A Phylogenetic Analysis of Sleep Architecture in Mammals: The Integration of Anatomy, Physiology, and Ecology

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abstract: Among mammalian species, the time spent in the two main "architectural" states of sleep—slow-wave sleep (SWS) and rapid-eye-movement (REM) sleep—varies greatly. Previous comparative studies of sleep architecture found that larger mammals, those with bigger brains, and those with higher absolute basal metabolic rates (BMR) tended to engage in less SWS and REM sleep. Species experiencing a greater risk of predation also exhibited less SWS and REM sleep. In all cases, however, these studies lacked a formal phylogenetic and theoretical framework and used mainly correlational analyses. Using independent contrasts and an updated data set, we extended existing approaches with path analysis to examine the integrated influence of anatomy, physiology, and ecology on sleep architecture. Path model structure was determined by nonmutually exclusive hypotheses for the function of sleep. We found that species with higher relative BMRs engage in less SWS, whereas species with larger relative brain masses engage in more REM sleep. REM sleep was the only sleep variable strongly influenced by predation risk; mammals sleeping in riskier environments engage in less REM sleep. Overall, we found support for some hypotheses for the function of sleep, such as facilitating memory consolidation or learning, but not others, such as energy conservation.

Keywords: independent contrasts, path analysis, predation risk, sleep architecture, sleep function, vigilance.

A mammal's decision of when and where to sleep can be influenced by many factors. The timing of sleep (reviewed in Tobler 1989; Ball 1992) can be affected by the activity patterns of other species (Fenn and Macdonald 1995), food availability (Tobler 1989), and digestive constraints (Saarikko and Hanski 1990), while the choice of where to sleep can be influenced by the thermal and predatory environments (reviewed in Anderson 1998; Lima et al. 2005). However, the factors influencing how to structure sleep and how long to sleep are much less clear (reviewed in Rechtschaffen 1998). The amount of time spent sleeping can vary intraspecifically due to season (Barre and Petter-Rousseaux 1988) or ontogeny (Lyamin et al. 1993; Carskadon and Dement 1994), but such variation is minor when compared to interspecific variation (Zepelin 2000). For example, over the course of a day, horses (*Equus caballus*) sleep only 3 h (Dallaire and Ruckebusch 1974*b*), whereas pocket mice (*Perognathus longimembris*) sleep up to 20 h (Walker et al. 1983*a*). In this article, our goal is to examine the roles of anatomy, physiology, and ecology in the maintenance of such interspecific variation in sleep. By doing so, we hope to reinvigorate evolutionary and ecological approaches to the study of sleep. Such approaches have disappeared almost entirely in the modern study of sleep (Lima et al. 2005), which is dominated by clinical work on rats and humans (but see Tobler 2005). Sleep is, in many ways, an unexplored frontier in behavioral ecology and in animal behavior in general.

The term "sleep architecture" is used by sleep researchers to describe the distribution and duration of time spent in various states of sleep. These states of sleep are often quantified as specific patterns of neuronal firing measured via electroencephalographic recordings (Tobler 1995). In mammals, the taxon for which the most electrophysiological data exist (Zepelin 2000), sleep is composed of two distinct states: slow-wave sleep (SWS) and rapid-eye-movement (REM) sleep. SWS is identified by high-amplitude, low-frequency waves in the electroencephalogram that result from synchronous neuronal activity in the cortex (Horne 1988). In some species, SWS is divided into stages, reflecting a continuum of intensity or depth of sleep (reviewed in Borbély and Achermann 2000; Tobler 2005). REM sleep is distinguished from SWS

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by low-amplitude, mixed-frequency neural activity similar to wakefulness and is accompanied by muscle atonia (except the extraocular and intercostal muscles) and rapid eye movements (Horne 2000). In all adult mammals studied to date, SWS is the predominant sleep state, with REM sleep usually occupying less than 25% of total sleep time (Zepelin 2000).

Previous comparative studies sought to understand the anatomical and physiological correlates of sleep architecture in mammals (Zepelin and Rechtschaffen 1974; Elgar et al. 1988; Siegel 2004, 2005; Zepelin et al. 2005; reviewed in Lesku et al. 2006). A few early studies also examined the ecological correlates of sleep architecture (Allison and Cicchetti 1976; Meddis 1983; reviewed in Lesku et al. 2006). These studies found that larger mammals, those with bigger brains and those with greater basal metabolic rates (BMR), showed lower durations of SWS and REM sleep over a 24-h period (Zepelin and Rechtschaffen 1974; Allison and Cicchetti 1976; Meddis 1983; Siegel 2004; Zepelin et al. 2005). Mammals with longer gestation periods (an index of brain and central nervous system maturity at birth) also engaged in less SWS and REM sleep (Zepelin and Rechtschaffen 1974; Allison and Cicchetti 1976; Siegel 2004, 2005; Zepelin et al. 2005). In addition, species subjected to a higher risk of predation had lower SWS and REM sleep times than those that were not (Allison and Cicchetti 1976; Meddis 1983). In all instances, however, these studies were conducted without an explicit phylogenetic and theoretical framework.

We have two objectives for our comparative study of sleep architecture. Our first objective is to control for shared evolutionary history among taxa using a formal phylogenetic framework. All of the previous comparative studies essentially treated each species or larger taxonomic unit (e.g., Elgar et al. [1988] worked at the family level) as an independent statistical unit. Hence, relationships identified by these studies may not hold up under the scrutiny of modern phylogenetic techniques (Felsenstein 1985; Garland et al. 1992, 2005; Martins 2000). Our second objective is to extend existing analyses by examining the integrated effects of anatomy, physiology, and ecology on sleep architecture using multivariate path analysis (Wright 1934; Mitchell 1992; Petraitis et al. 1996) and an updated data set. Path analysis has several advantages over simple correlation or regression. For instance, path analysis, like multiple regression, can examine the simultaneous influence of several variables on a dependent variable. Unlike multiple regression, however, path analysis allows for greater specification of the relationships among variables, using mediators (variables through which the effect of a predictor variable is channeled) and covariance among predictors (Baron and Kenny 1986). In addition, path analysis, as a form of structural equation modeling, can assess the degree to which a model reflects the observed data (Bentler 1990; Mitchell 1992). The structure of our path models was based on several hypotheses for the function of sleep and is developed in detail in the following section.

Path Models of Sleep Architecture

Our analyses included variables that have traditionally been included in correlational studies of sleep architecture (e.g., Zepelin and Rechtschaffen 1974; Allison and Cicchetti 1976). Specifically, we examined the influence of body mass, brain mass, basal metabolic rate, and gestation period on sleep architecture. In addition, we included two ecological variables, sleep exposure index and trophic position, that were measures of predation risk based on those used in Allison and Cicchetti (1976). Predation-related variables measured two facets of predation risk: the risk associated with sleep site (from burrow to open grassland) and the risk associated with a species' location on the trophic continuum from carnivore to herbivore. These two predation-related indexes are described in detail in "Methods," as are the sources for the aforementioned constitutive variables.

Two path models were created to examine the influence of the aforementioned variables on, first, the duration of time spent in SWS and REM sleep (model 1; fig. 1*a*) and, second, total sleep time (SWS time + REM sleep time) and the percentage of total sleep time allocated to REM sleep (%REM sleep; model 2; fig. 1*b*). The structure of the two models is very similar. We included the second model (fig. 1*b*) because paths to %REM sleep allowed us to examine the relative importance of SWS and REM sleep for a given nonsleep variable since an increase in %REM sleep must necessarily come at the expense of %SWS. In model 2, total sleep time was substituted for SWS time because all hypotheses considered for the functions of SWS also relate to sleep in general. Paths originating from each variable were derived concurrently for models 1 and 2 as follows (see also table 1).

