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The effects of REM sleep deprivation on the metabolic rate of the rat

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the rat**

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San Jose State University, 1989

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THE EFFECTS OF REM SLEEP DEPRIVATION
ON THE METABOLIC RATE OF THE RAT

A Thesis
Presented to
the Faculty of the Department of Psychology
San Jose State University

In Partial Fulfillment of the
Requirements for the Degree
Master of Arts

by
Jacqueline Puentes
May, 1989

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I would like to dedicate this thesis to my family for their continued love, support and encouragement.

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The Effects of REM Sleep Deprivation on the Metabolic Rate of the Rat

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Running head: RSD AND METABOLIC RATE IN RATS

Footnotes

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Abstract

The effects of REM Sleep Deprivation (RSD) on the estimated metabolic rate of rats both during deprivation and recovery were measured. Eighteen male Sprague-Dawley rats (45 days old) were randomly assigned to one of the following groups: 4-day RSD, wet control (WC) and dry control (DC). Metabolic rate was estimated from oxygen consumption and scaled to body weight. RSD was achieved by placing the rats on small (6.5 cm) platforms surrounded by water. The WC condition utilized large (16.5 cm) platforms surrounded by water, and the DC rats were on large platforms surrounded by wood shavings. Oxygen consumption was measured for each animal for 15 minutes between 1130 and 1330 hours on 10 consecutive days (2 baseline days, 4 treatment days, 4 recovery days). A 3 (treatments) by 10 (days) ANOVA with repeated measures on days was computed. Results show a significant increase in metabolic rate for both RSD and WC groups ($p < .005$). This effect returned to baseline during recovery.

The Effects of REM Sleep Deprivation on the Metabolic Rate of the Rat

One of the first hypotheses that was advanced to explain the effects of REM sleep (RS) on behavior was Dement's notion (Dement, 1965) that REM sleep deprivation (RSD) increased drive level. Subsequently, Vogel (1979) detailed the literature which supported this hypothesis and asserted that RS was the mechanism that controlled level of energy or drive. He stated that "...the theory holds that prevention of periodic disinhibition and neural discharge during RSD increases the waking excitability of a drive's neural substrate and thereby increases the waking drive. Inversely, the theory holds that disinhibition and neural discharge during RS reduce the waking excitability of the drive's neural substrate and thereby reduce the waking drive." (p. 235) Vogel argued that RSD "...increases such drive motivated behaviors as sex, aggression, pleasure seeking, food seeking and grooming." In testing this hypothesis, the RS drive-motivated behavioral relationship that has received the greatest attention is the relationship between RS and eating. Several studies have reported that RSD increases food intake in rats (e.g., Elomaa & Johansson, 1980; Kushida, Bergmann, Everson, Winter & Rechtschaffen, 1984). Elomaa and Johansson (1980) observed the eating behavior of male rats. Using a five-day baseline period followed by five days of RSD they found that the RSD rats ate

smaller meals (i.e., < 5 g) but their overall food intake (FI) increased by 32% (i.e., went from 673 g at baseline to a total of 891 g after treatment). However, these investigators did not measure FI during the recovery from RSD, and did not have an explanation for the increase in FI in the RSD rats. In separate studies, Bergmann (Bergmann, Kushida, Hennessey, Winter & Rechtschaffen, 1984) and Kushida et al (1984) found that total food consumption in RSD rats was 186.2% of baseline compared to 127.5% for their controls. In this study, the method used for RSD involved placing the animal on a horizontal disk above water, which was divided in half with a "yoked" control on one side and the RSD animal on the other. At the onset of RS in the RSD animal the disk would rotate forcing the animal to awaken and move to avoid falling into the water. They further reported that RSD rats body weight declined 21% more than controls. RSD rats also showed an increase in FI of 65.6% more compared to controls. In a more recent paper, Kushida, Bergmann, Fang, Leitch, Obermeyer, Refetoff, Schoeller and Rechtschaffen (1985) attempted to explain the increased FI of RSD rats by estimating that the maximal daily energy expenditure of the RSD rats was 3.2 times baseline compared to a 1.9 fold increase for the control rats. Here it should be noted that these estimates were not derived from direct measurements of energy expenditure. Rather their assertion, which is relevant to the RSD-drive hypothesis, was

a conclusion drawn from the differences in FI between RSD rats and controls that they observed.

