How to approach the study of syndromes in macroevolution

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ABSTRACT

Syndromes, wherein multiple traits evolve convergently in response to a shared selective driver, form a central concept in ecology and evolution. Recent work has questioned the utility and indeed the existence of some of the classic syndromes, such as pollination and seed dispersal syndromes. Here, we discuss some of the major issues that have plagued research into syndromes in macroevolution. First, observation of co-evolving traits (sometimes called “trait syndromes") is often used as evidence of adaptation to a particular driver, even when the link between traits and adaptation is not well-tested. Second, the study of syndromes often uses a biased sampling approach, focusing on the most extreme examples, which may obscure significant continuous variation between traits. Finally, researchers often focus on the traits that are easiest to measure even though these may not be the most directly relevant to adaptive hypotheses. We argue that these issues can be avoided by combining macroevolutionary studies of trait variation across entire clades with explicit tests of adaptive hypotheses, and that taking this approach will lead to a better understanding of syndrome-like evolution and its drivers.

INTRODUCTION

One of the most striking and commonly studied phenomena in biology is that of convergent evolution, whereby distantly related species evolve similar phenotypes as adaptations to similar selective pressures (e.g., Darwin 1859; Ollerton et al. 2009; Waser et al. 2011). When this convergence involves multiple traits, it is often called a “syndrome”. Many syndromes have been described in the literature, especially in plants (e.g., pollination syndromes, dispersal syndromes, succulent syndromes; Janson 1983; Waser et al. 1996; Ogburn and Edwards 2009) but also in animals where they are sometimes referred to by other terms (e.g., *Anolis* lizard ecomorphs, Beuttell and Losos 1999). In addition to its breadth of application across taxonomic groups, the concept of a “syndrome” is applied to a wide variety of scenarios, including traits fixed in a population or species (e.g., the repeated loss of eyes and pigmentation in cave fish; Strecker et al. 2012), polymorphic traits (such as behavioral syndromes or personalities; Sih et al. 2004) and plastic traits (e.g., plant defense syndromes; Agrawal and Fishbein 2006). Furthermore, syndromes can be restricted to a single lineage or a small number of lineages (e.g., tree lobsters; Buckley et al. 2009) or may have evolved many times across diverse clades of the tree of life (e.g., flight; Rayner 1988; Dudley 2002).

Despite this abundance of interest in syndromes in ecology and evolution, building a broad and balanced understanding of the role of selective agents in driving convergent, multi-trait evolution has been challenging due to the diversity of ways in which researchers approach the subject. First, discrete and well-defined syndromes are often presupposed to be present, leading researchers to focus sampling on extreme examples that comport with preconceived ideas about the existence of syndromes, e.g. a “hummingbird flower” versus a “bee flower”. This circular thinking ignores the many species that occur in between these ends of the continuum. A continuum of variation exists in flowers (Ollerton et al. 2009), fleshy fruits (Janson 1983; Sinnott-Armstrong et al. 2018), dry fruits (Wojewódzka et al. 2019), dispersal traits of terrestrial animals (Stevens et al. 2014), migration-associated traits in birds (Piersma et al. 2005), and body morphology in sea snakes (Sanders et al. 2013), among others, suggesting that continuous distributions in traits associated with syndromes are common. Second, the traits chosen for study in the context of a hypothesized syndrome tend to vary across studies. For example, one study of pollination syndromes might focus on color and flower size while another might examine corolla tube length and nectar sugar content. While many traits could be involved in a syndrome, this variation in traits studied raises the possibility that traits are cherry-picked to conform with the preconceived syndromes rather than selected to test for syndromes. Inconsistency of studied traits occurs in a variety of syndromes, including pollination syndromes (Ollerton et al. 2009), seed dispersal syndromes (Valenta and Nevo 2020), pace-of-life syndromes (Royauté et al. 2018), island syndromes (Raia et al. 2010; Jameson 2020), and others.

An equally challenging issue with studies of syndromes is the prevalence of ‘just-so’ stories, where adaptive significance is assigned without concrete evidence (Gould and Lewontin 1979; Olson and Arroyo-Santos 2015). Much of the research on syndromes focuses on the search for a pattern in traits (e.g., correlated evolution) but fails to collect data pertaining to a link between those traits and the putative evolutionary driver. Much of the allure of syndromes comes from the desire to infer adaptation from easily-observed traits, especially when the work of determining the evolutionary driver is challenging, as it usually is. However, observations of a pattern of correlated traits is not sufficient evidence that those traits are in fact adapted to a proposed, but untested, evolutionary driver — and, critically, the correlated evolution of *multiple* traits does not necessarily increase confidence in the inference of adaptation unless those traits evolve independently.