Paths from body mass and interpretation of mediator variables. Previous correlational studies of sleep architecture identified a strong negative correlation between body mass and the time spent in SWS and REM sleep (e.g., Allison and Cicchetti 1976; Zepelin et al. 2005). However, there are no hypotheses that address the basis of this relationship. Instead, we suggest that body mass affects sleep variables indirectly through four mediator variables: brain mass, BMR, sleep exposure index, and gestation period (fig. 1). A consequence of body mass acting through mediator variables is that paths from the mediator variables themselves essentially represent the influence of residual or "mass-controlled" values, although the variance in body

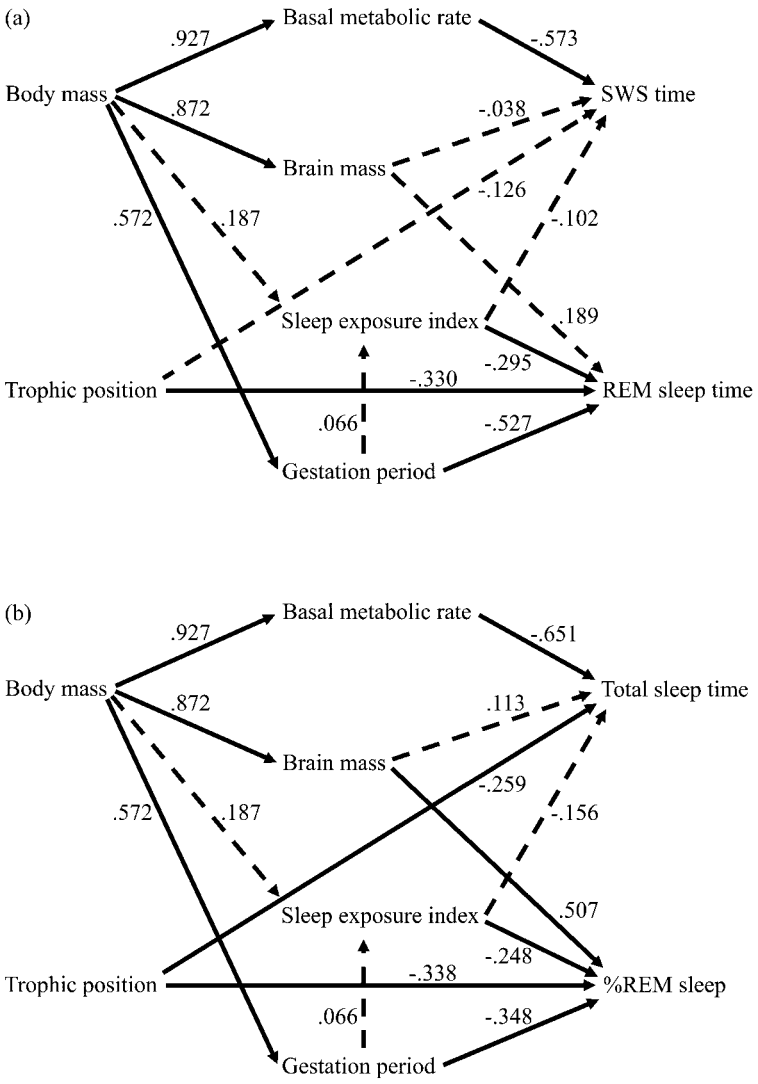

Figure 1: Path models depicting relationships among anatomical, physiological, and ecological variables and their hypothesized influence on slow-wave sleep (SWS) and rapid-eye-movement (REM) sleep times (*a*) and total sleep time and %REM sleep (*b*). Both models were analyzed using phylogenetically corrected contrast data. In each model, body mass and trophic position are independent variables; brain mass, basal metabolic rate, gestation period, and sleep exposure index are set as mediator variables, and sleep variables are treated as dependent variables. Justification for each path is given in the text (see also table 1). Solid and dashed paths denote significance and nonsignificance at $\alpha = 0.05$, respectively. The number above each path is the standardized regression coefficient (β), which quantifies the magnitude and direction of each relationship.

mass is partitioned among all mediator variables rather than a single variable, as in partial correlation (Baron and Kenny 1986). This nature of paths from mediator variables follows from the fact that a direct path between body mass and dependent (sleep) variables is nonsignificant in our two path models (Baron and Kenny 1986; see also "Results").

This "residual status" of paths from mediator variables influences the interpretation of key paths in our model. For instance, paths from brain mass to sleep variables are conceptually similar to the encephalization quotient (Jerison 1985). The encephalization quotient quantifies a species' brain mass as relatively larger or smaller than that expected for its body mass and has been used as an index of interspecific cognitive ability (Marino 1998, 2002; Williams 2002; Siegel 2004; Zepelin et al. 2005). Likewise, paths from BMR are essentially residual (relative) BMR. Paths from gestation period conceptually represent a continuous measure of a species' location along the altricial/precocial gradient (see also Eisenberg 1981); longer relative

Table 1: Overview of the paths in model 1 and model 2

Path from	Path to	Hypothesis and prediction	Reference
Body mass	Brain mass, BMR, gestation period, sleep exposure index	Larger species tend to have larger brains, higher gross BMRs, and longer gestation periods, and they sleep in riskier sites	Allison and Cicchetti 1976; McNab 1988; McNab and Eisenberg 1989
Gestation period	Sleep exposure index	Species bearing precocial young tend to sleep in riskier sites than those with altricial young	Allison and Cicchetti 1976
Brain mass	SWS, REM sleep, %REM sleep, total sleep	Species with larger relative brain masses are expected to engage in more sleep (both states) because SWS and REM sleep may facilitate learning and memory consolidation	Walker and Stickgold 2004
BMR	SWS, total sleep	Species with higher relative BMRs are expected to exhibit more total sleep (specifically SWS) because SWS/sleep may facilitate energy conservation	Berger and Phillips 1995; Zepelin 2000
Gestation period	REM sleep, %REM sleep	Species with longer relative gestation periods are expected, as adults, to engage in less REM sleep since REM sleep may serve as endogenous stimulation for developing brains	Roffwarg et al. 1966; Marks et al. 1995
Sleep exposure index	SWS, REM sleep, %REM sleep, total sleep	Species sleeping in exposed locations are expected to engage in less sleep (both states) because sleep is dangerous	Lima et al. 2005
Trophic position	SWS, REM sleep, %REM sleep, total sleep	Species in lower trophic positions are expected to engage in less sleep (both states) because sleep is dangerous	Lima et al. 2005

Note: BMR = basal metabolic rate, SWS = slow-wave sleep, REM = rapid-eye-movement sleep.

gestation times are associated with relatively precocial young.

Paths from brain mass. Several researchers hypothesize that SWS facilitates learning (Ambrosini and Giuditta 2001; Huber et al. 2004; Deregnaucourt et al. 2005) and/or the consolidation of new memories (Gais and Born 2004; Huber et al. 2004) and that REM sleep also facilitates memory consolidation (Stickgold 1998; Walker and Stickgold 2004; Muzur 2005; but see Siegel 2001; Vertes 2004). The prevalence of these learning/memory hypotheses warrants a path between some aspect of cognitive ability and sleep variables. Thus, we have a path from brain mass (functionally, the encephalization quotient) to both SWS time and REM sleep time (fig. 1*a*). Similarly, the path between brain mass and total sleep time (fig. 1*b*) reflects the hypothesis that both SWS and REM sleep are required to experience the full benefits of memory consolidation (Wagner et al. 2004; Walker and Stickgold 2004). A significant relationship between brain mass and %REM sleep (fig. 1*b*) would suggest that one sleep state is more important for relatively larger-brained mammals because an increase in %REM sleep would necessarily come at the expense of %SWS.

Paths from basal metabolic rate. Several hypotheses implicate various metabolic processes in the function of sleep. The energy conservation hypothesis of Berger and Phillips (1995) posits that sleep reduces BMR below levels achieved by immobility alone. Indeed, endotherms can reduce energy expenditure by as much as 10%–15% while asleep (Shapiro et al. 1984), partly due to the synchronous firing of neurons in the cortex during SWS (Siegel 2003; Cirelli et al. 2004). The energy conservation hypothesis implies that mammals with a higher BMR for a given body mass will engage in more SWS to offset the costs of increased BMR (fig. 1*a*). We therefore created a path between BMR and SWS time (fig. 1*a*). A variant of the energy conservation hypothesis suggests that sleep conserves energy by enforcing rest (Zepelin 2000). This version of the hypothesis is not specific to one sleep state but relates to overall sleep. We therefore created a path between BMR and total sleep time in model 2 (fig. 1*b*). Several additional hypotheses suggest a prominent role for cerebral metabolism as a determinant of sleep architecture (McGinty and Szymusiak 1990; Benington and Heller 1995; Maquet 1995; Siegel 2003, 2005). These energy-related hypotheses also suggest that a mammal's need for total sleep, or SWS specifically, is positively related to its relative metabolic rate. Alternatively, Elgar et al. (1988) proposed that foraging time may limit total sleep time. This idea suggests that mammals with relatively higher BMRs should sleep less due to increased foraging requirements to meet increased metabolic demands.

Paths from sleep exposure index. Exposure to predators should strongly influence sleep architecture. A sleeping mammal is essentially unconscious, and the stimulus strength necessary to awaken an animal (the arousal threshold; Tobler 2005) is much larger than that needed to alert an awake animal. Thus, mammals (and animals in general) are relatively vulnerable to predation while asleep (Lima et al. 2005). We therefore added a path from sleep exposure index to total sleep time (fig. 1*b*).