Subsequently, in an attempt to validate the assumption made by Kushida and his colleagues in their 1985 paper, Martinez and Mendieta (1986) directly measured the relationship between RSD and metabolic rate and they reported a 25% increase in metabolic rate (as measured by oxygen consumption) in the RSD rats, as compared to their untreated controls. While these data are provocative and appear to support the general RS - Energy Hypothesis, because of certain imperfections in the apparatus that they used, their results may be confounded by possible stress effects and thus must be viewed with caution. To explain, the apparatus they used to measure oxygen consumption required placing their animals inside small chambers and then submersing these chambers under water. Thus, it may well be the case that this technique acted as a stressor by immobilizing these animals in a highly novel environment. This is important because as Hicks and Sawrey (1978) demonstrated, RSD animals show substantially higher levels of susceptibility to immobilization-induced stress than untreated control animals.

The specific purpose of this study was to replicate and extend the Martinez and Mendieta (1986) study, using an oxygen analyzer that was free of the imperfections in their apparatus that confounded interpretation of their results. In doing this, the effects

of RSD on the estimated metabolic rate of groups of rats both during deprivation and recovery was studied. Consistent with the data sighted above, it was predicted that relative to the untreated controls, RSD would increase oxygen consumption.

Methods

Animals Eighteen male Sprague-Dawley rats were used in this study. Rats were 35 days old when obtained from the supplier and individually housed in specially designed cages with woodshavings and a large platform. After ten days the rats were assigned to one of the three treatment conditions: a dry control (DC) group, a wet control (WC) group, and a REM sleep deprivation (RSD) group. Three animals were run at one time with one in each condition and six groups total.

REM Sleep Deprivation RSD was achieved using the water tank procedure (Hicks & Moore, 1979). Throughout the experiment, all of the rats were housed in 18.9 liter buckets with food and water available ad lib from a feeder and water bottle positioned through the side of the bucket. During the treatment period these buckets were used for RSD and to achieve the necessary controls.

During the last three days of the adaptation period oxygen consumption baseline measures for all rats were taken. The adaptation period was followed by a four-day treatment period. All the animals continued to be housed in their 18.9 liter bucket but

with the changes required by the treatments that are outlined below. During the treatment period the animals assigned to the RSD group were placed on small (diameter=6.5 cm) platforms surrounded by water. This procedure selectively deprives animals of RS because when animals enter this stage of sleep they lose muscle tone causing the animals on the small platforms to make more frequent contact with the water which arouses them and causes them to lose RS.

During the treatment period, animals in the WC group were placed on large (diameter=16.5 cm) platforms surrounded by water. These platforms were designed to minimize contact with the water at the onset of RS and, thus the loss of RS. Therefore, the WC group served as a relatively undeprived control for exposure to the wet environment. (Throughout the treatment period, the water level for the WC and RSD conditions was maintained at one-cm below the top surface of the platform.)

The DC group remained in the same housing conditions with a large platform, during the four-day treatment period. The original buckets were used for all conditions. The DC group was served as an untreated baseline condition and as a control for "systematic" changes that might have occurred throughout the study. After the treatment period all the animals were returned to the original housing conditions for a four-day recovery period.

As a check on the possibility of differential stress levels induced, the animals were weighed on a daily basis between 1130 and 1330 hours throughout the entire period of this experiment.

