Journal of Phylogenetics & Evolutionary Biology

Spudeit, J Phylogen Evolution Biol 2014, 2:2
DOI: 10.4172/2329-9002.1000132

Research Article Open Access

Behavioral Sequence of Satiety: A Comparative Approach between Birds and Mammals

William Anderson Spudeit*

Department of Pharmacy and Pharmaceutical Management- Fluminense Federal University,24241-000, Niteroi, RJ , Brazil

Abstract

This approach on the behavioral satiety sequence (BSS) on the historical point of view, the relationship between behaviors that are part of this sequence, shaped as it is used in mammals and eventually brings a new model of how this sequence is made utilizer poultry using experimental animal allowing the use of index s to facilitate the study of the relationship between sequence and behaviors that are part of it.

Keywords: Feeding; Drinking; Sleep; Behavioral satiety sequence

A Behavioral Sequence of Satiety BSS

Behavioral Satiety Sequence we know under that name today is the result of physiological changes resulting eating behavior resulting in an organized and remarkable sequence of behaviors [1,2]. This sequence is composed of two phases, the first phase prandial composed by ingestion of food, and the second postprandial phase, composed by drinking behavior, behavior maintenance (Preening) and sleep behavior or attitude being typical sleep [1,2]. As can be seen in the Figure 1 below.

History

The discovery and description of the sequence of behaviors was only possible because of work, such as Barnet(1956) [3], Grant end Mackintosh (1963) [4] eScott (1966) [5]. These works brought a new look at behavioral pharmacology criticized the studies in the 50s and 60s abide by if only because no consequence to what happened, what behavior the animal had during the time of the experiment. From this new look, there was a substantial change on this type of experiment behavioral pharmacology including the study of food intake, by passing to observe and give more importance to behavior than just the total consumption of food after applying a drug. An example is the work of Bolles (1960) [6], studying the self-cleaning behavior in rats after ingestion of food.

In the year 1960, Richterend Bolles, first observed a sequence of behaviors performed by rodents that were presented to the food after being fasted for 24 hours .This sequence of behaviors was composed by drinking, then for a short period of preening and finished with a long period of inactivity with typical sleep posture. In 1974 arises, the work of Smith et al, the idea that cholecystokinin could be a natural satiety signal and triggering behavior of this sequence. Based on this idea and using behavioral observation and assessment tool, Antin and his colleagues came to confirmation that satiety was reflected through a sequence of behaviors [1,7].

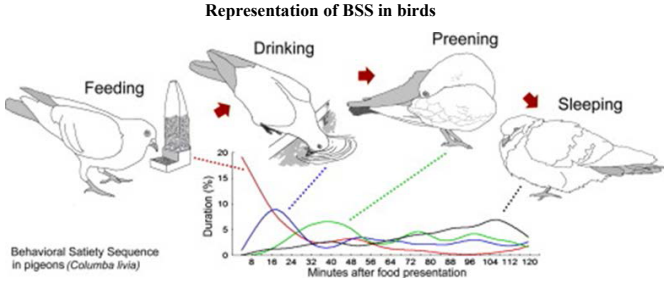

Figure 1: Statement of behaviors that are part of the SCS pigeons after adding a seed mixture . Accessed SPUDEIT, 2013).

Relationship Between the Behaviors that Make Up the BSS

Food intake

The intake has a direct relation to intake water in many species such as, for example, rodent, 70% of water consumption consumption occurs 24 hours from ten to thirty minutes after the feed intake. Already in pigeons, 70% of water consumption takes three minutes after the meal. In these species, a positive correlation between the size of the meal and the amount of water intake [8-14] is observed. The interdependence between the intake of feed and water intake becomes more evident when their consumption are evaluated in the absence of one or the other [11]. This leads to reduction in both feed intake and water in many species, and in invertebrates [15] in rodents [6] and birds [8,10]. This variety of species with similar responses suggests that the mechanisms of interdependence among 27 eating and drinking are preserved phylogenetically

Preening

The preening behavior is part of a set of stereotyped behaviors that are related to the body and are phylogenetically conserved maintenance, since being found in arthropods up in birds and mammals [16,17]. What differentiates this behavior between species is its function. While in arthropods has the primary function of care of legs and antennae, in mammals plays an important role, in addition to hygiene in the establishment of social relationships, such as tolerance to feed along with another individual, mutual assistance regarding the aggressive behavior of other groups [18,19]. Besides the functions mentioned, the wiper has been implicated as a marker of low levels of stress in primates and rodents, and birds [18,20-22].

