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Sex chromosomes and speciation in birds and other ZW systems

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Abstract

Theory and empirical patterns suggest a disproportionate role for sex chromosomes in evolution and speciation. Focusing on ZW sex determination (females ZW, males ZZ; the system in birds, many snakes, and lepidopterans), I review how evolutionary dynamics are expected to differ between the Z, W and the autosomes, discuss how these differences may lead to a greater role of the sex chromosomes in speciation and use data from birds to compare relative evolutionary rates of sex chromosomes and autosomes. Neutral mutations, partially or completely recessive beneficial mutations, and deleterious mutations under many conditions are expected to accumulate faster on the Z than on autosomes. Sexually antagonistic polymorphisms are expected to arise on the Z, raising the possibility of the spread of preference alleles. The faster accumulation of many types of mutations and the potential for complex evolutionary dynamics of sexually antagonistic traits and preferences contribute to a role for the Z chromosome in speciation. A quantitative comparison among a wide variety of bird species shows that the Z tends to have less within-population diversity and greater between-species differentiation than the autosomes, likely due to both adaptive evolution and a greater rate of fixation of deleterious alleles. The W chromosome also shows strong potential to be involved in speciation, in part because of its co-inheritance with the mitochondrial genome. While theory and empirical evidence suggest a disproportionate role for sex chromosomes in speciation, the importance of sex chromosomes is moderated by their small size compared to the whole genome.

KEYWORDS
hybridization, sex chromosomes, sexual antagonism, speciation, W chromosome, Z chromosome

1 | INTRODUCTION

Life is vastly more interesting because of the presence of sex. Although far from universal, sexual reproduction based on the uniting of two types of gametes is ubiquitous throughout most of the tree of life (Bachtrog et al., 2014), likely because of the evolutionary advantages of segregation and recombination (Otto & Gerstein, 2006). In many groups, sex is highly genetically determined (e.g., birds, mammals, many insects and some plants), often to the degree that there are distinct sex chromosomes (Bachtrog et al., 2014; Charlesworth, 2002). Most sex chromosome systems fall into two categories: ZW systems (e.g., birds, butterflies and moths, many snakes), in which females are ZW ("heterogametic") and males are ZZ ("homogametic"); and XY systems, in which females are XX and males are XY (e.g., mammals, beetles, fruit flies). In both cases, the sex chromosome that is found in only one sex (W or Y) usually has fewer functional genes and is often smaller than the sex chromosome that is found in both sexes (Z or X). There is usually no recombination between the two types of sex chromosomes within a species, except for within a pseudoautosomal region (PAR) that is

thought to be necessary for proper segregation of the sex chromosomes during meiosis (Otto et al., 2011; Smeds et al., 2014).

The predominant theory explaining these sex chromosome patterns relies in part on sexually antagonistic selection (Bachtrog et al., 2014; Bull, 1983; Ellegren, 2011a; Fisher, 1931; Rice, 1987; Zhou et al., 2014). We start by imagining a new sex-determining locus (SDL), with two alleles (Z and W), appearing on an autosome, determining sex such that ZW are female and ZZ are male (the reasoning also works for an XY pattern). Now imagine another locus on the same chromosome, with two alleles: one allele increases female fitness but decreases male fitness, and the other has the opposite effects. Finally, imagine a mutation (e.g., an inversion is one possibility; Kirkpatrick, 2010) that causes suppressed recombination between the sex-determining locus and the sexually antagonistic locus. This can result in the female-beneficial allele being linked to the W allele of the SDL, and the male-beneficial allele being linked to the Z allele of the SDL. These recombination-suppressed chromosomes have higher mean fitness than the original recombining chromosomes, resulting in them spreading through the population. This process can occur iteratively, increasing the length of the nonrecombining part of the chromosomes, a process that has apparently resulted in the observed pattern, in birds and snakes, of distinct "strata" of successively shallower evolutionary distances between segments of the Z and W chromosomes as one moves away from the sex-determining locus (Ellegren & Carmichael, 2001; Handley, Ceplitis, & Ellegren, 2004; Nam & Ellegren, 2008; Vicoso, Emerson, Zektser, Mahajan, & Bachtrog, 2013; Wright, Harrison, Montgomery, Pointer, & Mank, 2014; Wright, Moghadam, & Mank, 2012; Zhou et al., 2014). One consequence of the suppressed recombination, however, is that the nonrecombining part of the W chromosome accumulates deleterious mutations and degrades (Bellott et al., 2017; Berlin & Ellegren, 2006; Charlesworth, 1978; Charlesworth & Charlesworth, 2000; Chen et al., 2014; Kirkpatrick, 2010; Muller, 1918).

The presence of well-developed sex chromosomes, with sex-specific inheritance patterns and suppressed recombination over much of their length, raises the possibility that their evolutionary dynamics will differ substantially from autosomes (Charlesworth, Coyne, & Barton, 1987; Dean & Mank, 2014; Ellegren, 2009a; Mank, 2009; Mank, Vicoso, Berlin, & Charlesworth, 2010; Rice, 1984; Vicoso & Charlesworth, 2009; Wright, 1933). A wide variety of ideas have been proposed regarding how sex chromosomes may differ from autosomes in terms of strength and forms of selection, the efficacy of selection relative to genetic drift (Caballero, 1995; Mank, Vicoso, et al., 2010; Vicoso & Charlesworth, 2009; Wright et al., 2015), and their exposure to sexual selection and sexually antagonistic selection (Albert & Otto, 2005; Charlesworth et al., 1987; Rice, 1984). A consensus of both theoretical and empirical work is that the sex chromosomes, especially the Z (or X), tend to have a faster rate of functional evolution than the autosomes, but the details as to exactly why are under much debate (Dean, Harrison, Wright, Zimmer, & Mank, 2015; Dhami, Joseph, Roshier, & Peters, 2016; Edwards et al., 2005; Mank, Hosken, & Wedell, 2014; Mank, Nam, & Ellegren, 2010; Meisel & Connallon, 2013; Vicoso et al., 2013; Wang et al., 2014; Wright et al., 2015).

Faster rates of evolution on sex chromosomes raise an intriguing possibility: might sex chromosomes play a disproportionate, perhaps even a dominant, role in causing the evolutionary splitting of a single species into two? For purposes of this article, I define speciation as the evolution of incompatibilities between populations that prevent interbreeding (i.e., prezygotic isolation) and/or produce genetic incompatibilities in hybrid offspring (i.e., postzygotic isolation). Birds have received much attention as subjects of speciation research (Price, 2007) as well as genomic analysis, and they also have a relatively stable sex chromosome system (ZW) compared to many similarly large taxonomic groups of equivalent evolutionary age. Hence, birds provide a rich body of empirical data to evaluate theory regarding the role of sex chromosomes in evolution and speciation. While my quantitative analysis focuses on birds, much of the theoretical discussion applies equally well to any ZW system; hence, I also include some references to groups such as snakes (Vicoso et al., 2013) and moths (Sackton et al., 2014).

Here, I first review theory regarding how sex chromosome evolution differs from autosomal evolution within ZW sex-determination systems and how those differences might lead to a greater role of sex chromosomes in speciation. I then turn to empirical examinations of a potential link between sex chromosomes and speciation. I summarize the debate in the literature regarding the commonly observed pattern of faster Z chromosome functional evolution ("Faster-Z evolution"): some studies attribute this to faster adaptation (e.g., Dean et al., 2015; Elgvin et al., 2017; Ellegren, 2009b; Ellegren et al., 2012; Lavretsky et al., 2015; Sackton et al., 2014), and other studies attribute it primarily to a higher rate of fixation of slightly deleterious alleles due to a greater role of genetic drift (e.g., Mank, Nam, et al., 2010; Vicoso et al., 2013; Wang et al., 2014; Wright et al., 2015). I point to several important results from the theoretical literature that will help resolve this debate and suggest that both drift and adaptation are likely important. Most interestingly, theory predicts more stable polymorphisms on the Z chromosome than on autosomes ((Rice, 1984; Albert & Otto, 2005), a result that needs integration into discussions of "Faster-Z evolution" (for a striking example of a stable polymorphism affecting much of the Z chromosome, see Kim et al. (2017) and Knief et al. (2017)). Finally, I compile available estimates of autosomal and Z chromosome within-population diversity and between-population differentiation and show that the Z often exhibits patterns consistent with some form of adaptive evolution as well as a disproportionate role in speciation.

2 | EVOLUTION WITHIN POPULATIONS: THEORY

2.1 | Basic consequences of sex chromosome inheritance

The distinct inheritance patterns of the sex chromosomes, their association with sex and their suppressed recombination have major consequences for their evolutionary dynamics in comparison with autosomes. Some of these consequences are equivalent for ZW and

XY systems (by just switching the sexes being considered), and some are vastly different. I focus on ZW systems here, but occasionally point out where the consequences differ strongly from XY systems. First, I summarize basic consequences of the inheritance patterns of the sex chromosomes in terms of patterns of neutral (nonselective) evolution (Box 1) and then consider how various forms of selection affect the sex chromosomes compared to the autosomes (Box 2).

The most obvious characteristic of sex chromosomes is their strong association with the sex in which they are found. All else being equal, this means that the evolutionary dynamics of W are directly dependent only on factors affecting females, whereas the evolutionary dynamics of Z are more dependent on factors affecting males. The W is always inherited from the mother, whereas the Z is inherited from fathers 2/3 of the time.

A related characteristic of sex chromosomes is their differing effective population sizes (N_e; Charlesworth, 2001; Vicoso & Charlesworth, 2009; Mank, Vicoso, et al., 2010) compared to each other and to autosomes. This results in differing levels of expected genetic variation, which is expected to be proportional to N_e (assuming mutation rate is constant). In a stable population with equal sex ratios and random reproduction among individuals, the Z chromosome has ¾ the N_e of autosomes, and W has ¼ the N_e of

BOX 1 Factors that differ between Z and W chromosomes and autosomes, independent of selection

The association between sex chromosomes and the sex of individuals in which they are found (ZW females; ZZ males) has a variety of consequences for their evolution (Table 1). In ZW systems, the Z is found in males 2/3 of the time and undergoes recombination only in males (except in the pseudoautosomal region (PAR), which tends to have a high recombination rate between Z and W in females; Otto et al., 2011). The W is found only in females and undergoes no recombination (except in the PAR). In contrast, autosomes are found equally in the two sexes and undergo recombination in both.