In the literature, three key criteria characterize syndromes across systems, scales, and clades: (1) convergent evolution of traits; (2) multiple traits; and (3) adaptation of those traits to a selective driver (Figure 1). However, studies of syndromes regularly do not address all three of these features. For instance, many studies rely on only a single trait as a proxy for a syndrome, such as color; and studies that do include multiple traits typically map them onto a phylogeny without testing for correlated evolution (e.g., Tripp and Manos 2008; de Vos et al. 2013; Gillespie et al. 2018). Additionally, a suite of co-evolving traits are often identified and an adaptive function proposed without an explicit test of that function (e.g., Buckley et al. 2009). Demonstrating adaptation of traits to a particular ecological function can be very challenging, and we do not suggest that every individual study of syndromes must include all three of these components. “Trait syndromes” consisting largely of a set of co-evolving traits with untested function (Aspinwall et al. 2013) are commonly described. Rather, we emphasize that the comprehensiveness of our knowledge about any particular syndrome should be carefully considered rather than assumed to be true.

In the context of these problems with the study of syndromes historically, we describe here an approach to studying syndromes that addresses these challenges and encourages more rigorous research into multi-trait evolution. First, we illustrate a hypothetical example that demonstrates a common approach to studying syndromes, which fails to adequately test for any of the features of a syndrome. Then, we argue that trait syndromes should be considered *hypotheses* of adaptation to a selective driver, rather than assumed to be *evidence* of adaptation. We describe how sampling biases can create the illusion of syndromes, and how appropriate selection of traits can illuminate evolutionary drivers. Finally, we propose an approach to studying syndromes that overcomes these problems and allows us to build evidence-based evolutionary narratives about syndromes of traits and adaptation.

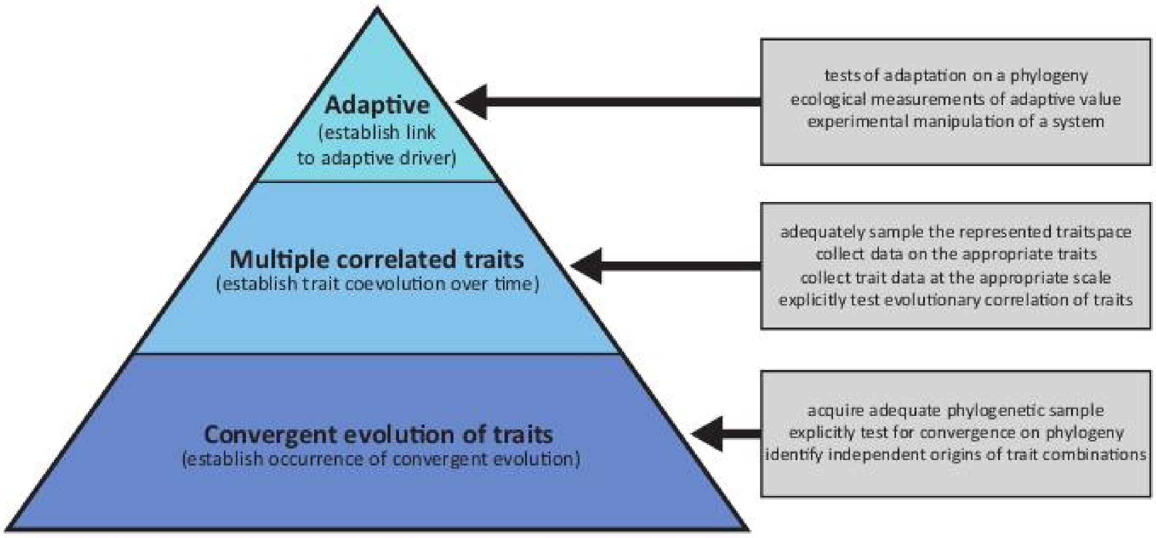

Figure 1 Schematic illustration of the three main features of syndromes (adaptation, multiple correlated traits, and convergent evolution of traits) as well as a sample of methods for demonstrating each of those features within a study system.

Setting the stage with a hypothetical case study

Let us travel, for a moment, to a hypothetical archipelago. On the first island, we find a small, iridescent beetle and a large, black beetle. The small iridescent beetle runs along branches and munches on leaves, while the large black beetle buries itself in the leaf litter and eats small insects. On the next island, we find a similar pair of beetle species — small and iridescent, large and black. We observe these beetles on each island for several months, and begin to suspect that the iridescence provides camouflage in variable light environments such as occur on exposed branches (Kjernsmo et al. 2020). Black color may provide camouflage against the leaf litter, enabling the large beetle to forage on the ground undetected. After wrapping up field work, we write it up: new beetle syndromes on islands, possibly adapted to foraging style!