The vulnerability to predators may also differ according to sleep state. REM sleep in particular is a relatively dangerous state. During REM sleep, arousal thresholds tend to be maximally elevated (Lima et al. 2005 and references therein). Although mammals aroused from REM sleep may actually be better able to respond to a threat than those awakened from SWS (Tolaas 1978; Voss 2004), mammals in REM sleep are less likely to detect an approaching threat (Dillon and Webb 1965; van Twyver and Garrett 1972). Moreover, many herbivores can engage in the lighter stages of SWS while standing (Tobler 1995) but must lay down to engage in REM sleep and the deeper stages of SWS (Tobler 1992; Tobler and Schwierin 1996) due to the loss of muscle tone. This eyes-closed, recumbent posture may signal vulnerability to predators, especially when prey are sleeping in the open (Lima et al. 2005). Consequently, mammals sleeping in riskier sleep sites are expected to have reduced amounts of REM sleep and %REM sleep; hence, paths project from sleep exposure index to these two dependent variables in our models (fig. 1*a*, 1*b*). A similar argument also applies to SWS (fig. 1*a*), the deeper stages of which might also be among the more dangerous forms of sleep (Lima et al. 2005).

Paths from trophic position. Trophic position should also reflect predation risk while asleep (Zepelin 1970). Species at the prey (herbivore) end of the trophic spectrum are likely to be more vulnerable to predation than carnivores (Allison and van Twyver 1970*a*). Prey species are thus expected to have reduced amounts of SWS and REM sleep relative to more predatory species, independent of risk exposure at the sleep site. Thus, we added paths between trophic position and SWS time and REM sleep time (fig. 1*a*). We also added paths between trophic position and total sleep time and %REM sleep (fig. 1*b*) because sleep in general is a dangerous behavior and REM sleep may be a particularly dangerous sleep state (Lima et al. 2005).

Paths from gestation period. The ontogenetic hypothesis for REM sleep posits that REM sleep provides artificial stimulation necessary for cortical development in utero and early in life (Roffwarg et al. 1966; Marks et al. 1995). This hypothesis is based (in part) on the observation that REM sleep is the predominant sleep state in fetuses and neonates (Marks et al. 1995; but see Frank and Heller 2003). Some researchers have used the high density of REM sleep in species with altricial young as support for the ontogenetic hypothesis (Horne 2000; Zepelin et al. 2005), but it is not clear why altricial species, as adults, would require more REM sleep than precocial species (Siegel 2004). Alternatively, Allison and Cicchetti (1976) suggested that gestation period is associated with the security of sleeping quarters. Species with relatively altrical young often develop in protected environments, such as burrows (e.g., Wolff 1997); as adults, they also would probably sleep in such safe quarters. Conversely, precocial species tend to live in exposed environments as adults and are thus expected to engage in less REM sleep (Lima et al. 2005). Based on both of these ideas, we included a direct path between gestation period (altricial/precocial gradient) and REM sleep time (fig. 1*a*) and %REM sleep (fig. 1*b*) and also a path from gestation period to sleep exposure index (fig. 1*a*, 1*b*).

Methods

Sources for Sleep Data

Our data set was created by an intensive literature search. We searched databases of primary literature (e.g., Web of Science, PubMed) using these keywords: EEG, electrophysiology, mammal, rapid-eye-movement, REM, sleep, slow-wave, and SWS and combinations thereof. A particularly important resource was the latest preexisting data set of comparative sleep architecture (Elgar et al. 1988) as updated by Elgar et al. (1990). We verified the majority of sleep quotas taken from Elgar et al. (1988, 1990) and adhered to the standards of Berger (1990) when adding species to our data set. In addition to the primary literature, data from book chapters and published meeting abstracts were also included. We note that sleep quotas for virtually all of the species in our data set came from laboratory recordings. Technological limitations have prevented such studies from being conducted in the field. Our analysis is thus based on the assumption that patterns of sleep in the laboratory reflect patterns of sleep in the wild.

Our final data set contained complete information on 54 species (table A1 in the online edition of the *American Naturalist*). Sleep data were included only from studies that quantified the time spent in SWS and REM sleep based on electrophysiology of adult mammals. When multiple electrophysiological sleep studies were available for a particular species, we calculated an average of sleep values weighted by respective sample sizes. As per Elgar et al. (1988), we excluded four species of cetacean (Shurley et al. 1969; Mukhametov and Polyakova 1981; Mukhametov et al. 1988; Lyamin et al. 2002*b*) and two species of manatee (Sokolov and Mukhametov 1982; Mukhametov et al. 1990, 1992)

because these aquatic mammals either do not exhibit REM sleep or do not exhibit REM sleep comparable to that of terrestrial mammals (Rattenborg et al. 2000; Lyamin et al. 2002*a*). We also excluded two species of monotreme for which electrophysiological sleep data were available (Siegel et al. 1999; Nicol et al. 2000) since ambiguity exists regarding the characterizing of sleep states in these mammals. Secondarily, we excluded electrophysiological data for several species for which either basal metabolic rate or brain mass were unavailable. The general lack of BMR data represents an important area of study necessary for future comparative work on mammalian sleep.

Sources for Constitutive Traits

Body masses of taxa came from the source study (as per Berger 1990) or from another published source (e.g., Crile and Quiring 1940; McNab and Eisenberg 1989; Nowak 1999) when unavailable in the source study. Brain masses for taxa were taken from the primary literature (e.g., Crile and Quiring 1940; McNab and Eisenberg 1989; Marino 1998; Bininda-Emonds 2000). The brain mass of the Mexican volcano mouse (*Neotomodon alstoni*) was provided by A. Castro and I. Villalpando (personal communication). Gestation periods were taken from Hayssen et al. (1993), except for three cases taken from the literature (star-nosed mole *Condylura cristata*, Kurta 1995; olive baboon *Papio anubis*, Herring et al. 1991; domestic dog *Canis familiaris*, Linde-Forsberg and Forsberg 1993). Basal metabolic rates were available in the literature (e.g., McNab 1988).

Sources for Ecological Variables

We included two measures of predation risk: sleep exposure index and trophic position. Unfortunately, the risk-related scoring system of Allison and Cicchetti (1976) was not described explicitly; thus, we generated our own indexes of risk. As in Allison and Cicchetti (1976), our scoring system was based on information gathered in *Walker's Mammals of the World* (Nowak 1999) and used discrete categories of risk; continuous measures of predation risk are unavailable at this scale of analysis (Lima 2002).

The sleep exposure index was based on a six-point integer scale that ranked the relative exposure of a given species' typical sleep quarters in the wild. Low values reflect safer sleep sites, and higher values reflect increasing exposure to predators. The rankings below reflect the idea that sleeping in hard-to-reach locations (e.g., burrows) is safer than sleeping in more exposed arboreal (e.g., tree canopy) or terrestrial locations (e.g., forest floor), which in turn are safer than sleeping in the open. The numerical rankings were assigned according to sleep site as follows: 1, cave ceilings, rock crevices, burrows, tree holes; 2, under logs or debris, hollow logs, dens, cave floors, hollow standing trees, sides of cliffs; 3, tree canopy or nest in tree; 4, well below the tree canopy at branch junctions; 5, forest floor or brush piles; 6, ground-level in open grasslands. Some species sleep at more than one level of our sleep exposure index; in such instances, we averaged values among levels. Implicit in these rankings is the assumption that the change in risk between adjacent ranks is constant, which is unavoidable given the complete lack of quantitative information on the risk of predation in almost every behavioral context (Lima 2002). An examination of the costs and benefits associated with particular sleep sites would be a valuable avenue for future work (Anderson 1998; Lima et al. 2005).

Trophic position was a four-point integer scale of relative risk based on diet, which we used as a surrogate for vulnerability to predators. We assumed that carnivores experience a lower risk of predation than those with a mixed animal-plant diet, which in turn experience a lower risk of predation than herbivores. Numerical rankings of diet were assigned as follows: 1, entirely vertebrates (lowest risk); 2, large invertebrates; 3, small invertebrates; 4, exclusively plants (highest risk). Like the sleep exposure index, some mammals had diets that spanned more than one level; in those instances, values were averaged between levels. However, if the primary constituent of diet was reported, then that specific diet category was given twice the weight of the others in the average. Elgar et al. (1988) included a variable similar to our trophic position in their analysis that they associated with foraging demands.