These different inheritance patterns mean that effective population size, which predicts the amount of diversity in a population (assuming constant mutation rate), differs between the Z, W and autosomes. If we assume a stable population, no selection and random mating of N^f females and N^m males each generation, then the effective population sizes (in terms of chromosomes, not individuals) of the autosomes ($N_{e,A}$), Z chromosome ($N_{e,Z}$) and W chromosome ($N_{e,W}$) are given by Wright (1933) and Pennell et al. (2015):

$$N_{e,A} = \frac{8N^f N^m}{N^f + N^m}$$

$$N_{e,Z} = \frac{9N^f N^m}{2N^f + N^m}$$

$$N_{e,W} = N^f$$

When equal numbers of females and males breed ($N^f = N^m$), then inspection of the above equations (see also Charlesworth, 2001) results in the ratio of effective population sizes of the Z compared to autosomes ($N_{e,Z}/N_{e,A}$) of ¾, and the W compared to autosomes ($N_{e,W}/N_{e,A}$) of ¼. These expectations change when the two sexes differ in their variances in reproductive success (Figure 1). Excess variance (compared to random mating) in male reproductive success (which reduces the effective number of mating males compared to mating females: $N^m < N^f$) results in $N_{e,Z}/N_{e,A}$ ratios below ¾ and $N_{e,W}/N_{e,A}$ ratios above ¼. With extreme variance in male reproduction (e.g., $N^m = 1 << N^f$), $N_{e,Z}/N_{e,A}$ ratios approach a minimum of 9/16 and $N_{e,W}/N_{e,A}$ ratios approach a maximum of $N^f/8$. Excess variance in female reproductive success, such that $N^f < N^m$, leads to $N_{e,Z}/N_{e,A}$ ratios higher than ¾ (approaching 9/8 in the extreme case of $N^f = 1 << N^m$) and $N_{e,W}/N_{e,A}$ ratios lower than ¼ (in the extreme case, approaching 1/8).

Sex chromosomes also differ from autosomes with regard to mutation rates, because the male germ line generally experiences higher mutation rates. The ratio of male mutation rate (μ^m) to female mutation rate (μ^f) has been estimated to range from 1.6 to 3.8 among 45 bird species surveyed (Wang et al., 2014). In the absence of selection, the substitution rate is predicted to be $\frac{2}{3}\mu^m + \frac{1}{3}\mu^f$ on the Z versus $\frac{1}{2}\mu^m + \frac{1}{2}\mu^f$ on the autosomes. Given this, the impact of a relatively large male bias in mutation rates is actually rather moderate: a male–female mutation rate ratio of 2 would produce a Z-to-autosome mutation rate ratio of roughly 1.11 (Axelsson et al., 2004; Oyler-McCance et al., 2015).

Females and males also differ in their gene dosage on the sex chromosomes, with males (ZZ) having twice the dose of females (ZW) of Z-linked genes. Birds tend to have only partial dosage compensation (i.e., moderation of expression levels in a way that partially balances out the otherwise two-fold difference), and it differs in degree throughout the genome (Ellegren et al., 2007; Itoh et al., 2007; Mank & Ellegren, 2009).

BOX 2 Differences in the way selection affects the Z chromosome and autosomes

For non-neutral mutations, interactions between the hemizygous state of the Z chromosome (in females) and the form of selection (beneficial vs. deleterious; dominant vs. codominant vs. recessive) can result in differences in the rate of substitution between the Z and autosomes. To summarize these dynamics, we can start with specification of how fitness depends on genotype (where A_2 represents a new mutant allele at a locus, and A_1 represents the pre-existing allele), the coefficient of dominance h, and the selection coefficient s, assuming perfect dosage compensation and no functional copy on the W chromosome (adapted from Charlesworth et al., 1987):

	Females			Males		
Autosomal						
Genotype	A_1A_1	A_1A_2	A_2A_2	A_1A_1	A_1A_2	A_2A_2
Fitness	1	$1 + hs$	$1 + s$	1	$1 + hs$	$1 + s$
Z-linked						
Genotype	A_1	A_2		A_1A_1	A_1A_2	A_2A_2
Fitness	1	$1 + s$		1	$1 + hs$	$1 + s$

For beneficial mutations, assuming $\mu^m = \mu^f$, the ratio of substitution rates on the Z vs. the autosomes is (Charlesworth et al., 1987):

$$R_{Z/A} \approx \frac{(2h+1)}{4h}$$

Hence, this ratio equals ¾ for completely dominant mutations ($h = 1$), increases as h declines, equals 1 when $h = 0.5$ (codominance) and increases to very high values as h declines towards zero (i.e., as recessivity of mutations increases).

For slightly deleterious mutations, the ratio of substitution rates on the Z vs. the autosomes is (Charlesworth et al., 1987):

$$R_{Z/A} = \frac{1}{\left(1 + \frac{1}{3}Ns\left(h - \frac{1}{2}\right)\right)},$$

where N is the effective population size (of individuals). Assuming s is negative (i.e., deleterious mutations), this ratio is above one (substitution rate on Z higher than on autosomes) when h is above ½ (i.e., mutations are dominant or partially dominant). In contrast, this ratio is below one (substitution rate on Z rate lower than on autosomes) when h is below ½, (i.e., mutations are recessive or partially recessive).

The above results for both beneficial and deleterious mutations depend on an assumption that the effective population size ratio of the Z to the autosomes ($N_{e,Z}/N_{e,A}$) is ¾. Higher variance in reproductive success in males compared to females reduces this ratio, whereas the opposite increases this ratio (see Box 1). For beneficial mutations, Vicoso and Charlesworth (2009) showed that lowering $N_{e,Z}/N_{e,A}$ (i.e., increasing variance of male reproductive success) lowers the relative Z-to-autosome substitution rates, for all levels of dominance, such that the level of dominance at which the Z and autosomes have equal substitution rates shifts from $h = 0.5$ to lower values (more recessive). Raising $N_{e,Z}/N_{e,A}$ (i.e., increasing variance of female reproductive success) does the opposite, raising the ratio of Z-to-autosome substitution rates for beneficial mutations and increasing the value of h at which the Z and autosomes have equal substitution rates. For deleterious mutations, the direction of these effects is opposite: lowering $N_{e,Z}/N_{e,A}$ dramatically increases the relative Z-to-autosome substitution rates, such that under some sets of parameters the substitution rate is higher on the Z for all levels of dominance (see figure 3 of Vicoso & Charlesworth, 2009).

While the above considered alleles with the same fitness effects in males and females, we can also consider sexually antagonistic alleles. On an autosome, a new mutation will tend to increase in frequency (when rare) whenever the average of its fitness effect on males and females is positive. When Z-linked, a new mutation can increase in frequency under a much wider range of fitness effects. We can specify the effects of the two alleles at a Z-linked locus as follows (following Parsons, 1961 and Rice, 1984), with A_1 referring to the pre-existing allele, A_2 referring to a new mutant allele. In the case when A_2 is female-benefitting, S refers to the fitness gain in females and T refers to the fitness cost in males. In the case when A_2 is male-benefitting, U refers to the fitness gain in males and V refers to the fitness cost in females:

	Females		Males		
Z-linked					
Genotype	A_1	A_2	A_1A_1	A_1A_2	A_2A_2
Fitness (female-benefitting)	1	$1 + S$	1	$1\text{-}hT$	$1\text{-}T$
Fitness (male-benefitting)	1	$1\text{-}V$	1	$1 + hU$	$1 + U$

When A_2 is female-benefitting and rare, then A_2 will tend to increase in frequency when this condition is met:

$$S > \frac{2hT}{1 - hT}$$

This means that if the new female-benefitting allele is sufficiently recessive (i.e., $h < 1/(2 + T)$), then it can tend to increase when rare even when the fitness gain to females is less than the cost to males.

When A_2 is male-benefitting and rare, then A_2 will tend to increase in frequency when this condition is met:

$$U > \frac{V}{h(2 - V)}$$

Hence, if the new male-benefitting allele is sufficiently dominant [i.e., $h > 1/(2\text{-}V)$], then it can tend to increase when rare even when the fitness gain to males is less than the cost to females.

Hence, there is much parameter space in which sexually antagonistic alleles increase in frequency when rare, even when the cost to one sex is greater than the gain to the other. This results in stable polymorphism (for equations describing the equilibrium allele frequencies, see Rice, 1984; for a related model of conditions that produce stable polymorphisms, see Otto et al., 2011).

Finally, Albert and Otto (2005) showed that alleles conferring a mating preference for a sexually antagonistic trait can spread and drive shifts in the equilibrium frequencies of the sexually antagonistic polymorphisms (Figure 2). If the preference is Z-linked then preferences for male-benefitting Z-linked traits will tend to rise in frequency, whereas if the preference is autosomal then preferences for female-benefitting Z-linked traits will tend to rise in frequency.

autosomes. These expectations are based on the assumption that the two sexes have the same variance in reproductive success. If they differ, for example because of one sex being more limited in reproductive output than the other (e.g., due to limited numbers of eggs per female in polygynous species, or conversely limited by the number of young a male can care for in polyandrous species), then these expectations differ to some degree (Box 1; Figure 1; Charlesworth, 2001; Vicoso & Charlesworth, 2009).

Another fundamental difference between autosomes and sex chromosomes is the influence of recombination. One consequence of the nonrecombining part of the W is that it is inherited as a single evolutionary unit through the matriline, in the same way as the mitochondrial genome in many taxa (including birds; Berlin & Ellegren, 2001; Smeds et al., 2015). Hence, the W and mitochondrial DNA are expected to have identical genealogies in these species (unlike in XY systems).

