>Cyprinus carpio</td>
<td>0.03</td>
<td>Scardinius erythrophthalmus</td>
<td>0.01</td>
<td>+</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Carassius auratus</td>
<td>0.18</td>
<td>Leuciscus pyrenaicus</td>
<td>0.22</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vimba vimba</td>
<td>0.05</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.09</td>
<td>Mean</td>
<td>0.12</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Characiformes</td>
<td>Hoplias malabaricus</td>
<td>-0.01</td>
<td>Triportheus guentheri</td>
<td>0.02</td>
<td>+</td>
<td>3</td>
</tr>
<tr>
<td>4 Loricariidae</td>
<td>Hypostomus ancylostoides</td>
<td>0.08</td>
<td>Loricariichthys platymetopon</td>
<td>0.05</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>5 Claridae/Heteropneustidae</td>
<td>Heteropeustes fossilis</td>
<td>0.07</td>
<td>Heterobranchus longifilis</td>
<td>0.63</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td>6 Salmoniformes/Esociformes</td>
<td>Oncorhynchus (7 spp.)</td>
<td>-0.04–0.10</td>
<td>Esox masquinongy</td>
<td>0.16</td>
<td>+</td>
<td>6</td>
</tr>
<tr>
<td>Mean</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 Poeciliidae</td>
<td>Limia perugiae</td>
<td>0.28</td>
<td>Gambusia nobilis</td>
<td>0.48</td>
<td></td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Gambusia punticulata</td>
<td>0.31</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Gambusia hurtadoi</td>
<td>0.22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.34</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 Gasterosteidae</td>
<td>Pungitius pungitius</td>
<td>0.06</td>
<td>Apeltes quadracus</td>
<td>0.17</td>
<td>+</td>
<td>8</td>
</tr>
<tr>
<td>9 Eleotridae</td>
<td>Dormitator maculatus</td>
<td>-0.47</td>
<td>Eleotris pisonis</td>
<td>-0.21</td>
<td>+</td>
<td>9</td>
</tr>
<tr>
<td>10 Gobiidae</td>
<td>Gobionellus shufeldti</td>
<td>-0.10</td>
<td>Boleophthalmus hodaerti</td>
<td>-0.02</td>
<td>+</td>
<td>10</td>
</tr>
<tr>
<td>11 Osphronemidae</td>
<td>Betta splendens</td>
<td>-0.21</td>
<td>Colisa fasciata</td>
<td>-0.11</td>
<td>+</td>
<td>11</td>
</tr>
<tr>
<td>12 Cynoglossidae</td>
<td>Symphurus plagiusa</td>
<td>0.02, 0.06</td>
<td>Cynoglossus puncticeps</td>
<td>0.18?</td>
<td>+</td>
<td>12</td>
</tr>
<tr>
<td>13 Pleuronectidae</td>
<td>Pseudopleuronectes yokohamae</td>
<td>0.30</td>
<td>Pleuronectes platessa</td>
<td>0.15</td>
<td>-</td>
<td>13</td>
</tr>
<tr>
<td>14 Scophthalmidae/Bothidae</td>
<td>Bothus podas</td>
<td>-0.17</td>
<td>Scophthalmus maximus</td>
<td>0.36 (weight)</td>
<td>+</td>
<td>14</td>
</tr>
<tr>
<td><strong>Turtles:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 Bataguridae</td>
<td>Siebenrockiella crassicollis</td>
<td>0.09</td>
<td>Kachuga smithii</td>
<td>1.1</td>
<td>+</td>
<td>15</td>
</tr>
<tr>
<td><strong>Geckos:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16 Gekkonidae</td>
<td>Gekko japonicus</td>
<td>0.03</td>
<td>Gekko hokouensis</td>
<td>0.08</td>
<td>+</td>
<td>16</td>
</tr>
</tbody>
</table>
Table 3: Independent comparisons test of the generality of the association between genetic sex determination (GSD) type and sexual size dimorphism (SSD): matched pairs differing in GSD type that are not predicted by the model to show a significant association

