**Why behavioural syndromes exist: testing the constraint and adaptive hypotheses**

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**Abstract**Suites of correlated, functionally different, behaviour (‘behavioural syndrome’) have been described in many animal populations across different taxa. Yet, the existence of behavioural syndromes are puzzling from an evolutionary view as the tendency to behave consistently in multiple integrated behavioural traits may lead to suboptimal, even seemingly maladaptive, actions. Two hypotheses have been proposed to why behavioural syndromes occur. The constraint hypothesis suggests that syndromes originate from a carryover effect of a genetic correlation, which is not easily eradicated by evolution, thus constraining adaptation. In contrast the adaptive hypothesis states that behavioural syndromes result from an adaptive advantage a correlation might provide in a suitable environment. Several studies have been able to reject the constraint hypothesis, without making clear how syndromes can be adaptive. I will provide a review of approaches which have been used to test the two hypotheses with the goal to find out why studies have not provided a solid explanation why behavioural syndromes exist.

**Introduction**

Behavioural ecologist occasionally encounter animal behaviour that seems to be maladaptive. This is puzzling from an evolutionary perspective. For example, precopulatory sexual cannibalism is considered to be maladaptive, because the female eats the male before mating and therefore prevents fertilisation. However, it is possible to explain this puzzling behaviour if the correlation with aggression is taken into consideration. One hypothesis suggest that the precopulatory cannibalism is a spillover from aggressiveness in a context where it is beneficial (Johnson and Sih, 2005). In the fishing spider (*Dolomedes triton*) female individuals, which have an increased tendency towards cannibalism, were also more voracious hunters as juveniles and subsequently had a higher growth rate and increased fecundity (Johnson and Sih, 2005). Hence, the benefits of being aggressive as a juvenile might outweigh the cost of sporadic cannibalism later in life.

This example illustrates the importance of taking into account the multidimensional aspect of phenotype. It shows how the emergence and maintenance of apparently maladaptive behaviour can be explained by looking at the structure of the behavioural phenotype (i.e. correlations among distinct behavioural traits), while also recognizing consistent individual differences across contexts. Individual variation in behaviour is omnipresent within animal populations. Previously however, behavioural ecologists might have considered individual differences as deviations from an adaptive mean (Krebs and Davies, 1978). Behaviour in animals was considered to be primarily plastic and optimal. One major step was to realise that plasticity in behaviour is far less pronounced than thought, and various behavioural traits are regularly repeatable and heritable within individuals (Huntingford, 1976; Riechert, 1992). Recent studies have shown how individual differences are often consistent across contexts situations and highly structured with multiple correlated behavioural traits (Sih, 2004). It is now well established that a behavioural syndrome is a suite of correlated functionally separate behaviours which differ among individuals consistently and is stable over time. Recently, there is growing interest to understand how these behavioural syndromes emerge and are maintained within population and over time.

Two main approaches have been designed to answer why behavioural syndromes exist. The first is called the constraint hypothesis, which mainly explains a proximate mechanism for behavioural syndromes, focussing on how it can persist over time in populations. It predicts that a consistent genetic correlation, that cannot be easily broken, is underlying a syndrome. The correlation originates from a proximal link between traits, which could be caused by e.g. pleiotropic effects of genes or multi-target hormones. Therefore the genetic correlation could be the source of constraint on the evolutionary response. If selection favours one trait, but not the other, it constrains evolution from adaptation towards optimal traits. The behavioural syndrome would not emerge easily, because changes in the structure of behaviour would require changes in genes or evolution in the hormonal machinery (Bell, 2005)

The second approach is an ultimate explanation for how behavioural syndromes emerge. The adaptive hypothesis states that correlations emerge as an adaptive response of selection favouring a syndrome. A syndrome might arise because a particular arrangement of behavioural tendencies is favourable in a given environment. Behavioural syndromes can arise from a trade-off, involving limits to plasticity. There are costs and benefits involved in the expression of certain behaviour, which is correlated with other behaviour. In the example of the fishing spider, an individual might invest in aggression as a juvenile, because the benefits of acquiring more food weighs out the cost of precopulatory cannibalism later in life (Johnson and Sih, 2005). The individual is not able to restrain from sporadically eating its potential mate, because of limited plasticity.