Mutation rate is another fundamental factor related to evolutionary dynamics and is often higher in males than females, presumably because the germ line in males undergoes more cell divisions before production of gametes (Axelsson, Smith, Sundström, Berlin, & Ellegren, 2004; Ellegren & Fridolfsson, 1997; Wang et al., 2014). This effect, termed "male-driven evolution," results in a higher mutation rate on the Z chromosome (in males 2/3 of the time) and a lower

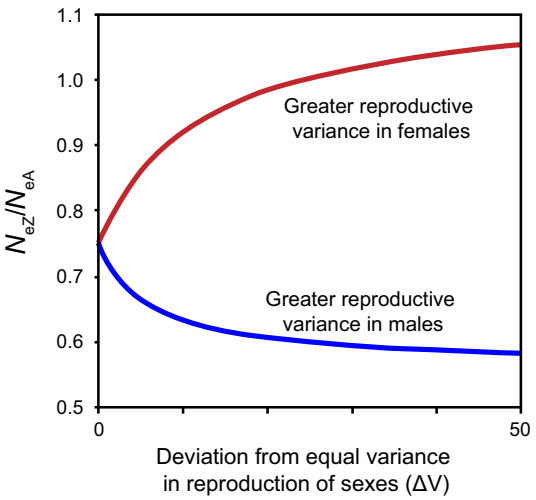

FIGURE 1 The ratio of effective population sizes of the Z chromosome and the autosomes equals ¾ in a stable panmictic population with no selection, but deviates from this expectation when the two sexes have different variance in reproductive success. For details regarding the horizontal axis scale, see Laporte and Charlesworth (2002). Adapted from Vicoso and Charlesworth (2009) with permission from Wiley

mutation rate on the W chromosome (only in females) compared to on autosomes, although the net effect is apparently rather mild (e.g., roughly 10% higher mutation rate on the Z than on autosomes; Box 1). Note that XY systems would be impacted differently by this male-driven evolution, with Y chromosomes having a higher, and X chromosomes a lower mutation rate compared to autosomes.

Finally, dosage compensation (Ellegren et al., 2007; Graves, 2015; Itoh et al., 2007) is a potentially important factor to consider when comparing the evolution of sex chromosomes and autosomes. All else being equal, the heterogametic sex is expected to express Z-linked genes at only half the level of the homogametic sex, because of one versus two copies of the Z chromosome (unless there are still functional versions of those genes on the W, as there are expected to be in the PAR or in recently evolved "strata" in the nonrecombining region). Hence, many groups have evolved mechanisms of dosage compensation. In mammals, this is commonly achieved through inactivation of one of the X chromosomes in the homogametic sex (females), directly resulting in similar expression levels of X-linked genes in males and females. Birds show a very different pattern, with most Z-linked genes showing ratios of male (ZZ) to female (ZW) expression that vary in the range of ~1.2–1.8 (Ellegren, 2013), showing that some dosage compensation occurs (as these ratios are <2, the expectation without compensation), but it is usually incomplete (as they are >1, the expectation with perfect compensation; although see Malone et al., 2012). The variability in these expression ratios among genes indicates a complex set of mechanisms determining dosage compensation, in a way that affects individual genes differently (Ellegren, 2013; Mank & Ellegren, 2009; Zimmer, Harrison, Dessimoz, & Mank, 2016).

2.2 | Sex chromosomes and selection

The above ways in which the evolutionary context of sex chromosomes differs from autosomes (different associations with sex, effective population sizes, recombination rates and mutation rates) can influence the potential for and effectiveness of selection on genetic variants. Here, I summarize a variety of ideas in the literature regarding the impact of specific types of selection and mutational input (Box 2). It should be noted that individual models (whether verbal, analytical or computational) in this field tend to focus on a restricted class of mutations in terms of their effect (beneficial vs. deleterious), their dominance and perhaps their sexual antagonism. Moving forward, the field would benefit from the development of models that integrate a wide variety of types of mutational input and look at the net effect on the pace and direction of evolution in sex chromosomes vs. autosomes.

Until very recently, most of the literature on population genomics of sex chromosomes in birds has focused on the Z chromosome, because relatively little genomic sequence was obtained from W chromosomes (most studies have sequenced males, so that the Z chromosome is sequenced at the same depth as the autosomes, but this results in no information for the W; for exceptions, see Berlin & Ellegren, 2004, 2006; Smeds et al., 2015; Bellott et al., 2017). I will first focus on the Z and provide some comments later regarding the W.

A consensus of much of the theoretical work on sex chromosome evolution is that the Z chromosome is expected to evolve faster than autosomes, both in terms of functional mutations (i.e., subject to selection) and neutral mutations. Part of this expectation is due simply to the higher mutation rate in males than females, resulting in an estimated ratio of mutation rates between the Z and autosomes of 1.1 (Box 1; Axelsson et al., 2004; Oyler-McCance, Cornman, Jones, & Fike, 2015), although this ratio is likely to vary between species. Even when this male-biased mutation is accounted for, most theoretical studies have concluded that the Z still evolves faster, but the reasons for this faster rate of evolution differ dramatically among models.

A central consideration in models of selection is that the Z (or X) chromosome is hemizygous (i.e., a single copy) in the heterogametic sex (i.e., females for ZW systems, males for XY systems): of all Z chromosomes in a population, 1/3 of them are in this hemizygous state. This means that recessive or partially recessive mutations are exposed more to selection when they are on the Z chromosome, compared to when they are on an autosome (Charlesworth et al., 1987). When the ratio of effective population sizes on the Z vs. the autosomes is ¾, recessive or partially recessive beneficial mutations experience higher substitution rates on the Z than on autosomes.

While the hemizygosity of the Z potentially leads to stronger effectiveness of selection on recessive or partially recessive beneficial alleles and a resulting higher substitution rate, a number of studies have emphasized an opposing effect of hemizygosity: it causes a lower effective population size (N_e) compared to autosomes, potentially leading to more genetic drift and reduced effectiveness of selection (Caballero, 1995; Wright, 1933). Hence, some studies have attributed Faster-Z functional evolution primarily to faster accumulation of deleterious mutations rather than faster accumulation of beneficial mutations (e.g., Mank, Nam, et al., 2010; Wright et al., 2015). This explanation is based largely on verbal models, whereas analytical models and simulations that incorporate N_e of each chromosome type have produced more complex predictions (Box 2). Vicoso and Charlesworth (2009) show that deleterious mutations have slower substitution rates on the Z (compared to the autosomes) when recessive or partially recessive, but they have higher substitution rates on the Z (compared to the autosomes) when dominant or partially dominant. However, when $N_{e,Z}/N_{e,A}$ ratio declines below ¾ (i.e., when males have higher variance in reproductive success than females), this expectation changes rapidly towards more accumulation of deleterious mutations on the Z, for all levels of dominance (figure 3 of Vicoso & Charlesworth, 2009). Increasing $N_{e,Z}/N_{e,A}$ (i.e., when females have higher variance in reproductive success than males) has the opposite effect, shifting towards more accumulation on the autosomes than the Z.

While the above considered alleles with the same fitness effects on males and females, the evolutionary dynamics of sex chromosomes become even more interesting with sexually antagonistic

alleles (Albert & Otto, 2005; Otto et al., 2011; Rice, 1984). These are thought to play a role in the origin of sex chromosomes (Bachtrog et al., 2014; Bull, 1983; Ellegren, 2011a; Fisher, 1931; Rice, 1987; Zhou et al., 2014), with male-benefitting alleles accumulating on the Z and female-benefitting alleles accumulating on the W. Now let us consider variation within the Z chromosome only, focusing on new mutations that are sexually antagonistic: they increase fitness of one sex and decrease fitness in the other sex. In autosomes, such alleles would tend to spread to fixation if their fitness advantage in one sex is larger than their fitness cost in the other. In the Z chromosome, the fate of such alleles is more complex. Rice (1984) showed that two types of Z-linked sexually antagonistic alleles will tend to spread when rare, even if the fitness cost to one sex is greater than the benefit to the other. First, alleles that benefit the heterogametic sex will spread if sufficiently recessive (see formula in Box 2). In the extreme case, a Z-linked mutation that gives a slight advantage to females will spread when rare if completely recessive, even if it causes lethality in males when homozygous. This is because when the allele is rare, there are no homozygous males, and the selective benefit to hemizygous females drives the frequency of the allele higher. Second, alleles that benefit the homogametic sex will spread if sufficiently dominant (formula in Box 2). This is because the dominance causes them to have a net larger average influence on phenotype in the homogametic sex compared to the heterogametic sex (assuming there is not also a functional copy on the W).

These two types of antagonistic alleles will spread when rare, but eventually the net selection in their favour declines. In the case of the spread of a recessive allele benefitting the heterogametic sex, the growing allele frequency causes increasing frequency of homozygotes in the homogametic sex, which are at a disadvantage. In the case of the spread of a dominant allele benefitting the homogametic sex, the advantage of dominance declines and the high frequency of heterogametic individuals with a fitness cost starts playing a major role. In both cases, the system approaches a stable polymorphic equilibrium between the original allele and the new sexually antagonistic allele (Albert & Otto, 2005; Otto et al., 2011; Rice, 1984).

Following the rise of such polymorphic sexually antagonistic loci, there are a variety of possible eventual outcomes. Rice (1984) proposes that the increase in frequency of sexually antagonistic alleles causes selection for sex-limited expression through changes in modifier loci, reducing the fitness cost in one sex and eventually resulting in fixation. Such a process would drive change in the Z chromosome in two ways: through change in both the sexually antagonistic locus and the modifier locus (if on the Z). Albert and Otto (2005) envision a polymorphic equilibrium at a Z-linked sexually antagonistic display trait locus and then analyse the consequences of a new preference allele (at a second locus) that causes females to prefer males carrying either the male-beneficial or female-beneficial trait allele. If the preference allele is Z-linked, preference for the male-beneficial trait spreads (Figure 2a). Depending on the fitness benefits and costs and the preference strength, the result is either the male-benefitting trait going to fixation or the preference becoming fixed and the equilibrium at the trait locus shifting towards the male-benefitting allele. In dramatic contrast, if the preference is autosomal, then preference for the female-beneficial trait spreads (Albert & Otto, 2005), either resulting in fixation of the female-beneficial trait or fixation of the preference and shifting the equilibrium towards the female-benefitting allele (Figure 2b).

