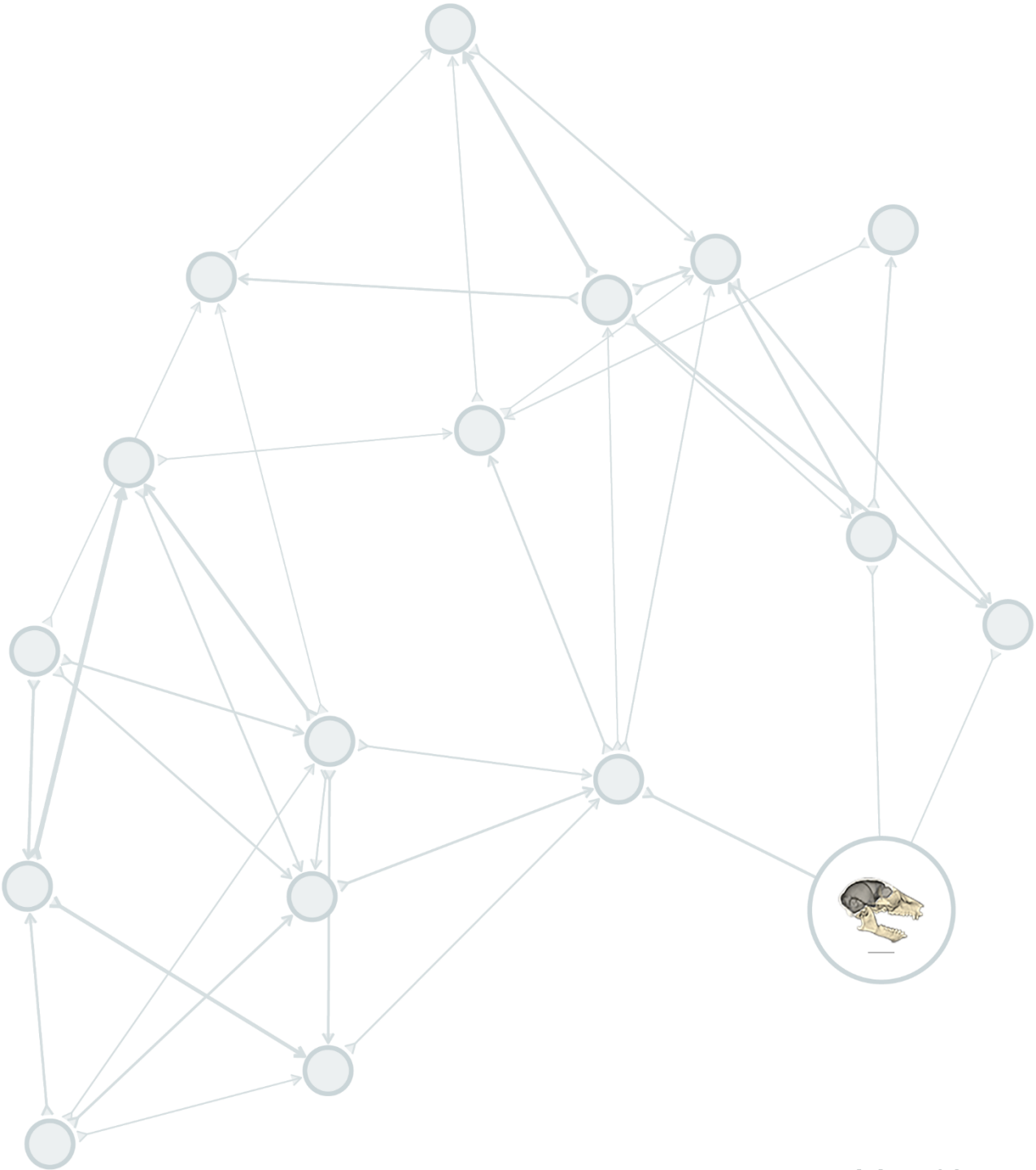
Understanding the Complex Causes of Primate Brain Evolution



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Understanding the Complex Causes of Primate Brain Evolution

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Abstract

Understanding the system of selection pressures and constraints that caused primates to evolve their distinctive brains is key to understanding primates as an order and ourselves as a species. As such, it has been a major goal in Biological Anthropology. However, I argue that the research approach that has traditionally been employed in the field is not capable of producing a realistic understanding of primate brain evolution because it cannot effectively model the complex causal systems that likely underlie it. In this thesis, I describe a new approach to understanding the complex causes of primate brain evolution that I have developed and tested. I demonstrate the effectiveness of this new approach by showing that it has been able to produce the first realistically complex models of the causes of primate brain evolution. These models both challenge our current understanding of primate brain evolution — including the social brain and frugivorous foraging hypotheses — and reveal previously unrecognised patterns, such as a core system of niche variables underlying primate brain evolution and unique systems of selection pressures and constraints operating in different primate clades.

Keywords: brain evolution, primates, allometry, observational-inductive, causal discovery, complex causal models

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NOTE

To enable interactivity, the figures and tables for this thesis are presented in an accompanying website. This can be accessed by following the links in the text (e.g., Figure 2.2.1) or at https://mkerney.shinyapps.io/UnderstandingPrimateBrainEvolution/

It is recommended to follow the links in the text, as these may lead to a specific view of a figure or table.

All the figures and tables, along with the code behind them, can also be viewed at https://github.com/MaxKerney/thesis-materials-and-methods

1. Introduction

Primates are distinctive for the diversity of brain sizes that they exhibit, which include some of the largest in the animal kingdom (Smaers et al., 2021). Understanding the causes of this diversity is, therefore, fundamental to understanding the primate order. Of course, us human primates also have a more personal motivation for wanting to understand the causes of primate brain evolution: it could help us to understand why we evolved our extraordinary brains that have done so much to shape our world and our experience of it.

But what would an understanding of the causes of primate brain evolution consist of? We can assume two things. The first is that brains are adaptations to the selection pressures and constraints imposed by an animal's niche — broadly defined here as their lifestyle, or what Darwin called their "line of life" (Darwin, 2006, p. 254; Pocheville, 2015) (Box 1.1). Therefore, an understanding of the causes of primate brain evolution will consist of some model of the causal relationships between niche variables and primate brains. The second thing that we can assume, given what we know about natural systems, is that this model will be complex (in the general sense of being multifaceted). It is likely to be complex for at least three reasons.

The first reason why any model of the causes of primate brain evolution is likely to be complex is that niche variables are often complex constructs that will require multifaceted representations in any model (Grace et al., 2012; Grace and Bollen, 2008). An example is "diet", which can encompass diet composition, breadth, quality, processing requirements, seasonal variation, etc. Another example is "sociality", which can encompass social system, group size, amount of social interaction, type of social interaction (competitive or cooperative), the mode and complexity of communication, etc.

The second reason why any model is likely to be complex is that it is likely to feature many niche variables that are densely interlinked. This is because natural phenomena often emerge from the additive effects of multiple variables or only emerge when variables interact (Hilborn and Stearns, 1982). Furthermore, variables could be interlinked by many different types of linear and nonlinear relationships. Many variables have already been suggested to be involved in primate brain evolution, including climate (van Woerden et al., 2014), home range size (Powell et al., 2017), diet (DeCasien et al., 2017), investment in offspring (Powell et al., 2019), and sociality (Dunbar, 1998). We can imagine how these variables could be interlinked: climate could affect diet by affecting the availability and distribution of plants and prey, which in turn could affect home range size; diet could affect the energy available to invest in offspring; home range size could affect the number of predators encountered, which could affect the size of social groups needed for defence; and, depending on diet, the size of social groups could affect the size of the home range needed to acquire enough food for all group members.

The third reason why any model of the causes of primate brain evolution is likely to be complex is that there is likely to be more than just one model. Primates are so diverse — ranging in size from the tiny mouse lemur (*Microcebus*) to the giant gorilla (*Gorilla*) and inhabiting almost every biome from tropical South American rainforest to snowy Asian mountains — that there are likely to be different systems of selection pressures and constraints acting in different taxa (Logan et al., 2018; Petter and Desbordes, 2013; Smaers et al., 2021; Walker et al., 2006).

Box 1.1 | Brains are too expensive to be nonadaptive

Although biological traits can be the product of neutral evolutionary processes such as mutation, recombination, and genetic drift (Kimura, 1991; Lynch, 2007), it is unlikely that brains are. Brains are almost certainly adaptations to positive selection pressures since they are so expensive that they would be quickly selected against if they were not adaptive. Brains impose considerable maternal, developmental, and metabolic costs and may increase vulnerability to extinction:

- Larger brains require extended gestation and lactation periods, increasing maternal energy demands and reducing reproductive rate (Barrickman et al., 2008; Isler and van Schaik, 2009).
- Larger brains require a prolonged maturation period, making young animals more vulnerable and delaying reproduction (Barrickman et al., 2008).
- Brains are metabolically costly to maintain. It has been estimated that primates use over 10% of their resting metabolism for their central nervous system (compared to an average of 5.3% in other vertebrates) (Mink et al., 1981) and that the daily caloric cost of primate brains can be as high as 180 kcal in orangutans and 516 kcal in humans (compared to <5 kcal in rodents) (Herculano-Houzel, 2011a).
- The high maternal energy demands and reduced reproductive rates associated with larger brains may make large-brained primate species less resilient to environmental changes and thus more vulnerable to extinction (Gonzalez-Voyer et al., 2016).

If this is what an understanding of the causes of primate brain evolution is likely to consist of — a complex causal model (or models) of the niche variables that have imposed selection pressures or constraints on primate brains — then, clearly, we must ensure that when investigating the subject we employ a research approach that is capable of producing such an understanding. My thesis, however, is that the approach that has traditionally been employed is ill-suited to developing complex causal models and thus is unlikely to be able to produce a realistic understanding of the causes of primate brain evolution.

The Hypothetico-Deductive Approach

The approach that has traditionally been employed is the hypothetico-deductive approach. It is the classic research approach that practically all science students are trained in, and, indeed, it is often regarded as being synonymous with science itself (Barnard et al., 2017; Elliott et al., 2016; Haufe, 2013; Hawkins, 2019; Mahootian and Eastman, 2009). The typical steps of the hypothetico-deductive approach are to: 1) develop, using researchers' knowledge, imagination, and intuition, a hypothetical model to explain the phenomenon of interest; 2) derive testable predictions from this model; 3) assemble the observational data necessary to test these predictions; and 4) test whether the data are consistent with the predictions of the hypothetical model. If they are, then the model can be accepted as being correct until an alternative hypothetical model is found to be a comparable or better fit to the data. This approach has so far been used to develop several hypothetical models of the causes of primate brain evolution, including the frugivorous foraging hypothesis, the social brain hypothesis, and the maternal investment hypothesis.

The frugivorous foraging hypothesis posits that primates' brain sizes are caused by the amount of fruit (and other reproductive plant parts such as flowers and seeds) in their diets. Harvey et al. (1980) and Milton (1981) were among the first to suggest that foraging for fruit requires a larger brain than foraging for other resources such as leaves because fruit is spatially and temporally patchy. Frugivorous species therefore need to have a greater capacity to learn and remember the location of food sources, how to navigate to them, and the times when they are available. Gibson (1986) added to this the idea that frugivorous diets also often require extractive processing (e.g., of nuts and seeds) which may also require a larger brain to be able to handle the increased sensorimotor demands. Later, Aiello and Wheeler (1995) offered a very different interpretation of the frugivorous foraging hypothesis — that the amount of fruit in the diet might not cause brain size by acting as a selection pressure, but by acting as an energetic constraint

on the size of the brain that can be grown. They suggested that the digestibility of a diet might affect the amount of energy available for brain growth, with a diet composed of larger amounts of highly digestible fruit allowing more energy to be reallocated from digestion to the brain. Whether the amount of fruit in the diet is thought to act as a selection pressure or a constraint, the frugivorous foraging hypothesis has been supported by evidence of positive correlations, both quantitative (e.g., Harvey et al., 1980) and qualitative (e.g., Milton, 1981), between frugivory and brain size across primates, including in a recent large-scale analysis of 140 species (DeCasien et al., 2017).

The social brain hypothesis suggests that primates' brain sizes are caused by the complexity of their social groups. It is thought that larger social groups that feature more, and potentially more complex, relationships create a selection pressure for larger brains that can both better manage those relationships and take advantage of the opportunities that they afford (Dunbar, 1998). Jolly (1966) emphasised the opportunities for social learning that are afforded by larger groups, which could allow individuals to acquire valuable knowledge about food sources, tool use, or potential predators. Humphrey (1976) similarly noted the potential benefits of social learning for individuals with the requisite cognitive capacity, but also suggested that social groups might provide opportunities for sufficiently capable individuals to manipulate other group members to their advantage. This idea was further developed by Byrne and Whiten (1988; 1992), who found a qualitative correlation between primates' brain sizes and the rate of observed incidents of tactical deception. The first quantitative support for the social brain hypothesis emerged in the 1990s, when Sawaguchi (1990) and Sawaguchi and Kudo (1990) found that relative brain size and relative neocortex size were positively associated with polygyny in anthropoids, and that relative neocortex size was positively associated with "troop-making" and troop size in prosimians. Dunbar (1992) similarly found evidence of a positive correlation between relative neocortex size and social group size in a sample of primate species, and also between relative brain size and group size (Shultz and Dunbar, 2007).

The maternal investment hypothesis suggests that primates' brain sizes are caused by the amount of energetic investment that species can make in the brain growth of offspring, in the sense that low levels of investment will constrain the brain sizes that can develop. Martin (1981, 1996) first brought attention to the costs of growing larger brains that species would have to meet by highlighting the positive association between brain size and energy turnover (as indicated by basal metabolic rate) across vertebrates, an association that has since been corroborated by Isler and van Schaik (2006). Armstrong (1985), too, suggested that growth of metabolically expensive neural tissue must be matched by increases in energy supply. As well as direct energy investments, later researchers have suggested that primate mothers must also invest more in terms of extended gestation and lactation periods in order to grow larger-brained offspring. Barrickman et al. (2008), for example, presented evidence of a positive correlation between neonatal brain size and gestation length across a sample of 27 primate species, while Barton and Capellini (2011) also found a positive correlation between post-natal brain growth and lactation duration across 128 mammals species.

Although they have achieved prominence in the literature, it is nevertheless clear that these three hypothetical models provide unrealistically simple explanations of primate brain evolution. They each involve only one niche variable (amount of fruit in the diet, social complexity, maternal investment) that is seen to have a direct and linear effect on brain evolution, and each model is generally assumed to apply universally across primates. The developers of these hypothetical models would likely be the first to admit that the real causal mechanisms underlying primate brain evolution are more complex than their models depict (e.g., Dunbar and Shultz, 2017), but they have been constrained by the hypothetico-deductive approach's inherently limited ability to model complex natural systems.

Limitations of the Hypothetico-Deductive Approach

The hypothetico-deductive approach's limited modelling ability is due, first and foremost, to its reliance on human cognitive abilities. The initial step of developing a hypothetical model relies on researchers' knowledge, imagination, and intuition. However, human knowledge, imagination and intuition are limited, especially when it comes to complex systems.

We are far from omniscient of the complexities of nature. In the case of primate brain evolution, researchers have little pre-existing knowledge of all the variables that may be relevant, how they may interact, and how variables and their interactions might change in different taxa. They therefore lack the information necessary to develop realistically complex hypothetical models to test.

Limitations in knowledge cannot be made up for by imagination since imagination is constrained by pre-existing knowledge. Imagination works by assembling objects stored in memory into novel combinations (Vyshedskiy, 2019), so having limited knowledge also limits researchers' ability to imagine realistically complex hypothetical models.

Finally, human intuition tends towards oversimplification. For the sake of efficiency, humans, like other animals, have evolved to use simplifying heuristics, or rules of thumb, when tackling cognitive tasks (Hutchinson and Gigerenzer, 2005; Johnson et al., 2013). These serve us well in day-to-day life but are prone to error when used for academic tasks such as hypothesis development. Two particularly problematic heuristics in this context are satisficing and social learning.

Satisficing is the heuristic that "the first reasonable solution is good enough" (Heuer, 2010). In the context of hypothesis development, this creates a tendency to fixate on the first reasonable hypothetical model that is developed (which, because of limited knowledge and imagination, is

inevitably a simple one) rather than systematically enumerating all the possible complex hypothetical models.

While social learning encompasses many nuanced sub-heuristics, or social learning strategies (Hoppitt and Laland, 2013, pp. 196–234), it can be broadly summarised as the heuristic that "other people probably have the right idea, so copy them". This creates a tendency to orbit and only incrementally build upon existing ideas in the literature rather than explore the entire space of complex possibilities.

As well as its reliance on human cognitive abilities, another limitation of the hypothetico-deductive approach is the statistical methods that it is based around. Regression methods are typically used to test the predictions of hypothetical models, but these methods impose restrictions on the complexity of the predictions that can be tested and therefore on the models that can be developed.

Generally, regression methods can only handle a small number of variables, otherwise they encounter issues of collinearity (Johnston et al., 2018), and variables usually cannot be of mixed types (i.e., continuous and factor). These methods also typically assume linear relationships between variables and only have limited ability to test for nonlinear relationships by including pre-specified polynomial terms. They can also only test for direct, and not indirect, relationships between variables. Perhaps most significantly, standard regression methods cannot directly test for causal relationships, only correlations. As a result, they can only ever provide correlational evidence to support hypothetical models, which — given the risk of spurious correlations (see http://tylervigen.com/spurious-correlations) — means that these methods cannot reliably establish causal models. More sophisticated methods such as path analysis and structural equation modelling (SEM) can be used to mitigate some of these issues (Grace et al., 2015; Shipley, 2016; von Hardenberg and Gonzalez-Voyer, 2014). Path analysis and SEM can, for instance, incorporate many variables, test for both direct and indirect relationships, and can

directly test for causal relationships. However, they still assume linear relationships and still rely on researchers' abilities to develop hypothetical models to test.

Indeed, these two limitations of the hypothetico-deductive approach — human cognitive abilities and restrictive statistical methods — are mutually reinforcing. Restrictive statistical methods encourage simplistic thinking and the development of simple hypothetical models, and the testing of simple hypothetical models perpetuates the use of restrictive statistical methods.

Even if the hypothetico-deductive approach was better able to model complex systems, it would be an inefficient and unreliable way of doing it. The approach relies on the repeated testing of alternative hypothetical models to converge on an accurate model, but for complex natural systems involving many variables there are many potential hypothetical models that would need to be tested. Assuming that the real system involved just five variables, there would be over one million possible models of the causal relationships between them (Shipley, 2016, p. 223). Even if most of these could be immediately discounted as implausible, many models would still remain that would need to be tested manually. Doing so would take considerable time, especially since in practice alternative models are typically developed and tested in separate publication cycles. Furthermore, due to the human cognitive limitations mentioned before, as well as practical issues around research funding and publication, there is no guarantee that all the possible hypothetical models would ever be developed and tested (Betini et al., 2017; Haufe, 2013).

Although the hypothetico-deductive approach is ill-suited to modelling complex systems such as those that likely underlie primate brain evolution, there is another approach available that is better suited to this task: the observational-inductive approach.

The Observational-Inductive Approach

Whereas the hypothetico-deductive approach is a "top-down" approach, in that it starts with a hypothetical model and tests whether it is a good fit to observational data, the observational-

inductive approach is "bottom-up", in that it starts with observational data and builds a model directly from it (Mahootian and Eastman, 2009). In its modern computerised form, the steps in the observational-inductive approach are: 1) assemble data on all variables potentially relevant to the phenomenon of interest; 2) computationally search this data set for patterns indicating causal relationships between variables; 3) provisionally accept the model of causal relationships that this search produces as the best explanatory model, subject to validation using other methods and until more data become available to develop a more accurate model.