Phylogenetic Analysis

To control for the nonindependence of taxa inherent in comparative analyses, we transformed our data set (table A1) into a set of independent contrasts (Felsenstein 1985). We first created a phylogenetic tree (fig. A1 in the online edition of the *American Naturalist*) based on the order-level tree of Murphy et al. (2001). Additional trees supplied the within-order and within-family topology for marsupials (Osborne et al. 2002), Xenarthra (Delsuc et al. 2003), Insectivora (Soricidae and Talpidae; Grenyer and Purvis 2003), Primates (Purvis 1995), Artiodactyla (Matthee et al. 2001), and Rodentia (Muridae; Herron et al. 2004; Jansa and Weksler 2004). No phylogeny was available for our three hyraxes (Hyracoidea), so we used the taxonomy by Roche (1972), which placed *Heterohyrax* as a subgenus within *Dendrohyrax*. Other taxonomic groups had either one or two representatives; thus, no additional phylogenetic information was required to establish their positions within the tree. Since our tree was an amalgam of trees derived using very different types of data and techniques, branch lengths were undeter-

mined and thus set arbitrarily to 1 (see Garland et al. 2005). We note that some disagreement exists as to the structure of the deeper nodes of the mammalian phylogenetic tree (e.g., Arnason et al. 2002). However, when based on the Arnason et al. (2002) phylogeny, our results were virtually identical to those based on Murphy et al. (2001). We therefore present only the results based on the latter phylogenetic tree.

Variables were log-transformed to meet the assumption of normality when necessary, as determined using a Shapiro-Wilks test (SPSS 2001). Percent REM sleep was arcsin square root transformed. Variables were then entered into COMPARE 4.6b (Martins 2004), a phylogenetically driven software package that uses the evolutionary relationships among taxa to transform comparative data into a set of independent contrasts (Felsenstein 1985). The resulting independent contrasts were then used for all analyses. We confirmed that contrasts were standardized by inspecting scatterplots of absolute values of contrasts versus standard deviation, which had (as required) slopes approximately equal to 0 (see Garland et al. 1992). We note that for each trait, standard errors were entered when known (table A1) and left at a default of 0 when unknown. However, statistical procedures such as independent contrasts and path analysis cannot currently account for standard errors (T. Garland, personal communication). Collectively, these statistical issues are important areas for future research.

Path Analysis

As outlined above, we constructed two path models based on various hypotheses for the function of sleep (see also table 1). Path models were analyzed using phylogenetically corrected contrasts in Amos 5.0 (SPSS 2003). Path models were forced through the origin, as is required when dealing with standardized data (see Garland et al. 1992).

Results

Models 1 and 2 explained the relationships among variables reasonably well. The comparative fit index, a measure of model fit ranging from 0 to 1, was 0.929 for model 1 and 0.959 for model 2, indicating good model fits (Bentler 1990). Given the complex nature of the models and our coarse ecological variables, the independent and mediator variables explained a relatively large proportion of the variance in sleep architecture: SWS time ($R^2 = 0.42$), REM sleep time ($R^2 = 0.44$), total sleep time ($R^2 = 0.44$), and %REM sleep ($R^2 = 0.36$). Despite these fits, however, not all of the proposed paths were supported by our analyses. Below, we consider the paths from all variables in turn. We note that standardized regression coefficients (β) in the path models range from -1 to 1 and quantify the magnitude and direction of a relationship between two variables within the context of each model. We also note that the interpretation of a contrast-based relationship differs somewhat from one based on nonphylogenetically corrected data in that contrast-based results reflect a relationship between the change in a given variable (e.g., SWS time) and the change in another variable (e.g., BMR; Felsenstein 1985).

Paths from body mass. We hypothesized that body mass influences sleep architecture only indirectly via mediator variables. The effects of body mass were indeed mediated through brain mass ($\beta = 0.872$, $P < .001$), BMR ($\beta = 0.927$, $P < .001$), and gestation period ($\beta = 0.572$, $P < .001$) but not through sleep exposure index ($\beta = 0.187$, $P = .255$). These relationships were numerically identical in the two path models. In both models, direct paths between body mass and sleep variables were not significant (paths not shown; SWS: $\beta = 0.159$, $P = .640$; REM sleep: $\beta = 0.216$, $P = .332$; total sleep time: $\beta = 0.383$, $P = .241$; %REM sleep: $\beta = 0.389$, $P = .098$). Therefore, paths emanating from the mediator variables of brain mass, BMR, and gestation period essentially represent the effect of residual values as outlined above.

Paths from brain mass. In our path model, %REM sleep was positively related to brain mass (conceptually relative brain mass, our encephalization quotient equivalent; fig. 1*b*; $\beta = 0.507$, $P < .001$). Thus, species with relatively larger brain masses allocated a significantly greater proportion of total time asleep to REM sleep. The relationship between (relative) brain mass and REM sleep was positive but nonsignificant ($\beta = 0.189$, $P = .116$). Species with relatively larger brain masses did not engage in more SWS (fig. 1*a*; $\beta = -0.038$, $P = .832$) or more sleep in general (fig. 1*b*; $\beta = 0.113$, $P = .522$).

Paths from basal metabolic rate. Our path models indicated a significantly negative relationship between (relative) BMR and SWS time (fig. 1*a*; $\beta = -0.573$, $P = .002$) and total sleep time (fig. 1*b*; $\beta = -0.651$, $P < .001$). That is, mammals with BMRs greater than expected for a given body mass tended to engage in less SWS and slept less altogether. BMR was the only variable significantly related to SWS time (fig. 1*a*).

Paths from predation variables. Our results suggested that predation risk influences sleep architecture primarily via REM sleep. Sleep exposure index related negatively to REM sleep time (fig. 1*a*; $\beta = -0.295$, $P = .005$) and %REM sleep (fig. 1*b*; $\beta = -0.248$, $P = .029$), indicating that species sleeping in riskier environments engage in less REM sleep and allocate less time spent asleep to REM sleep. The negative relationships between sleep exposure index and SWS time (fig. 1*a*; $\beta = -0.102$, $P = .344$) and total sleep time (fig. 1*b*; $\beta = -0.156$, $P = .141$) were nonsignificant.

Trophic position also related significantly and negatively to REM sleep time (fig. 1*a*; $\beta = -0.330$, $P = .001$), %REM sleep (fig. 1*b*; $\beta = -0.338$, $P = .002$), and total sleep time (fig. 1*b*; $\beta = -0.259$, $P = .012$), indicating that more herbivorous species engage in less overall sleep and less REM sleep and allocate less time asleep to REM sleep. The path between trophic position and SWS time was not significant (fig. 1*a*; $\beta = -0.126$, $P = .233$).

Paths from gestation period. The path models indicated that, in relatively precocial species, adults have significantly lower REM sleep times (fig. 1*a*, $\beta = -0.527$, $P < .001$) and less %REM sleep (fig. 1*b*, $\beta = -0.348$, $P = .007$) than species with altricial young. We also hypothesized that the influence of gestation period on REM sleep might be mediated through the sleep exposure index, but we found no support for this idea (fig. 1*a*, 1*b*; $\beta = 0.066$, $P = .686$).

Discussion

Our phylogenetically based path models represent a view of sleep architecture that is more comprehensive than those offered by previous correlational studies. Our basic results can be summarized as follows. In addition to finding that body mass is not directly correlated with sleep architecture, we found that species with higher relative BMRs engage in less SWS. Species with larger relative brain masses allocate a greater proportion of total sleep time to REM sleep. Species sleeping under higher risks of predation engage in less REM sleep and allocate less time asleep to REM sleep. As adults, precocial species also tend to engage in less REM sleep than more altricial species. Below, we interpret our results with respect to both existing hypotheses for the function of sleep and previous comparative studies.

Many correlations identified by previous studies (e.g., Zepelin and Rechtschaffen 1974; Allison and Cicchetti 1976) were probably confounded by body mass effects because body mass is strongly correlated with several constitutive variables (McNab 1988; McNab and Eisenberg 1989). Our path models indicate that body mass does not have a direct influence on any sleep variable, which is in accordance with the literature in that no existing hypothesis addresses the mechanism behind a direct relationship between body mass and sleep architecture without incorporating a third variable. Thus, the statement "larger mammals sleep less" is true but is not informative, in that larger mammals sleep less because body mass is related to other variables, such as BMR and risk associated with sleep site, which in turn influence sleep architecture.

The negative relationship between BMR (conceptually relative BMR) and SWS time/total sleep time does not support either form of the energy conservation hypothesis (Berger and Phillips 1995; Zepelin 2000) or many other metabolism-oriented hypotheses for the function of sleep, which would predict a relationship in the opposite direction. Our result might suggest that the speed or efficiency of the restorative aspects of sleep are dependent on BMR such that mammals with relatively higher BMRs engage in less SWS and less overall sleep because these restorative processes occur more quickly. Alternatively, this negative relationship supports the suggestion of Elgar et al. (1988) that mammals with relatively higher BMRs sleep less so that they can allocate more time to foraging to the meet the demands of increased metabolic rate. Although we did not find comparative support for the energy conservation hypotheses, intraspecific evidence suggests that energy conservation can influence sleep architecture under stressful conditions (Walker et al. 1980, 1983*b*; Rashotte et al. 1998) and that the timing of sleep may be adaptive for conserving energy (Saarikko and Hanski 1990).