This last result has received relatively little attention in the literature, but has large implications: First, a new autosome-based preference allele for a female-benefitting Z-based trait can spread to fixation, causing a selective sweep on that autosomal region and possibly a selective sweep on the Z region near the trait locus (Albert & Otto, 2005). Second, because the autosomal genome is so much larger than the Z chromosome, we might expect most

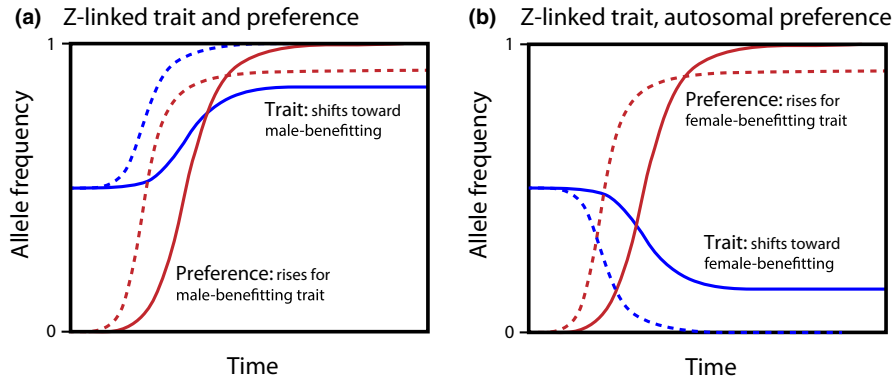

FIGURE 2 Simulation results from Albert and Otto (2005) showing initial stability of a sexually antagonistic polymorphism at a trait locus on the Z chromosome (Rice, 1984), followed by spread of a preference allele and the possible outcomes. (a) When the preference locus is Z-linked, a preference allele for the male-benefitting trait will spread, resulting in either fixation of the male-benefitting trait allele (dotted lines) or a new stable trait polymorphism (solid lines) shifted in favour of males. (b) When the preference locus is autosomal, a preference allele for a female-benefitting trait allele will spread, resulting in either fixation of the female-benefitting trait allele (dotted lines) or a new stable trait polymorphism (solid lines) shifted in favour of females. Such dynamics could drive rapid divergence and speciation between populations. Figure adapted from Albert and Otto (2005), with permission from AAAS

preference mutations to arise on autosomes, leading to most preferences evolving for female-benefitting traits and female-benefitting traits spreading. While much of the literature emphasizes differences between males and females, many bird species have remarkably similar traits of males and females in terms of appearance, parental care and other characteristics. Perhaps the spread and fixation of sexual preferences for female-benefitting traits is a force that keeps males and females more similar than they otherwise would be.

While fixation of either a sex-expression modifier locus and/or a preference locus can result in the elimination of stable polymorphisms, other antagonistic alleles will continue to arise through mutation (either at the same or different loci), such that we can expect some steady state of sexually antagonistic polymorphisms on the Z chromosome. Furthermore, the possibility that preference loci will arise on both autosomes and the Z chromosome means that there can be simultaneous increase in preferences (at different preference loci) for male-benefitting and female-benefitting alleles, either at the same or different trait loci. These interactions can lead to very complex evolutionary dynamics of Z-linked sexually antagonistic traits. Some aspects of these interactions lead to increase or maintenance of diversity on the Z (the stable polymorphisms and simultaneous preference for male-benefitting and female-benefitting traits in males), while some lead to reduced diversity (the sweeps of expression modifier loci or preference alleles and possible resolution of polymorphisms in favour of an allele benefitting one sex or the other).

In sum, theoretical models regarding the rate of Z chromosome vs. autosome evolution vary widely depending on the type of genetic variation being examined. I offer a few broad conclusions here (see also Table 1):

1. The neutral substitution rate tends to be slightly higher on the Z than the autosomes (due to the higher mutation rate in males).
2. The substitution rate for beneficial mutations is higher on the Z than autosomes if recessive or partially recessive, but lower on the Z if dominant or partially dominant (when the ratio of effective population sizes is ¾). Reducing the effective population size ratio shifts these relative rates towards autosomes.
3. Deleterious mutations accumulate faster on the Z than on the autosomes under a broad range of conditions applicable to birds: when dominant or partially dominant when there is equal variance in reproduction in females and males, and under all levels of dominance when there is sufficiently high variance in male reproductive success compared to females.
4. Sexually antagonistic polymorphisms arise on the Z under a wide range of conditions, and preference alleles for antagonistic traits are predicted to arise and spread, with Z-linked preferences favouring male-benefitting traits and autosome-linked preference favouring female-benefitting traits.

3 | EVOLUTION BETWEEN POPULATIONS: THEORY

There are a variety of ways the different evolutionary dynamics of the Z chromosome and autosomes could affect their relative rates

TABLE 1 Factors that differ between the Z, W, and autosomes. This table applies to the parts of the sex chromosomes that do not recombine between the Z and W (i.e., not the PAR)

Factors	Z	W	Key references
Association with sex	In males 2/3 of time	In females all of the time	
Hemizygosity	In hemizygous state (ZW) 1/3 of time (in females)	In hemizygous state all of the time	
Recombination	Only in males	No recombination	
Inheritance	Females inherit from father only; males inherit one from each parent	Through matriline only (same as mtDNA)	
Effective population size (N_e) in idealized population	3/4 that of autosomes (i.e., $N_{e,Z}/N_{e,A} = ¾$)	1/4 that of autosomes (i.e., $N_{e,W}/N_{e,A} = ¼$)	(Charlesworth, 2001)
Variance in male reproductive success	Reduces $N_{e,Z}/N_{e,A}$ towards a minimum of 9/16	Raises $N_{e,W}/N_{e,A}$ ratio towards a maximum of $N^f/8$	(Charlesworth, 2001)
Variance in female reproductive success	Raises $N_{e,Z}/N_{e,A}$ towards a maximum of 9/8	Lowers $N_{e,W}/N_{e,A}$ towards a minimum of 1/8	(Charlesworth, 2001)
Mutation rate	Higher than autosomes[a]	Lower than autosomes[a]	(Axelsson et al., 2004; Wang et al., 2014)
Dosage	Males (ZZ) have twice the gene dosage of females (ZW)	W genes only expressed in females	
Dosage compensation	Partial in birds: Most Z genes are expressed 1.2-1.8 times higher in males (ZZ) than females (ZW)	n/a	(Ellegren, 2013)

[a]Because of the higher genomewide mutation rate in males (i.e., "male-driven evolution").

of evolution of genetic incompatibilities. First, the higher rate of neutral substitutions in the Z may play a role, as an allele resulting from a series of substitutions that are each neutral to selection in one population's genetic background can nonetheless result in incompatibilities with another population. Second, the broad range of conditions under which slightly deleterious alleles accumulate faster on the Z could result in more incompatibilities evolving on the Z, as alleles that are mildly deleterious in one population might have much larger deleterious effects when interacting with the genome of another population. Third, the higher rate of accumulation of recessive beneficial mutations on the Z could contribute to faster evolution of incompatibilities between populations, although dominant beneficial mutations should accumulate faster on autosomes. Fourth, the accumulation of sexually antagonistic polymorphisms on the Z, and the subsequent sweeps of either expression modifiers (that limit expression to one sex) or preference alleles (either on the Z, in favour of male-benefitting traits, or on autosomes, in favour of female-benefitting traits) can lead to strong differences in sexual traits and preferences between populations as well as genetic incompatibilities in hybrids. While the Z plays a vital role in this last process, it is not clear whether the Z or autosomes are predicted to have a resulting higher net contribution to incompatibilities between populations, as sexually antagonistic polymorphisms on the Z can be stable and because some types of modifier alleles and preference alleles can sweep on the autosomes as well as the Z. Considering all these factors together, I conclude that the weight of evidence is that the Z is likely to have a disproportionate role in the evolution of incompatibilities, but the role may not be dramatically larger than the role of autosomes. More comprehensive modelling is needed to incorporate all of these types of genetic variation into a single framework.

While the above considered the evolution of incompatibilities while populations are geographically isolated, we can also consider the case of secondary contact between partially differentiated populations and examine whether the rate of exchange between populations might differ between Z-linked and autosomal loci. Here, the fitnesses of various classes of hybrids (F1's, F2's, backcrosses, etc.) and the dependence of those fitnesses on specific genotypes are centrally important. If the Z chromosome is disproportionately involved in the evolution of incompatibilities between populations, then it is expected to play a large role in reducing the fitness of specific hybrid combinations, resulting in less flow of the Z chromosome across a hybrid zone compared to autosomes. This difference should be strengthened by linkage of multiple incompatibility loci on the Z chromosome (e.g., a male-benefitting trait allele at one locus and an allele conferring preference for that trait at another locus), along with the lack of recombination across much of the Z chromosome in females. The resulting association between incompatibility loci results in net stronger selection against introgression of the Z (relative to autosomes), likely resulting in a steeper cline (i.e., transition in frequency across the hybrid zone) for the Z chromosome than autosomes. The relative isolation of the Z compared to autosomes would then allow proportionally more differentiation between populations to build up on the Z, enhancing their differentiation compared to autosomes.

However, there are also situations in which the Z chromosome might preferentially flow between populations that are interbreeding. A new globally advantageous allele on the Z of one population could sweep through the second as well. Conversely, if one population has accumulated more Z-linked deleterious alleles than the other, the less deleterious version of the Z from the second population could selectively spread into the first. Sexually antagonistic polymorphisms, and perhaps preferences for them, could also selectively spread between populations. These sorts of processes presumably occur more when there is relatively little differentiation between populations, before many incompatibilities have developed.

4 | EMPIRICAL PATTERNS

4.1 | Role in speciation

Perhaps the earliest empirical observation suggesting a disproportionate role for sex chromosomes in speciation is Haldane's Rule: the strong tendency for heterogametic hybrids (i.e., females in ZW systems; males in XY systems) to show lower fitness (i.e., higher rates of infertility and inviability) than homogametic hybrids (Haldane, 1922; Presgraves, 2002; Turelli & Orr, 1995). Haldane's observation has stood the test of time, with the pattern being supported in a wide range of both ZW and XY systems (Schilthuizen, Giesbers, & Beukeboom, 2011). Birds show an especially strong pattern (Price & Bouvier, 2002), with a recent review (Schilthuizen et al., 2011) reporting 99% of cases examined (215 of 217) show more hybrid inviability in females compared to males. Two main explanations have been proposed for Haldane's rule. One, which was developed as an explanation in XY systems, is that greater sexual selection on males causes faster evolution of male-expressed genes, producing more resulting incompatibilities in males (Wu & Davis, 1993). This explanation is not applicable to ZW systems, in which female hybrids have lower fitness. The other explanation is that deleterious recessive (or partially recessive) alleles on the Z (or X) have a larger average impact on fitness in the heterogametic sex (when they are hemizygous) than in the homogametic sex (Turelli & Orr, 1995). This explanation directly implicates the Z (or X) chromosome in lowering hybrid fitness and appears to be the most likely explanation for the very strong Haldane's rule pattern seen in birds. Other possible factors contributing to this pattern include incompatibilities between the Z of one population and the W of the other, or between the Z of one population and the mitochondrial genome of the other (Presgraves, 2002).