Although its modern computerised incarnation may be relatively new, the observational-inductive approach *sensu lato* has just as long a history of use in the natural sciences as the hypothetico-deductive approach. Indeed, in the form of natural history observation — *à la* von Humboldt (1769–1859), Darwin (1809–1882), and Wallace (1823–1913) — it is what evolutionary biology was founded upon (Elliott et al., 2016; "Humboldt's legacy," 2019; Kell and Oliver, 2004; Tewksbury et al., 2014; Willson and Armesto, 2006). Its modern computerised form has developed over the last thirty years and has been applied successfully in many fields, including genomics, ecology, epidemiology, space physics, clinical medicine, and neuroscience (Bielczyk et al., 2018; Glymour et al., 2019b).

Advantages of the Observational-Inductive Approach

The first reason why the observational-inductive approach is better suited than the hypothetico-deductive approach to modelling complex systems is that it does not rely on researchers' abilities to specify models. Researchers still have the more minimal responsibility of identifying potentially relevant variables to input, but thereafter computerised methods can find whatever relationships exist in the data regardless of whether they are something that researchers could have conceived.

Because these computerised methods need to be amenable to finding whatever relationships may exist in real-world data sets, they are designed to impose minimal restrictions compared to traditional statistical methods. They can accommodate a practically arbitrary number of variables

and variables of mixed types; they can find nonlinear and indirect relationships; and, crucially, they can directly infer causal relationships from data (Glymour et al., 2019a; Hochachka et al., 2007).

Finally, computer automation means that modelling using the observational-inductive approach can be systematic, fast, and comprehensive.

Project Goal

The goal of my project was to develop and test an implementation of the observational-inductive approach that could be used to investigate the causes of primate brain evolution. This implementation was not intended to produce a definitive understanding of the complex causes of primate brain evolution but provide an initial proof-of-concept of the potential of the observational-inductive approach to bring us closer to such an understanding.

The following chapters describe the different steps of the implementation that I developed and the results that it produced. Chapters 2 and 3 describe how I assembled a large set of data on primate brain evolution and niche variables to analyse. I then conducted a computational search for causal relationships in this data set in two stages. First, the data set was searched for correlations between variables (described in Chapter 4) to identify which variables are related at all. Then the resulting correlation models were searched for specific patterns of correlation that indicate how variables are causally related (described in Chapter 5). This process produced complex causal models of primate brain evolution of the sort envisioned at the beginning of this chapter — the first produced in the field. As a final step, I compared the different causal models to identify any recurring patterns that could provide a deeper understanding of primate brain evolution (described in Chapter 6). I conclude the thesis by discussing the extent to which my implementation of the observational-inductive approach was able to produce a more realistic understanding of the causes of primate brain evolution and how it could be further developed.

2. Data I: Allometric Grades

There are many ways in which primate brains can evolve. Brain evolution can involve changes in brain shape (Heuer et al., 2019; Sansalone et al., 2020); changes in the presence and size of different substructures (Smaers et al., 2017; Smaers and Vanier, 2019); changes in neuronal connectivity (Ardesch et al., 2019); changes in the number and ratios of different cell types (Herculano-Houzel, 2011b); changes in neuromodulatory systems (Agnati et al., 2010); changes in patterns of brainwave oscillation (Anderson, 2014, pp. 110–111); and changes in gene expression (Khrameeva et al., 2020; Sousa et al., 2017). However, data on such fine-grain aspects of primate brain evolution are currently only available for a few model species — too few for a primate-wide analysis (though this is changing, see Milham et al., 2018; Navarrete et al., 2018). Therefore, as alluded to at the beginning of the previous chapter, in this project I focussed on investigating the diversity of brain size that has evolved in the primate order (Box 2.1). The first step of my implementation of the observational-inductive approach was to acquire some measure of primate brain size that could become the dependent variable in subsequent analyses. There are several measures of brain size that have precedent in the literature, including absolute brain size, encephalisation, and brain-body allometry.

Box 2.1 | What does brain size mean?

Brain size is often assumed to reflect general cognitive ability or "intelligence". However, there is no clear correlation (Deacon, 1990; Fichtel et al., 2020; Logan et al., 2018). Many organisms without large brains exhibit what most would regard as intelligent behaviour. For example, bees learn which flowers are the most profitable to visit, and at what times of day, and adjust their foraging behaviour accordingly (Chittka, 2017); ants engage in both arable and pastoral farming (McGhee, 2011, pp. 223–228); and predatory salticid spiders take elaborate detour routes to ambush their prey (Barrett, 2015, p. 61). Intelligent behaviour is even found in organisms with no brain at all. Some bacteria apparently engage in pack hunting (Berleman and Kirby, 2009); slime molds can navigate complex environments (Reid et al., 2012); and plants act in ways that are self-aware (Calvo Garzón and Keijzer, 2011).

What brain size seems to reflect more specifically is the capacity for physiological regulation and behavioural flexibility. The organisms described above do not need large brains because their bodies and the niches that they inhabit are relatively simple. Consequently, they have limited need for a centralised organ such as the brain to coordinate physiological systems in the performance of behaviour and they can often leverage regularities in their environments to evolve non-neural, embodied solutions to cognitive tasks (Barrett, 2015; Keijzer et al., 2013; Sterling and Laughlin, 2017). For instance, salticid spiders evolved a particular sensorimotor coupling between their eyes and their legs that exploits the regular occurrence of horizontal surfaces in their environment to produce their sophisticated stalking behaviour (Barrett, 2015, pp. 61–70). Similarly, slime molds have evolved sensors that detect contact with the slime that they reliably leave in their environments, enabling them to avoid areas that they have previously explored and try new paths instead (Reid et al., 2012).

However, when bodies become more complex there is a greater need for centralised coordination, and when niches become more complex and variable embodied cognitive systems are not flexible enough to produce adaptive behaviour. In these situations, larger brains provide greater capacity to effectively manage physiology and greater capacity to learn and store in memory an arsenal of mental models that enable an organism to respond adaptively to myriad situations (Anderson, 2014; Godfrey-Smith, 2017; Keijzer et al., 2013; Sterling and Laughlin, 2017). The more complex the body and niche, the larger brains need to be.

Therefore, in studying the evolution of brain size in primates, we are not so much studying the evolution of "intelligence" but more the evolution of primates' capacity for physiological regulation, learning, and memory. This is underscored by the fact that primate brain size has been shown to be mainly a product of the size of the cortico-cerebellar system, a system particularly involved in learning and memory (Benagiano et al., 2018; Smaers et al., 2018; Smaers and Vanier, 2019).

Although absolute brain size has been used in some studies (e.g., Deaner et al., 2007; Marino, 2006), it has a serious limitation when it comes to studying brain evolution, in that it is confounded by the allometric scaling relationship between brain size and body size — the fact that brain size can vary simply because of variation in the overall size of animals (Gilbert and Jungers, 2017). Just as larger animals will have larger hearts and livers than smaller animals simply as a product of their overall larger body size, so too will they have larger brains (Jerison, 1973, p. 57). By not taking overall body size into account, a measure of absolute brain size makes it impossible to specifically identify — and therefore study — brain evolution in isolation from the evolution of overall size, making it an unsuitable measure to use in this project. What is needed to study brain evolution is a measure of brain size relative to body size.

Attempts to quantify relative brain size began in the 19th century, particularly with the work of Snell (1891) and Dubois (1897), who introduced the idea that relative brain size could be represented by deviations away from the "baseline" allometric relationship between brain size and body size. If a species' brain size is larger than that expected from the baseline brain-body allometry alone, then this could be interpreted as the species having evolved a relatively larger brain size, whereas if a species' brain size is smaller than expected this could be interpreted as the species having evolved a relatively smaller brain size. Because such deviations were assumed to isolate brain size evolution from the evolution of body size they came to be known as measures of "encephalisation" (Harvey and Krebs, 1990). Although the principle has remained the same, the methods for quantifying brain size deviations and encephalisation have changed in the century since Snell and Dubois. Originally, brain size deviations were calculated by manually fitting a theoretically derived allometric regression line to a sample, for instance one that featured a 2/3 scaling relationship (derived from theories of body surface area scaling) or a 3/4 relationship (derived from metabolic theory), and the differences between the brain size values estimated from this line and species' observed brain sizes were calculated (Deacon, 1990; Harvey and Krebs, 1990; Jerison, 1973, p. 49). However, more recent studies have taken advantage of

developments in statistics to use quantitative line-fitting methods to establish the most appropriate baseline brain-body allometry to calculate deviations from for any given sample (Harvey and Krebs, 1990). Early studies also tended to quantify deviations from brain-body allometry in terms of a ratio of the observed brain size of a species relative to the brain size expected for them from the allometry, a measure known as the Encephalisation Quotient (EQ) (Jerison, 1973, p. 61). However, more recent studies have typically favoured quantifying brain size deviations in terms of the absolute residual deviation between species' observed brain sizes (log-transformed for the allometric regression) and those estimated from the fitted allometry (e.g., Shultz and Dunbar, 2007), a measure which is in fact equivalent to log-transformed EQ (Gilbert and Jungers, 2017).

The methods typically used to calculate encephalisation, and measures of encephalisation themselves, do have a major issue, though: they do not take into account or represent the fact that brain-body allometries themselves evolve (both in terms of the intercept and slope of the allometry) and are indeed relatively labile over evolutionary time scales (Frankino et al., 2005; Ksepka et al., 2020; Smaers et al., 2021; Stillwell et al., 2016; Voje et al., 2014). From a methods standpoint, failing to account for the evolution of allometry means that species' encephalisation values are at risk of being invalid if they were calculated from a single global allometry for a sample when in fact different allometries evolved in and apply to different subgroups of the sample (Harvey and Krebs, 1990; Smaers et al., 2021). Indeed, species' levels of encephalisation could be grossly under- or overestimated. But even if the evolution of multiple allometries was accounted for in the calculation of encephalisation measures to ensure that species' values were accurate, encephalisation measures still do not capture any information that would allow one to investigate why particular brain-body allometries evolved in the first place — why particular "baseline" brain sizes for given body sizes should exist, and why these should vary between taxonomic groups. Given that brain-body scaling relationships ultimately determine most of the variation in brain sizes between species (Wartel et al., 2019), the greatest part of understanding

primate brain evolution would seem to lie in understanding why these allometries evolved, rather than understanding relatively minor deviations from them.

As a solution to the issues with encephalisation measures, Smaers et al. (2021) have recently proposed that brain-body allometries themselves should be used as measures of brain evolution. The authors suggest that if species are categorised into grades that exhibit distinct brain-body allometries, then these grades provide a categorical measure of the macroevolution of relative brain size. Differences between the grades in terms of allometric intercept would represent the evolution of different mean relative brain sizes in species in the different grades, while differences in allometric slope would represent the evolution of different scaling relationships between relative brain size and body size among the species within each grade (Shingleton and Frankino, 2012; Vea and Shingleton, 2021). While such a brain-body allometry-based measure is focused on the macroevolutionary scale, precluding the study of individual species' brain evolution, it does overcome the issues with the other measures of brain evolution discussed above, arguably making it the best measure of brain evolution currently available. For this reason, this is the measure of brain evolution that I decided to use as the dependent variable in my project.

Unhelpfully, both encephalisation measures and measures of brain-body allometry are often referred to generically in the literature as measures of relative brain size, which can make it unclear as to which measure is being used in a study. To avoid ambiguity in the rest of this thesis, I will use the following terminology. I will use Encephalisation Quotient (EQ) to refer to a measure of encephalisation that is derived from ratios; logEQ to refer to a measure of encephalisation that is derived from residuals; and allometric relative brain size (aRBS) to refer to both the mean relative brain size of species in an allometric grade or clade, as represented by an allometric intercept (grade/clade aRBS), and also the relative brain sizes of species within a grade that scale with body size in accordance with an allometric slope (species aRBS).

In publishing their ideas on using brain-body allometry as a measure of brain evolution, Smaers et al. (2021) presented a categorisation of mammal species — including primates — into different allometric grades, providing a categorical variable that could be used in a study of primate brain evolution. However, this data was not available at the time that I was assembling my data set, so in order to obtain a categorisation of primate species into allometric grades to use as the dependent variable in my project, I had to conduct my own allometric analyses.

Materials & Methods

Data

I gathered data on species' mean brain size (Endocranial Volume, ECV) and body size (body mass) for the allometric analyses primarily from Powell et al. (2017). This is the most comprehensive and high-quality source of ECV and body mass data currently available for primates. Most of the Powell et al. (2017) data were gathered directly from museum specimens of adult animals, multiple specimens were sampled per species, and wild-caught specimens were favoured. Also, when they calculated the mean brain and body sizes for species, Powell et al. (2017) only used data from female specimens if species were sexually dimorphic (which they defined as having >10% size difference) to avoid unrealistic mean values being derived from divergent male and female samples.

I supplemented the Powell et al. (2017) data with data from Smaers et al. (2021, but unpublished at the time), Myhrvold et al. (2015), and Kamilar & Cooper (2013). When incorporating these other data, I followed Powell et al. (2017) by only including data derived from female specimens for sexually dimorphic species. The data from Smaers et al. (2021) included data for 25 fossil species whose ECVs had been measured from actual or virtual endocasts and whose body sizes had been estimated from craniodental or postcranial measurements using predictive equations built from extant samples (for details, see the references cited in Smaers et al., 2021). Although

these fossil species could not be included in later analyses in the project (because there are no data available on their niches), including fossil species in allometric analyses can help to improve their accuracy (Ho and Ané, 2014; Slater et al., 2012).

To mitigate the possibility of errors in my data set — which could have been introduced during the assembly process or been present in the original sources — I cross-checked the brain and body size values for each species against the morphological description of the species given in Mittermeier et al. (2013), an authoritative reference on primates. My final set of brain and body size data can be viewed in Table 2.1.1.

In addition to the brain and body size data, the allometric analyses required data on the phylogenetic relationships between species in order to identify when and where on the primate evolutionary tree grade shifts occurred. For this, I used the primate branch of the mammal phylogeny developed by Smaers et al. (2018) from Faurby & Svenning's (2015) mammalian supertree.

Data Set Evaluation

Coverage

Together, the brain and body size data and phylogeny cover 192, or 37.8%, of the 508 extant primate species recognised at the time that I assembled the data set ("Integrated Taxonomic Information System (ITIS)," n.d.) (Figure 2.1.2). Of the 80 recognised genera, 71 are covered. The mean coverage of each genus is 55%, though the standard deviation is 35.6%, showing that there is considerable variation in coverage.

Twenty-five genera have 100% coverage with, in general, the subfamily Homininae and family Lemuridae being particularly well sampled. Consequently, any results from the allometric analyses that pertain to these taxa can be interpreted with relatively high confidence.

Nine genera, however, have 0% coverage. *Mico*, *Piliocolobus*, and *Tarsius* are particularly significant here because in absolute terms they are missing coverage for 14, 17, and 11 species, respectively (42 species between them). *Pithecia*, *Microcebus*, and *Plecturocebus* also have notably low coverage: 6.3%, 8.3%, and 13.6%, respectively. In absolute terms, *Pithecia* is missing data for 15 species, *Microcebus* 22 species, and *Plecturocebus* 19 species (56 species between them).

Furthermore, low coverage appears to be clustered in specific taxa, for instance the Pitheciidae and Cheirogaleidae families. I confirmed this by testing for phylogenetic signal in the missing coverage. I aligned my data set with a taxonomic tree of all 508 recognised primate species that I built using the Taxize R package (Chamberlain and Szöcs, 2013). I then used the miss.phylo.d function from the sensiPhy package (Paterno et al., 2018) to binary code the data set: 1 if data were missing for a species, 0 if they were not. The function then calculated the *D* statistic (Fritz and Purvis, 2010), a measure of phylogenetic signal in binary data. The test showed that the distribution of the gaps in coverage is indeed nonrandom — i.e., significantly different from random ($p < 0.001$). In fact, the *D* value is 0.61, which suggests that the gaps in coverage exhibit a moderate phylogenetic signal (the value is about mid-way between 0, indicating a full Brownian motion distribution, and 1, indicating an entirely random distribution). It may be that particular taxa were under-sampled in the literature because they possess certain traits that make them more difficult to gather data on (Garamszegi and Møller, 2011; González-Suárez et al., 2012). For instance, the Cheirogaleidae are all small (they are the smallest strepsirrhines), solitary, and nocturnal (Fleagle, 2013, p. 58). Whatever the reason, any results pertaining to these under-sampled taxa should be interpreted cautiously, as improved samples could affect the allometric regressions for these groups.

Distributions

Brain and body size data must be log-transformed for allometric regression analyses, to linearise the relationship between them (Vea and Shingleton, 2021). Histograms and QQ plots of the log-transformed data show that the data only deviate slightly from normality (Figure 2.1.3) (Zuur et al., 2010). Such minor deviations from normality would be unlikely to affect the analyses since linear regressions are reasonably robust against violations of the normality assumption (Mundry, 2014, p. 139).

Both variables, even after transformation, exhibit a small number of both low and high global outliers (Figure 2.1.3, *Bottom panel*). Again, though, such global outliers would be unlikely to affect the allometric analyses since, in order to identify shifts between groups, the analyses are conducted locally within groups rather than across the whole sample.

Allometric Analyses

To identify shifts in brain-body allometry between different primate grades — signified by shifts in the intercept and slope of allometric regressions — I used an Ornstein-Uhlenbeck (OU) modelling approach.

OU models depict species on an adaptive landscape, clustered around particular adaptive peaks that represent optimal trait values (Nunn, 2011, pp. 103–104; O'Meara and Beaulieu, 2014, pp. 381–393). They can be used to infer whether grade shifts in trait values occurred during the evolution of a taxon by testing whether a sample is best described as being clustered around a single adaptive optimum or multiple optima. An OU model specifying a single optimum and an OU model specifying multiple optima can be fitted to species' data, and if the multi-optima model is a better statistical fit, then it can be inferred that grade shifts to different optimal trait values probably occurred in that taxon. Similarly, OU models can be used to infer when and where on a phylogenetic tree grade shifts took place. Different models specifying different locations for shifts

can be fitted to the data, and the best fitting model can be taken to describe the most likely evolutionary history (e.g., Ksepka et al., 2020; Smaers et al., 2021).

Historically, OU models could only be used to infer grade shifts in traits with single values, making them unsuitable for inferring shifts in complex traits such as brain-body allometry which has more than one value associated with it (intercept and slope) (Uyeda et al., 2017). Also, models had to be fitted manually by researchers according to particular pre-specified hypotheses, with all the associated issues of the hypothetico-deductive approach that I discussed in Chapter 1. More recently, methods were developed to automatically find the best-fitting OU model for a data set using a more observational-inductive approach, but these methods could still only be used for traits with single values (O'Meara and Beaulieu, 2014, pp. 384–387). However, the most recent methodological innovation has now made it possible to fit OU models for bivariate traits using an observational-inductive approach.

The new method is called Ornstein-Uhlenbeck reversible jump Markov Chain Monte-Carlo (OUrjMCMC) (Uyeda et al., 2017; Uyeda and Harmon, 2014). OUrjMCMC automatically explores all possible specifications and placements of bivariate trait optima on a phylogeny and converges on the most likely scenario to have occurred given species' observed trait values. This is the method that I used for my analyses to identify shifts in brain-body allometry in primates.