In a previous analysis, Zepelin and Rechtschaffen (1974) reported a positive correlation between "relative" BMR and SWS time/total sleep time (see also Siegel 2004, 2005; Zepelin et al. 2005). The discrepancy between this positive result and the negative relationship seen in our study probably reflects methodological differences in the statistical control of body mass. Our path models controlled for body mass effects in BMR using residuals (as per Elgar et al. 1988) rather than mass-specific ratios (as per Zepelin and Rechtschaffen 1974; Siegel 2004; Zepelin et al. 2005). A mass-specific ratio (e.g., BMR/body mass) is an efficient way to control for the effects of body mass when BMR varies as a constant proportion of body mass (Packard and Boardman 1988, 1999). However, this assumption of constant proportionality does not hold, and thus the mass-specific value will still correlate with body mass. In such instances, residuals can be a more effective statistical control (Packard and Boardman 1988, 1999). The above diametrically opposed results are apparent in our raw (noncontrast) data, in which the correlation between mass-specific BMR (BMR/body mass) and SWS time was indeed significant and positive ($r = 0.298$, $P = .029$), as per Zepelin and Rechtschaffen (1974) and Zepelin et al. (2005). Mass-specific BMR, however, still correlated strongly with body mass ($r = -0.864$, $P < .001$), indicating that the control of body mass in the mass-specific measure was incomplete. Conversely, residual BMR correlated significantly and negatively with SWS time (raw data: $r = -0.355$, $P = .008$) but did not correlate with body mass at all ($r = 0.000$), as is the case when the assumptions of normality of residuals and homoscedasticity are met. Beaupre and Dunham (1995) also reported qualitatively dissimilar results when using residuals as opposed to mass-specific values in their analysis.

The positive relationship between (relative) brain mass

and %REM sleep suggests that mammals with relatively greater encephalization allocate more time asleep to REM sleep. However, given that ambiguity exists regarding exactly what facet of cognitive ability is captured by the encephalization quotient, we do not know whether these relationships reflect learning, memory consolidation (see also Walker and Stickgold 2004), or something else. For instance, our results might suggest that REM sleep is important in the maintenance or restoration of neural tissues, substrates, or metabolites (Benington and Heller 1995) associated with cognitive abilities. We note, however, that some species with the highest amounts of REM sleep (e.g., opossums) may not possess great cognitive abilities (Siegel 2000). We also note that Siegel (2004) and Zepelin et al. (2005) found that species with a higher encephalization quotient engaged in less %REM sleep. A similar negative correlation is also apparent in our raw data (between residual brain mass and %REM sleep: $r = -0.281$, $N = 54$, $P = .039$) but switches sign (and is nonsignificant) when phylogenetic effects are taken into account ($r = 0.092$, $N = 53$, $P = .512$).

Using conservative measures of predation risk, we found that mammalian species sleeping in riskier environments and those at the prey end of the trophic spectrum have lower REM sleep times and allocate a lower proportion of time spent asleep to REM sleep than those sleeping in more secure quarters and carnivorous species (see also Allison and Cicchetti 1976; Meddis 1983). We also identified a tendency for prey species to sleep less overall. These results provide support for the idea that REM sleep is a dangerous state (see also Lima et al. 2005). Recent experimental work bolsters this conclusion in that the expectation of electric shocks (i.e., a simulated threat) has a suppressive effect on REM sleep in rats (Sanford et al. 2001, 2003).

Perhaps consistent with expectations under the ontogenetic hypothesis for REM sleep (Roffwarg et al. 1966; Marks et al. 1995), relatively precocial species tend to engage in less REM sleep as adults, in both absolute and relative measures, than more altricial species (see also Siegel 2004; Zepelin et al. 2005). Again, however, it is not clear why, under this hypothesis, altricial species would engage in more REM sleep as adults than precocial species. The influence of (relative) gestation period was not mediated strongly through our sleep exposure index.

Our path models shed relatively little light on the determinants of SWS time. Only (relative) BMR associated significantly with SWS time, and even then, the relationship was not in the direction expected, given current hypotheses. In addition, the lack of significant relationships between our measures of predation risk and SWS time is surprising. We note, however, that SWS can respond to predation risk in a way not described by our data set. Multiple stages (or depths) of SWS have been reported in several species of mammal reflecting a continuum in the depth of sleep (Borbély and Achermann 2000; Tobler 2005). SWS in species sleeping under a higher risk of predation may be lighter than in those sleeping under lower predation risk (Lima et al. 2005). Thus, while the time spent in SWS may not respond strongly to predation risk, the depth of SWS may do so. SWS intensity may also be the more appropriate measure of SWS to address some of the hypotheses above, such as energy conservation. Unfortunately, studies rarely report information on the depth of SWS, which is clearly an area needing further research.

Although it is a common practice for researchers to hypothesize a primary function of sleep, our path models suggest that one overriding function may not exist. Rather, sleep architecture is probably influenced by multiple factors and probably reflects many functions. For instance, our models implicate REM sleep in the maintenance of some facet of the brain, possibly related to cognitive ability (Walker and Stickgold 2004). This idea complements another hypothesis for REM sleep involving neural ontogenesis and plasticity (Marks et al. 1995). Concurrent with these possible neurological roles for REM sleep, predation risk has a suppressive effect on REM sleep and favors shifts to safer sleep states (Lima et al. 2005). Other existing hypotheses for the function of sleep are also compatible with the hypotheses discussed here, such as sleep having a role in host immunological defense (Opp and Toth 2003). However, these functions may not necessarily have the same weight in each species. Species-specific ecological histories are expected to determine (in part) the relative importance of a given function and its subsequent effect on sleep architecture. Incorporating such ideas into strategic models of "behavioral shutdowns" (e.g., Pravosudov and Lucas 2000) should also prove fruitful.

Future studies should expand on our comparative data set to further illuminate pressures that may influence mammalian sleep architecture. Our current understanding of sleep in mammals comes largely from studies on rodents and, to a lesser extent, primates (Tobler 1995). Little is known about the electrophysiological correlates of sleep in the larger mammals. There is also a decided need to create analogous data sets on nonmammalian species (e.g., Campbell and Tobler 1984). Such data sets could also be expanded to include other potentially salient sleep variables such as the stages (or depth) of SWS, duration of sleep stage episodes, and the timing of sleep within the 24-h day (Campbell and Tobler 1984). It is also imperative that future studies be conducted in the field as well as in the laboratory environment. Such endeavors may illuminate additional functions of sleep and yield much in-

sight into the evolutionary factors that influence when, where, and how to sleep.

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Literature Cited

Affanni, J. 1972. Observations on the sleep of some South American marsupials and edentates. Perspectives in the Brain Sciences 1:21–23.

Affanni, J. M., C. O. Cervino, and H. J. A. Marcos. 2001. Absence of penile erections during paradoxical sleep: peculiar penile events during wakefulness and slow wave sleep in the armadillo. Journal of Sleep Research 10:219–228.

Allison, T., and D. V. Cicchetti. 1976. Sleep in mammals: ecological and constitutional correlates. Science 194:732–734.

Allison, T., and H. van Twyver. 1970*a*. The evolution of sleep. Natural History 79:56–65.

———. 1970*b*. Sleep in the moles *Scalopus aquaticus* and *Condylura cristata*. Experimental Neurology 27:564–578.

Allison, T., S. D. Gerber, S. M. Breedlove, and G. L. Drydon. 1977. A behavioral and polygraphic study of sleep in the shrews *Suncus murinus*, *Blarina brevicaudata* and *Cryptotis parva*. Behavioral Biology 20:354–366.

Ambrosini, M. V., and A. Giuditta. 2001. Learning and sleep: the sequential hypothesis. Sleep Medicine Reviews 5:477–490.

Anderson, J. R. 1998. Sleep, sleeping sites, and sleep-related activities: awakening to their significance. American Journal of Primatology 46:63–75.

Arnason, U., J. A. Adegoke, K. Bodin, E. W. Born, Y. B. Esa, A. Gullberg, M. Nilsson, et al. 2002. Mammalian mitogenomic relationships and the root of the eutherian tree. Proceedings of the National Academy of Sciences of the USA 99:8151–8156.

Astic, L., D. Saucier, and D. Megirian. 1979. Sleep circadian rhythm in rat kangaroo (*Potorous apicalis*): effect of food distribution. Physiology and Behavior 22:441–446.

Ball, N. J. 1992. The phasing of sleep in animals. Pages 31–49 *in* C. Stampi, ed. Why we nap. Birkhäuser, Boston.

Balzamo, E., and J. Bert. 1975. Sleep in *Papio anubis*: its organisation and lateral geniculate spikes. Sleep Research 4:138.