A second commonly cited "rule" of speciation (Coyne & Orr, 1989; Edwards et al., 2005), related to Haldane's rule and also implicating sex chromosomes, is the "large-X effect," originally described as an observed disproportionate contribution of the X chromosome in causing inviability and/or sterility in heterogametic F1 hybrids

between *Drosophila* species. Turelli and Orr (1995) explain that both Haldane's rule and the large-X(Z) effect are well explained by a tendency for the X(Z) to accumulate incompatibility alleles that act (at least partially) recessively. In taxa such as birds that are more difficult to breed in captivity and have fewer genetic resources than *Drosophila*, direct measurements of the contribution of the Z on reduced hybrid fitness are challenging. Rather, the possibility of an analogous "large-Z effect" has mostly been evaluated indirectly, in two ways.

First, the Z may have reduced gene flow between differentiated populations compared to autosomes (Elgvin et al., 2017; Sætre et al., 2003; Storchová, Reif, & Nachman, 2010). Patterns consistent with reduced gene flow at the Z (e.g., steeper clines, and/or inference of reduced gene flow using coalescent modelling) have been shown across a variety of avian hybrid zones, including *Passerina* buntings (Carling & Brumfield, 2008; Carling, Lovette, & Brumfield, 2010), *Luscinia* nightingales (Storchová et al., 2010), *Anas* ducks (Dhami et al., 2016; Lavretsky et al., 2015), *Aquila* eagles (Backström & Väli, 2011) and *Parus* chickadees (Taylor, Curry, White, Ferretti, & Lovette, 2014; Taylor, White, et al., 2014). One complication of such analyses is that, because of the smaller effective population size of the Z, two populations are expected to harbour less standing variation and be more subject to genetic drift on the Z than the autosomes—upon secondary contact, the Z will then tend to have higher relative differentiation than autosomes, perhaps leading to inference of narrower clines and less gene flow (as the models used often do not account for a history of isolation and secondary contact).

Second, a faster rate of functional evolution in Z-linked genes than autosomal genes ("Faster-Z evolution"; Ellegren, 2009b; Meisel & Connallon, 2013) may suggest a large-Z effect on reduced hybrid fitness. In a particularly convincing demonstration of an association between faster functional evolution and the Z chromosome, Ellegren (2009b) examined a set of 228 genes that show faster rates of evolution in birds compared to the orthologous genes in mammals (birds and mammals have nonhomologous sex chromosomes) and found that these were twice as likely to occur on the Z chromosome than the genomic proportion of Z-linked genes would predict. A variety of studies have compared ratios of nonsynonymous (d_N) to synonymous (d_S) substitution rates between the Z and autosomes: $(d_{N,Z}/d_{S,Z})/(d_{N,A}/d_{S,A})$; note that this controls for differences in mutation rates between the two types of chromosomes. A faster-Z effect (a ratio above 1, indicating faster functional evolution of genes on the Z) has been observed in a variety of bird groups (Borge, Webster, Andersson, & Saetre, 2005; see also Elgvin et al., 2017; Mank, Nam, et al., 2010; Wright et al., 2015; Zhang et al., 2014;) as well as silkmoths, which also have a ZW system (Sackton et al., 2014).

4.2 | Causes of faster-Z evolution

While faster-Z evolution is a reasonably strong and consistent pattern, the best explanation for it is debated. Some studies have emphasized adaptive evolution in explaining the faster rate of functional evolution on the Z (e.g., Dean et al., 2015; Elgvin et al., 2017; Ellegren, 2009b; Sackton et al., 2014), a consequence of the many conditions in which the Z may be exposed to higher selection intensity (e.g., when universally beneficial mutations tend to be recessive, when there are sexually antagonistic mutations and when there are preference alleles for male-beneficial Z-linked traits). Other studies have concluded that the effect is primarily explained by a higher rate of fixation of mildly deleterious mutations on the Z chromosome, due to less effective selection against them because of the smaller effective population size of the Z compared to autosomes (e.g., Mank, Nam, et al., 2010; Mank, Vicoso, et al., 2010; Wang et al., 2014; Wright et al., 2015). This is a reasonable possibility, as the small ratios of d_N/d_S, approximately 0.11–0.14 (Wright et al., 2015), that are typically observed suggest that most functional mutations are deleterious, on both the autosomes and Z. However, theory predicts that when the Z has ¾ the N_e of autosomes (i.e., sexes have equal variance in reproductive success), then only dominant or partially dominant deleterious mutations have a higher substitution rate on the Z—recessive or partially recessive mutations have a higher substitution rate on the autosomes than the Z (Box 2). Corl and Ellegren (2012) and Wright et al. (2015) propose that sexual selection on male birds causes high variance in male mating success compared to females, reducing the $N_{e,Z}/N_{e,A}$ ratio below ¾ and shifting the relative substitution rates of deleterious alleles (due to drift) towards the Z. Another possibility is that sexual selection has a direct effect on one or more Z-linked loci, causing hitchhiking of mildly deleterious alleles at other loci and leading to their greater accumulation. The stochasticity leading to their accumulation may be due more to the randomness of the mutational process and whether they are linked to genes under sexual selection, rather than drift per se.

While an increased role of drift on the Z almost certainly influences its evolution, it is unlikely to be the complete explanation for the faster-Z pattern, for several reasons. First, estimates from the literature of ratios of $N_{e,Z}/N_{e,A}$ based on standing variation in the two chromosomal classes are often far below the lower theoretical limit of 9/16 (Box 1; Charlesworth, 2001) that results purely from high reproductive variance of males (e.g., Dhami et al., 2016; Elgvin et al., 2017; Storchová et al., 2010; Sundström, Webster, & Ellegren, 2004), suggesting selective sweeps may also be involved in further reducing variation in the Z chromosome. Second, theory (Box 2) produces an expectation of Z-linked loci with semi-stable polymorphisms, presumably increasing levels of standing variation and calling into question the inference of N_e from levels of nucleotide diversity. Third, autosomes are likely also subject to some level of selection, such that variation on them may be reduced compared to neutral expectations; hence, an estimated $N_{e,Z}/N_{e,A}$ ratio of 0.75 does not necessarily indicate no selection on the Z.

Much of the empirical evidence against adaptive evolution as a cause of faster-Z evolution is based on a prediction regarding relative rates of evolution of genes expressed in differing levels in males and females (Mank, Nam, et al., 2010; Mank, Vicoso, et al., 2010; Wright et al., 2015): if faster-Z evolution were due to increased

efficiency of selection for beneficial recessive mutations in the hemizygous state, then faster-Z evolution would be expected to be largest for female-expression-biased genes, followed by unbiased and then male-biased genes. Such a pattern was not observed (Mank, Nam, et al., 2010; Mank, Vicoso, et al., 2010; Wright et al., 2015), casting doubt on the idea that the faster-Z effect is driven primarily by adaptation via recessive mutations in female-expression-biased genes. However, the relative ratio of dominance and recessivity in beneficial mutations is not well established (Payseur, 2014), and dominant mutations might be under more effective selection when male-biased and Z-linked (as Z-linked genes are in males 2/3 of the time, whereas autosomal genes are in males only ½ of the time). The net effect of the two types of beneficial mutations (recessive female-biased ones accumulating faster on the Z than autosomes; dominant male-biased ones accumulating faster on the Z than autosomes) might result in the observed no net difference in the faster-Z effect between male-biased and female-biased genes. Furthermore, Z-linked sexually antagonistic alleles and Z-based preference alleles are expected to resolve in favour of preferences for male-benefitting alleles, such that we might expect a faster rate of Z evolution in male-biased genes (see also Ellegren, 2011b regarding the tendency of genes with male-biased expression to move to the Z, whereas those with female-biased expression tend to move away from the Z onto autosomes).

4.3 | Sexual antagonism and sexual selection

While the above discussion focuses on broad comparisons between the Z and autosomes as a whole, there are many studies implicating particular Z-linked loci in speciation. Before turning to a summary of those studies, recall from above two broad conclusions from theory: first, the sexually asymmetric inheritance pattern of the Z should cause it to accumulate sexually antagonistic polymorphisms, preference alleles for male-biased traits and incompatibility alleles at faster rates than autosomes. Second, large nonrecombining regions of sex chromosomes are thought to arise because they isolate sexually antagonistic alleles of benefit to one sex and not the other, creating a situation where further mutations on the sex chromosomes can be expected to have a greater probability (compared to mutations on the autosomes) of being involved in sexual selection, have sex-specific functional effects, and/or have sexually antagonistic effects. These two factors might act together to produce an overrepresentation of such traits on the Z.

In fact, there is much evidence for such a pattern. Price (2002) reviewed 161 cases in which the genetic linkage pattern of traits that distinguish breeds of domesticated birds has been established. Of these, 22% (36/161) are Z-linked, many more than the 2.7% expected from the size of the Z chromosome as a percentage of the whole genome (Edwards et al., 2005; Price, 2002). While I am not aware of a similar survey in wild birds, a variety of studies show that the Z can be linked to traits, preference and incompatibilities. Two colour morphs of Gouldian Finches (*Erythrura gouldiae*) provide a particularly elegant example (Pryke, 2010). Head colour (red versus black) is inherited as a single Z-linked locus with red being dominant (i.e., male heterozygotes are red). Both males and females prefer mates of the same colour type as themselves, and the preference is also Z-linked. Mixed matings result in offspring with lower fitness (e.g., 34% higher inviability), with daughters of mixed matings having higher mortality than sons, a pattern consistent with Haldane's rule and implicating the Z chromosome (Pryke, 2010). In *Ficedula* flycatchers, three species-specific male plumage traits are Z-linked, whereas non-sex-limited plumage differences are autosomal (Sætre et al., 2003), and sexual preferences of females for those male traits are also Z-linked (Sæther et al., 2007). In both of these cases, the Z-linkage of both plumage traits and preferences for those traits can enhance selection intensity, resulting in a particularly strong role of the Z in premating isolation and further differentiation.