Typical sleep behavior

The sleep behavior after the meal appears at the end of the BSS is a competitor behavior and food intake, or both can not happen at the same time. Like other components of the BSS, the relationship

***Corresponding author:** William Anderson Spudeit, Department of Pharmacy and Pharmaceutical Management- Fluminense Federal University, 24241-000, Niteroi, RJ, Brazil, Tel: 88040-900; E-mail: willian.spudeit5@gmail.com

Received August 22, 2014; **Accepted** September 22, 2014; **Published** September 25, 2014

Citation: Spudeit WA (2014) Behavioral Sequence of Satiety: A Comparative Approach between Birds and Mammals. J Phylogen Evolution Biol 2: 132. doi:10.4172/2329-9002.1000132

between sleep after a meal seems to be evolutionarily preserved. This is because its occurrence is reported in both invertebrate (C. elegans) as in vertebrates such as rodents, for example. In C. elegans, sleep after the meal is characterized by a total period of immobility after the consumption of food, and there is a relationship between sleep time with the nutritional value of the food [23]. Already in rodents, this postprandial sleep does not seem to be so dependent on the nutritional value but the amount of food ingested, as shown by the work of [24,25]. Pigeons Columba livia, which were refed after fasting for 96 hours, showed an increase of three times the duration of slow-wave sleep and the emergence of paradoxical sleep [26]. Such a relationship between eating and sleep was also observed in humans and appears to result from a combination involving stimulation of vagal afferent pathways, increased body temperature, glucose concentration, the more the hormones that are released by the gastric system, for example, cholecystokinin (CCK), which acts on the central nervous system areas involved in control of sleep [27-29].

Use of the Behavioral Satiety Sequence as Pharmacological Research Tool

With the advent of new technologies, especially linked to registration by means of cameras, it became possible refinement studies on the BSS to the point of being able to better observe the behaviors - how they are expressed and the links between them. What made possible the experiments calibration of BSS. Among them, the experiments involving pre-charging of feed offered to the animal before the start of observation, experiment involving the tampering of the palatability of the diet with added quinine or lithium that are used to help differentiate making part of the natural process of satiation eating interrupting the unpleasant taste because of nausea or because of tampering with rations. BSS calibration with the use of drugs as a tool to better understand the sequence of attributes, such as whether or not BSS is sensitive to changes in food intake, and perhaps most importantly, allow it was possible to discern what is really a satiety caused by a decrease in drug intake because of its side effects such as, nausea, dizziness, increased locomotion that are caused by certain drugs. Finally, the pharmacological experiments have shown the participation of some neurotramissores the control of satiety sequence, such as serotonin and NPY [1,2,30,31] this sense, the attributes of the BSS, as the onset, duration, frequency of feeding behavior and its microstructure, have been repeatedly used as safer hipofagiantes drug effects in rodents [2,7,32,33] pre-clinical model. In addition, measures of changes in the temporal and sequential between components of BSS relations can dissect functional interactions between the systems related to energy homeostasis, hidrossalino balance and sleep-wake states

In mammals, the methods used to cause SCS lay hold of prolonged food deprivation (24-48 hours) or systematic dietary restriction, followed by reintroduction of food. The intake and postprandial behavior caused by the fast switching are recorded separately into small segments (five minutes) in order to describe the temporal behavior of each route in detail over 30-40 minutes after meal. The effects of drugs applied before the restatement of food or test diets of varying palatability with different nutritional and sensory properties, are examined by comparing the resulting curves of different behaviors simultaneously in a qualitative way [2].