Balzamo, E., R. J. Bradley, D. M. Bradley, G. V. Pegram, and J. M. Rhodes. 1972. Sleep ontogeny in the chimpanzee from birth to two months. Electroencephalography and Clinical Neurophysiology 33:47–60.

Balzamo, E., G. Vuillon-Cacciuttolo, J. J. Petter, and J. Bert. 1978. States of vigilance in two Lemuridae: EEG patterns and organisation obtained using telemetry. Waking and Sleeping 2:237–245.

Balzamo, E., P. van Beers, and D. Lagarde. 1998. Scoring of sleep and wakefulness by behavioral analysis from video recordings in rhesus monkeys: comparison with conventional EEG analysis. Electroencephalography and Clinical Neurophysiology 106:206–212.

Baron, R. M., and D. A. Kenny. 1986. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology 51:1173–1182.

Barre, V., and A. Petter-Rousseaux. 1988. Seasonal variations in sleep-wake cycle in *Microcebus murinus*. Primates 29:53–64.

Beaupre, S. J., and A. E. Dunham. 1995. A comparison of ratio-based and covariance analyses of a nutritional data set. Functional Ecology 9:876–880.

Bell, F. R., and T. Itabisashi. 1973. The electroencephalogram of sheep and goats with special reference to rumination. Physiology and Behavior 11:503–514.

Benington, J. H., and H. C. Heller. 1995. Restoration of brain energy metabolism as the function of sleep. Progress in Neurobiology 45: 347–360.

Bentler, P. M. 1990. Comparative fit indexes in structural models. Psychological Bulletin 107:238–246.

Berger, R. J. 1990. Relations between sleep duration, body weight and metabolic rate in mammals. Animal Behaviour 40:989–991.

Berger, R. J., and N. H. Phillips. 1995. Energy conservation and sleep. Behavioural Brain Research 69:65–73.

Berger, R. J., and J. M. Walker. 1972. A polygraphic study of sleep in the tree shrew *Tupaia glis*. Brain, Behavior and Evolution 5:54–69.

Bert, J. 1973. Similarities and differences in the sleep of two baboons, *Papio hamadryas* and *Papio papio*. Electroencephalography and Clinical Neurophysiology 35:209–212.

Bert, J., E. Balzamo, M. Chase, and V. Pegram. 1975. The sleep of the baboon, *Papio papio*, under natural conditions and in the laboratory. Electroencephalography and Clinical Neurophysiology 39:657–662.

Bininda-Emonds, O. R. P. 2000. Pinniped brain sizes. Marine Mammal Science 16:469–481.

Borbély, A. A., and P. Achermann. 2000. Sleep homeostasis and models of sleep regulation. Pages 377–390 *in* M. H. Kryger, T. Roth, and W. C. Dement, eds. Principles and practice of sleep medicine. 3rd ed. Saunders, Philadelphia.

Brebbia, D. R., and E. T. Pyne. 1972. Environmental hypothermia and cerebral electrical activity in the little brown bat, *Myotis lucifugus*. Psychophysiology 9:122.

Campbell, S. S., and I. Tobler. 1984. Animal sleep: a review of sleep duration across phylogeny. Neuroscience and Biobehavioral Reviews 8:269–300.

Carskadon, M. A., and W. C. Dement. 1994. Normal human sleep: an overview. Pages 16–25 *in* M. H. Kryger, T. Roth, and W. C. Dement, eds. Principles and practice of sleep medicine. 2nd ed. Saunders, Philadelphia.

Cirelli, C., C. M. Gutierrez, and G. Tononi. 2004. Extensive and divergent effects of sleep and wakefulness on brain gene expression. Neuron 41:35–43.

Crile, G., and D. P. Quiring. 1940. A record of the body weight and

certain organ and gland weights of 3,690 animals. Ohio Journal of Science 40:219–259.

Crofts, H. S., S. Wilson, N. G. Muggleton, D. J. Nutt, E. A. M. Scott, and P. C. Pearce. 2001. Investigation of the sleep electrocorticogram of the common marmoset (*Callithrix jacchus*) using radiotelemetry. Clinical Neurophysiology 112:2265–2273.

Dallaire, A., and Y. Ruckebusch. 1974*a*. Rest-activity cycle and sleep patterns in captive foxes *Vulpes vulpes*. Experientia 30:59–60.

———. 1974*b*. Sleep patterns in the pony with observations on partial perceptual deprivation. Physiology and Behavior 12:789–796.

Delsuc, F., M. J. Stanhope, and E. J. P. Douzery. 2003. Molecular systematics of armadillos (Xenarthra, Dasypodidae): contribution of maximum likelihood and Bayesian analyses of mitochondrial and nuclear genes. Molecular Phylogenetics and Evolution 28:261–275.

de Moura Filho, A. G., S. E. Huggins, and S. G. Lines. 1983. Sleep and waking in the three-toed sloth, *Bradypus tridactylus*. Comparative Biochemistry and Physiology A 76:345–355.

Deregnaucourt, S., P. P. Mitra, O. Feher, C. Pytte, and O. Tchernichovski. 2005. How sleep affects the developmental learning of bird song. Nature 433:710–716.

Dillon, R. F., and W. B. Webb. 1965. Threshold of arousal from activated sleep in the rat. Journal of Comparative and Physiological Psychology 59:447–449.

Edgar, D. M., W. C. Dement, and C. A. Fuller. 1993. Effect of SCN lesions on sleep in squirrel monkeys: evidence for opponent processes in sleep-wake regulation. Journal of Neuroscience 13:1065–1079.

Eisenberg, J. F. 1981. The mammalian radiations: an analysis of trends in evolution, adaptation, and behavior. University of Chicago Press, Chicago.

Elgar, M. A., M. D. Pagel, and P. H. Harvey. 1988. Sleep in mammals. Animal Behaviour 36:1407–1419.

———. 1990. Sources of variation in mammalian sleep. Animal Behaviour 40:991–995.

Felsenstein, J. 1985. Phylogenies and the comparative method. American Naturalist 125:1–15.

Fenn, M. G. P., and D. W. Macdonald. 1995. Use of middens by red foxes: risk reverses rhythms of rats. Journal of Mammalogy 76: 130–136.

Frank, M. G., and H. C. Heller. 2003. The ontogeny of mammalian sleep: a reappraisal of alternative hypotheses. Journal of Sleep Research 12:25–34.

Gais, S., and J. Born. 2004. Declarative memory consolidation: mechanisms acting during human sleep. Learning and Memory 11:679–685.

Garland, T., P. H. Harvey, and A. R. Ives. 1992. Procedures for the analysis of comparative data using phylogenetically independent contrasts. Systematic Biology 41:18–32.

Garland, T., A. F. Bennett, and E. L. Rezende. 2005. Phylogenetic approaches in comparative physiology. Journal of Experimental Biology 208:3015–3035.

Grenyer, R., and A. Purvis. 2003. A composite species-level phylogeny of the "Insectivora" (Mammalia: Order Lipotyphla Haeckel, 1866). Journal of Zoology 260:245–257.

Haskell, E. H., J. M. Walker, and R. J. Berger. 1979. Effects of cold stress on sleep of a hibernator, the golden-mantled ground squirrel (*C. lateralis*). Physiology and Behavior 23:1119–1121.

Hayssen, V., A. van Tienhoven, and A. van Tienhoven. 1993. Asdell's patterns of mammalian reproduction: a compendium of species-specific data. Cornell University Press, Ithaca, NY.

Herring, J. M., J. D. Fortman, R. J. Anderson, and B. T. Bennett. 1991. Ultrasonic determination of fetal parameters in baboons (*Papio anubis*). Laboratory Animal Science 41:602–605.

Herron, M. D., T. A. Castoe, and C. L. Parkinson. 2004. Sciurid phylogeny and the paraphyly of Holarctic ground squirrels (*Spermophilus*). Molecular Phylogenetics and Evolution 31:1015–1030.

Horne, J. A. 1988. Why we sleep: the functions of sleep in humans and other animals. Oxford University Press, Oxford.

———. 2000. REM sleep—by default? Neuroscience and Biobehavioral Reviews 24:777–797.

Huber, R., M. F. Ghilardi, M. Massimini, and G. Tononi. 2004. Local sleep and learning. Nature 430:78–81.

Ibuka, N. 1984. Ontogenesis of circadian sleep-wakefulness rhythms and developmental changes of sleep in the altricial rat and in the precocial guinea pig. Behavioural Brain Research 11:185–196.

Jansa, S. A., and M. Weksler. 2004. Phylogeny of muroid rodents: relationships within and among major lineages as determined by IRBP gene sequences. Molecular Phylogenetics and Evolution 31: 256–276.