Metabolic Rate Metabolic rate was estimated by measuring oxygen consumption. To do this, air was drawn through the animal chamber using the house vacuum. This chamber, which was much larger than those used by Martinez and Mendieta (1986) in their study, was a plexiglass cylinder measuring 30.48 cm in length, and 35.56 cm in circumference. Moisture was extracted from the air as it passed through Drierite desiccant before it reached a Mass Flow Meter (Model FM-360, Tylan), the flow meter was then connected to an output box (RO-32, Tylan). The flow meter was used to measure a constant rate of approximately 1 l./min. of air through the system. Using a "T" fitting, a sample of the air (100 ml/min) was drawn through an Oxygen Sensor (N-22M Applied Electrochemistry, AMETEK) connected to an Oxygen Analyzer (S-3A/1, Applied Electrochemistry, AMETEK) which read out the percent of oxygen in the air. The 100 ml/min of sample air was controlled using a Flow Control (R-1, Applied Electrochemistry, AMETEK). The pressure of the air going to the O₂ sensor was also measured using a manometer to ensure consistency. During measurement analog outputs from the O₂ sensor and the flow meter were digitized and stored on an Apple IIe computer. The actual oxygen consumption ($\dot{V}O_2$) of each animal was calculated using standard equations (Withers, 1977) and was similar

to the procedures used by Phillips and Berger (1986a, b) to measure oxygen consumption during shallow torpor in pigeons.

The system took 1-sec samples and printed out the mean for every 15-sec. Each animal was tested for a 15-min period which was divided into 3-min segments. The first three minutes of each test were disregarded and looked upon only as adapting time to the chamber. An overall mean for the last 12-min was used as the animals oxygen consumption score for a given test.

Procedure When they were received from the supplier, the animals were acclimated to the lab for ten days prior to the beginning of the treatment period and were approximately 45-days old when the treatments were initiated. Throughout the entire experimental period each rat was handled for a 10-min period for the first seven days of the adaptation period. During this 10-min period rats were weighed, handled, and placed in the metabolic chamber (two-min for the first four days and five-min for days five, six and seven). The last three days of the adaptation period (days 8-10) were used to obtain the baseline $\dot{V}O_2$ measures for each animal. To do this, an animal was placed in the oxygen consumption apparatus for a 15-min period. Day 8 for one of the animals was lost and so only days 9 and 10 were used as baseline in the results. Animals were assigned to one of the three treatment conditions after testing on the last baseline day. Following their four-day exposure to either the WC,

RSD or DC treatments all rats were returned to the original housing conditions for a four-day recovery period. Throughout treatment and recovery $\dot{V}O_2$ was measured every day for 15 minutes for each animal between the hours of 1130 -1330. The colony/test room temperature was maintained at 22 ± 2 °C and on a 12:12 hour light:dark cycle with the lights on at 0800 hrs (P.S.T.).

RESULTS AND DISCUSSION

As a rough check on the possibility of differential levels of treatment-induced stress, the weights of the animals, which are summarized in Table 1 for each group for each day of the $\dot{V}O_2$ measurement period, were analyzed by computing a two factor ANOVA with repeated measures in the second factor (days). The main effect for treatments was not significant, $[F(2,15)=2.10]$.

TABLE 1

Means and Standard Deviations for Body Weights for Each Day for Each Group

Group		Baseline		Treatment				Recovery			
		1	2	3	4	5	6	7	8	9	10
DC	X	204.47	210.83	218.82	225.42	232.00	241.38	245.05	253.55	257.93	265.47
	SD	11.06	11.70	10.19	12.02	11.28	12.04	12.23	12.22	11.42	11.47
WC	X	201.67	207.58	209.77	214.33	220.85	226.70	230.90	237.65	242.85	250.43
	SD	14.57	15.48	15.76	14.20	13.62	15.07	14.02	14.45	15.60	16.15
RSD	X	209.68	214.52	214.08	209.23	212.67	212.58	215.43	227.68	232.90	242.12
	SD	11.81	12.15	16.62	17.18	19.45	16.56	19.12	17.60	16.51	16.12

The $\dot{V}O_2$ means and standard deviations for each treatment group for each measurement day are listed in Table 2 and presented graphically in Figure 1.