<table>
<thead>
<tr>
<th>Pair</th>
<th>Family</th>
<th>Species with XX/XY GSD</th>
<th>SSD</th>
<th>Species with ZZ/ZW GSD</th>
<th>SSD</th>
<th>XY&lt;ZW for this pair?</th>
<th>Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Clariidae</td>
<td><em>Clarias fuscus</em></td>
<td></td>
<td><em>Clarias batrachus</em></td>
<td>.33 (weight)</td>
<td>+</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>Poeciliidae</td>
<td><em>Gambusia holbrooki</em></td>
<td>.35, .67</td>
<td><em>Gambusia affinis</em></td>
<td>.28, .65</td>
<td>−</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Poeciliidae</td>
<td><em>Poecilia reticulata</em></td>
<td>.26</td>
<td><em>Poecilia sphenops</em></td>
<td>.23</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Poecilia latipinna</em></td>
<td>.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Poeciliidae</td>
<td><em>Xiphophorus andersi</em></td>
<td>.22</td>
<td><em>Xiphophorus helleri</em></td>
<td>−.07</td>
<td>−</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>Cichlidae</td>
<td><em>Oreochromis niloticus</em></td>
<td>−.07, −.01</td>
<td><em>Oreochromis aureus</em></td>
<td>−.08, −.06</td>
<td>−</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Oreochromis karongae</em></td>
<td>.03</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>6</td>
<td>Cichlidae</td>
<td><em>Tilapia zillii</em></td>
<td>−.08</td>
<td><em>Tilapia mariae</em></td>
<td>−.18</td>
<td>−</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>Proteidae/Sirenidae</td>
<td><em>Necturus beyeri</em></td>
<td>.09</td>
<td><em>Siren intermedia</em></td>
<td>−.21</td>
<td></td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Necturus maculosus</em></td>
<td>−.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Necturus alabamensis</em></td>
<td>.01</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>8</td>
<td>Salamandridae</td>
<td><em>Triturus</em> (7 spp.)</td>
<td>.02−.17</td>
<td><em>Pleurodeles waltl</em></td>
<td>−.09</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Plethodontidae</td>
<td><em>Hydromantes ambrosii</em></td>
<td>.10</td>
<td><em>Anides ferreus</em></td>
<td>.05</td>
<td>−</td>
<td>9</td>
</tr>
<tr>
<td>10</td>
<td>Bombinatoridae/Alytidae</td>
<td><em>Bombina orientalis</em></td>
<td>.05</td>
<td><em>Discoglossus pictus</em></td>
<td>.00</td>
<td>−</td>
<td>10</td>
</tr>
<tr>
<td>11</td>
<td>Leptodactylidae</td>
<td><em>Eleutherodactylus rivieri</em></td>
<td>.60, .57</td>
<td><em>Eleutherodactylus euphronides</em></td>
<td>.44</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Eleutherodactylus johnstonei</em></td>
<td>.25, .31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Ranidae/Mantellidae/Rhacophoridae</td>
<td><em>Rana</em> (12 spp.)</td>
<td>−.05−.30</td>
<td><em>Hoplobatrachus tigerinus</em></td>
<td>.13</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Buergeria buergeri</em></td>
<td>.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Bufonidae</td>
<td><em>Bufo viridis</em></td>
<td>.03</td>
<td><em>Bufo bufo</em></td>
<td>.22</td>
<td></td>
<td>13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>Bufo marinus</em></td>
<td>.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sources:
- H11001
- H11002
Sources: Source citations and body lengths, where F = female, M = male, are as follows. Where no means could be located, length ranges or maximum lengths are given. All lengths are mm. 1 = GSD types: Pandey and Lakra 1997; Galbusera et al. 2000; Devlin and Nagahama 2002. Sizes: Qin et al. 1998 (C. fuscus, no length reported, F mean weight 246 g, M mean weight 306 g); Srivastava 2012 (C. batrachus, no length reported, F mean weight 160 g, M mean weight 120 g). 2 = GSD types: Black and Howell 1979. Sizes: McPeek 1992 (G. holbrooki, F maximum 40, M 17–29); Bisazza 1993 (G. affinis, SSD 0.28, G. holbrooki, SSD 0.35); Vargas and de Sostoa 1996 (G. holbrooki, F 17–63, M 13–32); Deaton 2008 (G. affinis, F 15–43, M 14–26). 3 = GSD types: Devlin and Nagahama 2002. Sizes: Qin et al. 1998 (C. fuscus, no length reported, F mean weight 246 g, M mean weight 306 g); Srivastava 2012 (C. batrachus, no length reported, F mean weight 160 g, M mean weight 120 g). 4 = GSD types: Black and Howell 1979. Sizes: McPeek 1992 (G. holbrooki, F maximum 40, M 17–29); Bisazza 1993 (G. affinis, SSD 0.28, G. holbrooki, SSD 0.35); Vargas and de Sostoa 1996 (G. holbrooki, F 17–63, M 13–32); Deaton 2008 (G. affinis, F 15–43, M 14–26). 