OUrjMCMC Analyses

Being a Bayesian method, OUrjMCMC accepts a prior to help reduce the space of possible optima that it needs to explore. As a prior for the range of allometric intercept and slope values that might plausibly have evolved in primates, I calculated 80% confidence intervals for standard deviations of intercept and slope values in my sample of species, following Smaers et al. (2021, but unpublished at the time). I used phylogenetic generalised least squares (pGLS) to calculate allometric regressions for my whole sample and six subsamples of major taxonomic groups (Lemuriformes, Lorisiformes, Platyrrhini, Cercopithecinae, Colobinae, and Hominoidea). I then

used the intercept and slope values from the whole sample regression (which are effectively the mean intercept and slope values for the sample) and the maximum intercept and slope values from across the subgroup regressions to calculate the standard deviation of intercepts and slopes at an 80% confidence level, following Smaers et al. (2021) (intercept SD = (maximum intercept – mean intercept) / 1.28 ; slope SD = (maximum slope – mean slope) / 1.28).

For the OUrjMCMC analysis itself, I developed tuning parameters for 10 Markov chains before running them in parallel for 3 million iterations each, again following Smaers et al. (2021, but unpublished at the time). Even using a prior to reduce the search space, OUrjMCMC analyses are computationally demanding, and the analysis took approximately 48 hours to run on a 32-core computer. Afterwards, the first 30% of each chain was discarded as burn-in before the results from all the chains were combined. For every grade shift that it infers, OUrjMCMC provides the posterior probability of the shift being valid, allowing unlikely shifts to be filtered out. I set a relatively inclusive threshold of 0.05 posterior probability and disregarded any proposed shifts with a lower probability than that. All of the allometric analyses were carried out in R (R Core Team, 2020). The code for the OUrjMCMC analyses can be viewed in the script 01-OUrjMCMC.R.

pANCOVA Analyses

OUrjMCMC is a new method and still has potential for further development. Currently, it is relatively susceptible to Type I and Type II errors. It may suggest grade shifts that are obviously incorrect or not suggest grade shifts that obviously occurred. Also, the method currently does not indicate the nature of the grade shifts that it infers — in this case, whether a shift involved a change of allometric intercept, slope, or both. As a solution to these issues, I followed the approach of Smaers et al. (2021, but unpublished at the time) of conducting phylogenetic Analysis of Covariance (pANCOVA) analyses (Smaers and Rohlf, 2016) on the results of the OUrjMCMC analysis. By translating the grades proposed by OUrjMCMC into groupings of species in a

pANCOVA, I could test whether groups were in fact significantly different, and in what way, and screen for Type I and Type II errors.

I began by plotting the allometries for the different grades proposed by OUrjMCMC and visually inspected these to see if any grade specifications seemed erroneous. For instance, if a proposed grade's allometry featured clearly distinct clusters of species this might indicate that multiple allometric grades should have been proposed instead of just one. Alternatively, if the allometries for different grades overlapped substantially this might indicate that only one grade should have been proposed instead of several. In suspicious cases such as these, I specified alternative group memberships for species (either splitting grades or merging them) and tested these alternative model specifications using pANCOVA. If the alternative model was found to be a better fit to the data, then I accepted this model in preference to the original OUrjMCMC model. For example, OUrjMCMC originally proposed that the cercopithecines and gibbons belonged to separate grades. However, visual inspection of the plotted allometries for these grades showed that they overlap substantially, suggesting that they may in fact constitute a single grade. I specified an alternative grouping for the taxa in these grades, merging the two grades together into one, and tested this alternative model using pANCOVA. The alternative model was found to be a significantly better fit than a model with the original OUrjMCMC specification (or rather, the original model was not found to be a significantly better fit than the alternative model; $F_{16,15} = 2.28$, $p = 0.133$; L.Ratio = 3.08, $p = 0.0791$, AICΔ = 1.08, AICw = 0.368) and so I treated the cercopithecines and gibbons as belonging to the same allometric grade for the rest of the project.

In a similar way, I specified and tested alternative models to establish whether grades were significantly different in terms of their intercepts, slopes, or both. For instance, to check whether two grades differed in terms of intercept, I specified and tested an alternative model in which they were considered to have the same intercept. If this alternative model was a worse fit to the data than the original model, then I concluded that the two grades did indeed differ in terms of intercept.

Finally, pANCOVA allowed me to validate the final model of grade shifts that I attained. I tested the final model against a null model specifying no grade shifts and a model specifying only shifts in intercept and statistically confirmed that primate brain evolution is best described by multiple allometric grade shifts featuring changes in both grade allometric relative brain size (aRBS) (intercept) and species aRBS scaling (slope). The code for the pANCOVA analyses, including details of all the alternative models that I tested, can be viewed in the script 02-pANCOVA.R.

Results

My allometric analyses indicated that 12 grades characterised by distinct brain-body allometries evolved in primates (Figure 2.2.1). Nine of these grades are extant; three are extinct. All 12 grades differ in grade aRBS (allometric intercept), while there are four different species aRBS scaling relationships (allometric slopes) exhibited across the grades (Figure 2.2.2). All but one of the extant scaling relationships are hypoallometric (slope <1), meaning that species aRBS decreases with increasing body size (or increases with decreasing body size) (Figure 2.2.3). The pANCOVA analyses confirmed that this description of primate brain evolution is a significantly better fit to the data than one suggesting that no grade shifts occurred ($F_{16,2}$ = 31.03, p < 0.001; L.Ratio = 266, p < 0.0001, AICΔ = 238, AICw = >0.999) and one suggesting that only changes in grade aRBS (intercept) occurred ($F_{16,13}$ = 31.14, p < 0.001; L.Ratio = 79.2, p < 0.0001, AICΔ = 73.2, AICw = >0.999).

The 12 grades are comprised of 17 clades: the ancestral clade plus 16 clades that shifted away from the ancestral brain-body allometry. These shifts in different clades involved 16 changes in grade aRBS and 6 changes in species aRBS scaling (Figure 2.2.1). The reason why only 12 grades resulted from 16 shifts is that some clades apparently evolved convergently towards the same brain-body allometry, and so became members of the same grade. There are also cases of convergent evolution towards just one aspect of allometry. For instance, a “lower” species aRBS

scaling relationship relative to that of the ancestral grade evolved independently 4 times (Figure 2.2.1).

As I said at the beginning of this chapter, the advantage of looking at a measure of relative brain size as opposed to absolute brain size is that it allows the evolution of brain size to be distinguished from the evolution of overall body size. The allometric analyses revealed that while most of the shifts in grade aRBS (12 of 16) involved increases in both mean brain size and mean body size, in almost all cases brain size changed more than body size, meaning that the shifts in grade aRBS primarily represent brain size evolution (Figure 2.2.4, *Table*). Many of the changes in mean brain size were considerable: there were 10 shifts where brain size more than doubled (Figure 2.2.4, *Table*).

Grade Description

Below I describe in detail each of the grades identified by the allometric analyses. I assigned the grades names that reflect their brain-body allometry. The letters in the names denote the type of species aRBS scaling relationship (allometric slope) that characterises a grade: M = “Medium” (the ancestral slope); L = “Low” (lower than the ancestral slope); H = “High” (higher than the ancestral slope). The numbers indicate the grade aRBS (allometric intercept) that characterises a grade: higher numbers indicate a higher grade aRBS within a given slope group. I start by describing the ancestral grade and all the grades that share the same species aRBS scaling relationship, proceeding roughly in the order in which the grades evolved (Figure 2.2.1). I then describe the grades that diverged from the ancestral scaling relationship by evolving “lower” or “higher” species aRBS scaling relationships. Figures 2.2.1 to 2.2.5 accompany the description of each grade.

It should be noted that while the terms “ancestral” and “descendant” grades/clades are used in the descriptions below, these are not meant in the strict phylogenetic sense of descendant species evolving from ancestral species. The terms are instead intended to convey the

evolutionary relationship between brain-body allometries. Thus, for example, describing clade L3_Hylobatidae as the ancestral clade of L4_Hominidae is not meant to imply that great apes are descended from gibbons, but to convey that the brain-body allometry exhibited by great apes is derived from the one that is today represented by gibbons. Also note that the mean brain and body sizes used to calculate the magnitude of changes in brain and body size in descendant clades are phylogenetic means. Therefore, differences in mean values do not represent differences between the extant members of ancestral and descendant clades (e.g., between gibbons and great apes) but between the estimated values of mean brain and body size at the root of each clade.

Grade M2 (Ancestral grade)

The brain-body allometry that characterises grade M2 is the original brain-body allometry in primates from which all others evolved. It features a small grade aRBS and what I am referring to as a "Medium" species aRBS scaling relationship relative to the rest of the grades (slope = 0.65). This scaling relationship seems to have been retained throughout much of primate evolution, making it the most prevalent. Grade M2 is comprised of 1 clade: M2_Ancestral.

> M2_Ancestral (dwarf lemurs, mouse lemurs, fork-marked lemurs, sifakas, woolly lemurs, sportive lemurs, tarsiers, ancestral simiiformes) (N = 28 species) — The members of this clade exhibit a small mean brain size (4.64 cm^3) and the smallest mean body size of the sample (348.45 g).

Some caution is warranted around grade M2. As it is comprised of only one clade, any changes to the estimated clade aRBS or species aRBS scaling of this clade would alter the characterisation of the grade. And it is possible that this could happen, since some taxa in clade M2_Ancestral were relatively under-sampled, as discussed above (e.g., *Tarsius*, *Microcebus*) (Figure 2.1.2). Additional data for these taxa could alter the results of the allometric analyses.

Grade M4

Grade M4 is characterised by a brain-body allometry that has the same species aRBS scaling relationship as the Ancestral grade (grade M2) but a larger grade aRBS. The grade is comprised of 3 clades: M4_Ancestral_Platyrrhini, M4_Daubentonia, and M4_Colobinae.

M4_Ancestral_Platyrrhini (night monkeys, howler monkeys, squirrel monkeys, saki monkeys, titi monkeys) (N = 16 species) — The members of this clade evolved the M4 grade aRBS by increasing their mean brain size by 5.63 times relative to their ancestral clade (M2_Ancestral) to reach 26.08 cm^3 while increasing their mean body size by 4.44 times to reach 1547.83 g. They obtained the M4 species aRBS scaling relationship by retaining the scaling relationship of their ancestral clade (M2_Ancestral).

M4_Daubentonia (Aye-Aye) (N = 1 species) — *Daubentonia madagascariensis* evolved the M4 grade aRBS by increasing its mean brain size by 9.64 times relative to its ancestral clade (M2_Ancestral) to reach 44.7 cm^3 while increasing its mean body size by 7.45 times to reach 2594.8 g. As M4_Daubentonia is a single species clade, an allometric slope cannot be estimated for it, meaning that it is not actually possible to tell whether the clade has the M4 species aRBS scaling relationship. However, I made the parsimonious assumption that it retained the scaling relationship of its ancestral clade (M2_Ancestral) and so does have the M4 species aRBS scaling relationship. If M4_Daubentonia turned out to have evolved a different scaling relationship, then the clade would exhibit a different brain-body allometry and so would belong to a different grade.

M4_Colobinae (colobus monkeys, leaf monkeys, snub-nosed monkeys, langurs) (N = 26 species) — The members of this clade evolved the M4 grade aRBS by increasing their mean brain size by 15.43 times relative to their ancestral clade (M2_Ancestral) to reach 71.51 cm^3 while increasing their mean body size by 19.56 times to reach 6815.36 g. They obtained the M4 species aRBS scaling relationship by retaining the scaling relationship of their ancestral

clade (M2_Ancestral). However, some caution is warranted around M4_Colobinae and its membership of grade M4, as some relevant taxa were relatively under-sampled (e.g., *Piliocolobus*, which is missing data for all 17 of its species) (Figure 2.1.2). If additional data were to change M4_Colobinae's estimated clade aRBS or species aRBS scaling relationship, then the clade may be found to belong to a different grade.

The clades that comprise grade M4 are not phylogenetically contiguous, meaning that they are an example of convergent evolution towards the same brain-body allometry (Figure 2.2.1). Indeed, these clades are so distantly related that they represent the most marked case of convergent evolution found here.

Grade M5

Grade M5 is characterised by a brain-body allometry that has the same species aRBS scaling relationship as the Ancestral grade and a larger grade aRBS than grade M4. It is comprised of 3 clades: M5_Atelinae, M5_Cacajao_Chiropotes, and M5_Cebus.

M5_Atelinae (spider monkeys, woolly monkeys) (N = 8 species) — The members of this clade evolved the M5 grade aRBS by increasing their mean brain size by 3.92 times relative to their ancestral clade (M4_Ancestral_Platyrrhini) to reach 102.3 cm^3 while increasing their mean body size by 5.12 times to reach 7921.15 g. They obtained the M5 species aRBS scaling relationship by retaining the scaling relationship of their ancestral clade (M4_Ancestral_Platyrrhini).

M5_Cacajao_Chiropotes (uakari, bearded saki) (N = 4 species) — The members of this clade evolved the M5 grade aRBS by increasing their mean brain size by 2.35 times relative to their ancestral clade (M4_Ancestral_Platyrrhini) to reach 61.36 cm^3 while increasing their mean body size by 1.7 times to reach 2632.43 g. The brain and body size data for the members of M5_Cacajao_Chiropotes are highly clustered, which precludes a valid estimate of the clade's

allometric slope. This means that it is not actually possible to tell whether M5_Cacajao_Chiropotes has the M5 species aRBS scaling relationship. However, I made the parsimonious assumption that it retained the scaling relationship of its ancestral clade (M4_Ancestral_Platyrhinni) and so does have the M5 species aRBS scaling relationship. Additional caution is warranted around this clade due to this region of the phylogeny being relatively under-sampled (e.g., *Pithecia*, *Plecturocebus*) (Figure 2.1.2). If the estimated clade aRBS or species aRBS scaling relationship of the clade were to change, then M5_Cacajao_Chiropotes may be found to belong to a different grade.

M5_Cebus (capuchin monkeys) (N = 6 species) — The members of this clade evolved the M5 grade aRBS by increasing their mean brain size by 2.55 times relative to their ancestral clade (M4_Ancestral_Platyrrhini) to reach 66.42 cm^3 while increasing their mean body size by 1.47 times to reach 2277.02 g. As with M5_Cacajao_Chiropotes, it is not actually possible to tell whether M5_Cebus has the M5 species aRBS scaling relationship since its members' brain and body size data are highly clustered, precluding a valid estimate of the clade's allometric slope. However, I made the parsimonious assumption that it retained the scaling relationship of its ancestral clade (M4_Ancestral_Platyrrhini) and so does have the M5 species aRBS scaling relationship. If M5_Cebus turned out to have evolved a different scaling relationship, then the clade would exhibit a different brain-body allometry and so would belong to a different grade.

The clades that comprise grade M5 are not phylogenetically contiguous, so they are another example of convergent evolution towards the same brain-body allometry. However, all being platyrrhines, they are a less extreme example than the clades that comprise grade M4 (Figure 2.2.1). M5_Cacajao_Chiropotes and M5_Cebus are notable, though, since they convergently evolved almost identical mean brain and body sizes (Figure 2.2.4).

Grade M3

Grade M3 is characterised by a brain-body allometry that has the same species aRBS scaling relationship as the Ancestral grade but a smaller grade aRBS than grade M4. It is comprised of 1 clade: M3_Callitrichidae.

M3_Callitrichidae (marmosets, tamarins) (N = 16 species) — The members of this clade are the only primates found to have experienced a decrease in both their mean brain size and mean body size after diverging from their ancestral clade (M4_Ancestral_Platyrrhini) (Figure 2.2.4). The Callitrichidae are indeed known for their phyletic dwarfism (Montgomery and Mundy, 2012). The members of M3_Callitrichidae evolved the M3 grade aRBS by decreasing their mean brain size by 0.35 times to reach 9.25 cm^3 while decreasing their mean body size by 0.27 times to reach 414.13 g (the smallest mean body size after M2_Ancestral). The clade obtained the M3 species aRBS scaling relationship by retaining the scaling relationship of its ancestral clade (M4_Ancestral_Platyrrhini).

Some caution is warranted around grade M3. As it is comprised of only one clade, any changes to the estimated clade aRBS or species aRBS scaling relationship of this clade would alter the characterisation of the grade. And it is possible that this could happen, since some taxa in clade M3_Callitrichidae were relatively under-sampled (e.g., *Mico*, which is missing data for all 14 of its species) (Figure 2.1.2). Additional data for these taxa could alter the results of the allometric analyses.

Grade M1

Grade M1 is an extinct grade that is characterised by a brain-body allometry that ostensibly has the same species aRBS scaling relationship as the Ancestral grade but a smaller grade aRBS — the smallest overall. It is comprised of 1 single-species clade: M1_Catopithecus.

M1_Catopithecus (*Catopithecus browni*) (N = 1 species) — This clade is one of only two found to have experienced a decrease in mean brain size but an increase in mean body size (Figure 2.2.4). *Catopithecus browni* evolved the M1 grade aRBS by decreasing its mean brain size by 0.69 times relative to its ancestral clade (M2_Ancestral) to reach 3.21 cm^3 while increasing its mean body size by 2.58 times to reach 900 g. As M1_Catopithecus is a single species clade, an allometric slope cannot be estimated for it, meaning that it is not actually possible to tell whether the clade has the M1 species aRBS scaling relationship. However, I made the parsimonious assumption that it retained the scaling relationship of its ancestral clade (M2_Ancestral) and so does have the M1 species aRBS scaling relationship.

There is a high degree of uncertainty around grade M1. As it is comprised of only one clade, any changes to the estimated clade aRBS or species aRBS scaling relationship of this clade would alter the characterisation of the grade. And such changes are highly likely to occur given that the clade is comprised of a single fossil species. However, this uncertainty does not affect the later analyses, as fossil grades are not included.

Grade L1

Grade L1 is characterised by a brain-body allometry that has a slightly larger grade aRBS than the Ancestral grade but a "lower" species aRBS scaling relationship (slope = 0.46). This means that relative brain size decreases more with increasing body size (or increases more with decreasing body size). Grade L1 is comprised of 1 clade: L1_Lorisiformes.

L1_Lorisiformes (galagos, angwantibos, lorises, pottos) (N = 18 species) — The members of this clade evolved the L1 grade aRBS by increasing their mean brain size by 1.57 times relative to their ancestral clade (M2_Ancestral) to reach 7.26 cm^3 while increasing their mean body size by 1.56 times to reach 544.05 g. The evolution of a lower species aRBS scaling relationship in L1_Lorisiformes seems to have been driven mainly by selection pressures

favouring a narrower range of absolute brain sizes (brain size variance decreased by 73% in the clade), which had the effect of suppressing relative brain size (Figure 2.2.5, *Table*).

Grade L2

Grade L2 is characterised by a brain-body allometry that has the same "low" species aRBS scaling relationship as grade L1 but a larger grade aRBS. It is comprised of 1 clade: L2_Lemuridae.

L2_Lemuridae (lemurs) (N = 15 species) — The members of this clade evolved the L2 grade aRBS by increasing their mean brain size by 4.99 times relative to their ancestral clade (M2_Ancestral) to reach 23.15 cm^3 while increasing their mean body size by 6.06 times to reach 2110.65 g. As with L1_Lorisiformes, the evolution of a lower species aRBS scaling relationship in L2_Lemuridae seems to have been driven mainly by selection pressures favouring a narrower range of absolute brain sizes (brain size variance decreased by 91% in the clade), which had the effect of suppressing relative brain size (Figure 2.2.5, *Table*).

We can have relatively high confidence in grade L1 since its constituent clade was well sampled, as discussed above (Figure 2.1.2).

Grade L3

Grade L3 is characterised by a brain-body allometry that has the same "low" species aRBS scaling relationship as grades L1 and L2 but a larger grade aRBS than grade L2. It is comprised of 2 clades: L3_Cercopithecinae and L3_Hylobatidae.