The studies that tested these two evolutionary hypotheses draw contradicting conclusions and mainly targeted the constraint hypothesis (Herczeg and Garamszegi, 2012). So the question why behavioural syndromes exist still remains. In this paper I will discuss some of the attempts made to answer this question, and will review their strength and weakness. I will describe possible approaches to draw conclusions on the most promising way of approaching this problem. Finally, I will provide recommendations for future research on this topic.

**Behavioural syndromes: definition**

I support the definition referring to a behavioural syndrome as a correlation of rank-order differences between two of more personality traits of individuals across time and through contexts (Bell, 2007). It is therefore a property of the population. In contrast, studies also use the term “behavioural type”, which refers to the specific arrangement of behaviour an individual expresses. A “personality trait” is a type of behaviour, in which psychological mechanisms are underlying the expression. To avoid confusion, I will refrain from using terms as ‘animal personality’ or ‘temperament’. Behavioural syndromes have been used analogously with these terms, despite the difference in substance. Temperament or animal personality are more often used to describe repeatable behaviour of the individual and variation within the population for a single behavioural trait. Evidently, the terms overlap in what they represent. Nonetheless, behavioural syndromes ought to focus on the correlation of multiple behavioural traits.

Formally, a behavioural syndrome exists when the average phenotypes of some individuals in one context/situation is correlated with the average phenotype of the same individuals in a different context/situation, in a manner that populations harbour consistent individual variation on suites of correlated behaviours (Dingemanse, Dochtermann and Nakagawa, 2012). When phenotypic correlations in a population are observed, they are composed from both within-individual variation and between-individual variation (Dingemanse, Dochtermann and Nakagawa, 2012). Between-individual variation measures how much individuals differ from each other in their average phenotype, whereas within-individual variances measure how much repeated responses differ from each other within the same individual. Therefore, behavioural syndromes are not composed of phenotypic correlation due to within-individual variation. A behavioural syndrome is a property of the population, thus represented by between-individual variation.

**Individual consistency**

Consistency of behaviour has multi-layered definitions in animal ecology. Behavioural consistency can be interpreted as consistent individual differences in suites of functionally different behaviour within a population. This interpretation fits the description of a behavioural syndrome, Behavioural consistency can also be considered as a form of limited plasticity: individuals consistently show the same behaviour across temporal or contextual changes (Garamszegi and Herczeg, 2012). This is often an important aspect within a behavioural syndrome. Limited plasticity often lays at the core of observed maladaptive behaviour, since it might restrict the animal from behaving optimally. Furthermore, when animals are limited in the plasticity of their behaviour it might explain why certain correlations between multiple behavioural traits exist. In line with Darwinian reasoning several suggestions have been elaborated from an adaptive perspective to explain why individual consistency, i.e. limited plasticity, could exist (Dall, Houston and McNamara, 2004). First, it might be best for an individual to be consistent in order to deal with environmental uncertainty (McElreath and Strimling, 2006). Second, being predictable can provide social benefits. Consistency might persist when selection favours trustworthiness (McNamara *et al.*, 2009). Third, animals behave consistently when their behaviour is tied to a stable state. Adaptive behaviour can be dependent on a state variable such as size, energy reserves or life-history type.(Nicolaus *et al.*, 2012; Luttberg, 2017)*.* Additionally, several costs (maintenance, production, developmental) have been attributed to phenotypic plasticity(Hendry, Kinnison and Dewitt, 1998).

**Measurements of personality traits**

To study behavioural correlations, behaviour needs to be measured on a continuous scale. Five different personality trait categories were distinguished: shy-boldness, aggression, exploratory behaviour, activity and sociability (Réale *et al.*, 2007). These distinguishable traits are important to avoid the mistake of measuring the same functional behaviour and therefore falsely identifying a behavioural syndrome. Intuitively, we see how multiple personality traits might be expressed at the same moment in the same environment. This model allows researchers to connect standardized methods and guidelines to the measurements of personality traits. For example, exploratory behaviour should be tested in connection to a novelty-environment, while the measurement of boldness requires a response to a risky situation, possibly a predator. These personality traits are ranges of phenotypes, which are represented on a continuous scale. The shy individual does not portray a different trait than the bold individual, it is only a different phenotype of the same trait (Réale *et al.*, 2007). These five personality traits are often correlated with each other and with other behavioural tendencies with different ecological tasks such as parental care, dispersal or mating strategies (Duckworth and Badyaev, 2007; Johnson and Sih, 2007; Sih and Del Giudice, 2012). It has proven to be challenging to ensure that the measurement of behaviour is a true representation of particular personality traits.