5 = GSD types: Baroiller et al. 1995; Devlin and Nagahama 2002. Sizes: Qin et al. 1998 (C. fuscus, no length reported, F mean weight 246 g, M mean weight 306 g); Srivastava 2012 (C. batrachus, no length reported, F mean weight 160 g, M mean weight 120 g). 6 = GSD types: Cnaani et al. 2008. Sizes: Ishikawa and Tachihara 2008 (T. zillii, F mean 76, M mean 82); Russell et al. 2012 (T. mariae, two sites, F means 222 and 213, M means 271 and 243, SSDs –0.22 and –0.14, mean SSD –0.18). 7 = GSD types: Hills and Green 1990. Sizes: Shoop 1965 (N. maculosus, F 127–180, M 130–192); Frese et al. 2003 (Sires, F mean 287, M mean 348); Moreno et al. 2006 (N. alabamensis, F mean 170, M mean 169); McDaniell et al. 2009 (N. bayeri, F mean 335, M mean 306). 8 = GSD types: King 1999; Schmid and Steinlein 2001. Sizes: Shine 1979 (Pleurodeles, lists male as the larger sex); Joly and Giacoma 1992 (Triturus alpestris, SSD 0.16, Triturus carnifex, SSD 0.11, Triturus italicus, SSD 0.15); Caetano and Leclair 1999 (Triturus boscai, SSD 0.17); Deaton 2008 (T. cristatus, SSD 0.06, Triturus vulgaris, SSD 0.03). 9 = GSD types: Hillis and Green 1990. Sizes: McKenzie 1969 (Aneides, F monthly means 54–65, M monthly means 49–63, SSDs –0.09–0.15, mean SSD 0.03); Salvadito and Bruce 2006 (H. ambrosii, F mean 66, M mean 60). 10 = GSD types: Hilits and Green 1990. Sizes: Capula and Corti 1993 (Discoglossus, two populations, F means 50, 51, M means 52, 49, SSDs –0.04 and 0.04, mean SSD 0.000); Zeyl and Laberge 2011 (Bombina, F mean 43, M mean 41). 11 = GSD types: Schmid et al. 2002; Schmid et al. 2003. Sizes: Ovaska 1992 (E. johnstonei, F 21–38, M 20–25); Lynch and La Marca 1993 (E. riveroi, F mean 32, F 30–36, M mean 20, M 16–23); Bourne 1997 (E. johnstonei, F mean 32, M mean 24); Ortega et al. 2005 (E. johnstonei, F mean 27, F 23–32, M mean 23, M 17–29); AmphibiaWeb 2013 (E. euphronides, F maximum 39, M maximum 27). 12 = GSD types: Hillis and Green 1990. Sizes: Woolbright 1983 (Rana clamitans, SSD 0.07); Teletka and Blackburn 1992 (Rana pipiens, SSD 0.08); Ueda et al. 1998 (Buergerea, two populations, F means 67, 50, M means 43, 37, SSDs, 0.56, 0.35, mean SSD 0.47); Khonsue et al. 2002 (Rana brevipoda, SSD 0.16); Monnet and Cherry 2002 (Rana catesbeiana, Rana temporaria, Rana rugosa, and Rana nigromaculata, SSDs 0.06, 0.07, 0.30, and 0.16, respectively); Gramapurohit et al. 2004 (Hyla arborea, F mean 153, M mean 135); Tryjanowski et al. 2006 (Rana esculenta and Rana ridibunda, SSDs 0.16 and 0.07, respectively); AmphibiaWeb 2013 (Rana esculenta, Rana japonica, and Rana t ypshimensis, SSDs –0.05, 0.13, and 0.29, respectively). 13 = GSD types: Abramyan et al. 2009; Stöckl et al. 2011. Sizes: Lee et al. 2001 (R. marinus, F mean 122, M mean 113); Monnet and Cherry 2002 (B. bufo, six sites, SSDs 0.19–0.26, mean SSD 0.22); Sinsch et al. 2007 (R. viridis, F mean 64, M mean 63); Kuttrup et al. 2011 (R. viridis, two populations, F means 79, 69, M means 77, 66, SSDs 0.03, 0.05). Note: P = .092, sign test (.146 if pair 5, where the SSDs are very close, is indicated with an equal sign). More than half the comparisons differ in the opposite direction from that predicted by the model for species with environmental sex determination (ESD) ancestry. The table contains comparisons that would not be expected to differ in the same direction as the pairs in table 2, because the GSD types are unlikely to have evolved from ESD. Where amphibians need to be paired with members of different families (because there was no within-family variation in GSD type), the phylogeny in Frost et al. (2006) was used to determine the most closely related family with a different GSD type. This phylogeny was also used to determine the pair within the Plethodontidae. SSDs and comparisons between SSDs are based on means when possible and on maximum lengths when no means can be located. SSDs based on means are shown in boldface. Where there are multiple sources for a species, the mean of the SSDs is given. Where there are multiple species in the XY or ZW half of the comparison, the species mean SSD is compared. * A plus sign = yes; a minus sign = no. ** The most closely related ZZ/ZZ congener according to the phylogeny of Pouyat et al. (2009) for which size dimorphism information could be found. *** The most closely related ZZ/ZZZ congener according to the phylogeny of Meyer (1997) for which size dimorphism information could be found.
selective pressures and molecular mechanisms for transitions from one GSD type to another, as do the Lake Malawi cichlids of the genus Metriaclima, which appear to have quite diverse and evolutionarily labile genetic sex-determining systems (Ser et al. 2010).