However the use of this model has been used in mammals in a way that may be affecting the results, for example when neglecting the drinking behavior as can be seen from the work [1,7]. The neglect of this behavior can lead to less reliable results of the composition of the sequence, since there is a relationship between the intake of food

Way to evaluate the data from the SCS in mammals

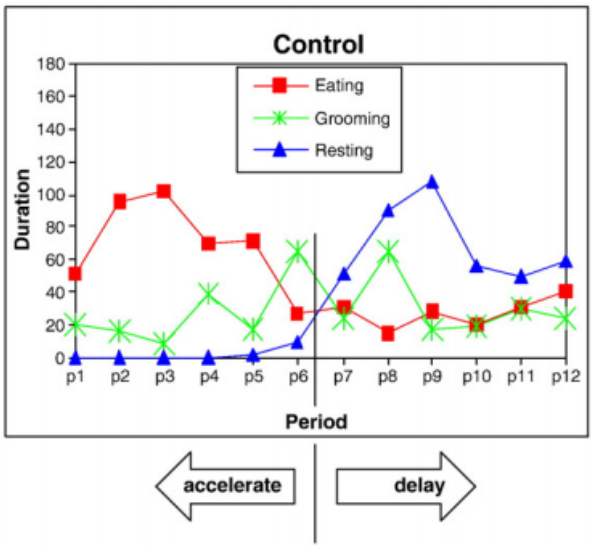

Figure 2: The behavioural satiety sequence (BSS) in undeprived adult male Lister hoodedrats tested for 1 h with palatable mash (n=10). Data shown are mean duration (s)scores for each of the three behaviours (eat, groom, rest) in each of 12 × 5 min timebinscomprising the 1 h test. As shown by the horizontal arrows, the vertical line(representing the transition from eating to resting i.e. behavioural satiety) wouldshift to the left (accelerate) with an anorectic agent and to the right (delay) with anappetite stimulant. Behavioural selectivity of treatment would be indicated bypreservation of the sequence despite acceleration or delay accessed RODGERS et al., 2010.

and drink so that the drink is part of the food intake as stated by [14]. Furthermore, it is worth noting that from the beginnings to the present day the results of experiments in which the employee is SCS are evaluated in a qualitative way down a line where it crosses a line to eat and sleep as we can see in Figure 2.

This form of evaluation is somewhat poor form and this can hide important data on the effects of the studied drugs, such as increased or decreased thirst or hunger.

Furthermore, it should be noted that although food deprivation is a common factor in the animal in its natural habitat, in experimental conditions it may introduce uncontrollable variables in the expression of the SCS, the impact of changing phenomenon in the post-ingestive sleep prandial. This fact occurs because food deprivation leads to modification of sleep patterns in both rodents as in birds. The difference between birds and rodents is that prolonged fasting in rodents increases wakefulness and reduces time spent in REM sleep, and increase the time spent on motor activities and to contribute to a substantial delay in circadian rhythms. Birds already in the starvation leads to increased as time spent sleeping, increased sleep time of light waves and paradoxical sleep [26,34].

Within this context, with the aim of improving the model of SCS and improve compression donated produced [35] SPUDEIT, 2013 proposed a model for studying the pigeons called SCS SM-BSS. The use of bird to study a model as studied in mammals was only possible because of works such as Takei et al. [36], Hoeller et al. [37] and Rial et al. [38] showing that the behaviors that are part of SCS are phylogenetically conserved.

News of the protocol SM-BSS

The first novelty presented by this new protocol was to reduce

the time that the animal needs to go without food so you can unleash the BSS. This reduction was 24 pm for 1h. That was enough to add a mixture of seeds (see for composition see [35]) that the animal likes to order the return of normal chow and water. This is an important finding because it avoids theeffects of prolonged fasting. This reduction in fasting time increased with this mix of seeds not only triggered the BSS as not married significant changes when compared with animals that were fasted for 24 hours as we can see by the Table 1 below.