An example of this problem and its consequences, is the study on behavioural syndromes in wild brown trout (*Salmo trutta*) by Adriaenssens and Johnsson (2013). The researchers performed three experiments in a fish tank, where individual brown trouts were exposed to 1) an empty field, 2) a novel object and 3) a mirror. Three personality traits were assigned to the expressed behaviour during the experiments: 1) activity 2) exploration and 3) aggression. Several features of the fish behaviour were scored during the tests, but eventually ‘average velocity’ was used for statistical analysis, because repeatability was highest for this measure. Several positive correlations were found between average velocity across the different tests. However, it is difficult to classify the measurement of the different experimental set-ups as separate types of behaviour. Rather, it shows correlations within a single trait, namely average swimming speed. Therefore, this leaves doubts to whether the experiment shows a behavioural syndrome in the wild or simply a variation in a personality trait which is highly consistent in individuals across contexts. This experiment should rather be categorised as an excellent study on animal temperament.

Repeatability represents individual consistency in a given behaviour. It is the proportion of the total phenotypic variation that is explained by variation among individuals. High repeatability is therefore observed by low within-individual variation relative to high between-individual variation. High repeatability does not instantly mean that behaviour is not plastic. When behaviour is optimally plastic, animals can theoretically still behave consistently, in accordance with a stable environment.

Repeatability usually gives indication to the heritability of a trait (Dochtermann, Schwab and Sih, 2015). However individual consistency can come from genetic and non-genetic resources: learning, maternal effects, environmental conditions. Repeatability does not allow a separation of the genetic and non-genetic components which explain the individual consistency. Heritability (h2) is a standard index determined as the proportion of the additive genetic variance as opposed to the total phenotypic variance (Réale *et al.*, 2007). The heritability of personality, referring to the proportion of personality variation attributable to additive genetic variance, has been shown to much higher than heritability of behaviour generally (Dochtermann, Schwab and Sih, 2015).

**Proximate mechanisms of behavioural syndromes**

The proximate explanation for behavioural syndromes is heavily associated with the constraint hypothesis, which proposes that the genetic correlation slow down or impede evolution as behavioural traits are not “free to evolve” (Bell, 2005). As a consequence, selection favouring a change in one type of behaviour might also alter a different type of behaviour.

To evaluate whether a syndrome structure can impose a constraint on adaptive evolution research was done on the genetic basis of the syndrome (Dochtermann and Dingemanse, 2013). An essential starting point to evolutionary research on behaviour was the heritability. A review of van Oers *et al.*, (2005) of behavioural studies shows that most traits are expected to be partly heritable. Next, to provide a better understanding on the evolvability of traits knowledge of the genetic structure of correlations is needed. The constraint hypothesis states genetic correlation between traits can constrain evolution as selection for one trait effects the selection response of the other. Genetic correlations could be due to pleiotropic effects. Pleiotropy occurs when a single gene influences the phenotypic expression of two of more traits. If the genetic correlations as the foundation of the behavioural syndromes are due to pleiotropy, selection will not be able to easily disintegrate the correlation. Therefore pleiotropy can be major cause of long-term evolutionary constraints (Ketterson and Nolan, 1999; Dochtermann and Dingemanse, 2013). Alternatively one could apply the view that maladaptive pleiotropic effects ought to be altered over time by selection through modifier genes (Ketterson and Nolan, 1999; Sih, 2004). However this is generally considered to be a coincidental and slow process, since it requires gene duplication or other major changes at molecular level (Dochtermann and Dingemanse, 2013)