A particularly striking example of genetic variation on the Z chromosome affecting sex-specific traits was recently provided by Kim et al. (2017) and Knief et al. (2017), who showed that variation in Zebra Finch (*Taeniopygia guttata*) sperm morphology is largely explained by an inversion polymorphism on the Z chromosome. Three major inversion haplotypes occur: one ancestral version (type A), one with a single major inversion (type C) and one with a second inversion (type B). These inversions span most of the Z chromosome, resulting in large regions of linked genes with recombination suppression between these haplotypes. The polymorphism appears to be maintained by heterozygote advantage, as heterozygous males have faster sperm and higher siring success than homozygous males. Kim et al. (2017) explain this situation as a result of the accumulation of different dominant alleles that benefit males (e.g., increased sperm velocity) on each inversion haplotype, such that heterozygotes have a combination of dominant alleles at different genes that together enhance sperm function. Knief et al. (2017) note that genes with male-biased expression have tended to move from autosomes onto the Z over the course of bird evolution (Ellegren, 2011b) and that inversion polymorphisms on the Z are known from a number of species related to the Zebra Finch. This example illustrates how adaptive evolution can shape variation on the Z chromosome and how recombination suppression (in this case through inversions) can extend the effects of that selection over broad genomic regions. It also shows that the net level of nucleotide diversity in a population can be the result of complex selective processes: variation is very low within each haplotype, presumably the result of selective sweeps, whereas variation is high between haplotypes, due to the stable polymorphism (Kim et al., 2017). It is certainly conceivable that these sorts of processes could proceed down different pathways in two populations, resulting in reproductive isolation between them.

It should be noted that large nonrecombining regions that could contribute to speciation are not necessarily confined to the sex chromosomes. A remarkably bizarre example is provided by the white-throated sparrows (Lowther, 1961; Thorneycroft, 1975; Tuttle, 2003; Tuttle et al., 2016), in which there is suppressed recombination (due to an inversion) between two forms of a very large region of chromosome 2 (approximately 100 Mbp, and 1,000

genes). The two versions of these supergenes confer different suites of traits related to plumage colour, mating behaviour and parental investment, affecting both sexes somewhat similarly. Remarkably, the two forms display nearly perfect dis-assortative mating (i.e., strongly preferring the other form as mates), and offspring from rare assortative mating have very low fitness. While the differences between the two versions of chromosome 2 produce almost the complete opposite of speciation (i.e., always mating with the other form, rather than having reproductive isolation), this case does illustrate that large nonrecombining regions of the genome can be hotspots for evolution of sexual traits, preferences and incompatibilities.

5 | EMPIRICAL COMPARISONS OF AUTOSOME AND Z CHROMOSOME VARIATION

A number of studies have compared patterns of differentiation at sex chromosomes and autosomes in small groups of related species (Corl & Ellegren, 2012; Oyler-McCance et al., 2015; Van Doren et al., 2017), but there has been no prior survey of patterns throughout birds as a whole. I examined studies of birds that have measured within-population levels of genetic diversity (π) at the Z chromosomes (π_Z) and at autosomes (π_A); and/or between-population relative differentiation (F_{ST}) at the Z and autosomes. I also compiled estimates of relative substitution rates between the Z and autosomes, which should be closely proportional to relative mutation rates; when combined with within-population genetic diversity, this would allow an estimation of relative effective population size (N_e). My goal was to examine quantitatively whether there tends to be (i) smaller estimated effective population sizes at the Z, and (ii) faster relative population differentiation at the Z, a pattern consistent with it playing a disproportionate role in speciation.

To be included in the survey, studies had to be based on sequence data from at least several unlinked autosomal genes and several Z-linked genes. However, most studies employed high-throughput sequencing of thousands of loci throughout the genome (i.e., whole-genome resequencing, or reduced-representation sequencing methods such as RAD-seq or GBS), such that nucleotide diversity and differentiation estimates are much more precise—I analysed both the whole data set as well as a more limited data set based on just the high-throughput sequencing studies. "Population" for purposes of this survey is defined as a distinct taxon (species/subspecies) and/or well-marked geographic unit that researchers viewed as distinct enough to warrant examination from a speciation research perspective.

Of 33 populations measured for within-population Z and autosome nucleotide diversity (Table 3), a nearly universal pattern is that within-population diversity is lower on the Z than at autosomes (average π_Z/π_A ratio of 0.674; *SD*: 0.465; range 0.149–3.0; Figure 3). The only ratio above 1 was an extreme outlier (a ratio of 3.0) that also has extremely low within-group diversity as well. This is a very small island population of zebra finch—a situation that could be subject to extreme stochasticity as well as varying gene flow from other populations (Balakrishnan & Edwards, 2009). The other cases all had ratios considerably below 1. Because of the very unusual situation of this zebra finch population, I do not include it in the further analysis.

I identified three independent estimates of the substitution rate ratio between Z and autosomes that I considered highly reliable, as they were based on large amounts of sequence data and compared species that were distantly related. These include a comparison of chicken and turkey (1.09; Axelsson et al., 2004), a comparison of pied/collared flycatchers to red-breasted flycatcher (1.12; Borge et al., 2005) and a comparison of MacGillivray's warblers to Townsend's warblers (1.10; Irwin et al., in review). The agreement in these three estimates enabled reasonably confident application of an estimate of 1.1 for the substitution rate ratio of Z vs. autosomes, which provides an estimate of the mutation rate ratio (in the absence of selection). Hence, for the purpose of expressing the relative diversity of Z and autosomes while controlling for the difference in mutation rates, I divided the diversity of Z by this mutation rate ratio to obtain adjusted Z diversity: $\pi_Z^* = (\pi_Z/1.1)$. I then took the ratio of adjusted Z diversity to autosome diversity as an estimate of the ratio of effective population sizes of the two chromosome types: $N_{e,Z}/N_{e,A} = \pi_Z^*/\pi_A$.

This estimated $N_{e,Z}/N_{e,A}$ ratio is well below 1 in all of the populations in the analysis, with an average of 0.547 (*SD*: 0.190) and a range of 0.135–0.806 (Table 2; Figure 4). Most of these estimates are below the theoretical minimum of 9/16 (i.e., 0.5625) that could be explained as a result of extreme variance in male mating success without invoking selection and/or population fluctuations (Box 1; Charlesworth, 2001). These lower values can easily be explained by a greater tendency for selective sweeps on the Z than on autosomes, although it is also possible that extreme population bottlenecks might also contribute (Pool & Nielsen, 2007). Another possibility is that there tends to be more autosomal than Z chromosome gene flow across hybrid zones, resulting in much higher diversity in autosomes than the Z chromosome; this explanation however also involves selection as the explanation of differing amounts of gene flow. Altogether, these low values of $N_{e,Z}/N_{e,A}$ are difficult to explain without invoking more intense selection on the Z than on autosomes.

On the other hand, some of the estimated $N_{e,Z}/N_{e,A}$ ratios are slightly above the value of 0.75 expected under neutrality with equal variance in mating success of the two sexes. Values higher than 0.75, if significantly so, are unlikely to be explained by higher variance in female than male reproductive success (most birds are expected to have the opposite), but could be explained through stable polymorphisms slightly elevating diversity on the Z (as predicted by sexual antagonism theory) and/or selection on autosomes reducing diversity there. Extreme population growth can also raise the Z-to-autosome diversity ratio above neutral expectations (Ellegren, 2009a; Pool & Nielsen, 2007).

TABLE 2 Summary of how substitution rates of various types of mutations differ between the Z chromosome and autosomes (assuming constant mutation rates). When referring to Z-linked mutations, the terms "dominant" and "recessive" refer to the effect in males (ZZ; because females are hemizygous [ZW] those terms are not applicable)

Mutation type	Outcome	Key references
Recessive (or partially recessive) beneficial mutations	Higher rate of substitution on Z than on autosomes, when $N_{e,Z}$ is ¾ that of $N_{e,A}$; lowering $N_{e,Z}/N_{e,A}$ ratio below ¾ shifts the relative rates of substitution towards autosomes	(Charlesworth et al., 1987)
Dominant (or partially dominant) beneficial mutations	Lower rate of substitution on the Z than on autosomes, when $N_{e,Z}$ is ¾ that of $N_{e,A}$; lowering $N_{e,Z}/N_{e,A}$ ratio below ¾ shifts the relative rates of substitution towards autosomes	(Charlesworth et al., 1987)
Recessive (or partially recessive) deleterious mutations	Lower rate of substitution on Z than on autosomes, when $N_{e,Z}$ is ¾ that of $N_{e,A}$; lowering $N_{e,Z}/N_{e,A}$ ratio below ¾ shifts the relative rates of substitution towards Z	(Charlesworth et al., 1987; Vicoso & Charlesworth, 2009)
Dominant (or partially dominant) deleterious mutations	Higher rate of substitution on Z than on autosomes, when $N_{e,Z}$ is ¾ that of $N_{e,A}$; lowering $N_{e,Z}/N_{e,A}$ ratio below ¾ shifts the relative rates of substitution towards Z	(Charlesworth et al., 1987; Vicoso & Charlesworth, 2009)
Female-benefitting sexually antagonistic mutations	If Z-linked and sufficiently recessive, will spread when rare	(Rice, 1984)
Male-benefitting sexually antagonistic mutations	If Z-linked and sufficiently dominant, will spread when rare	(Rice, 1984)
Preference mutation for Z-linked sexually antagonistic trait	If Z-linked preference allele, then preference for male-beneficial trait spreads, driving male-benefitting trait allele to higher frequency; If autosomal preference allele, then preference for female-beneficial trait spreads, driving female-benefitting trait allele to higher frequency (Figure 2)	(Albert & Otto, 2005)

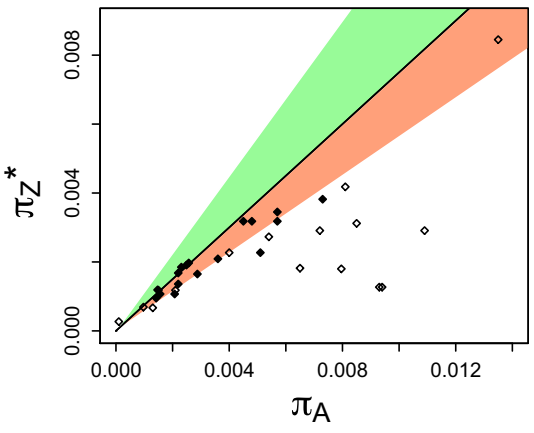

FIGURE 3 Comparisons (from studies of birds; Table 3) of within-population nucleotide diversity on the autosomes (π_A) versus the Z chromosome (π_Z^*; after correcting for higher mutation rate: $\pi_Z^* = \pi_Z/1.1$) estimated from either large-scale genomic sequencing (filled diamonds) or sequences from a small number of genetic loci (open diamonds). The black line is the theoretical expectation of π_Z^*/π_A = ¾ when there is equal variance of reproductive success in the two sexes. The salmon area shows π_Z^*/π_A values expected when variance in reproductive success is higher in males than females; whereas the green area show values expected when females have higher variance in reproductive success than males

A particularly surprising result is that as the average effective population size increases, the estimated $N_{e,Z}/N_{e,A}$ ratio tends to decline (Figure 4). This suggests that larger population sizes tend to have lower ratios of Z diversity to autosomal diversity. I interpret this pattern as being a result of selection being more effective (compared to drift) in larger populations: if selective sweeps tend to occur more often on the Z than autosomes, the signature of such selection (a low estimated $N_{e,Z}/N_{e,A}$ ratio) would be most apparent in larger populations. However, there may also be methodological differences contributing to this pattern: as shown in Figure 4, the cases with high genetic diversity and low $N_{e,Z}/N_{e,A}$ ratio tend to have been studied using estimates from a small number of genomic loci, whereas cases with lower genetic diversity and higher $N_{e,Z}/N_{e,A}$ ratios tend to have been studied using large-scale genomic sequencing. I look forward to future case studies to be added to this analytical framework, such that the possible contributions of methodology versus a true biological relationship can be inferred with more confidence.