Models for the evolution of GSD types from simultaneous hermaphroditism can also be developed, but most animal genetic sex-determining systems probably did not evolve from simultaneous hermaphroditism (Bull 1983; Charlesworth and Mank 2010). Living members of basal vertebrate lineages are not hermaphroditic.

Instead, the most common form of hermaphroditism in vertebrates, which occurs in some perciform teleost fishes, is successive hermaphroditism, a form of ESD. Most cases appear to be derived from GSD rather than ancestral to it and so are not a test of our model of the origin of GSD from ESD (Weibel et al. 1999; Ota et al. 2000; Devlin and Nagahama 2002). An interesting exception is found in the groupers (epinepheline serranids), where a recent phylogeny suggests that GSD of an unknown type has evolved at least three times from protogynous (ESD) ancestry (Erisman et al. 2009). In successive hermaphrodites, because size tends to increase with age, the final sex (males in protogynous species) is larger than the initial sex. This would set the stage for the evolution of XY systems in clades with sex change from female to male once increased male size becomes selected and the evolution of ZW systems in clades with sex change from female to male once increased female size becomes selected. As more unknown GSD types become characterized, they will provide additional tests of the model’s predictions.

More generally, as phylogenies become better resolved, it will be possible to compose a sufficient number of ESD-GSD comparisons (including turtles and lizards as well as fishes) to further test the model (Mank et al. 2006). Where the genetic sex-determining systems of vertebrate groups containing ESD are not yet known, the model allows prediction in advance of what they might be, as in the grouper example above, where the prediction is ZW if females are larger in the GSD than in the protogynous species. As modern molecular genetic approaches are extended to a greater diversity of vertebrates, allowing discovery of sex-determining systems in the absence of heteromorphic sex chromosomes, there should be increasing opportunities for testing such predictions. Potentially interesting test cases include those groups where body size dimorphism is extreme, such as ceratioid anglerfish of the Lophiiformes and other examples of dwarf vertebrate males discussed by Parker (1992).

In conclusion, a change from ESD to genetic sex determination of type XY or ZW (from one adaptive peak to either of another two) is facilitated by sexual size dimorphism. GSD type has important implications for what sexual selection can produce (Roldan and Gomendio 1999; Kirkpatrick and Hall 2004) and introduces a major bias in which sex will evolve a novel extreme trait such as cooperative breeding or “fancy” ornaments (Reeve and Pfennig 2003). The possibility of important consequences for developmental processes such as sexual differentiation has been recognized for some time but remains to be explored formally and statistically (Witschi 1959; Jost 1960; Mittwoch 1975; Adkins-Regan 1981).

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Origin of Sex-Determination Systems


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from an island and a mainland population in Giresun, Turkey. Journal of Animal and Veterinary Advances 10:1469–1472.


Associate Editor: Jean-Michel Gaillard
Editor: Judith L. Bronstein

A tokay gecko (*Gekko gecko*) clinging to a wall in the Calgary Zoo. This species is XX/XY, but it has close relatives that are ESD and ZZ/ZW. Photo © 2005 Jeff Whitlock, The Online Zoo, used with permission.