Jerison, H. J. 1985. Animal intelligence as encephalization. Philosophical Transactions of the Royal Society B: Biological Sciences 308:21–35.

Karadzic, W. 1967. Polygraphic stages of the activity rhythm of the blind mole rat *Spalax leucodon* Nordman, 1840. Säugetierkunde Mitteilungen 2:167–170.

Karmanova, I. G., F. M. Maksimuk, L. N. Murav'eva, Y. F. Pastukhov, and V. S. Sazonov. 1979. Specific features of the cycle "wakefulness-sleep" in the arctic lemming *Dicrostonyx torquatus*. Journal of Evolutionary Biochemistry and Physiology 15:190–195.

Kilduff, T. S., and M. G. Dube. 1976. Sleep characteristics of the cotton rat (*Sigmodon hispidus*). Sleep Research 5:97.

Kurta, A. 1995. Mammals of the Great Lakes region. University of Michigan Press, Ann Arbor.

Lesku, J. A., N. C. Rattenborg, and C. J. Amlaner. 2006. The evolution of sleep: a phylogenetic approach, Pages 49–61 *in* T. L. Lee-Chiong, ed. Sleep: a comprehensive handbook. Wiley, Hoboken, NJ.

Lima, S. L. 2002. Putting predators back into behavioral predator-prey interactions. Trends in Ecology & Evolution 17:70–75.

Lima, S. L., N. C. Rattenborg, J. A. Lesku, and C. J. Amlaner. 2005. Sleeping under the risk of predation. Animal Behaviour 70:723–736.

Linde-Forsberg, C., and M. Forsberg. 1993. Results of 527 controlled artificial inseminations in dogs. Journal of Reproduction and Fertility 47:313–323.

LoPresti, R. W., and D. J. McGinty. 1970. Sleep in the phalanger (*Trichosurus vulpecula*): an Australian marsupial. Psychophysiology 7:304.

Lyamin, O. I., A. I. Oleksenko, and I. G. Polyakova. 1993. Sleep in the harp seal (*Pagophilus groenlandica*): peculiarities of sleep in pups during the first month of their lives. Journal of Sleep Research 2:163–169.

Lyamin, O. I., O. V. Shpak, E. A. Nazarenko, and L. M. Mukhametov. 2002*a*. Muscle jerks during behavioral sleep in a beluga whale (*Delphinapterus leucas* L.). Physiology and Behavior 76:265–270.

Lyamin, O. I., L. M. Mukhametov, J. M. Siegel, E. A. Nazarenko, I. G. Polyakova, and O. V. Shpak. 2002*b*. Unihemispheric slow wave sleep and the state of the eyes in a white whale. Behavioural Brain Research 129:125–129.

Maquet, P. 1995. Sleep function(s) and cerebral metabolism. Behavioural Brain Research 69:75–83.

Marino, L. 1998. A comparison of encephalization between odontocete cetaceans and anthropoid primates. Brain, Behavior and Evolution 51:230–238.

———. 2002. Convergence of complex cognitive abilities in cetaceans and primates. Brain, Behavior and Evolution 59:21–32.

Marks, G. A., J. P. Shaffery, A. Oksenberg, S. G. Speciale, and H. P. Roffwarg. 1995. A functional role for REM sleep in brain maturation. Behavioural Brain Research 69:1–11.

Martins, E. P. 2000. Adaptation and the comparative method. Trends in Ecology & Evolution 15:296–299.

———. 2004. COMPARE. Computer programs for the statistical analysis of comparative data. Version 4.6b. Department of Biology, Indiana University, Bloomington.

Matthee, C. A., J. D. Burzlaff, J. F. Taylor, and S. K. Davis. 2001. Mining the mammalian genome for artiodactyl systematics. Systematic Biology 50:367–390.

McGinty, D., and R. Szymusiak. 1990. Keeping cool: a hypothesis about the mechanisms and functions of slow-wave sleep. Trends in Neurosciences 13:480–487.

McNab, B. K. 1988. Complications inherent in scaling the basal rate of metabolism in mammals. Quarterly Review of Biology 63:25–54.

McNab, B. K., and J. F. Eisenberg. 1989. Brain size and its relation to the rate of metabolism in mammals. American Naturalist 133: 157–167.

McNew, J. J., R. C. Howe, and W. R. Adey. 1971. The sleep cycle and subcortical-cortical EEG relations in the unrestrained chimpanzee. Electroencephalography and Clinical Neurophysiology 30: 489–503.

Meddis, R. 1983. The evolution of sleep. Pages 57–95 *in* A. Mayes, ed. Sleep mechanisms and functions in humans and animals. Van Nostrand Reinhold, Wokingham.

Mitchell, R. J. 1992. Testing evolutionary and ecological hypotheses using path analysis and structural equation modeling. Functional Ecology 6:123–129.

Mukhametov, L. M., and I. G. Polyakova. 1981. EEG investigation of the sleep in porpoises (*Phocoena phocoena*). Journal of Higher Nervous Activity 31:333–339.

Mukhametov, L. M., A. I. Oleksenko, and I. G. Polyakova. 1988. Quantification of ECoG stages of sleep in the bottlenose dolphin. Neurophysiology 20:532–538.

Mukhametov, L. M., O. I. Lyamin, I. S. Chetyrbok, A. A. Vassilyev, and R. Dias. 1990. Sleep and wakefulness in an Amazonian manatee. Pages 119–122 *in* J. Horne, ed. Sleep 1990. Proceedings of the 10th European Congress on Sleep Research, Strasbourg, France. Pontenagel, Bochum.

Mukhametov, L. M., O. I. Lyamin, I. S. Chetyrbok, A. A. Vassilyev, and R. P. Diaz. 1992. Sleep in an Amazonian manatee, *Trichechus inunguis*. Experientia 48:417–419.

Murphy, W. J., E. Eizirik, W. E. Johnson, Y. P. Zhang, O. A. Ryderk, and S. J. O'Brien. 2001. Molecular phylogenetics and the origins of placental mammals. Nature 409:614–618.

Muzur, A. 2005. Toward an integrative theory of sleep and dreaming. Journal of Theoretical Biology 233:103–118.

Nicol, S. C., N. A. Andersen, N. H. Phillips, and R. J. Berger. 2000. The echidna manifests typical characteristics of rapid eye movement sleep. Neuroscience Letters 283:49–52.

Nowak, R. M. 1999. Walker's mammals of the world. Johns Hopkins University Press, Baltimore.

Opp, M. R., and L. A. Toth. 2003. Neural-immune interactions in the regulation of sleep. Frontiers in Bioscience 8:D768–D779.

Osborne, M. J., L. Christidis, and J. A. Norman. 2002. Molecular phylogenetics of the Diprotodontia (kangaroos, wombats, koala, possums, and allies). Molecular Phylogenetics and Evolution 25: 219–228.

Packard, G. C., and T. J. Boardman. 1988. The misuse of ratios, indices, and percentages in ecophysiological research. Physiological Zoology 61:1–9.

———. 1999. The use of percentages and size-specific indices to normalize physiological data for variation in body size: wasted time, wasted effort? Comparative Biochemistry and Physiology A 122:37–44.

Perachio, A. A. 1971. Sleep in the nocturnal primate *Aotus trivirgatus*. Proceedings of the 3rd International Congress of Primatology 2: 54–60. Karger, Basel.

Petraitis, P. S., A. E. Dunham, and P. H. Niewiarowski. 1996. Inferring multiple causality: the limitations of path analysis. Functional Ecology 10:421–431.

Pravosudov, V. V., and J. R. Lucas. 2000. The costs of being cool: a dynamic model of nocturnal hypothermia by small food-caching birds in winter. Journal of Avian Biology 31:463–472.

Prudom, A. E., and W. R. Klemm. 1973. Electrographic correlates of sleep behavior in a primitive mammal, the armadillo *Dasypus novemcinctus*. Physiology and Behavior 10:275–282.

Purvis, A. 1995. A composite estimate of primate phylogeny. Philosophical Transactions of the Royal Society B: Biological Sciences 348:405–421.

Rashotte, M. E., I. F. Pastukhov, E. L. Poliakov, and R. P. Henderson. 1998. Vigilance states and body temperature during the circadian cycle in fed and fasted pigeons (*Columba livia*). American Journal of Physiology 275:R1690–R1702.

Rattenborg, N. C., C. J. Amlaner, and S. L. Lima. 2000. Behavioral, neurophysiological and evolutionary perspectives on unihemispheric sleep. Neuroscience and Biobehavioral Reviews 24:817–842.

Rechtschaffen, A. 1998. Current perspectives on the function of sleep. Perspectives in Biology and Medicine 41:359–390.