Syndromes are commonly discovered and first described in this fashion, with covarying traits across species and observations of potential drivers. Often, a body of literature is built on the assumption of an adaptive connection. However, there are problems with this approach. As described above, syndromes have three primary features: convergent evolution, multiple traits, and adaptation to an evolutionary driver (Figure 1). In this hypothetical example, we have not explicitly tested for any of these features. First, we have not tested for convergence of the traits we observed. Without knowing the relationships between species, we cannot say that convergence has happened. Second, we have not established that changes in the traits are correlated in a phylogenetic context, in part because we have not sampled species beyond the few pairs that were the focus of our hypothetical study. Third, we did not test the association between traits and potential drivers. Simply observing a set of traits that cluster in trait space is not evidence that an adaptive connection exists without additional data. Below, we describe three downstream problems with the sloppy approach to describing new syndromes that have occurred in our hypothetical example.

Correlated traits alone are weak evidence of adaptation

One of the major problems in our hypothetical example is that we did not test explicitly for convergence of traits along a phylogeny. Because traits may be shared due to common ancestry rather than adaptation, failing to test for convergence can give the impression of a strong relationship between traits and adaptation that are not a result of selection acting on independent lineages. For instance, we might travel to a third island and observe another, similar pair of beetles. We might assume that, because they share a similar morphology, they also have the same foraging styles as our original species pairs. However, without other data — including how many times this set of correlated traits evolved, and more explicit testing of the adaptive function — the simple correlation between traits is only weak evidence of adaptation to foraging style. In building an adaptive story from the limited observation and testing of only a small number of species pairs, we may simply be testing whether these different species are different (Garland Jr. and Adolph 1994) — not whether they are adapted to the driver that we propose.

Making this leap from trait covariation to adaptive explanation may seem far fetched, but it is common practice. Many syndrome studies rely on data about adaptation that is a mix of peer-reviewed papers, personal observations, and inferred evolutionary drivers based on an organism's traits (see, e.g., Bruneau 1997; Hingston and McQuillan 2000; Whittall and Hodges 2007; Lomáscolo et al. 2008; Goolsby 2017; Valenta et al. 2018). The more iterations of this type of unsubstantiated inference, the more difficult it becomes for downstream users of the information to determine the quality of evidence supporting the putative syndrome. Recent examples illustrate how some phenomena, long believed to be true, were based largely on limited observations that turn out to be incorrect or incomplete when examined more comprehensively (e.g., migratory syndrome in birds, Piersma et al. 2005; territoriality in lizards, Kamath 2017). Personal observations contribute vital information to evolution and ecology, but should be treated as observations, not generalizable principles.

Demonstrating adaptation is often challenging and time-consuming, and consequently a variety of methods and approaches can be used to study adaptation. Repeated evolution —especially across multiple traits — is one line of evidence for adaptation (Pagel 1999), but in our view explicit tests of adaptation are crucial. Due to the varied nature of syndromes and their selective drivers, a variety of methods for demonstrating adaptation are valid, including field observations, laboratory experiments, phylogenetic comparative methods, and other approaches. For instance, finding clustering in trait space that corresponds with the putative selective driver (e.g., Ollerton et al. 2009; Agrawal 2020), especially if distinct gaps are observed between optima, is one line of evidence that the syndrome is adaptive to that driver. Identifying correlated evolution along a phylogeny is another line of evidence (Pagel 1994, 1999) — if the trait syndrome itself is not used to infer the driver.

Selecting appropriate traits

A second major problem with our hypothetical example is that we did not carefully select the traits we measured. Without careful consideration of our hypothesis about the relationship between our traits and putative driver/s, we may not choose to study the traits that are most relevant to testing for a link between those traits and that putative driver. In our hypothetical example, let us say that we have looked at the community of beetles on our two islands and observed that beetle size is correlated with habitat (branches vs. leaf litter) but color is not (perhaps we have since found iridescent beetles in the leaf litter and black beetles in variable light environments). We spend several years observing these beetles in their natural habitats, and eventually notice that beetles foraging on branches (which we once thought were camouflaged with iridescent coloring) have a peculiar behavior: they tap the branch several times before scurrying out to munch on leaves, upside down. This new observation leads us to consider other traits that might be adapted to this foraging lifestyle. We find that foot morphology, behavior, body size, and some vascular features do reliably differentiate between branch-foraging and litter-foraging beetles. In fact, these other traits are highly predictive of foraging style across beetles of a variety of colors.