Turning to between-population relative differentiation, in almost every case (one exception) F_{ST} is higher for the Z chromosome than for autosomes (Figure 5). This finding is certainly consistent with the Z playing a larger role in speciation than autosomes. However, this finding is expected as a result of either greater rates of adaptive evolution on the Z or simply lower effective population size resulting

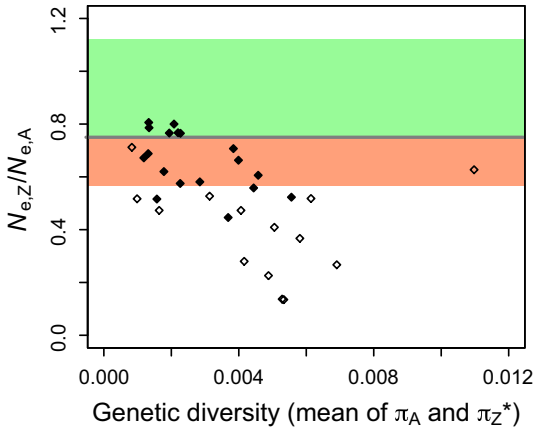

FIGURE 4 Estimated Z-to-autosome ratios of effective population sizes ($N_{e,Z}/N_{e,A}$) tend to decrease with increasing within-pop variation, suggesting greater relative influence of diversity-reducing selection on the Z as population size increases. The horizontal axis represents the average of autosomal and Z-linked within-population diversity (the mean of π_A and π_Z^*), and the vertical axis ($N_{e,Z}/N_{e,A}$) is estimated by π_Z^*/π_A. Filled diamonds indicate estimates from large-scale genomic sequencing, whereas open diamonds indicate estimates from sequences of a small number of genetic loci. To test significance of this apparent inverse relationship while ensuring it is not due to spurious correlation between variables influencing both axes, I standardized both π_A and π_Z^* by dividing by the standard deviation of each, and then tested whether the average of these standardized values was correlated with the ratio between them. This resulted in a statistically significant inverse relationship (Pearson's correlation: $r = -.378$, $df = 30$, $p = .033$; Spearman's rank correlation: $\rho = -0.538$, $p = .0017$)

in lower within-group diversity. The simultaneous observation of lower $N_{e,Z}/N_{e,A}$ ratios than expected under neutrality gives more credence to selection playing an important role.

While this broad survey confirms patterns suggesting an important role of selection on the Z in population differentiation, detailed examinations of "differentiation landscapes" (as measured by F_{ST}) across chromosomes provide a more complex picture. Patterns of differentiation in many cases (e.g., Delmore et al., 2015; Ellegren et al., 2012; Irwin, Alcaide, Delmore, Irwin, & Owens, 2016; Poelstra et al., 2014; Toews, Brelsford, Grossen, Milá, & Irwin, 2016; Toews, Taylor, et al., 2016) show high heterogeneity across most chromosomes, suggesting that other factors (aside from just sex linkage) play a more dominant role in affecting differentiation. While F_{ST} tends to have a higher mean (or median) across the Z than across autosomes, the highest F_{ST} peaks are often on autosomes rather than the Z (e.g., Irwin et al., 2016, in review; Poelstra et al., 2014). These observations lead to some tempering of the conclusion that Z tends to play a key role in speciation. Rather, it appears that the Z has a tendency to be subject to greater selective forces as well as drift, but because autosomes make up most of the genome and tend to have a higher effective population size (rendering equally strong selection more effective), autosomes may in many cases also play a key role.

6 | THE W CHROMOSOME

Most theoretical and observational research on the potential roles of sex chromosomes in speciation has focused on the Z (or X), but it is conceivable that the nonrecombining part of the W (or Y) may play an even greater role. The fewer functional genes, lower mutation rate (because not passed through males) and usually smaller size of the W likely contribute to less interest from researchers compared to the Z. Because of its absence in males, the W is perceived as less subject to sexual selection, often invoked as a driving force in speciation. Furthermore, its lower effective population size (1/4 that of autosomes, all else being equal) is expected to shift the relative influence of drift vs. selection in the direction of drift. However, the little observational work that has been performed to date suggests the W may be highly involved in speciation, and there are in fact strong theoretical reasons to think it should be.

In a particularly thorough (and perhaps only) analysis of population genomic variation of the W within and between sister species, Smeds et al. (2015) analysed four species of *Ficedula* flycatchers and showed that the nonrecombining part of the W chromosome has much higher F_{ST} and vastly lower within-species nucleotide diversity (π) compared to both the Z chromosome and the autosomes. Even after accounting for the lower effective population size and lower mutation rate on the W, levels of within-population variation on the W were nine to 13 times lower than on autosomes (Smeds et al., 2015; see Berlin & Ellegren, 2004 for a similar observation in the chicken W chromosome). Positive selection for particular variants on the W can easily account for such a pattern, as selective sweeps of just a single point mutation would affect the whole nonrecombining part of the W. It is also possible that background (i.e., negative) selection (Charlesworth, Morgan, & Charlesworth, 1993) contributes to reduced variation on the W (Smeds et al., 2015), although simulations of background selection to date fail to show an effect sizeable enough to account for this dramatic a reduction in diversity (Charlesworth et al., 1993; Zeng & Charlesworth, 2011; Zeng & Corcoran, 2015). There is strong evidence for purifying selection maintaining the functions and expression levels of essential W genes (Bellott et al., 2017; Smeds et al., 2015; Wright et al., 2014), but there could still be occasional mutations that are favourable and cause selective sweeps (Berlin & Ellegren, 2004). The high F_{ST} between populations may then be explained by selective sweeps that are local, responding to different selection in the two populations.

The potential for selective sweeps affecting the nonrecombining part of the W is further enhanced by the evolutionary dynamics of the mitochondrial genome. These two genomic elements are not physically linked but in birds and many other groups are nonetheless perfectly co-inherited through the matriline, causing the two to have identical genealogies (Berlin & Ellegren, 2001; Smeds et al., 2015). Hence, any favourable mutation on either the W or the mitochondrial genome can cause parallel sweeps through the population, reducing diversity at both genomic elements. Building evidence indicates that genes encoded on the mitochondria can differ functionally between different populations (Ballard & Whitlock, 2004; Hill, 2016;

TABLE 3 Comparisons of Z chromosomes and autosomes in terms of estimated within-population diversity (π) and/or between-population relative differentiation (F_{ST}), produced by a literature survey of available avian population genetic/genomic data

Species	π_Z	π_A	π_Z/π_A	π_Z^*	π_Z^*/π_A	$F_{ST,Z}$	$F_{ST,A}$	Method	References
Mallard, *Anas platyrhynchos*	0.0038	0.0057	0.667	0.00345	0.606	0.088	0.017	ddRAD	(Lavretsky et al., 2015)
Mexican duck (USA pop.), *A. diazi*	0.0035	0.0057	0.614	0.00318	0.558				
Grey teal, *Anas gracilis*	0.0014	0.0094	0.149	0.00127	0.135	0.281	0.0056	loci: 7 Z, 17 A	(Dhami et al., 2016)
Chestnut teal, *A. castanea*	0.0014	0.0093	0.151	0.00127	0.137				
Sage grouse, *Centrocercus urophasianus*						0.73	0.43	ddRAD	(Oyler-McCance et al., 2015) [c]
Gunnison grouse, *C. minimus*									
Chicken, *Gallus gallus*	0.0020	0.0065	0.308	0.00182	0.280			loci: 13 Z, 14 A	(Sundström et al., 2004)
Greater spotted eagle, *Clanga clanga*	0.00074	0.00130	0.569	0.00067	0.517	0.37	0.27	loci: 15 Z, 36 A	(Backström & Väli, 2011)
Lesser spotted eagle, *C. pomarina*	0.00076	0.00097	0.784	0.00069	0.712				
Ruff, *Calidris pugnax*	0.0013[a]	0.0021[a]	0.52		0.473			loci: 13 Z, 15 A	(Corl & Ellegren, 2012; Oyler-McCance et al., 2015)
Least sandpiper, *C. minutilla*	0.0030[a]	0.0054[a]	0.52		0.473			loci: 13 Z, 15 A	(Corl & Ellegren, 2012; Oyler-McCance et al., 2015)
White-rumped sandpiper, *C. fuscicollis*	0.0032[a]	0.0072[a]	0.45		0.409			loci: 13 Z, 15 A	(Corl & Ellegren, 2012; Oyler-McCance et al., 2015)
Pectoral sandpiper, *C. melanotos*	0.0046[a]	0.0081[a]	0.57		0.518			loci: 13 Z, 15 A	(Corl & Ellegren, 2012; Oyler-McCance et al., 2015)
Western sandpiper, *C. mauri*	0.0025[a]	0.0040[a]	0.58		0.527			loci: 13 Z, 15 A	(Corl & Ellegren, 2012; Oyler-McCance et al., 2015)
Red-necked phalarope, *Phalaropus lobatus*	0.0093[a]	0.0135[a]	0.69		0.627			loci: 13 Z, 15 A	(Corl & Ellegren, 2012; Oyler-McCance et al., 2015)
Carrion crow (in Spain), *Corvus corone*	0.00105	0.00142	0.739	0.00095	0.672	0.2760	0.1554	WGS	(Poelstra et al., 2014)
Hooded crow (in Poland), *C. cornix*	0.00118	0.00156	0.756	0.00107	0.688				
Greenish warbler (*viridanus* form), *Phylloscopus trochiloides viridanus*	0.00118	0.00207	0.567	0.00107	0.516	0.52	0.33	GBS	(Irwin et al., 2016)
Two-barred [greenish] warbler, *P. [t.] plumbeitarsus*	0.00182	0.00288	0.632	0.00165	0.575				
Swainson's thrush (west coast), *Catharus ustulatus ustulatus*						0.17	0.09	WGS	(Delmore et al., 2015)
Swainson's thrush (interior), *C. u. swainsoni*									
Pied flycatcher, *Ficedula hypoleuca*	0.0023	0.0036	0.639	0.00209	0.581	0.22	0.12	WGS	(Van Doren et al., 2017; based on data from Smeds et al., 2015)
Collared flycatcher, *F. albicollis*	0.0035	0.0045	0.778	0.00318	0.707				