Robert, S., and A. Dallaire. 1986. Polygraphic analysis of the sleep-wake states and the REM sleep periodicity in domesticated pigs (*Sus scrofa*). Physiology and Behavior 37:289–293.

Roche, J. 1972. Systématique du genre *Procavia* et des damans en général. Mammalia 36:22–49.

Roffwarg, H. P., J. N. Muzio, and W. C. Dement. 1966. Ontogenetic development of the human sleep-dream cycle. Science 152:604–619.

Ruckebusch, Y. 1972. The relevance of drowsiness in the circadian cycle of farm animals. Animal Behaviour 20:637–643.

Saarikko, J., and I. Hanski. 1990. Timing of rest and sleep in foraging shrews. Animal Behaviour 40:861–869.

Sanford, L. D., A. J. Silvestri, R. J. Ross, and A. R. Morrison. 2001. Influence of fear conditioning on elicited ponto-geniculo-occipital waves and rapid eye movement sleep. Archives Italiennes de Biologie 139:169–183.

Sanford, L. D., X. D. Tang, R. J. Ross, and A. R. Morrison. 2003. Influence of shock training and explicit fear-conditioned cues on sleep architecture in mice: strain comparison. Behavior Genetics 33:43–58.

Shapiro, C. M., C. C. Goll, G. R. Cohen, and I. Oswald. 1984. Heat production during sleep. Journal of Applied Physiology 56:671–677.

Shurley, J. T., E. A. Serafetinides, R. E. Brooks, R. Elsner, and D. W.

Kenney. 1969. Sleep in cetaceans. I. The pilot whale, *Globicephala scammoni*. Psychophysiology 6:230.
Siegel, J. M. 2000. Phylogenetic data bearing on the REM sleep learning connection. Behavioral and Brain Sciences 23:1007.
———. 2001. The REM sleep-memory consolidation hypothesis. Science 294:1058–1063.
———. 2003. Why we sleep. Scientific American 289:92–97.
———. 2004. Sleep phylogeny: clues to the evolution and function of sleep. Pages 163–176 *in* P. H. Luppi, ed. Sleep: circuits and functions. CRC, Boca Raton, FL.
———. 2005. Clues to the functions of mammalian sleep. Nature 437:1264–1271.
Siegel, J. M., P. R. Manger, R. Nienhuis, H. M. Fahringer, T. Shalita, and J. D. Pettigrew. 1999. Sleep in the platypus. Neuroscience 91: 391–400.
Snyder, F. 1974. Sleep-waking patterns of Hyracoidea. Sleep Research 3:87.
Snyder, F., N. Bugbee, and T. C. Douthitt. 1972. Telemetric studies of 24-hour sleep-waking patterns in some primitive mammals. Psychophysiology 9:122.
Sokolov, V. E., and L. M. Mukhametov. 1982. Electrophysiological investigation on the sleep in the manatee *Trichechus manatus*. Journal of Evolutionary Biochemistry and Physiology 18:191–193.
SPSS. 2001. SPSS for Windows, release 11.0.1. SPSS, Chicago.
———. 2003. Amos, release 5.0.1. SPSS, Chicago.
Stickgold, R. 1998. Sleep: off-line memory reprocessing. Trends in Cognitive Sciences 2:484–492.
Susic, V., and G. Masirevic. 1986. Sleep patterns in the Mongolian gerbil, *Meriones unguiculatus*. Physiology and Behavior 37:257–261.
Tauber, E. S., F. Michel, and H. P. Roffwarg. 1968. Preliminary note on the sleep and waking cycle in the desert hedgehog (*Paraechinus hypomelas*). Psychophysiology 5:201.
Tobler, I. 1989. Napping and polyphasic sleep in mammals. Pages 9–30 *in* D. F. Dinges and R. J. Broughton, eds. Sleep and alertness: chronobiological, behavioral, and medical aspects of napping. Raven, New York.
———. 1992. Behavioral sleep in the Asian elephant in captivity. Sleep 15:1–12.
———. 1995. Is sleep fundamentally different between mammalian species? Behavioural Brain Research 69:35–41.
———. 2005. Phylogeny of sleep regulation. Pages 77–90 *in* M. H. Kryger, T. Roth, and W. C. Dement, eds. Principles and practice of sleep medicine. 4th ed. Saunders, Philadelphia.
Tobler, I., and T. Deboer. 2001. Sleep in the blind mole rat *Spalax ehrenbergi*. Sleep 24:147–154.
Tobler, I., and K. Jaggi. 1987. Sleep and EEG spectra in the Syrian hamster (*Mesocricetus auratus*) under baseline conditions and following sleep deprivation. Journal of Comparative Physiology A 161:449–459.
Tobler, I., and B. Schwierin. 1996. Behavioural sleep in the giraffe (*Giraffa camelopardalis*) in a zoological garden. Journal of Sleep Research 5:21–32.
Tobler, I., P. Franken, and K. Jaggi. 1993. Vigilance states, EEG spectra, and cortical temperature in the guinea pig. American Journal of Physiology 264:R1125–R1132.
Tolaas, J. 1978. REM sleep and the concept of vigilance. Biological Psychiatry 13:135–148.
Toutain, P. L., and Y. Ruckebusch. 1975. Arousal as a cyclic phenomenon during sleep and hibernation in the hedgehog *Erinaceus europaeus*. Experientia 31:312–314.
Ursin, R. 1968. The two stages of slow wave sleep in the cat and their relation to REM sleep. Brain Research 11:347–356.
van Twyver, H. 1969. Sleep patterns of five rodent species. Physiology and Behavior 4:901–905.
van Twyver, H., and T. Allison. 1970. Sleep in the opossum *Didelphis marsupialis*. Electroencephalography and Clinical Neurophysiology 29:181–189.
———. 1974. Sleep in the armadillo *Dasypus novemcinctus* at moderate and low ambient temperatures. Brain, Behavior and Evolution 9:107–120.
van Twyver, H., and W. Garrett. 1972. Arousal threshold in the rat determined by "meaningful" stimuli. Behavioural Biology 7:205–215.
Vertes, R. P. 2004. Memory consolidation in sleep: dream or reality. Neuron 44:135–148.
Voss, U. 2004. Functions of sleep architecture and the concept of protective fields. Reviews in the Neurosciences 15:33–46.
Wagner, U., S. Gais, H. Haider, R. Verleger, and J. Born. 2004. Sleep inspires insight. Nature 427:352–355.
Walker, J. M., and R. J. Berger. 1980. The ontogenesis of sleep states, thermogenesis, and thermoregulation in the Virginia opossum. Developmental Psychobiology 13:443–454.
Walker, J. M., S. F. Glotzbach, R. J. Berger, and H. C. Heller. 1977. Sleep and hibernation in ground squirrels (*Citellus* spp.): electrophysiological observations. American Journal of Physiology 233: R213–R221.
Walker, J. M., E. H. Haskell, R. J. Berger, and H. C. Heller. 1980. Hibernation and circannual rhythms of sleep. Physiological Zoology 53:8–11.
Walker, J. M., L. E. Walker, D. V. Harris, and R. J. Berger. 1983*a*. Cessation of thermoregulation during REM sleep in the pocket mouse. American Journal of Physiology 244:R114–R118.
Walker, L. E., J. M. Walker, J. W. Palca, and R. J. Berger. 1983*b*. A continuum of sleep and shallow torpor in fasting doves. Science 221:194–195.
Walker, M. P., and R. Stickgold. 2004. Sleep-dependent learning and memory consolidation. Neuron 44:121–133.
Wexler, D. B., and M. C. Moore-Ede. 1985. Circadian sleep-wake cycle organization in squirrel monkeys. American Journal of Physiology 248:R353–R362.
Williams, M. F. 2002. Primate encephalization and intelligence. Medical Hypotheses 58:284–290.
Wolff, J. O. 1997. Population regulation in mammals: an evolutionary perspective. Journal of Animal Ecology 66:1–13.
Wright, S. 1934. The method of path coefficients. Annals of Mathematical Statistics 5:161–215.
Zepelin, H. 1970. Sleep of the jaguar and the tapir: a prey-predator contrast. Psychophysiology 7:305–306.
———. 2000. Mammalian sleep. Pages 82–92 *in* M. H. Kryger, T. Roth, and W. C. Dement, eds. Principles and practice of sleep medicine. 3rd ed. Saunders, Philadelphia.
Zepelin, H., and A. Rechtschaffen. 1974. Mammalian sleep, longevity, and energy metabolism. Brain, Behavior and Evolution 10:425–470.
Zepelin, H., J. M. Siegel, and I. Tobler. 2005. Mammalian sleep. Pages 91–100 *in* M. H. Kryger, T. Roth, and W. C. Dement, eds. Principles and practice of sleep medicine. 4th ed. Saunders, Philadelphia.

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