In addition, linkage disequilibrium can also attribute to genetic correlations. Linkage disequilibrium is a non-random association of alleles at different loci in a population. This is influenced by many factors including selection and genetic drift and is therefore not restricted to only genetic linkage. Past correlational selection can result in so-called selection-induced linkage disequilibrium. (Dochtermann and Dingemanse, 2013). Thus, in this case, the correlations would be a result from adaptation to selective forces from the environment and would not be a constraint on evolution. Behavioural correlations should quickly dissolve when antagonistic directional selection is applied, if the correlations only emerged from selection-induced linkage disequilibrium. In an experiment this could be tested when artificial selection favouring a specific configuration of traits (Delph *et al.*, 2011). For example, in a population with highly aggressive and bold individuals, artificial selection favouring high level of aggression and shyness should break the syndrome if selection-induced linkage disequilibrium is the primary source of the genetic correlations.

A genetic basis is not always underlying a correlation among traits, as extragenetic inheritance can be a cause of a stable behavioural syndrome. The earliest environment of every animal is always influenced by its parents (Rossiter, 1996). All parental effects on phenotype, other than transmission genetic material, are encompassed by extragenetic inheritance (Stamps, 2003). Heritability studies have shown that multiple personality traits can be genetically inherited (Dochtermann, Schwab and Sih, 2015). However, research on rodents shows how maternal behaviour affects the development of a suite of correlated traits in their young (Meaney, 2001). A particular example involves the effect of parental care, amount of licking and nursing, on the syndrome of stress reactivity of juvenile rodents (Meaney, 2001). Furthermore, cross-fostering studies females raised by more intensive nursing mothers, also turn into mothers, which spend more time nursing (Fleming et al., 2002). This create a system in which a trait syndrome of high-licking and low-reactivity is maternally inherited in extragenetic ways.

**Testing the hypotheses**

Rejecting the constraint hypothesis directly implies the behavioural syndromes result from adaptive evolution. Otherwise it would exist by coincidence or a process yet unknown. However, when the constraint hypothesis is rejected, it is still unclear how the correlation is adaptive. As Bell (2005) described it: “a behavioural syndrome might be an integrated suite of behaviour that is the result of selection, not a constraint on it”. The correlation, itself, is a consequence of adaptive evolution of individuals within a population. To prove this idea, it is necessary to know the mechanisms of the adaptation.

A pioneer study of Huntingford (1976) on the threespined stickleback (*Gasterosteus aculeatus*) had major influence on consecutive behavioural syndrome studies. This study was first to show that individual were consistent in aggression and boldness and to explicitly describe the significant correlation in social aggression and antipredator behaviour, often referred to as the aggression-boldness syndrome. Individuals who were bold towards predators, were also territorially aggressive towards conspecifics. Huntingford speculated that the aggression and boldness should functionally co-vary during the breeding season as conspecifics prone to cannibalism pose a comparable threat as the carnivorous predators. Predation could serve as the acting selection pressure. A different antipredator strategy in high levels of predation might be behaving timidly and shy, minimising the chance of being noticed. During low levels of predation it would allow higher levels of aggression, as aggression prolongs courtship in the stickleback. This example already shows how predation risk can influence behavioural syndromes with varying degrees of adaptive behaviour.

Since Huntingford’s study, accumulating evidence suggests that increased selective pressures may favour the emergence of behavioural syndromes (Bell and Sih, 2007; Dingemanse *et al.*, 2007; Adriaenssens and Johnsson, 2013). According to the adaptive hypothesis, the correlation is expected to have a positive effect on the fitness of the individuals. Yet, fitness consequences have very rarely been included in studies on behavioural syndromes. This is strange, to say, since it will surely advance the field towards more solid inferences on the constraint and adaptive hypotheses. I will specify several fruitful approaches, which can be used to determine how fitness is related to a syndrome.