L3_Cercopithecinae (mangabeys, macaques, baboons) (N = 47 species) — The members of this clade evolved the L3 grade aRBS by increasing their mean brain size by 17.82 times relative to their ancestral clade (M2_Ancestral) — the second-largest increase in mean brain size found — to reach 82.62 cm^3 while increasing their mean body size by 15.71 times to reach 5474.86 g. Again, the evolution of a lower species aRBS scaling relationship in

L3_Cercopithecinae seems to have been driven mainly by selection pressures favouring a narrower range of absolute brain sizes (brain size variance decreased by 56% in the clade), which had the effect of suppressing relative brain size (Figure 2.2.5, *Table*).

L3_Hylobatidae (gibbons) (N = 9 species) — The members of this clade evolved the L3 grade aRBS by increasing their mean brain size by 29.29 times relative to their ancestral clade (M2_Ancestral) — the largest increase in mean brain size found — to reach 135.78 cm^3 while increasing their mean body size by 25.44 times — the largest increase in mean body size found — to reach 8865.37 g. As with L3_Cercopithecinae, the evolution of a lower species aRBS scaling relationship in L3_Hylobatidae seems to have been driven mainly by selection pressures favouring a narrower range of absolute brain sizes (brain size variance decreased by 88% in the clade), which had the effect of suppressing relative brain size (Figure 2.2.5, *Table*).

Some caution is warranted around the large changes in mean brain and body size found in L3_Cercopithecinae and L3_Hylobatidae. These changes were calculated on the basis of the brain-body allometry of these two clades being derived from the M2_Ancestral allometry, as is currently proposed by the OUrjMCMC analyses, but it is possible that additional data from fossil species in the future may change what brain-body allometry is inferred for basal catarrhines. If it turned out that the brain-body allometry for basal catarrhines was more like that exhibited by M4_Colobinae, and L3_Cercopithecinae's and L3_Hylobatidae's allometries were derived from this instead, then brain evolution in these clades may not have been so dramatic. Indeed, the issue of the characterisation of ancestral allometries changing in the future with the addition of new data, and this changing the inferred extent of evolution in descendant clades, is something that could affect all of the clades described here. However, any changes to the descriptions other clades' brain evolution are unlikely to be as significant as those that could be seen in L3_Cercopithecinae and L3_Hylobatidae.

Grade L4

Grade L4 is characterised by a brain-body allometry that has the same "low" species aRBS scaling relationship as grades L1, L2, and L3 but a larger grade aRBS than grade L3. It is comprised of 1 clade: L4_Hominidae.

L4_Hominidae (gorillas, chimpanzees, orangutans) (N = 8 species) — This clade is one of only two clades — and is the only extant clade — found to have experienced a disproportionate change in mean body size (Figure 2.2.4, *Table*). The members of L4_Hominidae evolved the L4 grade aRBS by increasing their mean brain size by 2.82 times relative to their ancestral clade (L3_Hylobatidae) to reach 382.94 cm^3 while increasing their mean body size by 4.4 times to reach 39012.56 g. The clade obtained the L4 species aRBS scaling relationship by retaining the scaling relationship of its ancestral clade (L3_Hylobatidae).

We can have relatively high confidence in grade L4 since its constituent clade was well sampled (Figure 2.1.2).

Grade H1

Grade H1 is an extinct grade that is characterised by a brain-body allometry that has a smaller grade aRBS than the Ancestral grade but a "higher" species aRBS scaling relationship (slope = 1.09). Indeed, its scaling relationship is hyperallometric (slope > 1), meaning that relative brain size increases with increasing body size (or decreases with decreasing body size). The grade is comprised of 1 clade: H1_Northarctidae_Adapidae.

H1_Northarctidae_Adapidae (*Notharctus tenebrosus, Adapis parisiensis*, *Smilodectes gracilis*, *Pronycticebus gaudryi*) (N = 4 species) — This is the only other clade along with L4_Hominidae found to have experienced a disproportionate change in mean body size (Figure 2.2.4, *Table*). The members of H1_Northarctidae_Adapidae evolved the H1 grade aRBS by increasing their mean brain size by 1.72 times relative to their ancestral clade

(M2_Ancestral) to reach 7.97 cm^3 while increasing their mean body size by 5.17 times to reach 1802.88 g. The evolution of a higher species aRBS scaling relationship in H1_Northarctidae_Adapidae seems to have been driven mainly by selection pressures favouring a narrower range of body sizes (body size variance decreased by 96% in the clade) while a wider range of absolute brain sizes was maintained (Figure 2.2.5, *Table*). This caused relative brain size to increase across the clade.

There is a high degree of uncertainty around grade H1. As it is comprised of only one clade, any changes to the estimated clade aRBS or species aRBS scaling relationship of this clade would alter the characterisation of the grade. And such changes are highly likely to occur given that the clade is comprised of only four fossil species. However, this uncertainty does not affect the later analyses, as fossil grades are not included in them.

Grade H3

Grade H3 is characterised by a brain-body allometry that has the largest grade aRBS found and an even higher species aRBS scaling relationship than grade H1 (slope = 1.22). Again, such a scaling relationship is hyperallometric (slope > 1), meaning that relative brain size increases with increasing body size (or decreases with decreasing body size). The grade is comprised of 1 clade: H3_Hominins.

H3_Hominins (australopiths, early Homo species, humans) (N = 9 species) — The members of this clade are the only primates found to have experienced an increase in mean brain size but a decrease in mean body size (Figure 2.2.4). They evolved the H3 grade aRBS by increasing their mean brain size by 1.35 times relative to their ancestral clade (L4_Hominidae) — the smallest increase in mean brain size found — to reach 518 cm^3 while decreasing their mean body size by 0.86 times to reach 33502.17 g. The shift in species aRBS scaling relationship in H3_Hominins was the most dramatic found here since it involved a change from a “low” scaling relationship in H3_Hominin’s ancestral clade (L4_Hominidae) to a “high”

scaling relationship. This shift seems to have been driven mainly by divergent selection on absolute brain size (brain size variance increased by approximately 4800% in the clade) while a narrower range of body sizes was maintained (Figure 2.2.5, *Table*). This caused relative brain size to increase across the clade.

It is interesting to note that what appears to have made the human lineage unique is not so much its grade aRBS — which only seems to have increased slightly relative to other primates — but the hyperallometric species aRBS scaling relationship that it evolved. This is what led to humans evolving their large relative brain size, by causing relative brain size to increase significantly with only small increases in body size.

Grade H2

Grade H2 is an extinct grade that is characterised by a brain-body allometry that ostensibly has the same species aRBS scaling relationship as grade H3 but a slightly smaller grade aRBS. It is comprised of 1 single-species clade: H2_Boisei.

H2_Boisei (*Paranthropus boisei*) (N = 1 species) — This clade is the only other clade along with M1_Catopithecus found to have experienced a decrease in brain size but an increase in body size (Figure 2.2.4). *Paranthropus boisei* evolved the H2 grade aRBS by decreasing its mean brain size by 0.94 times relative to its ancestral clade (H3_Hominins) to reach 486.13 cm^3 while increasing its mean body size by 1.38 times to reach 46400 g (the largest mean body size found). As H2_Boisei is a single species clade, an allometric slope cannot be estimated for it, meaning that it is not actually possible to tell whether the clade has the H2 species aRBS scaling relationship. However, I made the parsimonious assumption that it retained the scaling relationship of its ancestral clade (H3_Hominins) and so does have the H2 species aRBS scaling relationship.

There is a high degree of uncertainty around grade H2. As it is comprised of only one clade, any changes to the estimated clade aRBS or species aRBS scaling relationship of this clade would alter the characterisation of the grade. And such changes are highly likely to occur given that the clade is comprised of a single fossil species. However, this uncertainty does not affect the later analyses, as fossil grades are not included in them.

Discussion

The 12 grades identified by the allometric analyses represent the macroevolution of relative brain size across primates. As such, they provided a categorical dependent variable that I could use in my observational-inductive analyses to understand the causes of primate brain evolution. However, because several of the grades were found to be comprised of polyphyletic clades, I decided to use the clades as the categorical dependent variable in my analyses instead. This afforded the possibility of understanding not only the causes of the different allometric grades but also why some clades evolved convergently to become members of the same grade.

3. Data II: Niche Variables

Along with data on primate brain evolution, I needed data on the niche variables that could have imposed the selection pressures and constraints that shaped this evolution. Then I could apply my implementation of the observational-inductive approach to uncover the causal relationships between these niche variables and primate brain evolution.

As discussed in Chapter 1, the idea of the observational-inductive approach is that researchers assemble data on as many potentially relevant variables as they can and let observational-inductive methods uncover the relationships between the variables. Therefore, I attempted to gather data on as many potentially relevant niche variables as I could. This included those niche variables that had previously been hypothesised to be relevant to primate brain evolution — e.g., diet, sociality, investment in offspring (see Chapter 1) — along with any other niche variables that I thought might be relevant. To ensure that I ended up with a comprehensive set of niche variables, I used Winemiller et al.'s (2015) niche theory as a guide. This theory suggests that all niches have 5 basic aspects — or dimensions — to them: habitat, trophic, life history, defence, and metabolic. In other words, the life of any organism is situated in a particular habitat, is sustained by a particular diet, follows a particular life history, requires particular threats to be countered, and a particular body to be operated. When assembling my data set of niche variables, I endeavoured to include variables related to each of these niche dimensions. However, I did add a sixth dimension — social — to accommodate the fact that sociality has been hypothesised to be particularly relevant to primate brain evolution (see Chapter 1).

Data Gathering

Data Gathering from Published Sources

I used the resources listed in Table 3.0 to find published compilations of primate niche data. I searched these resources using terms such as "primates AND habitat", "primates AND diet", "primates AND life history", etc.

TABLE 3.0 | RESOURCES USED TO FIND PUBLISHED DATA COMPILATIONS

Resource	Access
Data Retriever	http://www.data-retriever.org/
DataONE	https://www.dataone.org/
Dryad Digital Repository	https://datadryad.org/
Ecological Data Wiki	https://ecologicaldata.org/
Global Biodiversity Information Facility (GBIF)	https://www.gbif.org/
Google Scholar	https://scholar.google.co.uk/
Knowledge Network for Biocomplexity (KNB)	https://knb.ecoinformatics.org/
Manuela Gonzalez	https://tinyurl.com/y8ej858l

Some species were not well-represented in the data compilations that I found, so I also used Google Scholar and "All the World's Primates" (Rowe and Myers, n.d.) to search for primary sources that could supplement the data compilations.

Humans are typically not included in primate data sets because we are an outlier in many respects and so create problems for the regression methods that are used in the standard hypothetico-deductive approach (e.g., DeCasien et al., 2017). However, outliers are less of a concern for the observational-inductive methods that I chose to use for my later analyses, and it is important to

include humans in analyses if we want to understand the causes of human brain evolution, which is arguably one of the main motivations for investigating primate brain evolution at all (as noted at the beginning of Chapter 1). The problem then becomes how to represent humans, who are extremely diverse. I chose to use data from the Hadza hunter-gatherer population to represent humans, which I sourced from Marlowe (2010). The rationale for this was that the hunter-gatherer niche is likely to be most representative of the niche in which human relative brain size — grade H3 in the context of this project — evolved, and the Hadza are regarded as being particularly representative hunter-gatherers (Marlowe, 2010, p. 209).

Primary Data Gathering

As well as gathering data from published sources, I also gathered some data on primate niche variables myself. Specifically, I gathered data on several variables relevant to the habitat and defence dimensions of primates' niches by conducting a series of geospatial analyses (i.e., Geographic Information System, GIS, analyses). I conducted these analyses in R (R Core Team, 2020), primarily using the sf (Pebesma, 2018) and raster (Hijmans, 2020) packages. The code for these analyses can be viewed in the script 03-Gathering-geospatial-data.R.

Habitat Type

The type of habitat that primate species live in is usually represented using relatively crude categories (e.g., forest, savannah) (e.g., Galán-Acedo et al., 2019). However, I was able to gather data on two continuous measures of habitat type: the percentage of land covered by trees and the variation in tree cover. These measures allow for a more fine-grained depiction of differences between species' habitats.

I downloaded a satellite imagery product depicting global tree cover (Dimiceli et al., 2015) from NASA's EarthData search service (National Aeronautics and Space Administration (NASA), n.d.). This is an annual product, and I downloaded the latest 10 years of data (2008-2017) to be able to create a representation of average tree cover in species' habitats. This was to mitigate the risk

of any individual years being unrepresentative due to issues with the imaging or droughts, fires, etc. The satellite data were provided in a relatively raw state, so I had to process them considerably before I could extract usable measures of tree cover. The data for each year was supplied in 170 separate raster files rather than as a single map, so I had to join these files together. The data were also not quality screened, so I had to use a coding scheme that accompanied the data (Townshend et al., n.d.) to filter out any potentially erroneous values caused by data collection issues (e.g., cloud cover affecting the accuracy of the satellite's sensors). Only after this could I aggregate the data for the different years to produce a composite map showing 10-year average tree cover.

To extract the measures of tree cover, I obtained data on primates' geographic ranges from the International Union for Conservation of Nature (IUCN, 2018), filtering these down to just include extant and native ranges. I then overlaid these ranges on the composite map and calculated the median percentage of tree cover and the Median Absolute Deviation (MAD) of percentage tree cover in each primates' range. Definitions of these variables and the details of how they were calculated can be seen in the metadata of Table 3.1.

Using geographic range data instead of data on the home ranges actually utilised by primates is not ideal, in that it could lead to misrepresentations of species' experienced habitats, for instance in cases where species are geographically located in a mosaic environment but only inhabit one part of it, such as forest patches. In such cases, the median percentage of tree cover in species' habitats may be estimated to be relatively low when in fact the species actually inhabit highly forested areas of that habitat. However, there are currently not enough data on primate species' actual home ranges to enable a large-scale analysis. It should be noted, though, that this issue also affects the categorical measures of habitat type that are traditionally used, which are also based on general geographic descriptions.

Biodiversity

The biodiversity of primates' habitats could potentially be relevant to their brain evolution. Primates might need to learn and memorise which other animals are friends or foes, or which plants are edible or not, and the more biodiverse their habitat, and the more there is for them to learn and remember, the larger their brains might need to be (see Box 2.1).

As a proxy for plant biodiversity (Gillman et al., 2015), I gathered data on the Net Primary Productivity (NPP) of primates' niches. The process was similar to that which I used to acquire the tree cover data. I downloaded data from NASA on global NPP for the years 2005-2014 (Numerical Terradynamic Simulation Group (NTSG), 2015). I created a single map for each year by joining together individual raster files and then composited these maps to create a single map of average NPP. I then overlaid the data on primates' geographic ranges onto the composite map and calculated the median NPP of species' ranges.

As a measure of how many other animal species primates might encounter in their habitats, I estimated the mammal, bird, and reptile species richness of primates' geographic ranges. I obtained data on the geographic ranges of mammals (IUCN, 2018), birds (BirdLife International and Handbook of the Birds of the World, 2018), and reptiles (Roll et al., 2017) and filtered these data to just include extant and native ranges. This resulted in a sample of 5427 mammal species (~90% of all species), 10936 bird species (~98% of all species), and 10064 reptile species (~99% of all extant terrestrial species). I then used the st_intersects function from the sf package (Pebesma, 2018) to count the number of overlaps between these species' range geometries and primates' range geometries.

Climate

The final habitat variables that I gathered primary data on were climate variables. I downloaded the Bioclimatic variables data set from WorldClim, which contains data on 19 climate variables that are commonly used for species distribution modelling (Fick and Hijmans, 2017). These data

are preprocessed and are already a multi-year average (1970-2000), so I could immediately extract data on species' median values for various climate variables. I decided that some of the 19 Bioclimatic variables were unlikely to be relevant to primate brain evolution (e.g., Mean Temperature of Wettest Quarter), so I only gathered data for 6 of the most relevant variables (annual mean temperature, mean diurnal range, temperature seasonality, temperature annual range, annual precipitation, and precipitation seasonality).

Predation Pressure

Predation has been recognised as being potentially relevant to primate evolution, but few quantitative data currently exist on it in the literature (McGraw and Berger, 2013; Miller and Treves, 2011). Because I had data on the geographic ranges of mammal, bird, and reptile species (described above), I thought that I could derive a quantitative estimate of the predation pressure that primates might experience from their most common predators — carnivores, raptors, snakes, and crocodiles (McGraw and Berger, 2013; Miller and Treves, 2011) — by estimating the species richness of these predators in primates' ranges. I filtered the mammal, bird, and reptile range data to just the ranges of carnivore (N = 253), raptor (N = 535), snake, and crocodile (N = 3438) species and counted the number of overlaps between these ranges and primates' geographic ranges. I did not attempt to further filter potential predator species to those most likely to predate on particular primate species because there are currently not enough data to be able to reliably do so. For instance, while one might assume that smaller predators would not be able to take a larger adult primate, they may still be relevant as a threat to infants. Similarly, while diurnal predators may not be thought to be relevant to nocturnal primates, there can be interactions between species with different activity patterns, and, indeed, diurnal raptors have been seen to attack the sleeping sites of nocturnal primates (McGraw and Berger, 2013). I chose to consider all predator species and not make any potentially restrictive assumptions until more data are available to be able to better inform which species should be considered in estimates of predation pressure on primate species.

Although observations of actual predation would of course be preferable, studies have shown that predator species richness is a valid indicator of predation pressure (Griffin et al., 2013). Furthermore, the number of predators that primates encounter in their habitats might provide a reasonable measure of *perceived* predation risk, which some have suggested may be just as important for driving evolution as actual predation (Miller and Treves, 2011).

Data Set Assembly

Once I had gathered data on niche variables from various published sources and my own analyses, I needed to assemble it all into a single data set. To do so, I had to standardise each data source to use common units, common variable names, and consistent species names. I resolved the species names used in each data source to a common taxonomy using the Global Names Resolver service ("Global Names Resolver," n.d.), which I accessed through the Taxize R package (Chamberlain and Szöcs, 2013). I chose to use the Integrated Taxonomic Information System (ITIS) taxonomy as the common taxonomy, as this is well-established and used by the Encyclopedia of Life and organisations such as the IUCN ("Integrated Taxonomic Information System (ITIS)," n.d.). The code used for this standardisation process can be viewed in the script 01-Preparing-the-donor-datasets.R.

There are many recognised issues with data quality in the primate literature. For example, estimates of traits that are difficult to measure (e.g., gestation length, social group size) may contain significant measurement error; average values for species may be unrepresentative because of high levels of intra-species variation; and data from different studies may be inconsistent or incompatible because different variable definitions or data collection methods were used (Borries et al., 2013; Garamszegi and Møller, 2010; Patterson et al., 2014). Although I could control the quality of the primary data that I gathered, there was little that I could do about the quality of the data in published sources except control what sources I used for my final data set.

I ranked the data sources that I found in order of perceived quality using a simple schema. If a source demonstrated an effort to ensure data quality (e.g., applied inclusion criteria, performed checks on the data) and was authored by primatologists (whom I assumed would be more likely to spot errors in the data), then I considered it to be "high quality". If a source *either* demonstrated an effort to ensure data quality *or* was authored by primatologists, then I considered it to be "medium quality". If a source demonstrated *neither* an effort to ensure data quality *nor* was authored by primatologists (e.g., it was a general mammal data set that included primates) or had other issues that called into question the quality of its data, then I considered it to be "low quality". Within this scheme, I also ranked sources by publication date, favouring more recent sources. When assembling my data set, I took data from the highest-ranked sources first and only took data from successively lower-ranked sources to fill any remaining gaps. The code used for the assembly process can be viewed in the script 04-Assembling-the-project-dataset.R.