	MS-BSS (n=6)	24-h Food deprivation (n=6)
Food Intake (g/100g bw)	3.65±0.64	3.99±1.17
Feeding (duration, s)	1601±335	818±349
Latency (s)	51±13	50±65
TTP (bins)	3(2,3)	2(1,3)
Water Intake (ml/100 g bw)	1.85±0.73	1.89±1.05
Drinking (duration, s)	35.88±18.97	43.74±24.99
Latency (s)	1342 (941, 1709)	822 (580, 3065)
TTP (bins)	17 (8,22)	5 (4,6)
Preening (duration, s)	229.16±97.81	608±772
Latency (s)	1989 (1152,2436)	986 (863,1025)
TTP (bins)	19 (17,21)	9.5 (7,12)
Sleep (duration, s)	1675±981	520±405
Latency (s)	2290 (1668,2916)	1854 (1263,2131)
TTP (bins)	18 (13,20)	10 (7,14)
IPI feeding/drinking (bins)	5 (2,20)	2.5 (1,4)
IPI feeding/preening (bins)	9 (8,9)	6.5 (5,10)
IPI feeding/sleep (bins)	15 (10,16)	8.5 (6,11)
ItS feeding/drinking (bins)	6,5 (3,9)	4 (3,4)
ItS feeding/preening (bins)	11 (9,12)	8.5 (7,9)
ItS feeding/sleep (bins)	10 (8, 13)	9.5 (7,14)

Table 1: Values of latency, inter-peak interval (IPI), time-to-peak (TTP) and intersections (ItS) are expressed as medians (minus 25% and plus 75%), while food/water intake and duration ofbehaviors as expressed as mean ± SEM.

Having such data note the importance the search for a palatable food that can add to food intake so that rodents can trigger BSS without the animal needs to undergo long periods of fasting.

Another innovation was the use of indexes that have made it possible to study the relationship between behaviors that are part of the sequence as well as determine precisely when the BSS and ends well know fact that the treatment accelerated or extended the sequence. Developed index were the peak time (TTP) which is the time it takes to reach each behavior at its maximum and is determined by the number of bins (bins- dividing the total time observed in interval of 4 minutes), the interval between peaks (IPI) shows that as the treatment may influence the distances between maximum durations, which is already an idea of stretching or acceleration of BSS and given by subtracting the values of the peak bins of eating for peak drinking, of eating for peak autolimpeza and peak to peak eat and sleep behaviors intersection (ItS), which demonstrates the exact time at which a sleep behavior becomes more important than another and thus determines the end of the BSS can be better visualized in the Figures 3 and 4 below Table 2.

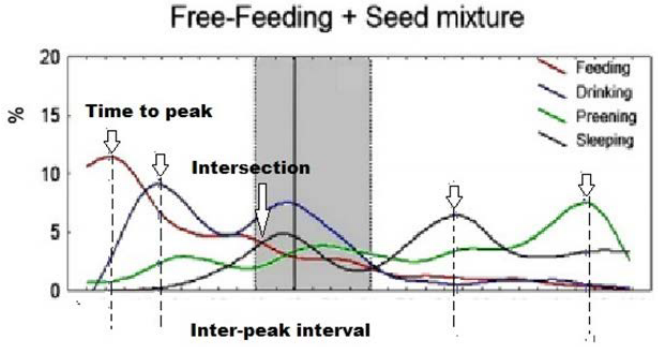

Figure 3: demonstration of the indices evaluated in the MS-BSS protocol.

It can be shown graphically as follows:

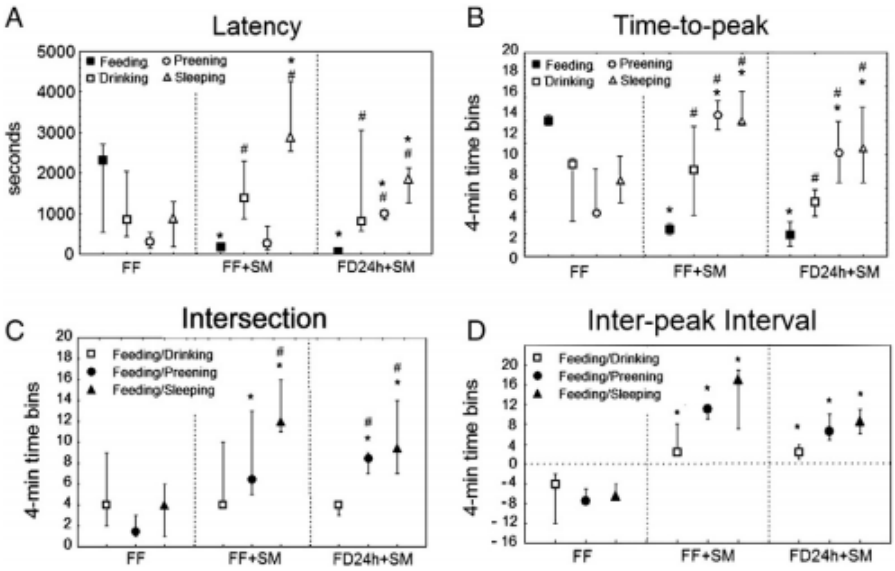

Figure 4 . Latency to thefirst occurrence (A), time-to-peak (B), intersection point (C), and inter-peak intervals (D) of ingestive and non-ingestive behaviors in free-feeding pigeons (FF) in freefeeding pigeons after food presentation to the seed mixture (FF + SM) and in 24-h-food deprived animals presented to the seed mixture and regular chow (FD24h + SM). Data wereexpressed as the median (symbols) plus the 75th percentile and minus the 25th percentile (whiskers). (*) pb0.05 compared to the FF data and (#) pb0.05 compared to the feedingscore in the same experimental condition.

	Free Feeding FF (n=6)	FF + Seed Mixture (n=6)	24-h deprivation (n=6)
Food intake (g/100g bw)			
1st hour	0.89 ± 034	2.88 ± 1.08[a,b]	3.99 ± 1.17[a]
2nd hour	0.75 ± 0.45	0.23 ± 0.20	0.23 ± 0.19
Feeding (duration s)			
1st hour	292 ± 142	1064 ± 75[a]	818 ± 349[a]
2nd hour	436 ± 369	118 ± 23[a]	50 ± 65[a]
Latency (s)	2321 (529,2710)[c]	185 (144,221)[a]	56 (39,61)[a]
TTP (bins)	12.5 (12,3)	2.5 (3,2)[a]	2 (1,3)[a]
Water intake (ml/100g bw)			
1st hour	1.11 ± 0.32	2.01 ± 1.31	1.89 ± 1.05
2nd hour	1.06 ± 1.08	0.46 ± 0.37	1.80 ± 1.00
Drinking (duration, s)			
1st hour	25.56 ± 8.46	48.31 ± 21.75[a]	43.74 ± 24.99[a]
2nd hour	17.06 ± 13.59	5.48 ± 5.05	24.66 ± 24.02
Latency (s)	863 (442,2053)	1387 (881,2306)	822 (580,3065)
TTP (bins)	8.50 (3.00, 9.00)	8.0 (4,12)	5 (4,6)
Preening (duration, s)			
1st hour	758 ± 219	311 ± 223	608 ± 772
2nd hour	715 ± 534	496 ± 369	374 ± 359
Latency (s)	304 (143,529)	251 (98, 694)	986 (863, 1025)[a]
TTP (bins)	4 (4,8)	13 (12, 14)[a]	9.5 (7, 12)[a]
Sleep			
1st hour	516 ± 125	276 ± 441	520 ± 405
2nd hour	470 ± 247	669 ± 599	1512 ± 807
Latency (s)	870 (184, 1281)	2880 (2533, 4560)	1854 (1263, 2131)[a]
TTP (bins)	7 (6, 1)	19.5 (16, 22)[a]	10 (7, 14)[a]
Exploratory			
1st hour	1908 ± 166	1783 ± 513	1733 ± 408
2nd hour	1845 ± 463	2062 ± 432	1415 ± 531
Alert Immobility			
1st hour	81 ± 53	0.0	26 ± 41
2nd hour	30 ± 57	0.0	95 ± 75
IPI feeding/drinking (bins)	=4 (=12, =2)	2.5 (2, 8)[a]	2.5 (1, 4)[a]
IPI feeding/preening (bins)	=7.5 (=8, =5)	11 (9, 12)[a]	6.5 (5, 10)[a]
IPI feeding/sleep(bins)	=6.5 (=7, =4)	17 (7, 19)[a]	8.5 (6, 11)[a]
ItS feeding/drinking (bins)	4 (2, 9)	4 (4, 10)	4 (3, 4)
ItS feeding/preening (bins)	1.5 (1, 3)	6.5 (5, 13)[a]	8.5 (7, 9)[a]
ItS feeding/sleep(bins)	4 (1, 6)	1(10, 16)[a]	9.5 (7, 14)[a]

Table 2: Ingestive and behavioural to fasting and palatable food in pegoins.