1) The first approach measures the fitness of behavioural types on a long term and combines this with detailed information on the ecology of the species. This combination of information can help explaining why a behavioural syndrome has evolved in a population. One long-term case study on the western bluebird (*Sialia mexicana*) provides an astonishingly complete description of how a particular behavioural syndrome, namely the dispersal syndrome, is manifested and maintained (Duckworth and Badyaev, 2007). It describes the positive correlation between aggression and dispersal during a range expansion, which appeared to be adaptive from a historical perspective. Only highly aggressive bluebirds were found in newly colonized habitat, whereas less aggressive population of bluebirds were found in older areas. The level of aggression, which was very consistent throughout a bluebird’s lifespan, slowly declined as the population aged. This was caused by selection; aggressive males produced less offspring and were therefore slowly replaced by less-aggressive individuals. They speculated that the ultimate causes of the coupling between aggression and dispersal originated from a trade-off between reproductive investment and ability to compete for breeding sites. Historically, the western bluebird relied on ephemeral habitat. Forest fires would suddenly and unpredictably provide new habitat. Then, only the most aggressive bluebirds were able to colonize the new breeding grounds. This study shows how long term fitness measurements of individuals with a specified behavioural type, together with a detailed view of the ecology of the species, can provide a likely explanation for a behavioural syndrome.

2) Recently Herczeg and Garamszegi (2012) proposed a new metric called ‘syndrome deviation’ which connects a value to the way an individual fits within the behavioural syndrome. It builds on the fact that a behavioural syndrome is defined as a rank-order correlation between two personality traits. Syndrome deviation required rank transformation of the two behavioural traits. The deviation is then calculated as the difference between its ranks and a perfect correlation. The syndrome deviation for an individual would be zero, if the ranks for both behaviours are the same. Herczeg and Garamszegi (2012) suggest that relating syndrome deviation to measures of fitness is a logical step to study the adaptiveness of a correlation. Based on the adaptive hypothesis, we can expect a non-zero syndrome deviation to be associated with negative fitness consequences. Thus, individuals with a higher syndrome deviation value should have a lower fitness. To my knowledge, syndrome deviation has not yet been used in recent studies. The concept have received criticism for not acknowledging the differences between phenotypic and between-individual correlations (representing behavioural syndromes), which reduces the applicability of syndrome deviation (Dingemanse *et al*., 2012). Also, the ecological meaning of syndrome deviation as a distinct phenotypic trait remains not fully explained.

3) The third approach involves an experiment in which the population is exposed a selection event (Bell and Sih, 2007)*.* When a population is exposed to, for example, predation, it is possible to formulate expectations according to the two hypotheses: constraint or adaptation (Bell and Sih, 2007). This approach will be discussed in paragraph ‘syndromes across populations’.

4)The fourth approach studies the development of individuals and measures related fitness. Also, a reaction norm experiment could be included where individuals develop in a range of environments. If the behavioural syndrome originates from a constraint, a correlation would be constant across development and similar in different environments. This approach will be discussed in paragraph ‘syndromes across development’.

**Syndromes across populations**

An useful method to test the constraint and adaptive hypotheses is comparison between populations in different environments (Bell, 2005; Dingemanse *et al.*, 2007; Pruitt *et al.*, 2008). A correlation between two traits is expected to be consistent among population if it originates from genetic linkage or pleiotropy. On the other hand, the adaptive hypothesis is supported when the correlations follows a predictable structure in accordance with the environmental gradient.

One specific example of a behavioural syndrome which appears to be widespread in various animal is the aggression-boldness syndrome (Huntingford, 1976; Dingemanse and Wolf, 2010). When this syndrome was studied in the threespined stickleback the researchers made predictions conforming to the two hypotheses (Bell, 2005). According to the constraint hypothesis levels of aggression and boldness should be positively correlated at the individual and population level, regardless of the environment. So, the population which is on average the most aggressive is also predicted to be the boldest. In contrast, according to the adaptive hypothesis, populations should not necessarily show the same syndromes and instead display correlations fitting to the environment. Depending on what selection has arranged, numerous scenarios are possible (Bell, 2005). 1) Two behavioural traits can be independent in individuals, however average behaviours are still positively correlated among populations (e.g. the more aggressive population is also boldest). 2) Traits can be positively correlated in one population, but negative in the next. 3) Behavioural traits can be independent in a given population, but positively correlated in another one.