As a further step to ensure data quality, after assembling my data set I cross-checked every data point against species' profiles in Mittermeier et al. (2013), an authoritative reference on primates. This caught several significant errors, such as weaning length in *Pongo pygmaeus* being described as ~2.7 years when really it is closer to 5 years; the diet of *Nomascus gabriellae* being described as being composed of 42% reproductive plant parts when it is really composed of around 90% reproductive plant parts; and the home range size of *Presbytis frontata* being described as 2km^2 when really it is nearer half this. The full list of errors that I found and the corrections that I applied can be viewed in the script 05-Dataset-validation.R.

My final data set can be viewed in Table 3.1. This shows the source of each data point and the definition of each niche variable included.

Data Set Evaluation

My final data set contains 30 measured variables. However, as discussed in Chapter 1, many niche variables are complex, conceptual constructs that require multiple measured variables to represent them (Grace et al., 2012; Grace and Bollen, 2008). So, while there are 30 measured variables in the data set, between them they represent 15 "conceptual" niche variables (e.g., "diet composition", "climate") (Figure 3.2).

There are undoubtedly other variables — both measured and conceptual — that may be relevant to primate brain evolution. However, either there are currently no data available on these variables in the literature or not enough data to be able to include them in a large-scale analysis. Examples include tool use, communication complexity, and basal metabolic rate (BMR). Indeed, I was not able to include any variables related to the metabolic niche dimension, so this dimension is not represented in the final data set.

Coverage

The niche data obviously had to be matched to those species with data on the evolution of their relative brain size, so the coverage of the final data set is the same as that of the data set used for the allometric analyses described in Chapter 2. The full data set covers 192, or 37.8%, of the 508 extant primate species recognised at the time that the data set was assembled (Figure 3.3). The data set covers 71 of the 80 recognised genera, and the mean coverage of each genus is 55% (SD = 35.6%).

The difference between the final data set and the data set that I used for the allometric analyses is that some species have missing data for some niche variables. Even going through my whole ranking of sources to try to fill the gaps, there were still some variables for some species that no published sources seemed to have data on. The methods that I used for the later observational-inductive analyses require complete case data, so missing values either have to be imputed or

cases with missing values have to be dropped (Nakagawa, 2015). I decided not to impute the missing values because the level of missingness in the data is low enough that I thought that the benefits of data imputation did not outweigh the risk of potentially introducing unrepresentative imputed values. Instead, I opted to drop cases with missing data when necessary for the observational-inductive analyses, which makes the coverage of the complete case (CC) version of the final data set relevant.

The CC version of the final data set covers 125, or 24.6%, of recognised primate species (67 fewer species than the full data set) (Figure 3.3). This constitutes a 13.2% reduction in coverage versus the full data set, although almost a quarter of primate species are still represented. Of the 80 recognised genera, 62 are covered in the CC data set (9 fewer genera than the full data set). The mean coverage of each genus is 40.1% (versus 55% in the full data set), and there is a similar amount of variation in coverage: the standard deviation of genus coverage is 37% in the CC data set versus 35.6% in the full data set.

Along with the 9 additional genera that have 0% coverage in the CC data set, 15 genera show a >= 50% reduction in coverage. However, almost half of genera (34) suffer no change in coverage in the CC data set, and 18 genera maintain 100% coverage. Furthermore, dropping species with missing data does not seem to introduce any additional sampling bias into the data set, which is typically the concern with case-wise deletion (Nakagawa, 2015). I tested for phylogenetic signal in the missing coverage of the CC data set using the same process I described in Chapter 2, and this revealed that the *D* value of the CC data set is 0.59, which is very similar to that of the full data set (0.61). So, while missing coverage remains clustered in particular taxa in the CC data set, case-wise deletion does not introduce much additional bias.

Missingness

To better understand what drives the changes in coverage in the CC data set, I evaluated missingness in the full data set.

As I mentioned, the level of missingness in the data set is reasonably low (Figure 3.4). Most of the variables (25 of 30) have <5% missingness. This means that the reduction in coverage in the CC data set is driven mainly by missing data for only a few variables. The 5 variables with >5% missingness are all life history or trophic variables (total maternal investment, % reproductive plant parts, % nonreproductive plant parts, % prey, and mating system). Furthermore, of the 67 species dropped in the CC data set, 52 are only missing data for 1 to 3 variables (28 are only missing data for 1 variable). Similarly, of the 9 genera lost in the CC data set, many were only lost due to missingness in one or two variables (total maternal investment for *Allochrocebus* and *Presbytis*; mating system for *Chiropotes*; and maximum longevity and extrinsic mortality rate for *Indri* and *Avahi*) (Figure 3.4, *By genus*).

Missingness appears to be distributed quite randomly in the data set (Figure 3.4, *By genus*) and tests for phylogenetic signal confirm this. Of the 5 variables that contribute most to missingness, 3 (the trophic variables) show no phylogenetic signal in their missingness and are thus missing at random (their D values are not significantly different from 1 (random distribution); $p > 0.05$). Missingness in the other two variables with the highest missingness (total maternal investment and mating system) is non-randomly distributed (the D values of these variables are 0.61 and 0.75, respectively, which are significantly different from 1 (random distribution); $p < 0.05$). However, the missingness in these two variables happens to be clustered on opposite ends of the primate phylogeny, so they balance each other out (Figure 3.4, *By genus*). The overall random distribution of missingness in the data set explains why dropping cases does not introduce additional sampling bias into the CC data set: cases are dropped quite evenly across taxa.

Distributions

Of the 30 measured variables in the final data set, 26 are continuous and 4 are factor (diel activity, substrate, mating system, social system). Even after transformation, most of the continuous variables (19 of 26) are non-normally distributed, and most (21 of 26) have global outliers (Figure

3.5). The situation is similar in the CC version of the data set: most continuous variables (17 of 26) are non-normally distributed after transformation, and most (17 of 26) have global outliers. Most of the factor variables (3 of 4) also exhibit unbalanced classes in both the full and CC data sets. These distributions illustrate how complex real-world data are and emphasise the necessity of using methods that do not make unrealistic assumptions, unlike typical hypothetico-deductive methods.

Comparison with Other Data Sets

The coverage of my full data set compares favourably to that of two of the largest data sets previously used to investigate the causes of primate brain evolution. Compared to my 192 species, the Shultz & Dunbar (2007) data set covers 43 species and the DeCasien et al. (2017) data set covers 144 species (Figure 3.3). Compared to my coverage of 71 genera, Shultz & Dunbar cover 40 and DeCasien et al. cover 70. The mean coverage of each genus is 15.4% in the Shultz & Dunbar data set and 42.7% in the DeCasien et al. data set compared to 55% in my data set.

The sampling bias in my data set is also no worse than that in these previously used data sets. These, too, have relatively high coverage of the Homininae and relatively low coverage of the Pitheciidae and Cheirogaleidae. A test for phylogenetic signal in missing coverage confirmed that the DeCasien et al. data set has a similar D value to mine (0.73 vs 0.61). The clustering of missing coverage is not actually statistically significant in the Shultz & Dunbar data set (its D value is 0.96, which is not significantly different from 1 (random distribution); $p = 0.24$), but this is probably due to the overall low coverage of the data set.

My data set contains 30 measured variables compared to the 5 in both the Shultz & Dunbar and DeCasien et al. data sets. This exemplifies the difference between a data set intended for modelling complex natural systems using an observational-inductive approach and data sets intended for simple hypothesis testing.

This comparison with two of the largest data sets in the field demonstrates that — in terms of both species coverage and number of variables — my data set is the most substantial yet assembled for a study of the causes of primate brain evolution.

4. Correlation Models

After assembling a set of data on primate brain evolution and niche variables, I could begin to apply my implementation of the observational-inductive approach to search this data set for patterns indicating causal relationships between the variables. As I said in Chapter 1, I conducted this search for causal relationships in two stages. First, I searched the data set for correlations between variables (described in this chapter) to identify which variables are related at all. Then I searched within the resulting correlation models for specific patterns of correlation that indicate how variables are causally related (described in Chapter 5).

The primary method that I used to test for correlations between variables was Multiscale Graph Correlation (MGC) (Vogelstein et al., 2019) (Box 4.1). This method is an elaboration of the classic Pearson correlation test (Pearson and Galton, 1895) that was designed specifically for observational-inductive analyses. Unlike traditional methods, it does not make restrictive assumptions about data (e.g., normal distributions, no outliers); it can be used on mixed data sets (both continuous and factor variables); and it can detect nonlinear correlations. It is able to do all this by analysing not the data points themselves but the distances between data points.

MGC works on the principle that if two variables are linearly correlated, then data points that have similar (close) values in one variable will have similar (close) values in the other variable, and data points that have dissimilar (distant) values in one variable will have dissimilar (distant) values in the other variable. Since distances can be calculated between the data points of different types of variables with the appropriate distance metric (e.g., Gower, 1971), such “distance correlations” can be detected in mixed data sets.

Box 4.1 | Phylogenetic Comparative Methods

It has become standard procedure in analyses testing for correlations between species' traits to use specialist Phylogenetic Comparative Methods (PCMs) (e.g., DeCasien et al., 2017; Powell et al., 2017). However, MGC and the other methods that I used for my observational-inductive analyses are not PCMs. This was deliberate, because the use of PCMs is based on an assumption that is likely not valid when investigating the causes of primate brain evolution.

The use of PCMs is based on the assumption that phylogenetic inertia is a major confounding factor in comparative analyses that causes more closely related species to exhibit correlated trait values simply because they have retained the traits of a common ancestor, not because there is an adaptive relationship between traits (Felsenstein, 1985). PCMs seek to remove the confounding effect of phylogenetic inertia by "down weighting" the contribution of data from closely related species to correlation analyses (Symonds and Blomberg, 2014, p. 110).

However, given how expensive brains are (see Box 1.1), it is unlikely that a neutral evolutionary process such as phylogenetic inertia would have a significant influence on primate brain evolution, especially over the sorts of million-year periods that separate primate species. Even Felsenstein (1985), the source most often cited to justify the use of PCMs (9350 citations to date), said that phylogenetic inertia would be "essentially absent" in traits that are very responsive, making PCMs unnecessary. Furthermore, when investigating the causes of primate brain evolution, we are typically testing for correlations with and among niche variables, which are not heritable traits and so cannot experience phylogenetic inertia. For these reasons, PCMs are likely unnecessary for studies investigating the causes of primate brain evolution.

Not only are PCMs likely unnecessary, but they could be counterproductive. If phylogenetic inertia is not responsible for closely related species having similar trait values, then selection pressures probably are. More closely related species may exhibit correlated trait values because they are adapted to similar niches. If this is the case, then applying PCMs and down weighting species' data would artificially weaken the very adaptive correlations we seek (Blomberg and Garland, 2002; Hansen, 2014; Losos, 2011; Uyeda et al., 2018; Westoby et al., 1995b, 1995a).

MGC is able to detect nonlinear correlations by testing for distance correlations within different-sized subsamples of neighbouring data points. The idea is that nonlinear relationships will still be linear at some scale. If significant distance correlations are detected within any size of neighbour subsample — i.e., at any scale — then there is some sort of correlation between the two variables,

either linear or nonlinear. The scale at which the correlation breaks down indicates how nonlinear the relationship between variables is. If the relationship is perfectly linear, then significant distance correlations will be detected through all scales, up to the size of the full sample.

Multiscale Graph Correlation (MGC) Analyses

Before the MGC analyses, I transformed the niche variable data. This was not a requirement of the analyses; I did it to focus on proportional — rather than absolute — variation in the niche variables, as proportional variation is likely to be more revealing (Sokal and Rohlf, 2012, p. 426). For example, species with a social group size of 1 and a social group size of 5 have the same absolute difference in group size as species with group sizes of 20 and 24. However, proportionally, the first species exhibit a 400% difference in social group size, whereas the second species only exhibit a 20% difference. I applied the appropriate transformations for each data type: log transformation for continuous data, square root transformation for count data, and logit transformation for percentage data (Sokal and Rohlf, 2012, pp. 430, 433, 532; Warton and Hui, 2011). The only exception was social group size which, while technically count data, I log transformed because the data are species means and so more closely resemble continuous data (e.g., mean group sizes of 3.5 or 7.8).

As I discussed at the end of Chapter 2, I decided to use the allometric clades — rather than the allometric grades — as the dependent variable in my observational-inductive analyses to be able to investigate why some clades convergently evolved the same brain-body allometry. I binary-coded clade membership for the MGC analyses so that, for each clade, members were assigned a value of 1 and nonmembers were assigned a value of 0. This meant that any detected correlations between niche variables and the clades would signify an association between those niche variables and being a member of a particular clade or not.

As described in Chapter 2, I identified 17 allometric clades. However, 3 of these clades are extinct (M1_Catopithecus, H1_Northarctidae_Adapidae, and H2_Boisei) so could not be included in the MGC analyses — or any subsequent analyses in this project — because there are no niche variable data available for them. Furthermore, 2 clades (M4_Daubentonia and H3_Hominins) consist of only one extant species, and correlation tests cannot be performed on single data points (though see below). Therefore, 12 clades were included in the MGC analyses.

I used MGC to test for correlations between every included clade and every niche variable in the data set (although I did exclude the niche variables total species richness and total predator species richness, as these are technically summary variables that are redundant if their component variables are included in analyses). I also tested for correlations between every niche variable and every other niche variable to establish which variables are related. In total, this process required 714 individual correlation tests. Furthermore, to ensure reliable *p*-values, I ran 10000 iteration permutation tests for each correlation test. This made the analysis process extremely computationally intensive, and it took approximately 16.5 hours to complete on a 48-core Amazon Web Services (AWS) EC2 instance. Afterwards, I adjusted the 714 *p*-values for multiple testing using the Benjamini-Hochberg procedure (Benjamini and Hochberg, 1995; Korthauer et al., 2019). The code of the MGC analyses can be viewed in the script 01-Correlation-models.R.

Novelty Detection

As I mentioned above, the clades M4_Daubentonia and H3_Hominins could not be included in the MGC analyses because they each contain only one extant species (*Daubentonia madagascariensis* and *Homo sapiens*, respectively). As an alternative means of establishing which niche variables are associated with these clades' distinct brain-body allometries, I tested whether their member species exhibit significantly distinct or novel values for any niche variables.

I used a nearest neighbours-based novelty detection method to do this, called *k*-NN (Ding et al., 2014; Goldstein and Uchida, 2016; Tax, 2001). This method is similar to MGC in that it also uses distance measurements. *k*-NN tests whether a data point is significantly distant from other data points in the sample; if it is, then the data point is considered to have a novel value. More specifically, *k*-NN measures the distances between a data point and its *k* nearest neighbours (i.e., data points with the closest values) and then measures the distances between these *k* nearest neighbours and *their k* nearest neighbours. If the average distance from the data point to its nearest neighbours is significantly greater (determined by a specified threshold value) than the "normal" average distance between the other neighbours, then the data point is considered to be abnormally different or novel.

I used the *k*-NN method to test which continuous niche variables *Daubentonia madagascariensis* and *Homo sapiens* — and therefore clades M4_Daubentonia and H3_Hominins — exhibit novel values for. For factor variables, I simply noted whether each species was the only one to exhibit a particular factor value. When specifying the value of *k* for each *k*-NN test, I followed the standard heuristic of using the square root of the sample size (minus the test data point) (Hassanat et al., 2014). For the threshold value, I considered a data point to be novel if the average distance from the data point to its nearest neighbours was 1.5 times the "normal" distance between neighbours. The code for the *k*-NN analyses can be viewed in the script 01-Correlation-models.R.

Results

Figure 4.0 shows the significant correlations that were found between the niche variables and each allometric clade ($p < 0.05$ for the correlations found with MGC; *k*-NN tests do not produce *p*-values). These correlation models are only an intermediate step in the process of developing causal models to understand the evolution of each clade, so it would be inappropriate to draw any conclusions from them. However, they do provide initial validation of several of the

assumptions made in Chapter 1 about how complex causal models of primate brain evolution might end up being.

The correlation models make it clear that there are likely to be many densely interlinked niche variables involved in primate brain evolution. All of the 28 niche variables analysed were found to be either directly or indirectly correlated with membership of the allometric clades, and many relationships were found among niche variables (the median number of correlations in each model is 285). Even if most of the correlations turned out to be spurious, it is still likely that many causal relationships would remain in the final causal models.

The finding that most of the correlations are weak (illustrated by the thickness of the edges in Figure 4.0) further supports the idea that multiple variables are likely involved in primate brain evolution, as it may only be through the additive effect of multiple variables that substantial selection pressures or constraints are produced. Although most of the correlations were found to be highly significant (median *p*-value = 0.0006, MAD *p*-value = 0.0004) the median correlation is only 0.106 (MAD = 0.071).

The correlation models also validate the assumption that multiple causal models will likely be required to understand primate brain evolution, as different clades were found to be correlated with different sets of niche variables (Figure 4.0, *e.g., M2_Ancestral and H3_Hominins*). This is exemplified by the fact that different clades have different numbers of niche variables directly correlated with them (the number of "direct nodes" in the models ranges from 1 to 24). This even includes clades within the same grade. For example, the constituent clades of grade M4 — M4_Ancestral_Playrrhini, M4_Daubentonia, and M4_Colobinae — are directly correlated with 12, 1, and 19 niche variables, respectively. Again, even if most of the correlations turned out to be spurious, these different sets of relationships are likely to translate into different causal models. For clades within the same grade, this raises the intriguing possibility that different sets of causal influences might result in the evolution of the same brain-body allometry.

Finally, the results of the correlation analyses demonstrate the effectiveness and efficiency of the observational-inductive approach (as discussed in Chapter 1). Within a matter of hours, the MGC and *k*-NN analyses were able to uncover hundreds of relationships potentially relevant to primate brain evolution, including nonlinear relationships and relationships between continuous and factor variables. To thoroughly screen a data set in this way could take years with the standard hypothetico-deductive approach.

The next step of my implementation of the observational-inductive approach was to determine how the correlated variables are actually causally related.

5. Causal Models

After establishing which niche variables are correlated with primate brain evolution (Chapter 4), the next step in my implementation of the observational-inductive approach was to search within the correlation models for specific patterns of correlation that indicate how the variables are causally related. For although correlation may not imply causation, causation does imply a specific pattern of correlation (Shipley, 2016).

Specifically, causation implies that if A causes B, A and B will still be correlated even under conditions where all other variables correlated with the pair are controlled for — either physically, as in controlled experiments, or statistically. Conversely, if A does not cause B, and any correlation between them is spurious, then A and B will not be correlated under conditions where all other variables correlated with the pair are controlled for (Glymour et al., 2019b; Pearl and Mackenzie, 2018, pp. 113–116). Causal discovery methods can be used to search data sets for such patterns of conditional correlation and conditional independence (Glymour et al., 2019b) (Box 5.1). The ability of these methods to reliably uncover true causal relationships has been demonstrated in numerous simulation studies and real-world applications (see Glymour et al., 2019a).

Broadly speaking, there are two types of causal discovery methods: constraint-based methods and score-based methods. Constraint-based methods test for conditional correlation and conditional independence between variables in a step-wise manner to reach a final overall model of causal relationships (Glymour et al., 2019b). Score-based methods, on the other hand, propose different patterns of conditional correlation and conditional independence and find which pattern(s) provide the best fit to the data using a score such as the Bayesian Information Criterion (BIC) (Glymour et al., 2019b; Heckerman et al., 1999; Huang et al., 2018).