TABLE 2

Means and Standard Deviations for Oxygen Consumption (ml O_2 /g*hr) for Each Day for Each Group

Group		Baseline		Treatment				Recovery			
		1	2	3	4	5	6	7	8	9	10
DC	$\bar{X}$	2.28	2.17	2.23	2.18	2.16	2.14	2.14	1.98	2.04	1.98
	SD	.08	.17	.19	.17	.14	.19	.17	.06	.11	.10
WC	$\bar{X}$	2.20	2.13	2.41	2.52	2.54	2.50	2.29	2.14	2.03	2.03
	SD	.23	.21	.12	.27	.21	.16	.21	.07	.26	.19
RSD	$\bar{X}$	2.27	2.16	2.67	2.61	2.62	2.70	2.30	2.02	2.11	1.98
	SD	.20	.20	.20	.27	.27	.12	.24	.99	.14	.13

FIGURE 1

Weight Specific Metabolic Rate for Each Group for Each Day

The data summarized in Table 2 were first analyzed by computing a three (treatments) by ten (days) factor ANOVA with repeated measures in the second factor (days). The results of this ANOVA are summarized in Table 3.

Table 3

The Summary of the ANOVA Computed to Analyze Oxygen Consumption
(ml O₂/g*hr) Data

<u>Source</u>	<u>df</u>	<u>SS</u>	<u>MSS</u>	<u>F</u>	<u>P</u>
Btwn Subj	17	2.84			
A(Treatments)	2	1.49	0.75	8.38	<.005
Subj w/ Grp	15	1.34	0.09		
W/in Subj	162	10.84			
B(Days)	9	5.50	0.61	22.62	<.001
AB	18	1.69	0.09	3.47	<.001
BXSubjwGroups	135	3.65	0.03		

In agreement with the general hypothesis that RSD increases energy expenditure and Martinez and Mendieta's (1986) observation that RSD increases metabolic rate, these data seem to indicate that RSD increases $\dot{V}O_2$ consumption significantly. As is shown in Table 3, I computed a significant main effect for treatments [$F(2,15)=8.38$, $p < .005$] and a significant treatments by days interaction [$F(18,135)=3.47$, $p < .001$].

To elaborate the significant main effect for treatments and the significant treatments by days interaction, post hoc analyses for each day were computed between each treatment group using the Newman-Keuls statistical procedure. As is shown in Table 4, the results of these analyses revealed that on the first treatment day, the $\dot{V}O_2$ increases that are apparent in Figure 1 for the RSD and WC groups, were both significantly higher than the untreated DC group. Further, this was true for each of the subsequent days of the treatment period. However, the differences between the RSD and WC groups were not significant during the treatment period. Except for one recovery day (i.e., Day 8), the two baseline days and the other three recovery days were not significant (see Table 4).

TABLE 4

The Levels of Significance Computed for Each Post hoc Analyses for $\dot{V}O_2$

Group	Baseline		Treatment				Recovery			
	1	2	3	4	5	6	7	8	9	10
DCvWC	N.S.	N.S.	0.05	0.05	0.01	0.01	N.S.	0.01	N.S.	N.S.
WCvRSD	N.S.	N.S.	0.1	N.S.	N.S.	0.05	N.S.	0.05	N.S.	N.S.
RSDvDC	N.S.	N.S.	0.01	0.05	0.01	0.01	N.S.	N.S.	N.S.	N.S.

There are several possible reasons for the lack of difference between the RSD and WC groups during the treatment period and these are discussed in the following paragraphs.