After Huntingford (1976), many studies have assessed the two hypotheses by studying the threespined stickleback. This species occurs over most of the Northern hemispheres and inhabits many different freshwater and coastal environments. Populations of threespined sticklebacks have been tested for correlations between aggressive behaviour and boldness and between aggression and exploratory behaviour in environments with different predation levels. Results show that behavioural correlations differ significantly among various populations according to their selective environments (Bell, 2005; Dingemanse *et al.*, 2007). Explicitly, it became clear that syndromes were present in populations, when predation risk was high (Bell, 2005). Logically, an experiment was needed to determine if high predation pressure could cause the emergence of the aggression-boldness syndromes. To reach that goal, sticklebacks were exposed to predation and marked individual were measured before and after the predation event, during which half of the sticklebacks had been eaten by trout (Bell and Sih, 2007). After the predation event the population showed an aggression-boldness syndrome. The study concluded that the syndrome resulted from a combination of selection and behavioural plasticity. Intriguingly, the selection was not consistent with the emerged phenotypic correlation. In fact, the selection pressures were antagonistic: individuals with the highest survival rate were shy individuals and aggressive individual. Therefore, the question to why and how predation favours the aggression-boldness correlation remains partially unexplained. Nonetheless, the method of comparative studies among populations has provided supportive evidence for the adaptive hypothesis that behavioural correlations probably are adaptation to the environment.

**Syndromes across development**

I have shown that behavioural correlations can differ among populations, representing spatial variation. However temporal variation in syndromes may also exist when syndromes vary within individuals throughout their lifespan. Differences in syndrome structure across developmental stages may occur when juveniles are exposed to different selective pressures.

Consistency of behavioural syndromes over lifetime is often implied or explicitly named (Réale *et al.*, 2007). Despite individuals often being consistent in behaviour over time and across situations, they may not maintain the same rank in aspects of the behaviour as they age. Even more so, behavioural correlations are not always securely present in the same individual over its lifespan. Correlations among behavioural traits may change, appear or disappear as individuals age. The influence of individual development is important for understanding the mechanisms and evolutionary consequences of behavioural syndromes. This line of reasoning has been elaborately described in a review on ontogeny by Stamps (2003).

Behavioural development is the change in mechanisms responsible for producing behaviour, which is ongoing throughout the animal’s life. These processes do not cease when maturity is reached. The development of behaviour relies on a combination of genes and the environment. An idealized experiment to see how much of the environment contributes to the phenotype, would let animals with same genotype develop in an infinite number of different environments (Stamps, 2003). The result of this idealized experiment shows the reaction norm: the array of phenotypes that can be produced by an individual genotype along an environmental gradient (Schlichting and Pigliucci, 1998). Many studies on development are a simplified version of the ideal reaction norm experiment, where very closely related individuals are exposed to a few different environments (Dingemanse *et al.*, 2010).

A problem with the reaction norm experiment is also that, animals are not only influenced by their environment but what the individual experiences can also be largely influenced by the individual itself (Davis and Stamps, 2004). The animal can choose or shape its environment. Early choices of the most suitable environment can explain the behavioural syndromes present in populations of a species living in heterogeneous environments (Stamps, 2003). Therefore, the environment could be the cause of the behavioural syndrome and also behavioural syndromes can develop as a consequence of selecting a suitable environment. This relevant aspect is, for reasons of convenience, disregarded in typical reaction norm experiments. Nonetheless, selection or modification of the environment is important to consider when assessing our two hypotheses. For example, when three different behavioural correlations are observed in three populations living in different environments, one could conclude that the behavioural syndromes do not arise from a genetic correlation, thus not a constraint. However, it is possible that the environments had been selected by juvenile individuals to match their behavioural type. Therefore, when selection of environment is disregarded, it could lead to falsely rejecting the constraint hypothesis.