I chose to use a score-based causal discovery method for my analyses, specifically a version of Greedy Equivalence Search (GES) (Chickering, 2002) that incorporates a novel cross-validated likelihood scoring function (Huang et al., 2018). After surveying the constraint-based and score-based causal discovery methods currently available, I decided that this GES method was the best suited to observational-inductive analyses. It does not make assumptions about the distribution of data, it can be used on mixed data sets (both continuous and factor variables), and it can detect nonlinear causal relationships. It has also been shown to be more accurate and reliable than other causal discovery methods, both on simulated data and on real-world data sets (Huang et al., 2018).

However, this method does assume that data are complete case (as mentioned in Chapter 3), that there are no feedback loops between variables, and that there are no latent variables in the analysis (i.e., variables that might be causally involved in the system of interest but that have not been included). These last two assumptions are unlikely to be fully met when modelling the causes of primate brain evolution and violating them could increase the Type I error rate of analyses (i.e., finding causal relationships to exist when they do not). However, as I said, I surveyed the causal discovery methods currently available and could find none that were better suited. Therefore, until even more capable methods are developed, the potential for spurious relationships will simply have to be kept in mind when interpreting the final causal models. As I said in Chapter 1, my goal was not to produce a definitive understanding of the causes of primate brain evolution in this project but simply to provide a proof-of-concept of the potential of observational-inductive methods to provide a better understanding than we have currently.

Box 5.1 | The C-word

Outside of the context of randomised controlled experiments, it is generally considered taboo to speak of "causation" (Hernán, 2018; Hernán et al., 2019; Shipley, 2016, pp. 5, 11). Statistics textbooks and undergraduate courses teach us that causation cannot be determined from observational data alone because statistical methods can only establish correlation, and correlation does not imply causation. Consequently, researchers would be naïve to make causal claims. However, this state of affairs is more the product of cultural evolution than statistical reality. Statistical methods for inferring causation from observational data have been available since the 1920s, when the renowned evolutionary biologist Sewall Wright invented path analysis (Wright, 1921). However, Wright's ideas were suppressed by the two dominant statistical schools of the time.

The first school was that led by Karl Pearson, the father of the correlation coefficient (Pearl and Mackenzie, 2018, p. 62). Pearson's school was based on a positivist philosophy of science which held that the universe is a product of human thought (Pearl and Mackenzie, 2018, p. 67). It was therefore believed that it was not possible to study causation at all because there could be no such thing as a cause that was external to the human mind (Pearl and Mackenzie, 2018, p. 67). According to this school, the goal of science was to mathematically describe the correlations between phenomena, not discover the underlying reasons for these correlations (Shipley, 2016, p. 58). Pearson aggressively enforced his beliefs, and he had considerable influence as head of the Biometrics Lab at University College London — the world centre for statistics at the time — and as editor of the statistics journal *Biometrika* (Pearl and Mackenzie, 2018, p. 68).

The second school was that led by Ronald A. Fisher, perhaps the greatest promoter of the idea that randomised controlled experiments were the only way to establish causation (Shipley, 2016, p. 59). Indeed, Fisher was so opposed to the idea that causation could be inferred from observational data alone that he wrote an entire book dismissing the claim that smoking was a cause of disease on the grounds that randomised controlled trials had not been conducted (Shipley, 2016, p. 60; Stolley, 1991). Like Pearson, Fisher was militant in his beliefs and feuded with anyone with differing views, including Sewall Wright. Although the focus of Fisher's feud with Wright was Wright's theory of genetic drift, path analysis was collateral damage (Pearl and Mackenzie, 2018, p. 85).

Researchers should not be pressured to either ignore causal questions or ashamedly obscure the true goals of their research simply because of the historical influence of a few dominant personalities. This only stifles open and progressive science (Hernán, 2018; Hernán et al., 2019). Valid methods exist to explicitly study causation using observational data, and researchers should not be afraid to adopt these methods and their associated language. It would be fitting for evolutionary biologists to lead the way in this, given that their field was the birthplace of causal analysis.

Greedy Equivalence Search (GES) Analyses

I used the same data for the GES analyses as I did for the correlation analyses, with niche variables appropriately transformed and allometric clade membership binary coded (see Chapter 4).

I provided the information on the correlations found between variables in the form of adjacency matrices, one for each correlation model. An adjacency matrix is a table with a row per variable and a column per variable where the value in a given cell indicates the relationship between the row variable and the column variable (Newman, 2018, pp. 106–108). In this case, a value of 1 indicated that a significant correlation ($p < 0.05$ for MGC correlations) had been found between the two variables, while a value of 0 indicated that no significant correlation had been found between the variables. The GES method was configured to only look for causal relationships among those variables that had been found to be significantly correlated.

Several causal relationships could be ruled out *a priori*. For example, the MGC analyses found a significant correlation between bird species richness and annual mean temperature (MGC = 0.23, $p < 0.001$), but common sense would suggest that bird species richness does not cause the temperature (although the opposite may, of course, be true). As a quasi prior for the analyses, I modified the adjacency matrices so that the GES method would not consider obviously implausible causal relationships such as these. A list of the causal relationships that I prohibited can be viewed in the file general-prior-knowledge.csv. The only other causal relationships that I prohibited in addition to the ones on this list were those directed from the allometric clades to the niche variables. Although the theory of niche construction makes it a matter of debate as to whether relative brain size could be a cause of certain niche variables (Kendal et al., 2011; Laland et al., 2016), for the purposes of these analyses (i.e., to avoid feedback loops) I prohibited clade membership from being a cause of any niche variables.

To mitigate the reduction in sample size due to the GES method's requirement for complete case data, I split the causal discovery process into two stages. First, I applied the GES method to the subsets of niche variables that were found to be directly correlated with each clade, to identify each clades' direct causes. Then I used the GES method to resolve the causal relationships among all the niche variables in the data set. Since the analyses to establish the direct causes of each clade only required data on those niche variables found to be directly correlated with each clade, the data for these analyses only had to be complete case across this subset of variables. The analysis to establish the causal relationships among all the niche variables was the only one that needed to be complete case across the full data set and so had the complete case sample size described in Chapter 3 (N = 125). Once both the direct causes of each clade and the causal relationships among all the niche variables had been established, I could join the results to produce full models of the direct and indirect causes of each clade.

As I mentioned above, the particular version of GES that I used incorporated a novel cross-validated likelihood scoring function. By default, this function uses 10-fold cross-validation, but as a further step to maximise sample sizes during the analyses, I changed this to leave-one-out cross-validation (Sammut and Webb, 2010). This way, instead of $1/10^{th}$ of the data set being left out of each round of cross-validation, only 1 case was left out.

It was not possible to include the clades M4_Daubentonia or H3_Hominins in the GES analyses for the same reason that it was not possible to include them in the MGC analyses described in Chapter 4. Each of these clades only contains one extant species, and it is not possible to perform (conditional) correlation tests on single data points. Consequently, it was not possible to resolve the direct causes of these clades. Therefore, in these clades' final causal models, the direct relationships between the niche variables and the clades continue to represent correlations.

The GES analyses were run in MATLAB (*MATLAB*, 2019) from R (R Core Team, 2020). The code can be viewed in the script 01-Causal-models.R.

Once I had the final causal models for each clade, I calculated several network statistics to evaluate the causal importance of each niche variable in each model. To evaluate whether niche variables are important as direct or indirect causes of clade membership, I calculated the degree of separation — or distance — between each niche variable and the clade variable in each model. To evaluate whether niche variables are important "mediators" of the causal influence of other variables, I calculated each variables' In-degree, which is the number of variables that cause it (Newman, 2018, p. 159). Similarly, I calculated each niche variables' Out-degree — the number of variables that it causes — as an indicator of how important each niche variable is as an "influencer" of other variables (Newman, 2018, p. 159). Finally, to evaluate whether niche variables are important by their association with important "mediator" or "influencer" variables, I calculated each variables' PageRank in each model (Gleich, 2014; Newman, 2018, pp. 165–167). A variable will have a high PageRank if it is connected to highly connected variables.

Results

The final causal models produced by the GES analyses can be seen in Figure 5.0. If, as I suggested at the beginning of Chapter 1, the end goal of research into the causes of primate brain evolution is to produce realistically complex models of the causal relationships between niche variables and primate brain evolution, then I would argue that the models depicted in Figure 5.0 represent the closest we have yet come to that goal.

Complex causal models are most effectively and efficiently described by the sorts of network diagrams presented in Figure 5.0. However, as a supplement to these diagrams, I provide below a brief verbal summary of each of the 14 models. For simplicity, I describe the models mostly with reference to the conceptual niche variables that they depict (see Figure 3.2). Also, in these descriptions, I refer to variables' average values in a clade and characterise these as "high", "medium"/"moderate", or "low" in relation to other clades' average values. Please see Figure 5.0

for full details of the measured niche variables in each model, their values and distributions, and their relationships with other variables.

I describe the final causal models for each clade in the same order in which I described the grades and clades in Chapter 2 (minus the 3 extinct grades/clades). I start by describing the ancestral clade and all the clades that share the same species aRBS scaling relationship (allometric slope), proceeding roughly in the order in which the clades evolved (Figure 2.2.1). I then describe the clades that diverged from the ancestral species aRBS scaling relationship by evolving "lower" or "higher" scaling relationships. Please refer to Chapter 2 and Figures 2.2.1 to 2.2.5 for details of the brain-body allometry of each clade.

Causal Model Summary

M2_ANCESTRAL (dwarf lemurs, mouse lemurs, fork-marked lemurs, sifakas, woolly lemurs, sportive lemurs, tarsiers)

The brain-body allometry that characterises M2_Ancestral appears to be directly caused by defence-related niche variables. Specifically, this clades' small clade aRBS and "medium" species aRBS scaling relationship appear to be a direct adaptation to high overall mortality risk, but low predation pressure from raptors and low levels of intraspecific conflict.

The clade's brain-body allometry seems to be indirectly caused by a generally warm but highly variable climate and an arboreal habitat that has moderate amounts of tree cover and plant biodiversity. A diet composed of low amounts of reproductive plant parts and prey also seems to play an indirect causal role. So too does a low investment in offspring and a social life that is either solitary or features small group sizes.

M4_ANCESTRAL_PLATYRRHINI (night monkeys, howler monkeys, squirrel monkeys, saki monkeys, titi monkeys)

M4_Ancestral_Platyrrhini has the same species aRBS scaling relationship as M2_Ancestral, but a larger clade aRBS. Habitat variables seem to be the main direct cause of this brain-body allometry. The members of M4_Ancestral_Platyrrhini are all arboreal and have small to medium-sized home ranges in habitats rich in bird species. As well as habitat variables, the clade's brain-body allometry appears to be a direct adaptation to moderate predation pressure from carnivores.

M4_Ancestral_Platyrrhini's brain-body allometry appears to be indirectly caused by diurnal activity in highly forested habitats that have moderate plant biodiversity and hot and moderately variable climates. Other indirect causes include a diet low in prey but with moderate amounts of reproductive plant parts and mostly monogamous mating systems that feature moderate to low investment in offspring. On top of predation pressure from carnivores, moderate to high predation pressure from raptors seems to be indirectly relevant to members of the clade, as does a moderate to high mortality risk. Species in M4_Ancestral_Platyrrhini exhibit either social monogamous or group living social systems that are characterised by small to moderate group sizes, and this also seems to contribute indirectly to the clade's brain-body allometry.

M4_Daubentonia (Aye-Aye)

Being members of the same grade, M4_Daubentonia has the same brain-body allometry as M4_Ancestral_Platyrrhini. However, as noted above, it was not possible to establish the direct causes of this brain-body allometry in M4_Daubentonia. All that it is possible to say is that it is *potentially* caused by low predation pressure from raptors. This low number of raptors appears to be due to a relatively cool but highly variable climate and a moderately forested habitat with high plant biodiversity.

M4_Colobinae (colobus monkeys, leaf monkeys, snub-nosed monkeys, langurs)

Again, being members of the same grade, M4_Colobinae has the same brain-body allometry as M4_Ancestral_Platyrrhini and M4_Daubentonia. However, this brain-body allometry appears to

have more direct causes in M4_Colobinae. Diurnality seems to play a direct role, as does a diet composed of low amounts of prey and high amounts of nonreproductive plant parts. Other direct causes include harem polygyny mating systems, moderate investment in offspring, group living social systems, and moderate group sizes. Finally, the clade's brain-body allometry also appears to be a direct adaptation to moderate predation pressure from raptors and high predation pressure from carnivores.

In addition to the direct effects of predation pressure, a moderate to high mortality risk seems to contribute indirectly to M4_Colobinae's brain-body allometry. Habitat variables also seem to have an indirect influence. Species in M4_Colobinae are mostly arboreal and live in moderately to highly forested habitats that have moderate biodiversity and relatively hot but not especially variable climates. Presumably because of its relationship with the other diet variables that were found to be direct causes, the percentage of reproductive plant parts in species' diets was also found to be an indirect cause of the clade's brain-body allometry.

M5_Atelinae (spider monkeys, woolly monkeys)

Clade M5_Atelinae is characterised by the same species aRBS scaling relationship as M2_Ancestral and a larger clade aRBS than the clades in grade M4. The clade's brain-body allometry appears to be directly caused by group living social systems, polygynandrous mating systems, and moderate to high investment in offspring. Moderate precipitation seasonality and high mammal species richness also seem to have a direct causal influence.

Indirect causes of M5_Atelinae's brain-body allometry include a relatively hot and invariant climate and a biodiverse, arboreal habitat with high tree cover. A diet high in reproductive plant parts and low in prey also seems to be relevant to the clade, as does a relatively large social group size. Finally, the clade's brain-body allometry seems to be indirectly caused by moderate to high predation pressure from raptors and carnivores and moderate overall mortality risk.

M5_Cacajao_Chiropotes (uakari, bearded saki)

M5_Cacajao_Chiropotes's brain-body allometry — which is the same as M5_Atelinae's — was found to be directly caused by moderate predation pressure from carnivores and moderate levels of intraspecific conflict. Diets high in reproductive plant parts and habitats rich in reptile species were also found to be direct causal influences.

Moderate mammal, bird, and plant biodiversity appear to play an indirect role in causing M5_Cacajao_Chiropotes's brain-body allometry. Accompanying this biodiversity is moderate predation pressure from raptors, snakes, and crocodiles and generally high mortality risk. All of this occurs within arboreal and highly forested habitats that have relatively hot and stable climates. Species in M5_Cacajao_Chiropotes have diets composed of moderate to low amounts of prey, they make moderate investments in their offspring, and they live in relatively large groups. All of these also indirectly contribute to the clade's brain-body allometry.

M5_Cebus (capuchin monkeys)

As they are part of the same grade, M5_Cebus has the same brain-body allometry as M5_Atelinae and M5_Cacajao_Chiropotes. In M5_Cebus, this brain-body allometry seems to be a direct adaptation to long lives lived in moderate to large social groups that feature moderate levels of intraspecific conflict. This level of conflict is accompanied by moderate mortality risk. Moderate levels of precipitation seasonality also seem to have a direct influence on the clade.

Other climate variables have a more indirect influence on M5_Cebus: members of the clade experience generally high temperatures with little climatic variation. Other indirect causes of the clade's brain-body allometry include species' arboreality, their highly forested habitats with moderate plant biodiversity, and diets composed of moderate amounts of reproductive plant parts and moderate to high amounts of prey. Group living social systems naturally accompany the large social groups that were found to be a direct cause of the clade's brain-body allometry, and

moderate to high predation pressure from raptors was also found to be related to these variables. Finally, the moderate investment in offspring made by species in M5_Cebus was found to be an indirect cause of the clade's brain-body allometry.

M3_Callitrichidae (marmosets, tamarins)

As described in Chapter 2, this clade was the only one found to have experienced a decrease in both mean brain size and mean body size. This left M3_Callitrichidae with a brain-body allometry that has the same species aRBS scaling relationship as clade M2_Ancestral and a smaller clade aRBS than the clades in grade M4. M3_Callitrichidae's brain-body allometry seems to have many direct causes. Species' diurnality and arborality seem to have a direct influence, as does their diet, which is composed of moderate to high amounts of prey. Other direct causes of the clade's brain-body allometry include a low investment in offspring, a moderate to high mortality rate, and monogamous social systems with moderate group sizes.

Indirectly, M3_Callitrichidae's brain-body allometry seems to be caused by moderately to highly forested habitats that have moderate plant biodiversity and hot and moderately variable climates. Within these habitats, members of the clade experience moderate to high predation pressure from raptors. Other indirect causes of the clades' brain-body allometry include the moderate amounts of reproductive plant parts that species consume and the monogamous or polyandrous mating systems that they exhibit.

L1_Lorisiformes (galagos, angwantibos, lorises, pottos)

L1_Lorisiformes was the first clade to exhibit a shift to a species aRBS scaling relationship "lower" than that of the ancestral grades'. However, the clade's clade aRBS is only slightly larger than M2_Ancestral's. This particular brain-body allometry appears to be a direct adaptation to a nocturnal and largely solitary lifestyle, spatial polygyny mating systems, and moderate to high predation pressure from raptors.

L1_Lorisiformes's brain-body allometry appears to be indirectly caused by low investment in offspring, high mortality risk, and diets high in prey and low in reproductive plant parts. Life in arboreal habitats with moderate tree cover, moderate to low plant biodiversity, and relatively warm and variable climates also appears to be indirectly causal.

L2_Lemuridae (lemurs)

L2_Lemuridae has a brain-body allometry that has the same "low" species aRBS scaling relationship as L1_Lorisiformes but a larger clade aRBS. The direct causes of this brain-body allometry appear to include a low investment in offspring, moderate mortality risk, and a diet composed mostly of reproductive plant parts and no prey. L2_Lemuridae's brain-body allometry also appears to be directly influenced by species' cathemerality in habitats that have low bird species richness and concomitantly low predation pressure from raptors.

As well as low predation pressure from raptors, members of L2_Lemuridae experience low predation pressure from carnivores, and this was found to be an indirect cause of their brain-body allometry. Also indirectly causative are species' mostly polygynandrous mating systems and social life in moderately sized groups. Warm, highly variable climates and moderately forested arboreal habitats with high plant biodiversity also seem to indirectly influence the clade.

L3_Cercopithecinae (mangabeys, macaques, baboons)

L3_Cercopithecinae has a "low" species aRBS scaling relationship and a larger clade aRBS than L2_Lemuridae. Its brain-body allometry seems to be directly caused by moderate investments in offspring, high levels of intraspecific conflict, moderate mortality risk, and a diet composed of moderate amounts of prey.

Moderate to high amounts of reproductive plant parts in the diet was found to be an indirect cause of the clade's brain-body allometry. Other indirect causes include large social groups, moderate to high predation pressure from raptors, and habitats that have moderate tree cover, low plant

biodiversity, and moderately warm and variable climates. The fact that species can be both arboreal and terrestrial also seems to contribute indirectly to L3_Cercopithecinae's brain-body allometry.

L3_Hylobatidae (gibbons)

Life history variables seem to be particularly important as direct causes of L3_Hylobatidae's brain-body allometry, which is the same as L3_Cercopithecinae's. Members of L3_Hylobatidae live long lives that feature monogamous mating systems and large investments in offspring. They also experience moderate to low mortality risk. The clade's brain-body allometry also appears to be a direct adaptation to small, socially monogamous social groups and moderate home range sizes.

The habitats that members of L3_Hylobatidae live in were found to be an indirect cause of the clade's brain-body allometry. These habitats generally have high amounts of tree cover and moderate plant biodiversity, as well as hot and moderately variable climates. Species in L3_Hylobatidae are arboreal and diurnal and consume a diet with moderate amounts of reproductive plant parts and prey. They also experience moderate predation pressure from raptors, which indirectly contributes to the clade's brain-body allometry.