Conclusion

Exposed before all is worth pointing out that the model of BSS employee in mammals brought great discoveries to science, such as the discovery of peptide colicistoquinina as part of the control of food intake as well as some neurotramissores which also has an effect on the control of intake e.g. with serotonin. However we believe the adequacy of the models used in mammals to model MS-BSS can be valuable because it can demonstrate more accurate results and better discriminated.

Disclosure of Conflict of Interest Statement

All authors here state that they have no actual or potential conflict of interest including any financial, personal or other relationships with other people or organizations that could inappropriately influence the work here submitted

Role of the Funding Source

The funding agencies CNPq and FAPESC had no influence or role in study design, in the collection/ analysis/interpretation of the data, in the writing of the report, or in the decision to submit the paper for publication.

References

1. Rodgers RJ, Holch P, Tallett AJ (2010) Behavioural satiety sequence (BSS): separating wheat from chaff in the behavioural pharmacology of appetite. Pharmacol Biochem Behav 97: 3-14.
2. Halford JC, Wanninayake SC, Blundell JE (1998) Behavioral satiety sequence (BSS) for the diagnosis of drug action on food intake. Pharmacol Biochem Behav 61: 159-168.
3. Barnett SA (1956) Behaviour components in the feeding of wild and laboratory rats. Behaviour 9: 24-44
4. Grant EC, Mackintosh JH (1963) A comparison of social postures of some common laboratoryrodents. Behaviour 21: 246-251
5. Scott JP (1966) Agonistic behavior of mice and rats: a review. Am Zool 6: 683-701.
6. BOLLES RC (1960) Grooming behavior in the rat. J Comp Physiol Psychol 53: 306-310.
7. Antin J, Gibbs J, Holt J, Young RC, Smith GP (1975) Cholecystokinin elicits the complete behavioral sequence of satiety in rats. J Comp Physiol Psychol 89: 784-790.
8. McFarland DJ (1965) The effect of hunger on thirst motivated behaviour in the Barbary dove. Anim Behav 13: 286-292.
9. Fitzsimons TJ, Le Magnen J (1969) Eating as a regulatory control of drinking in the rat. J Comp Physiol Psychol 67: 273-283.
10. Zeigler HP, Green HL, Lehrer R (1971) Patterns of feeding behavior in the pigeon. J Comp Physiol Psychol 76: 468-477.
11. Normile HJ, Barraco RA (1984) Relation Between Food and Water Intake in the Pigeon (Columba livia). Journal of Comparative Psychology V.98: 76-90.
12. de Castro JM (1988) A microregulatory analysis of spontaneous fluid intake by humans: evidence that the amount of liquid ingested and its timing is mainly governed by feeding. Physiol Behav 43: 705-714.
13. Morgan CA, Emmans GC, Tolkamp BJ, Kyriazakis I (2000) Analysis of the feeding behavior of pigs using different models. Physiol Behav 68: 395-403.
14. Zorrilla EP, Inoue K, Fekete EM, Tabarin A, Valdez GR, et al. (2005) Measuring meals: structure of prandial food and water intake of rats. Am J Physiol Regul Integr Comp Physiol 288: R1450-1467.
15. Raubenheimer D, Guide J (1994) Hunger-thirst Interactions in the Locust, Locustamigratoria. J fnsecf Physiol v. 40: 631-639.
16. Radford AN, Plessis MAD (2006) Dual function of allopreening in the cooperatively breeding green woodhoopoe, Phoeniculuspurpureus. Behav Ecol Socio biol v.61: 221-230.
17. Hosoda N, Gorb SN (2011) Friction force reduction triggers feet grooming behaviour in beetles. Proc Biol Sci 278: 1748-1752.
18. Spruijt BM, van Hooff JA, Gispen WH (1992) Ethology and neurobiology of grooming behavior. Physiol Rev 72: 825-852.
19. Ventura R, Majolo B, Koyama NF, Hardie S, Schino G (2006) Reciprocation and interchange in wild Japanese macaques: grooming, cofeeding, and agonistic support. Am J Primatol 68: 1138-1149.
20. Aureli F, Yates K (2010) Distress prevention by grooming others in crested black macaques. Biol Lett 6: 27-29.
21. Shutt K, MacLarnon A, Heistermann M, Semple S (2007) Grooming in Barbary macaques: better to give than to receive? Biol Lett 3: 231-233.
22. Nin MS, Couto-Pereira NS, Souza MF, Azeredo LA, Ferri MK, et al. (2012) Anxiolytic effect of clonazepam in female rats: grooming microstructure and elevated plus maze tests. Eur J Pharmacol 684: 95-101.