How can behavioural syndromes change over time when it is caused by a strong genetic correlation? A genotype-age interaction (GAI) affecting the expression of genes can change the underlying genetic correlation of a behavioural syndrome (Class and Brommer, 2015). When GAI occurs in one trait, it is likely that the genetic correlation between suites of traits changes accordingly, unless the expression of the traits are structured through pleiotropy. Alternatively, (independent of GAI) selection may cause a change in the presence of behavioural syndromes. For example correlational selection can be disruptive for adults and convergent in juveniles. Selection may favour the presence of a behavioural syndrome in juveniles, as not all survive, adulthood selection may eradicate the correlation. Selection can thus change through stages of developmental. Class and Brommer (2015) performed a long-term study to answer the question whether a change in behavioural syndromes during development is caused by selection or GAI. The aim was to compare the genetic correlation of two traits, handling aggression and breath rate of nestlings and adults of a Finnish blue tit (*Cyanistes caeruleus*) population. They hypothesised that if selection alters the magnitude of the correlation between these two traits, the behavioural syndrome of first year breeders should be similar to the adult and different from non-recruiting juveniles. The results showed that the strong genetic correlation in juveniles where handling aggression is negatively correlated with breath rate, was absent in adults. There was no significant difference in nestling that recruited and nestling which did not recruit. Since selection on the genetic correlation was not significant, GAI’s was likely the cause of the developmental change in the behavioural syndrome.

In conclusion, development is in multiples ways involved in the maintenance of behavioural syndromes. The environment juveniles are exposed to can steer individuals towards a certain behavioural phenotype. This environment might be selected by the individuals. When genetic correlation are underlying a behavioural syndrome, genotype-age interactions as well as changing correlational selection may lead to ontogenetic variation. Measurements of behavioural syndrome ought to include a lifetime perspective, because assuming these correlations are stable across development can lead to an overestimation of the strength of evolutionary constraints.

**Recommendations for future research**

Two main hypotheses are proposed to explain the existence of behavioural syndromes. Importantly, the constraint and adaptive hypotheses are not mutually exclusive. In practice, a behavioural syndrome could very well be the result of both adaptive evolution and be affected by genetic constraints. However, research has noticeably focussed on testing the constraint hypothesis, resulting in a gap in our understanding of behavioural syndromes.

One of the following two conditions need to be confirmed before accepting the adaptive hypothesis.

1. The syndrome occurs within a population as expected with its ecological situation.
2. The syndrome leads to fitness benefits.

The first condition has been tested repeatedly. However, it remains difficult to predict what syndrome should evolve in a given ecological situation. The dispersal syndrome in the American blue bird is the best example of an adaptive behavioural syndrome, of which the mechanisms for persistence are quite known (Duckworth and Badyaev, 2007). Mainly a trade-off situation is expected, where investment in a certain trait results in a correlation among multiple traits. An individual’s behavioural type may determine what kind of investment is made in a particular behavioural trait. For example, aggressive bluebirds have high competitive ability for breeding sites, but lower reproductive success (Duckworth and Badyaev, 2007). The developmental environment might determine the individuals behavioural type and thereby influencing the trade-off (Stamps, 2003). It is important to describe the components of the trade-off to explain how a correlation exists from an adaptive perspective. Furthermore, there is convincing evidence that a high selective regime can induce behavioural syndromes (Bell, 2005; Bell and Sih, 2007; Dingemanse *et al.*, 2007). However the exact underlying processes of the phenomenon are not yet fully developed. Which can obscure clear conclusions on the main question.

The second condition has, to my knowledge, not been tested (with the exception of the blue bird study by Duckworth and Badyaev (2007)). The main problem with answering whether a syndrome has fitness benefits, lays in the fact that a behavioural syndrome is a property of the population, instead of a phenotypic trait. However measurement of fitness, survival and reproductive success, ought to be more common in syndrome studies, as this may lead to revealing how the syndrome emerged and is maintained. Researchers have often tried to reject the constraint hypothesis by demonstrating how a behavioural syndrome can be broken or appear in different populations or environments. This has been successfully established in multiple species. Nevertheless, this does not prove that the behavioural syndrome is an adaptation, as it does not show the adaptive mechanism.

Behavioural syndromes has largely influenced the view on animal behaviour. A syndrome can include correlations with e.g. dispersal, predator-prey strategies and mating behaviour, therefore affecting key components of population functioning (Sih, 2004). Studying behavioural syndromes provides a more holistic insight into the functioning of a population than measurements on only one behavioural trait. Most importantly, solving the puzzle of how behavioural syndromes emerge and are maintained can help to better understand how evolution works.

This thesis has provided guidelines for studying behavioural syndromes and made recommendations to advance the field. I have described multiple approaches, shortcomings and strong points, which can be used to improve our knowledge on why behavioural syndromes exist.

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