L4_Hominidae (gorillas, chimpanzees, orangutans)

As described in Chapter 2, L4_Hominidae's brain-body allometry is notable for having evolved through a disproportionate change in mean body size. It is characterised by a "low" species aRBS scaling relationship and a larger clade aRBS than the clades in grade L3. This brain-body allometry seems to be directly caused by high levels of maternal investment, low mortality risk, and long lifespans in large home ranges.

Species' mating systems and moderate group sizes both contribute indirectly to L4_Hominidae's brain-body allometry. The large home ranges in the clade occur in highly forested habitats with moderate plant biodiversity and reasonably hot and stable climates. Members of L4_Hominidae

are diurnal and traverse their habitats using both arboreal and terrestrial substrates, encountering moderate predation pressure from raptors. A final indirect cause of L4_Hominidae's brain-body allometry is a diet composed of moderate to high amounts of reproductive plant parts and moderate to low amounts of prey.

H3_Hominins (humans)

As noted above, it was not possible to establish the direct causes of H3_Hominins's brain-body allometry, which is distinctive for its large clade aRBS and hyperallometric species aRBS scaling relationship. All that it is possible to say is that the clade's brain-body allometry is *potentially* caused by low mortality risk, long lifespans, high maternal investment, and large home ranges in habitats with low tree cover and low precipitation.

A diet relatively high in prey and with moderate to low amounts of reproductive plant parts may be an indirect cause of H3_Hominins's brain-body allometry. Other potential indirect causes include moderate to high predation pressure from raptors, diurnality, and terrestrial activity in habitats that have low plant biodiversity and relatively cool, variable climates. Large social groups and monogamous mating systems are also potentially relevant to H3_Hominins' brain-body allometry.

Discussion

As I said in Chapter 1, once causal models have been produced using the observational-inductive approach, these models can be provisionally accepted as being the current best explanatory models, subject to validation using other methods and until more data (and better methods) become available to develop more accurate models.

I think there is good reason to provisionally accept the models described above and in Figure 5.0 as the current best models of the causes of primate brain evolution. As I described in Chapter 1,

other models in the field — developed with the traditional hypothetico-deductive approach — are unrealistically simple. Models such as the frugivorous foraging hypothesis, social brain hypothesis, and maternal investment hypothesis suggest that primate brain evolution is driven by single selection pressures or constraints that exert a direct and linear influence on primates' brains, and they make no provision for selection pressures or constraints being different in different taxa. Furthermore, these models are supported solely by correlations, whereas the models I have described here are explicitly causal. Although, as discussed above, there is a risk of spurious relationships in these causal models, there is an even greater risk of spurious relationships in correlation-based models. This is starkly demonstrated here in the differences between the correlation models for the clades shown in Figure 4.0 and the clades' final causal models shown in Figure 5.0. It can be seen that most correlations were ultimately found to be spurious, as far fewer causal relationships remain in the final causal models. Indeed, while the correlation models feature an average of 285 correlations, the causal models feature an average of only 50 causal relationships.

In this chapter, I have given an overview of the causal models that were produced by my implementation of the observational-inductive approach. In the next chapter, I explore these models further to extract from them a deeper understanding of the causes of primate brain evolution.

6. Causal Model Comparison

The models described in the previous chapter and in Figure 5.0 provide a detailed understanding of what caused the evolution of each brain-body allometry exhibited by the different primate clades. However, by comparing across these causal models, it is also possible to identify recurring patterns that provide a deeper understanding of the causes of primate brain evolution in general.

Comparing across the causal models reveals two key recurring patterns. The first is that there is a core system of niche variables that underlies primate brain evolution. The second is that — despite the presence of this core system — every clade is adapted to a unique set of causal influences.

Pattern 1: Core System

There are 15 measured niche variables that are common to all the causal models (if you exclude M4_Daubentonia's unusual model, which is based on a single correlation) (Figure 6.1.1). To better understand and visualise the variation in these variables across the clades, I performed a Multiple Factor Analysis (MFA) (Bécue-Bertaut and Pagès, 2008).

MFA is a version of Principal Components Analysis (PCA) that is more appropriate when variables are organised into groups, as they are here with the niche variables being grouped into different dimensions (i.e., habitat, trophic, life history, defence, and social) (Figure 3.2). MFA applies weights to the different variable groupings to prevent particular groups from dominating analyses simply because they contain more variables (e.g., in this case, the habitat dimension, which contains many more variables than the others).

For the MFA, I used the common variables' average values in each clade (median for continuous variables; mode for factor variables), normalised between 0 and 1 so that all the variables were

on the same scale. I also included as supplementary variables in the analysis the mean brain size of each clade and the slope group to which they belong to see how the common variables might be related to these. I carried out the MFA in R (R Core Team, 2020) using the FactoMineR package (Lê et al., 2008). The code for the analysis can be viewed in the script 02-Causal-model-comparison.R.

The MFA revealed that, of the 15 measured niche variables common to all the causal models, 12 in particular seem to be of general relevance to primate brain evolution. This is because 6 of these variables seem to be generally associated with variation in mean brain size across the clades (Dimension 1 of the MFA, which is significantly correlated with mean brain size; $r = 0.927$, $p < 0.001$), while 7 of the variables seem to be generally associated with variation in species aRBS scaling across the clades (Dimension 2 of the MFA, which is significantly correlated with slope group; $r^2 = 0.716$, $p < 0.05$) (Figure 6.1.2, *Dim. Descrip.*). The variables generally associated with variation in mean brain size are raptor species richness, social system, social group size, extrinsic mortality rate, total maternal investment, and substrate. Substrate is also one of the variables generally associated with variation in species aRBS scaling, along with median tree cover, temperature seasonality, precipitation seasonality, annual mean temperature, percent reproductive plant parts, and percent prey. The 3 variables that are common to all the causal models but were not found to be generally relevant to primate brain evolution are temperature annual range, median Net Primary Productivity, and mean diurnal range. The MFA found temperature annual range to not vary significantly between the clades and only found median Net Primary Productivity and mean diurnal range to vary significantly between the clades L1_Lorisiformes and L2_Lemuridae (Dimension 3 of the MFA) (Figure 6.1.2).

Together, the 12 common niche variables highlighted by the MFA could be seen to constitute a core system underlying primate brain evolution (Figure 6.1.3). Many of these variables have previously been suggested to be important to primate brain evolution, which validates the ability

of the GES causal discovery method to uncover realistic causal relationships directly from data. What the models developed in this project can provide beyond previous research, however, is the first explicitly causal understanding of the importance of each variable and the complex ways in which they interact with each other and with primate brain evolution. Below, I describe the insights that the models can provide into the causal importance of each variable and their interaction as a core system.

Raptor Species Richness

Causal Importance

The MFA found that raptor species richness is positively correlated with mean brain size across the allometric clades (specifically, it is positively correlated with Dimension 1 of the MFA — r = 0.590, $p < 0.05$ — which is in turn correlated with mean brain size) (Figure 6.1.2). However, although raptor species richness is a direct cause of 4 clades' brain-body allometries (M2_Ancestral, M4_Colobinae, L1_Lorisiformes, L2_Lemuridae), it seems that its influence on primate brain evolution is relatively indirect (its median Distance in the causal models is 2) (Figure 6.1.1, *View table*). This suggests that dealing with predation pressure from raptors might not be directly cognitively challenging for most primates. Instead, raptor species richness seems to be important as an "influencer" of other niche variables that are involved in primate brain evolution. It causes an average of 4 other niche variables in every causal model (median Out-degree = 4) (Figure 6.1.1).

Most notably, raptor species richness was found to cause social group size (Figure 6.1.3), which accords with prior theory that suggests that social groups form as a defence against predators (Miller and Treves, 2011, p. 530). It is thought that groups provide increased ability to detect predators, reduce individual risk through dilution effects, and enable animals to mob and fend off attackers (Miller and Treves, 2011, p. 530). Indeed, the finding that raptor species richness

causes social group size supports previous research suggesting that it is raptor predation in particular that drives social group formation in primates (see McGraw and Berger, 2013).

However, the relationship found here between raptor species richness and social group size is nonlinear. While initially positive as predicted by the predator defence theory, the correlation between the variables dissipates as raptor species richness and group sizes increase. This challenges the idea that predation pressure from raptors has been a universal driver of group size across primates. Instead, it is more consistent with the idea that raptor predation may only be important in certain taxa (Miller and Treves, 2011, p. 530). Specifically, the positive relationship between raptor species richness and social group size found here is strongest in the clades M2_Ancestral (Figure 5.0, *M2_Ancestral*) and L2_Lemuridae (Figure 5.0, *L2_Lemuridae*), suggesting that raptor predation may only be important as a driver of social group size in the Lemuriformes. It may be that the smaller social groups exhibited by these clades are adaptive, but larger groups do not confer any additional protection or might even become counterproductive by presenting a bigger target (Miller and Treves, 2011, p. 532).

As well as social group size, raptor species richness was found to influence total maternal investment and the percentage of prey in species' diets (Figure 6.1.3). Again, though, while raptor species richness is positively related to these variables, the relationships break down outside of the clades M2_Ancestral and L2_Lemuridae. There is no pre-existing theory for why predation pressure from raptors might affect total maternal investment and diet composition — and specifically in Lemuriformes — but this presents a potential new avenue for research.

Finally, raptor species richness was found to influence other measures of predation pressure and biodiversity (carnivore, snake, crocodile, mammal, bird, and reptile species richness) (e.g., Figure 5.0, *M5_Cacajao_Chiropotes*), as well as diel activity, with primates that encounter higher raptor species richness tending to be diurnal (e.g., Figure 5.0, *M4_Colobinae*).

Interaction with Other Core Variables

As discussed above, raptor species richness was found to be a cause of social group size, total maternal investment, and percent prey, which are all also core variables (Figure 6.1.3). Raptor species richness is itself caused by the core variable precipitation seasonality, with which it has a nonlinear relationship (initially positive, then negative) (Figure 6.1.3).

Social System & Social Group Size

Causal Importance

Because they are closely interrelated, I will discuss social system and social group size together here. The MFA found both social system and social group size to be positively correlated with mean brain size: larger-brained species tend to have group living social systems, and those with larger social groups tend to have larger brains (correlations with Dim. 1 of the MFA: social system: $r = 0.679$, $p < 0.05$; social group size: $r = 0.829$, $p < 0.001$) (Figure 6.1.2). On the surface, this finding is in line with the social brain hypothesis discussed in Chapter 1, which posits that group living primates require larger brains to be able to manage the complexities and exploit the opportunities of social life.

However, the social brain hypothesis considers sociality to impose a direct selection pressure on primates' brains, whereas the causal models suggest that the social variables actually have a more indirect influence on primate brain evolution. The median Distance of both social system and social group size in the causal models is 2, and they are only a direct cause of clades' brain-body allometries in less than half of the models (6 of 14) (Figure 6.1.1, *View table*). Thus, the results of this project challenge the social brain hypothesis and are more supportive of those who have argued that sociality is not necessarily directly cognitively demanding (Barrett et al., 2007; Boyer and Ramos-Fernandez, 2018). The results suggest that sociality might be more consistently important as an indirect cause of primate brain size through its influence on other niche variables. Both social system and social group size were found to have high Out-degrees

in the models, indicating that they are important "influencers" of other niche variables (Figure 6.1.1). Indeed, social group size is the most important "influencer" of all the niche variables analysed, causing an average of 7 other variables in each causal model.

Social group size was found to cause extrinsic mortality rate, with increased group size seemingly decreasing mortality (Figure 6.1.3). This would have fit with the idea of social groups being a predator defence strategy had social group size been found to be universally caused by raptor species richness. However, since it was not, this relationship implies that there is some other way in which social groups decrease mortality. One possibility is that sociality might protect against or mitigate the effects of disease (Ezenwa and Worsley-Tonks, 2018; Lucas and Keller, 2020). Social group size was also found to cause substrate, home range size, and body mass dimorphism, with larger social groups tending to be more terrestrial, have larger home ranges, and higher levels of intraspecific conflict (e.g., Figure 5.0, *M4_Ancestral_Platyrrhini*; Figure 5.0, *L3_Cercopithecinae*).

Both social group size and social system were found to affect total maternal investment, with group living and larger group sizes causing increased investment in offspring (Figure 6.1.3). Both social variables also seem to affect diel activity and mating system, with larger groups tending to be diurnal and have harem polygyny or polygynandrous mating systems (e.g., Figure 5.0, *L2_Lemuridae*).

Again, the finding that the social variables influence so many other niche variables and are less often a direct cause of clades' brain-body allometries themselves suggests that the true importance of sociality in primate brain evolution might be in creating the right ambient conditions for larger brains to evolve.

Interaction with Other Core Variables

Almost by definition, the two social variables are causally related to one another, since if species have a large mean group size this will cause them to be classified as group living. This is reflected

in the fact that social group size was found to cause social system (Figure 6.1.3). As described above, social group size also causes fellow core variables extrinsic mortality rate and substrate, and both social group size and social system cause total maternal investment.

Social system was found to be caused in the core system by the diet variable percent prey, with lower amounts of prey in the diet being associated with increased sociality (Figure 6.1.3). Social system was also found to be caused by substrate. Because substrate is in turn caused by social group size, there may be some sort of feedback relationship between sociality and substrate, or else the causal relationships between these variables may simply not have been well-resolved by the GES analyses.

Social group size is caused by raptor species richness in the core system (Figure 6.1.3), but, as discussed above, the relationship between these variables only seems to hold in certain taxa. No other causes of social group size were found in the causal models — not even other forms of predation pressure — so the main causes of social group size remain unclear here.

Extrinsic Mortality Rate

Causal Importance

Extrinsic mortality rate has long been considered to be an important indirect cause of primate brain evolution through its effect on life history (Dunbar and Shultz, 2007; Jones, 2011; van Schaik et al., 2012). Life history theory suggests that the sorts of long, slow life histories necessary for large brains to develop can only evolve if extrinsic mortality is sufficiently low. If it is not, then natural selection favours a risk-mitigating strategy of having a larger number of "lower quality" offspring (so-called *r*-selection) instead of a strategy of investing heavily in the development of only a few (*K*-selection) (Lewin and Foley, 2004, pp. 155–157). The results of this project support this theory with evidence that lower extrinsic mortality rates are linked to higher total maternal investment and larger mean brain sizes (correlation with Dim. 1 of the MFA: $r = -0.744$, $p < 0.05$) (Figure 6.1.3, Figure 6.1.2). Also ostensibly corroborating life history theory is the finding that

extrinsic mortality rate is a cause of maximum longevity (Ricklefs, 2010) (e.g., Figure 5.0, *L3_Hylobatidae*), though here this most likely simply reflects the fact that extrinsic mortality rate was calculated from maximum longevity.

More novel is the finding that extrinsic mortality rate may have a more direct influence on primate brain evolution than previously recognised. Its median Distance in the models is 1 (Figure 6.1.1), suggesting that — as well as its indirect influence via life history — extrinsic mortality rate also commonly has a direct influence on clades' brain-body allometries. It may be that the level of risk in species' environments affects how exploratory they are and, therefore, how much of a requirement they have for a neural substrate for learning and memory. Species living in lower-risk environments might be more exploratory and so require larger brains. Ostensibly supporting this idea is the finding that a lower extrinsic mortality rate also causes increased amounts of reproductive plant parts in the diet and larger home ranges (e.g., Figure 5.0, *M4_Ancestral_Platyrrhini*). Species in lower-risk environments might be more exploratory and willing to search for more distributed food resources.

Interaction with Other Core Variables

Two of the variables that extrinsic mortality rate was found to cause — total maternal investment and percent reproductive plant parts — are also core variables (Figure 6.1.3). Extrinsic mortality rate itself is caused by social group size in the core system. Indeed, this is the only cause of extrinsic mortality rate that was found, which — given extrinsic mortality rate's importance to primate brain evolution — emphasises the potential indirect importance of sociality.

Total Maternal Investment

Causal Importance

Total maternal investment is most frequently a direct cause of clades' brain-body allometries (median Distance = 1) (Figure 6.1.1) and is positively correlated with mean brain size (correlation

with Dim. 1 of the MFA: $r = 0.848$, $p < 0.001$) (Figure 6.1.2). This is consistent with the life history theory discussed above: larger brains can only evolve if extended pre-natal and post-natal periods are available for them to grow and mature (Barrickman et al., 2008; Isler and van Schaik, 2009; Powell et al., 2019).

Indeed, the results of this project suggest that total maternal investment may be of central importance to primate brain evolution. This is because many niche variables seem to affect primate brain evolution through their influence on how much investment species can make in their offspring. Total maternal investment's In-degree across the models is 5, meaning that it mediates the influence of 5 other niche variables in every causal model (Figure 6.1.1). Lower extrinsic mortality rates, group living and larger group sizes, increased predation pressure from raptors (at least in Lemuriformes), and reduced amounts of prey in the diet all affect primate brain evolution by causing total maternal investment to increase.

As well as being a direct cause of clades' brain-body allometries and mediating the effects of other niche variables, total maternal investment was also found to affect body mass dimorphism (positive relationship) (e.g., Figure 5.0, *L3_Cercopithecinae*), diel activity (species that invest more in offspring tend to be diurnal), and home range size (positive relationship) (e.g., Figure 5.0, *L4_Hominidae*).

Interaction with Other Core Variables

All of the causes of total maternal investment listed above are core variables, and these core variables are the only causes of total maternal investment across the causal models. Indeed, the entire core system could be seen to be oriented around determining the amount of investment that primates can make in their offspring (Figure 6.1.3). This may be the primary function and significance of the core system.

Substrate

Causal Importance

The MFA found substrate to be generally associated with mean brain size across the clades, with increased terrestriality seemingly leading to larger mean brain sizes (correlation with Dim. 1 of the MFA: $r = 0.585$, $p < 0.05$) (Figure 6.1.2). Substrate was also found to be associated with variation in species aRBS scaling across the clades (specifically, it is correlated with Dimension 2 of the MFA — $r = -0.688$, $p < 0.05$ — which is in turn correlated with slope group). All the "medium" slope clades are predominantly arboreal, some "low" slope clades are more terrestrial, and the one "high" slope clade is exclusively terrestrial (Figure 6.1.1). However, the association between substrate and species aRBS scaling is most likely an artefact of substrate's association with mean brain size, since the clades that drive the association with species aRBS scaling — L3_Cercopithecinae, L4_Hominidae, and H3_Hominins — are also the clades that drive the association with mean brain size (Figure 6.1.1).

The models suggest that substrate's influence on primate brain evolution is relatively indirect. The median Distance of the variable in the causal models is 2 (Figure 6.1.1). Substrate seems to exert its influence in part through diel activity and diet composition: all terrestrial species are diurnal and tend to consume fewer nonreproductive plant parts (e.g., Figure 5.0, *M4_Colobinae*). As mentioned above, substrate was also found to influence social system, but social group size was in turn found to influence substrate, so the relationships between substrate and sociality are ambiguous. Either the relationships between these variables were not well resolved by the analyses, or there might be some sort of feedback loop operating. It is plausible that increased terrestriality might require larger groups — perhaps to increase foraging efficiency (Markham et al., 2015) — and in turn larger groups might force a commitment to terrestriality if large groups cannot be maintained in trees.

Interaction with Other Core Variables

In the core system, substrate ostensibly causes social system and is caused by social group size, as discussed (Figure 6.1.3). Unsurprisingly, it is also caused by median tree cover, with higher levels of tree cover causing species to be more arboreal and lower levels of tree cover causing them to be more terrestrial.