First, as was mentioned, both the RSD and WC groups require that animals be housed on platforms surrounded by water. While the platform diameters differ, both of these treatments cause loss of REM sleep that is greater in the RSD group. Furthermore, even though they are maintained in the same chambers throughout, the addition of water creates a novel environment that is typically associated with the so-called "first-night effect" (i.e., suppression of REM during the first night and possibly the second night when sleeping in a novel environment). Thus, it may be the case that loss of REM sleep was similar for both the RSD and WC groups. Since I did not include the electrophysiological measurement in the design of this study this possibility cannot be verified. However, as is shown in Table 5, it should be noted that Martinez and Mendieta (1986) reported a similar pattern of metabolic rate changes during their treatment period but the 4-day RSD group was significantly higher than the WC group.

Table 5

$\dot{V}O_2$ Differences Between Baseline Days and Subsequent Treatment and Recovery Days as Reported by Martinez and Mendieta (1986)

Group	Treatment Days				Recovery Days			
	1	2	3	4	5	6	7	8
4d RSD	13.05	20.85	24.85	13.45	4.95	-22.55	-27.35	18.35
2d RSD	6.80	6.90	2.20	5.00	4.60	-29.70	-29.20	26.30
WC	2.05	2.35	-0.75	-9.45	-10.95	-34.15	-34.05	29.65
DC	6.85	-12.15	-14.55	-19.05	-23.55	-40.55	-41.55	33.55

A second possibility is, that like the procedure used by Martinez and Mendieta to measure the metabolic rate, my procedure for measuring $\dot{V}O_2$ may have been stressful and interacted with RSD to obscure any differences between the RSD and the WC groups during treatment. While this is a possibility, it seems an unlikely one for these reasons. The main effect for treatments on the weights of these animals was not significant and by inspecting

Table 1, it can be seen that during the treatment period, the RSD and WC groups showed patterns of weight change that are inconsistent with comparable levels of stress for these groups. In addition, by inspecting the $\dot{V}O_2$ means in Table 2, it can be seen that the animals in all groups did not show any signs of an adaptation effect from the baseline period to the recovery period as was the case in the Martinez and Mendieta study (see Table 5). Thus, I feel that the increases in the $\dot{V}O_2$ means during the treatment period cannot be attributed to differential stress but rather are a consequence of loss of REM and/or exposure to the treatment environments.

The final possibility is that lack of difference between the RSD and WC groups and the significant difference between these groups and the DC group maybe associated, at least in part, with the unique features of their treatment environment. To explain, both the RSD and WC groups were exposed to water during the four-day treatment period, and exposure to water may increase heat loss, which in turn, may increase metabolic rate. In this regard, Roussel and Bittel (1979) found that after depilating rats and measuring oxygen consumption, their $\dot{V}O_2$ increased compared to normal animals. Although the depilated animals were not subjected to water, as mine were, both groups may have lost use of their insulation. That is, animals in water lose heat quicker because water is a good conductor of heat, and when the animals in Roussel

and Bittel's study lost their fur, their ability to retain heat was diminished. Thus both the RSD and WC groups have needed to increase energy consumption to remain warm. This possibility should be considered in future research.

Another limitation of this study that may have affected its outcome is that the animals I used were tested for a 15-min period each day at the same time during the middle of the light cycle. Since rats are nocturnal, these animals are typically much less active during the light period. Therefore, the time of day that the tests were conducted may have effected the oxygen consumption values that were derived from these animals. One can only speculate that had these animals been tested at a different time of day, for a longer period, the results may have been different. Clearly, further research is needed to elaborate this possibility.

Conclusions: When these data are considered together with those provided by Martinez and Mendieta, it seems fair to conclude that reductions in RS act to increase metabolic rates of rats. However, because the method used to deprive rats of RS in both of these studies may have introduced a differential heat loss in the RSD animals it is not prudent to argue that RSD was the only factor contributing to the changes in metabolic rate during the treatment period that were observed in both of these studies. Before a clear assessment of the relationship between RS and metabolic rate can

be made, research which takes into account temperature changes and/or uses a "dry" method of RSD will be required. Further, this research should be designed to measure metabolic rate over extended periods of time.

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