(Continues)

TABLE 3 (Continued)

Species	π_Z	π_A	π_Z/π_A	π_Z^*	π_Z^*/π_A	$F_{ST,Z}$	$F_{ST,A}$	Method	References
Thrush nightingale, *Luscinia luscinia*	0.00343	0.0085	0.404	0.00312	0.367	0.783	0.298	loci: 4 Z, 8 A	(Storchová et al., 2010)
Common nightingale, *L. megarhynchos*	0.00198	0.00796	0.249	0.00180	0.226				
African stonechat, *Saxicola torquatus*	0.0015	0.0022	0.682	0.00136	0.620	0.35[b]	0.27[b]	WGS	(Van Doren et al., 2017)
Siberian stonechat, *S. maurus*	0.0035	0.0048	0.729	0.00318	0.663				
House sparrow, *Passer domesticus*	0.0042	0.0073	0.575	0.00382	0.523	0.45	0.33	WGS	(see also Elgvin et al., 2011, 2017)
Spanish sparrow, *P. hispaniolensis*	0.0025	0.0051	0.490	0.00227	0.446				
Zebra Finch (mainland population), *Taeniopygia guttata castanotis*	0.0032	0.0109	0.294	0.00291	0.267	0.238	0.117	loci: 5 Z, 4 A	(Balakrishnan & Edwards, 2009)
Zebra finch (small island population), *T. g. guttata*	0.0003	0.0001	3.000	0.00027	2.727				
MacGillivray's warbler, *Geothlypis tolmiei*	0.00210	0.00249	0.842	0.00191	0.767	0.091	0.107	GBS	(Irwin et al., in review; see also Porter, 2015)
Mourning warbler, *G. philadelphia*	0.00216	0.00257	0.839	0.00197	0.765				
Audubon's warbler (northern), *Setophaga auduboni*	0.00184	0.00220	0.840	0.00168	0.766	0.099	0.088	GBS	(Irwin et al., in review; see also Toews, Brelsford, et al., 2016)
Myrtle warbler, *S. coronata*	0.00203	0.00231	0.878	0.00185	0.800				
Townsend's warbler, *Setophaga townsendi*	0.00129	0.00150	0.862	0.00118	0.786	0.133	0.105	GBS	(Irwin et al., in review; see also Kenyon, Alcaide, Toews, & Irwin, 2017)
Black-throated green warbler, *S. virens*	0.00130	0.00147	0.884	0.00119	0.806				
Other relevant cases									
Blue-winged/Golden-winged warblers, *Vermivora cyanoptera/V. chrysoptera*						Two of six peaks on Z		WGS	(Toews, Taylor, et al., 2016)
Capuchino seedeaters (multiple species), *Sporophila* spp.						10 of 25 peaks on Z		WGS	(Campagna et al., 2017)
Red-breasted/Yellow-bellied sapsuckers, *Sphyrapicus ruber/S. varius*						Three of nine peaks on Z		GBS	(Grossen, Seneviratne, Croll, & Irwin, 2016)
Black-capped/Carolina chickadee, *Poecile atricapillus/P. carolinensis*						Nine of 20 peaks on Z		GBS	(Taylor, Curry, et al., 2014)

[a]Nucleotide diversity estimated from θ, a statistic based on the number of segregating sites, rather than π, the mean number of pairwise differences (Corl & Ellegren, 2012).
[b]Estimated by eye from figure S1 of Van Doren et al. (2017).
[c]Nucleotide diversity was reported, but estimated without incorporating invariant sites, so I do not include those values in this analysis.

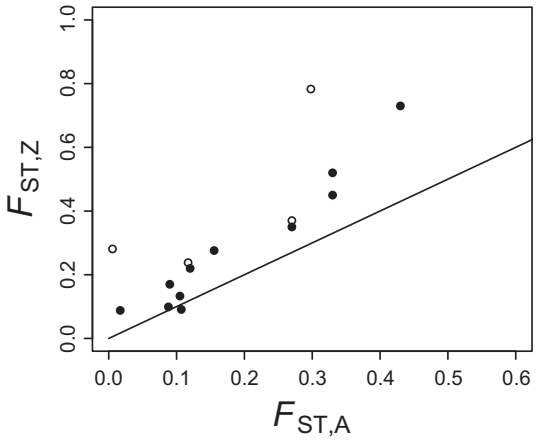

FIGURE 5 In studies of genetic differentiation between closely related bird populations, relative genetic differentiation tends to be higher on the Z chromosome ($F_{ST,Z}$) than on the autosomes ($F_{ST,A}$). Filled circles represent studies that used large-scale genomic sequencing, whereas open circles represent studies that used sequencing of a small number of loci. All but one case have larger F_{ST} on the Z (sign test: 14 of 15 ratios positive, $p = .00098$). The 1:1 line is shown

Irwin, 2012; Lee-Yaw, Jacobs, & Irwin, 2014; Morales, Pavlova, Joseph, & Sunnucks, 2015; Pavlova et al., 2013; Sunnucks, Morales, Lamb, Pavlova, & Greening, 2017; Toews, Mandic, Richards, & Irwin, 2014), suggesting that selective sweeps in mtDNA (and therefore the W) have occurred (this is further suggested by cases of apparent adaptive introgression; Toews & Brelsford, 2012). A further consequence is that such sweeps will occasionally carry along mildly deleterious alleles (i.e., the phenomenon of hitchhiking), contributing to functional degradation and raising the potential for subsequent sweeps of beneficial mutations that correct the function. In a similar way, evolutionary changes to genes on autosomes or on the Z chromosome, whether a result of advantageous or deleterious mutations, might induce compensatory changes in selection on the W and mitochondrial genomes. It is not difficult to imagine such processes occurring in two populations in different ways and leading to genetic incompatibilities between them.

7 | CONCLUSION

The sex-linked inheritance of sex chromosomes causes them to have different evolutionary dynamics compared to the autosomes and potentially allows them a greater role in speciation. A disproportionate role for the Z chromosome in speciation (compared to the autosomes) is predicted by multiple factors: a higher mutation rate (due to the Z being in males more than females), higher density of genes encoding sex-related traits, greater probability of fixation for some types of mutations, the potential for the evolution of sexually antagonistic loci and the potential for the evolution of mate preferences related to Z-linked traits. This conclusion is consistent with the observed tendency for the Z to show higher F_{ST} than autosomes (Figure 5), as well as the many case studies in which traits important to speciation have been linked to the Z.

While the evidence is reasonably strong for a disproportionate role of the Z in evolution and speciation, the jury is still out regarding the relative importance of deleterious vs. advantageous mutations in this process. Many of the most reliably estimated $N_{e,Z}/N_{e,A}$ ratios (those from high-throughput sequencing; Figure 4) are reasonably consistent with those predicted under neutrality, but there are a variety of ways that beneficial mutations could still be involved in the process (most simply, if they affect both the Z and autosomes) while not resulting in a very low $N_{e,Z}/N_{e,A}$ ratio. Specific case studies reviewed above (e.g., the Gouldian Finches; Pryke, 2010) have indicated positive selection on some Z-linked loci. I think the key question is not whether beneficial or deleterious mutations are involved in Z chromosome evolution (surely they must both be), but rather their relative importance and impact on within-population evolution and on the speciation process. Intriguingly, theory predicts the evolution of stable sexually antagonistic polymorphisms (Figure 2), a result which has received little attention in discussions of the Faster-Z affect; I emphasize that this these stable polymorphisms may preserve diversity on the Z, counteracting the reduction through low effective population size and selective sweeps.

While the Z chromosome receives more research attention than the W, there are reasons to think the W may in fact be more important in the speciation process. Because of the lower effective population size, drift presumably plays a dramatically larger role, such that deleterious mutations have a higher fixation rate. However, the few remaining genes on the nonrecombining part of the W (e.g., in the Collared Flycatcher *Ficedula albicollis*, 46 genes on the nonrecombining W vs. approximately 600 on the Z, plus 20 in the PAR of both; Smeds et al., 2015) appear to play very important functional roles (i.e., that is why they have been conserved), suggesting mutations could have large fitness effects. In the few cases studied, levels of variation in the nonrecombining part of the W are far lower than predicted based on neutrality, suggesting frequent selective sweeps. Because of the complete co-inheritance of the W and the mitochondrial genome, the ultimate cause of diversity-reducing selective sweeps could be on the mitochondrial genome rather than on the W.

The conclusion that the sex chromosomes are likely to play disproportionate roles in speciation must be tempered by one important fact: they make up a relatively small proportion of the genome. The large size of the autosomes (all together) implies that autosomal variation can also be very important in the speciation process, and case studies of speciation genomics often show high F_{ST} peaks on the autosomes. The literature on the theory of sex chromosome evolution tends to emphasize the ways that sex chromosomes may evolve faster or play a larger role in speciation than the autosomes, but this literature has also revealed ways the autosomes might be more important. For example, dominant or partially dominant beneficial mutations accumulate faster on the autosomes than on the Z, and autosomal preference genes for a W-linked sexually antagonistic trait will tend to fix for an allele conferring preference for the female-benefitting trait. Altogether, there is

much evidence indicative of a disproportionate influence of the Z (and perhaps W) in speciation but that still leaves much room for the autosomes to also play important roles.

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AUTHOR CONTRIBUTION

D.E.I. designed the study, conducted the literature review, analyzed the data, and wrote the paper.

DATA ACCESSIBILITY

All data presented in the article is contained in Table 3 and/or the text.

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