Median Tree Cover

Causal Importance

Tree cover was found to be associated with species aRBS scaling across the clades (correlation with Dim. 2 of the MFA: $r = 0.881$, $p < 0.001$) (Figure 6.1.2), with tree cover generally being higher in the "medium" slope clades, lower in the "low" slope clades, and lowest in the "high" slope clade (Figure 6.1.1). This nonlinear relationship between tree cover and species aRBS scaling — with decreasing tree cover at first favouring a lower slope and then a higher slope — can be explained by a tipping point between habitat types. The level of tree cover in the "low" slope clades represents a relatively patchy forest habitat, whereas the level of tree cover in the "high" slope clade (H3_Hominins) represents a savannah-type habitat. Such distinct habitats might be expected to require fundamentally different species aRBS scaling relationships.

Tree cover does not seem to influence primate brain evolution directly (its median Distance in the models is 3) (Figure 6.1.1), but through its effect on other niche variables. Indeed, it is one of the most important "influencer" variables, affecting an average of 4 other niche variables in every model (Figure 6.1.1). As mentioned above, tree cover affects substrate, with higher levels of tree cover causing primate species to be more arboreal and lower levels of tree cover causing them to be more terrestrial (Figure 6.1.3). Naturally, tree cover also affects the climate and biodiversity of habitats. It was found to have a negative relationship with temperature seasonality, a negative nonlinear relationship with mean diurnal range, positive nonlinear relationships with annual precipitation and median Net Primary Productivity, a positive relationship with bird species

richness, and a negative relationship with carnivore species richness (e.g., Figure 5.0, *M4_Ancestral_Platyrrhini*).

Tree cover itself was not found to have any causes in the causal models — its In-degree is always 0 (Figure 6.1.1). This, coupled with the finding that it is an indirectly causal, highly influential variable, suggests that tree cover may be one of the ultimate drivers of primate brain evolution.

Interaction with Other Core Variables

Two of the variables that tree cover causes — substrate and temperature seasonality — are also core variables (Figure 6.1.3). Again, because of its indirect influence and the fact that it does not have any causes inside or outside of the core system, tree cover could be seen to be one of the ultimate drivers of the core system.

Temperature Seasonality, Precipitation Seasonality, and Annual Mean Temperature

Causal Importance

Along with tree cover, climate variables may also be ultimate causes of primate brain evolution. They were found to have some of the most indirect influences on clades' brain-body allometries (Figure 6.1.1).

Temperature seasonality, precipitation seasonality, and annual mean temperature were all found to be associated with variation in species aRBS scaling across clades (correlations with Dim. 2 of the MFA: temperature seasonality: $r = -0.613$, $p < 0.05$; precipitation seasonality: $r = -0.618$, $p < 0.05$; annual mean temperature: $r = 0.818$, $p < 0.05$) (Figure 6.1.2). Temperature seasonality and precipitation seasonality both increase from "medium" to "low" to "high" slope clades, whereas annual mean temperature consistently decreases across these groups (Figure 6.1.1). As with tree cover, the nonlinear relationships between the climate variables and species aRBS scaling

are likely indicative of a tipping point between different habitat types that have distinct climates that favour distinct species aRBS scaling relationships.

It is notable that — out of all the climate variables in this project's data set — two of the three that were found to be generally associated with primate brain evolution relate to climate variability (i.e., temperature seasonality and precipitation seasonality). This corroborates previous research that has suggested that it is climate variability in particular that is relevant to primate brain evolution. Climate variability is thought to lead to periodic dietary restrictions that either constrain the energy available to grow and maintain brains or make foraging more cognitively demanding (Potts, 2004; van Woerden et al., 2014).

The data were not available to be able to include a measure of periodic dietary restriction in the analyses to establish whether the climate variables do influence primate brain evolution via this mechanism, but what the analyses were able to establish is that the core climate variables seem to influence primate brain evolution through their effect on other climate and habitat variables. Annual mean temperature was found to affect mean diurnal range and median Net Primary Productivity, exhibiting a nonlinear relationship with both (initially positive, then negative); temperature seasonality was found to affect temperature annual range (positive relationship); and precipitation seasonality was found to affect annual precipitation (negative relationship) and raptor species richness (nonlinear relationship; initially positive, then negative) (e.g., Figure 5.0, *H3_Hominins*).

Interaction with Other Core Variables

Within the core system, temperature seasonality is caused by median tree cover (negative relationship) (Figure 6.1.3). The core climate variables are also all interrelated. Decreasing annual mean temperature causes increased temperature and precipitation seasonality, and temperature seasonality also independently affects precipitation seasonality, with there being a positive nonlinear relationship between the two variables.

Percent Reproductive Plant Parts & Percent Prey

Causal Importance

The amount of reproductive plant parts and prey in species' diets was found to be associated with species aRBS scaling across the clades (correlations with Dim. 2 of the MFA: percent repro plant parts: $r = 0.637$, $p < 0.05$; percent prey: $r = -0.563$, $p < 0.05$) (Figure 6.1.2). The amount of reproductive plant parts in species' diets seems to generally decrease between the "medium", "low", and "high" slope clades, whereas the amount of prey in their diets increases (Figure 6.1.1). Again, these nonlinear relationships probably reflect shifts to fundamentally different habitats that have different food availabilities.

The finding that percent reproductive plant parts is generally associated with primate brain evolution is ostensibly in line with the frugivorous foraging hypothesis discussed in Chapter 1. However, the fact that percent reproductive plant parts is associated with species aRBS scaling and not directly with mean brain size challenges the idea that it is the cognitive demands of foraging that affect primate brain evolution. Instead, the results are more consistent with the idea of diet being an indirect energetic constraint on primate brain evolution (Aiello and Wheeler, 1995; Fish and Lockwood, 2003). Both percent reproductive plant parts and percent prey are quite indirectly related to clades' brain-body allometries in the causal models (they have a median Distance of 3 and 2, respectively) (Figure 6.1.1). They seem to influence primate brain evolution by affecting what social systems species can evolve and how much investment they can make in their offspring (Figure 6.1.3).

Interaction with Other Core Variables

As would be expected, the two core diet variables are closely interlinked (Figure 6.1.3). Indeed, they are so closely interlinked that the causal discovery analyses may have struggled to resolve the exact relationships between them and other niche variables. So, while percent prey was found

to be negatively related to social system and total maternal investment in the core system, it may be that, in reality, these variables are positively related to percent reproductive plant parts.

The core diet variables are themselves caused by the core variables extrinsic mortality rate (negative relationship with percent reproductive plant parts) and raptor species richness (positive relationship with percent prey, at least in Lemuriformes) (Figure 6.1.3).

Discussion

The MFA highlighted an interesting pattern of some niche variables — mostly defence, social, and life history variables — being associated with mean brain size across clades while other variables — habitat and trophic variables — are associated with species aRBS scaling. This pattern helped to establish which niche variables are of general importance to primate brain evolution and thus helped to identify the core system. However, the meaning of the pattern itself remains unclear. The pattern is only correlational and cannot tell us how — or even if — different sets of niche variables separately cause mean brain size and species aRBS scaling across primates. The causal models cannot inform our understanding of this because they were built to determine the causes of each clade's brain-body allometry, not determine the causes of the separate allometric components (i.e., grade aRBS and species aRBS scaling) across primates. Additional causal analyses would be needed to do this, but these were not feasible within the time constraints of this project. Investigating this pattern does, however, present an interesting avenue for future research.

Pattern 2: Clade Uniqueness

The second key recurring pattern that emerges when comparing across the causal models is that — despite the presence of the core system in every model — every clade's overall causal model is unique. Different clades have different relationships with the core system, and every clade has a varying number of other niche variables that are relevant to them in addition to the core ones.

For example, while the core variables social system and social group size are most often indirect causes of clades' brain-body allometries, as discussed above, there are 6 clades in which one or another of these variables is a direct cause (e.g., L3_Hylobatidae and M3_Callitrichidae) (Figure 6.1.1, *View table*). Similarly, while the core diet variables tend to be only indirectly causal, percent prey was found to be a direct cause of 4 clades' brain-body allometries (M3_Callitrichidae, L2_Lemuridae, M4_Colobinae, and L3_Cercopithecinae) (Figure 6.1.1, *View table*). Even variables that could be considered ultimate causes in the core system can sometimes be direct causes in clades. For instance, precipitation seasonality was found to be a direct cause in M5_Atelinae and M5_Cebus (Figure 6.1.1, *View table*).

All the causal models also feature between 4 and 9 niche variables in addition to the core ones, and these variables might be just as or even more important to clades' brain evolution. For example, home range size and maximum longevity may be particularly important in the evolution of mean brain size because they appear almost exclusively in the models of the largest-brained clades (L3_Hylobatidae, L4_Hominidae, and H3_Hominins), and they show a positive association with mean brain size (Figure 6.1.1). Furthermore, both home range size and maximum longevity are direct causes of clades' brain-body allometries in the models in which they appear, and home range size has one of the highest ln-degrees (median = 4) (Figure 6.1.1) suggesting that it is an important mediator of the effects of other niche variables.

The extent to which each clade has a unique set of causal variables and relationships can be assessed by formally comparing the structures of the different causal models. I calculated the Jaccard distance (Jaccard, 1912) between the sets of niche variables (nodes), directly causal niche variables (direct nodes), and causal relationships (edges) in each causal model to determine how similar or dissimilar the causal models are in these respects. This revealed that, while some clades have similar models (e.g., the three largest brained clades: L3_Hylobatidae, L4_Hominidae, and H3_Hominins), and some clades even share the same set of causal niche

variables (e.g., M3_Callitrichidae and L1_Lorisiformes; M2_Ancestral and L3_Cercopithecinae), no clades have identical causal models with exactly the same sets of niche variables, directly causal niche variables, and causal relationships (Figure 6.2.1, Figure 6.2.2). Not even clades within the same grade have identical causal models, and indeed they can be quite dissimilar (e.g., M4_Ancestral_Platyrrhini, M4_Daubentonia, and M4_Colobinae; M5_Atelinae, M5_Cacajao_Chiropotes, and M5_Cebus; and L3_Cercopithecinae and L3_Hylobatidae) (Figure 6.2.1). This substantiates the possibility raised in Chapter 4 that different causal influences might lead to the convergent evolution of the same brain-body allometry. It certainly flags this as an area for further investigation.

The uniqueness of each causal model reinforces the idea put forward in Chapter 1 that primate brain evolution has been complex. It suggests that while we may be able to use comparative studies to identify broad patterns across taxa — such as the core system discussed above — we will likely not be able to fully understand primate brain evolution without acknowledging the diversity of the primate order and studying different taxa individually within the context of their unique niches (see Logan et al., 2018).

7. Discussion

I suggested in Chapter 1 that primate brain evolution is likely to have been complex for at least three reasons. Firstly, it is likely to have been caused by niche variables that are themselves complex, multifaceted constructs. Secondly, it is likely that many such variables interacted in various linear, nonlinear, direct, and indirect ways to affect primate brain evolution. Thirdly, it is likely that different causal systems operated in different primate taxa. I said that any research approach used to investigate the causes of primate brain evolution must be capable of modelling this sort of complexity if it is to produce a realistic understanding of it. I argued that the traditional hypothetico-deductive approach is not suited to building complex causal models and that an observational-inductive approach would be better. The subsequent chapters then presented the particular observational-inductive approach to understanding primate brain evolution that I developed. Here, I discuss the extent to which this approach has been able to produce a realistic understanding of the causes of primate brain evolution and how it could be further developed.

Limitations

I have said from the beginning that my implementation of the observational-inductive approach was not intended to produce a definitive understanding of the causes of primate brain evolution but provide an initial proof-of-concept for the application of the observational-inductive approach to this field of research. It is clear that the realism of the final causal models presented in Chapter 5 and Figure 5.0 is currently still limited.

That being said, the limited realism of the final causal models is not primarily due to the observational-inductive approach itself or my implementation of it, but to a fundamental lack of data that is limiting all research in the field. Most of the data sets currently available in the literature were developed to serve the traditional hypothetico-deductive approach, so they tend to contain

a minimal amount of data — enough to test simple hypotheses but not enough to build realistically complex models. Even pooling many of these data sets together as I did to assemble the largest data set yet for a study of the causes of primate brain evolution (see Chapter 3), the data were still a limiting factor.

For example, the available data limited the extent to which different aspects of complex, multifaceted niche variables could be represented. I mentioned in the previous chapter how it was not possible to represent the periodic restriction aspect of diet, which might be important for understanding how climate variability affects primate brain evolution. It was also not possible to represent potentially important aspects of sociality, such as the complexity of relationships, social learning and culture, or communication complexity.

Similarly, the lack of data limited the number of niche variables that could be included in the analysis. There are many variables potentially relevant to primate brain evolution that could not be evaluated here, including mode of locomotion (Povinelli and Cant, 1995), water foraging (Sharma et al., 2016), self-medication behaviour (Huffman, 2017), sleep (Fruth et al., 2017; Nunn and Samson, 2018), cooperative breeding (Isler and van Schaik, 2009), and long-term intermittent variables such as droughts that could produce fallback adaptations (Constantino and Wright, 2009). Indeed, as I said in Chapter 3, it was not possible to include any variables related to the metabolic niche dimension. However, the sorts of physiological, developmental, and genetic variables that could come under this heading would be important to include to be able to understand the mechanistic links between niche variables and primate brain evolution (Charvet et al., 2011; Gilbert et al., 2005).

Finally, data availability limited the level of diversity that could be modelled. I focussed on clade-level diversity in primate brain evolution because insufficient data were available to estimate brain-body allometry for individual species. However, it is evident from any of the detailed distributions of niche variables in Figure 6.1.1 that there is considerable species-level variation within the

identified clades, suggesting that a more realistic understanding of primate brain evolution might be produced by modelling individual species' niches. Indeed, the amount of variation that can occur even within species might mean that population-level models are needed for a realistic understanding of primate brain evolution, as recommended by Logan et al. (2018). It has even been suggested that different systems of selection pressures and constraints might apply to the different sexes within populations (Lindenfors et al., 2007). While clearly there is a limit to the level of complexity that it would be useful to model (Prusinkiewicz, 1998), the clade-level models that it was possible to develop here are likely still too coarse-grained.

Ultimately, the current restrictions imposed by a lack of data will only be alleviated by a campaign to gather data for their general observational-inductive value, not just to test specific hypotheses. Prioritising data gathering in this way would require a major cultural shift, especially in the attitudes of funding bodies and journal publishers (Haufe, 2013). Indeed, it would require nothing short of a renaissance in natural history observation (Greene, 2005). Such a renaissance could today be facilitated by new animal-borne sensor technology that is making it possible to gather large amounts of detailed, objective data on animals and their environments, and by new machine learning methods that make it possible to extract meaningful patterns from such data (Fehlmann and King, 2016; Valletta et al., 2017; Wilmers et al., 2015). Ideally, any future data gathering efforts would be orchestrated by a centralised body that could provide direction and coordination and establish and enforce data quality standards (e.g., Borries et al., 2013).

Although a lack of data was the primary factor limiting the realism of the final causal models, I also discussed in Chapter 5 how the Greedy Equivalence Search (GES) method that I used to establish the causal relationships between variables also imposed certain limitations. The somewhat unrealistic assumptions of GES that there will be no latent variables or feedback loops in the analysis could have introduced spurious relationships into the final causal models, limiting their reliability. However, as I discussed, this causal discovery method is the best that is currently

available. Given how young the field of causal discovery is, it is likely that better methods will be developed in the future (e.g., Forré and Mooij, 2018), and these could easily be swapped in to improve this step of the modelling process.

Capabilities

While the observational-inductive approach may currently be limited by available data and methods, what is more pertinent is whether it is likely to be a more productive approach to pursue than the traditional hypothetico-deductive approach. I believe that this project has clearly demonstrated how much more capable the observational-inductive approach is compared to the traditional approach when it comes to modelling complex causal systems.

The implementation of the observational-inductive approach that I developed was able to produce what the traditional hypothetico-deductive approach could not: the first realistically complex models of the causes of primate brain evolution (Figure 5.0). Even with the limitations discussed above, these models are far more realistic than the simple correlation-based models currently in the literature. As such, they arguably represent our current best understanding of the causes of primate brain evolution.

The ability of observational-inductive methods to model directly from data the linear, nonlinear, direct, and indirect — and explicitly causal — interactions between large numbers of variables (both continuous and factor) has already produced a more realistic understanding of the importance of various niche variables in primate brain evolution. As discussed in the previous chapter, the causal models have challenged the idea that predation pressure is an important driver of sociality across primates, as well as the idea that sociality is an important direct cause of primate brain size. Instead, the models suggest that sociality might be more important as an indirect cause of brain size through its influence on other niche variables. Similarly, diet is often thought to have a direct influence on primates' brains, but the causal models suggest that diet is

more likely to be important as an indirect energetic constraint on the brain size that species can evolve. The models have backed up life history theory with causal evidence that extrinsic mortality rate and total maternal investment play important roles in primate brain evolution, and they have highlighted how total maternal investment might in fact be of central importance. They have also established that habitat and climate variables might be the ultimate drivers of primate brain evolution.

Finally, the fact that my implementation of the observational-inductive approach was able to produce individual causal models for 14 primate clades made it possible to identify and describe a core system of niche variables underlying primate brain evolution (Figure 6.1.3). At the same time, it revealed that every clade appears to be subject to a unique system of causal influences — even clades within the same grade — which questions the validity of the sort of broad comparative studies that are currently popular and potentially adds to our understanding of convergent evolution.

That even a proof-of-concept implementation of the observational-inductive approach could produce such potentially significant new insights into the causes of primate brain evolution clearly demonstrates how productive an observational-inductive approach could be in this field. Furthermore, the approach could be extended to produce even greater levels of understanding.

Extensions

This project's scope was limited to establishing structural models of the causes of primate brain evolution — models depicting the variables involved and how they are causally related. However, once established, these structural models could be parameterised to create dynamic simulation models (González-Forero and Gardner, 2018; Mobus and Kalton, 2015, pp. 645–698; Muthukrishna et al., 2018). Such simulation models could be used to validate the causal relationships proposed in the structural models by testing whether the interaction between

variables actually produces realistic dynamics (Servedio et al., 2014). The dynamic aspect of simulation models could also allow us to investigate complex phenomena such as thresholds or emergence. We could, for example, potentially identify the points at which changing niches begin to favour different brain-body allometries, or better understand how many weakly influential niche variables combine to have a significant emergent effect on primate brain evolution. Finally, once simulation models were sufficiently developed and validated, they could be used to ask counterfactual questions about primate brain evolution or make predictions, for instance about how climate change might affect primate species (Grace et al., 2015, pp. 193–195; Mobus and Kalton, 2015, pp. 658–659; Pearl and Mackenzie, 2018, pp. 219–297).

Of course, there is nothing about my implementation of the observational-inductive approach that restricts it to being used for studying primate brain evolution, and it could easily be extended to study other taxa or to investigate other complex causal systems. For instance, it could be used to study the causes of brain evolution in other mammalian groups or the selection pressures and constraints that have shaped the evolution of other biological traits.

Conclusion

My goal in this project was to demonstrate the potential of the observational-inductive approach to produce a more realistic understanding of the causes of primate brain evolution. The results of even this initial proof-of-concept study suggest that the observational-inductive approach has enormous potential and should be further explored and developed. Hopefully, this potential will provide the motivation to address the data availability issues that are currently the main factor limiting all research in the field. At the very least, I hope that the results will prompt researchers investigating the causes of primate brain evolution to reflect on the approach that they are currently employing and whether it is likely to provide them with the understanding that they seek.

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