What happened in this extension of our hypothetical example? The obvious trait —coloration — is easily measured. But, the important traits are more difficult to quantify, requiring behavioral assays and microscopic analyses of foot morphology and blood vessel diameter. This may seem obvious from the safety of our office chairs, but measuring the relevant traits, at the appropriate scale, is critical to evaluating adaptation. A classic example of this need occurs in UV nectar guides in flowers: humans cannot see UV, but bees can. Until the scientific community could measure UV reflectance, UV nectar guides remained unknown, yet are central to the ability of bees to find and pollinate flowers (Hansen et al. 2012). As another example, nectar viscosity (which is partly a function of its sugar content) affects the rate at which nectar can be sucked up by pollinators, and consequently has a strong influence on the rate at which energy is acquired from that nectar (Pattrick et al. 2020) but is measured less often than other floral traits (Parachnowitsch et al. 2019). Thus, assessing adaptation requires measuring the relevant traits, rather than simply traits that are easy for humans to observe and measure.

While it is clear that studying systems at a natural and relevant scale is essential for understanding syndrome evolution, the method for identifying the appropriate scale is not always obvious, and some exploration will usually be useful (Agrawal 2017, 2020). Iterative work, building on previous hypotheses and evidence, is usually necessary to identify the relevant traits (Olson and Arroyo-Santos 2015). Measuring the appropriate traits in the appropriate context is critical, both in terms of the relevant traits for adaptation as well as the relevant biological scale (Endler 1990). Thus, we encourage readers to carefully consider the biological scale of analysis, and the scale at which convergence occurs, that will yield the most insight into their syndromes of interest.

Mitigating sampling bias

A third major issue in our example is that our dataset is biased to detect differences, rather than to test for an adaptive link between traits and putative drivers. Our hypothetical example illustrates a common story behind the description of a new syndrome. However, this path to describing a new syndrome ignores the sampling bias inherent in such a study, namely, focusing on species with clear differences rather than sampling agnostically to those differences (e.g. by sampling broadly evolutionarily or ecologically). In our example, each putative syndrome (iridescent and small, black and large) may be inherited from a common ancestor rather than independently adapted to a foraging lifestyle (Felsenstein 1985). Observing multiple species pairs with these differences erroneously increases our confidence in the adaptive value of the syndromes without knowledge of the evolutionary history of these species and traits. Consequently, more comprehensive sampling, at the clade level and/or community level, is needed to provide strong evidence of syndrome-like evolution and adaptation.

Broader, more comprehensive sampling enables the detection of multiple origins of a collection of traits, increases confidence in the inferred evolutionary patterns, and provides power for testing adaptive hypotheses. For example, *Anolis* lizard ecomorphs have evolved multiple times, but always on islands — continental *Anolis* species differ in morphology from island species (Pinto et al. 2008). By studying the evolution of *Anolis* morphology on a phylogeny, the multiple origins are identifiable and the differing patterns on islands vs. mainland becomes clear. In flowers, pollination syndromes are often thought of as discrete, multivariate optima, but more complete sampling reveals that floral traits rarely match up exactly with the platonic “ideal” of discrete optima (Smith et al. 2008, 2009; Tripp and Manos 2008; Ollerton et al. 2009). Broad phylogenetic sampling reveals these trait continua, and also enables the identification of independent origins in order to count the true sample size — which is necessary to determine the strength of the evidence across multiple sampled species. Identifying independent origins is important for avoiding Darwin’s scenario, wherein correlated trait variation is taken as evidence for adaptive significance when it is simply due to common ancestry (Maddison and Fitzjohn 2015). Although it is not impossible to assess adaptive hypotheses for trait combinations that have arisen only a single time, phylogenies large enough to capture multiple origins of a syndrome will have much greater power to move toward assessing causal relationships (Uyeda et al. 2018).

Studies of syndromes in their ecological context can address similar questions about trait variation and adaptation. By sampling entire communities, we can identify traits that co-vary and the degree to which those trait combinations are associated with putative selective drivers. Indeed, early evidence for the classic mammal and bird fruit dispersal syndromes is based on ecological surveys (Snow 1981; Janson 1983), although community-level studies have frequently been the source of evidence against the syndromes (Hingston and McQuillan 2000; Wang et al. 2020). It's important to note that even in a community context, trait variation is shaped by evolutionary history, which accordingly should be incorporated when estimating the strength of correlated evolution and its relation to potential drivers (Webb et al. 2002; Cavender-Bares et al. 2009).