23. You YJ, Kim J, Raizen DM, Avery L (2008) Insulin, cGMP, and TGF-beta signals regulate food intake and quiescence in C. elegans: a model for satiety. Cell Metab 7: 249-257.

24. Danguir J, Nicolaidis S (1985) Feeding, metabolism and sleep: Peripheral and central mechanisms of their interaction. In: McGinty DJ., et al., Brain mechanisms of sleep. Raven Press, New York, USA, pp. 321-340.

25. De Saint Hilaire Z, Nicolaïdis S (1992) Enhancement of slow wave sleep parallel to the satiating effect of acidic fibroblast growth factor in rats. Brain Res Bull 29: 525-528.

26. Dario AJ, Lopes PR, Freitas CG, Paschoalini MA, Marino-Neto J (1996) Electrographic patterns of postprandial sleep after food deprivation or intraventricular adrenaline injections in pigeons. Brain Res Bull 39: 249-254.

27. Zammit GK, Ackerman SH, Shindledecker R, Fauci M, Smith GP (1992) Postprandial sleep and thermogenesis in normal men. Physiol Behav 52: 251-259.

28. Orr WC, Shadid G, Harnish MJ, Elsenbruch S (1997) Meal composition and its effect on postprandial sleepiness. Physiol Behav 62: 709-712.

29. Wells AS, Read NW, Uvnas-Moberg K, Alster P (1997) Influences of fat and carbohydrate on postprandial sleepiness, mood, and hormones. Physiol Behav 61: 679-686.

30. Blundell JE, Rogers PJ, Hill AJ (1985) Behavioural structure and mechanisms of anorexia: calibration of natural and abnormal inhibition of eating. Brain Res Bull 15: 371-376.

31. López-Alonso VE, Mancilla-Díaz JM, Rito-Domingo M, González-Hernández B, Escartín-Pérez RE (2007) The effects of 5-HT1A and 5-HT2C receptor agonists on behavioral satiety sequence in rats. Neurosci Lett 416: 285-288.

32. Blundell JE (1986) Serotonin manipulations and the structure of feeding behaviour. Appetite 7 Suppl: 39-56.

33. Gibbs J, Smith GP (1982) Gut peptides and food in the gut produce similar satiety effects. Peptides 3: 553-557.

34. Borbély AA (1977) Sleep in the rat during food deprivation and subsequent restitution of food. Brain Res 124: 457-471.

35. Spudeit WA, Sulzbach NS, Bittencourt Mde A, Duarte AM, Liang H, et al. (2013) The behavioral satiety sequence in pigeons (Columba livia). Description and development of a method for quantitative analysis. Physiol Behav 122: 62-71.

36. Takei Y (2000) Comparative physiology of body fluid regulation in vertebrates with special reference to thirst regulation. Jpn J Physiol 50: 171-186.

37. Hoeller AA, dos Santos TS, Bruxel RR, Dallazen AR, Silva HTA, et al. (2013) Serotonergic control of ingestive and post-ingestive behaviors in pigeons (Columba livia): The role of 5-HT1A receptor-mediated central mechanisms. Behavioural Brain Research 236: 118– 130.

38. Rial RV, Akaârir M, Gamundí A, Nicolau C, Garau C, et al. (2010) Evolution of wakefulness, sleep and hibernation: from reptiles to mammals. Neurosci Biobehav Rev 34: 1144-1160.