Connecting to evolutionary processes

In our opinion, the popularity of syndromes in ecology and evolution is in large part due to their connection to adaptation, which makes it all the more surprising that this connection regularly remains untested. Even if the pattern of repeated, convergent evolution reflects adaptation (Pagel 1999), additional evidence is required to support particular drivers and to reject others (Smith et al. 2008). There are diverse approaches for probing the connection between syndromes and selective drivers, ranging from field observations to manipulative experiments and phylogenetic comparative analyses. As noted above, finding clustering in trait space or correlated evolution along a phylogeny (Pagel 1994, 1999) that correlates with the putative driver (e.g., Ollerton et al. 2009; Agrawal 2020) can support an adaptive connection. Demonstrating whether syndromes can predict their putative driver is another path to collecting evidence pertaining to the question of adaptation. For example, observing that carnivorous plants only occur in low-nutrient environments is evidence that carnivory may be especially adapted to low-nutrient environments (Ellison and Adamec 2018). In our hypothetical beetle example, if we observe that the small and iridescent beetles always occur in the same habitat, and never occur in other habitats, that is evidence that particular phenotypes are adapted to certain habitats. Confirming this, however, requires seeking data to test the hypothesis of exclusive occurrence of that phenotype in that habitat — by examining other habitats as well.

It is equally important to recognize that patterns of trait variation are shaped by many factors beyond adaptation, which can complicate but also enrich the study of syndromes. For example, historical contingency is well known to shape adaptive trajectories, having a strong effect on phenotypic outcomes at various scales (Blount et al. 2008; Harms and Thornton 2014; McGlothlin et al. 2016; Xie et al 2021). These historical effects can also limit the trait space that is accessible to a lineage, and result in incomplete convergence (Grossnickle et al. 2020). Even when convergence is complete, the coordinated evolution of multiple traits can occur without coordinated selection on those traits. For example, traits like flower length and width are genetically correlated, which could explain, at least in part, their coordinated evolution at the macroevolutionary scale (Smith 2016; Wessinger and Hileman 2016). Dissecting the interplay of genetic architecture and the multi-trait response to selection has a long history in quantitative genetics (Lande 1979; Chevreud 1984; Saltz et al. 2017), and merits greater integration with the study of classic syndrome traits.

CONCLUSIONS

Despite widespread interest in syndromes among evolutionary biologists, their study has been haphazard, unsystematic, and rife with circularity. Here, we outline three major problems with the ways that syndromes have been treated in evolutionary biology. (1) Trait syndromes have been used as evidence of adaptation, but should be considered hypotheses that must be tested. (2) Traits that are easy to measure are often used in place of traits with direct relevance to the proposed adaptive driver. And (3), syndromes are identified based on biased samples of the most morphologically divergent species without consideration of whether those trait combinations represent true optima. Together, these issues have meant that syndromes of traits are regularly described and attributed to an adaptive driver with little evidence actually linking the two.

Through studying syndromes more rigorously, many questions about syndrome evolution, convergence, and adaptation become accessible. What is the evolutionary trajectory of different syndromes — do traits evolve in the same order, or in different orders, as a syndrome is assembled? To what extent do pleiotropy and other genetic linkage mechanisms explain the emergence of syndromes? To what extent are traits forming syndromes convergent across scales (e.g., genetic, protein, cellular, morphological, etc.) and to what extent do species evolve unique solutions to adaptive problems? Some theoretical questions about syndromes also remain. For instance, are there synergistic interactions between individual traits of a syndrome such that their combined contributions are greater than the sum of its parts? As traits related to a syndrome accumulate in a lineage, do they offer diminishing returns, such that a subset of traits is sufficient for adaptation to the selective driver? How does the relative size of fitness contributions from individual syndrome traits affect evolutionary trajectories and derived phenotypes? These kinds of questions, and more, are facilitated by rigorous study of syndromes.

CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

DATA ACCESSIBILITY STATEMENT

Any data associated with this manuscript will be uploaded to a permanent online repository such as Zenodo or Dryad.

AUTHOR CONTRIBUTIONS

CP, MSA, LCW, SL, RD, JH, and ADW conceived the review. LCW and MSA led the conceptual development of the content. MSA, LCW, CP, RD, SL, JH, and ADW conducted background research and wrote the first draft. MSA, RD, LCW, and SDS revised subsequent drafts. MSA and LCW wrote the final draft and generated the figure.

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