

# **Physiological Effects of Sleep Deprivation on Acute Stress Reactivity**

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## **Abstract**

Sleep deprivation affects a large population of students on college campuses and is associated with heightened sympathetic nervous system arousal. The aim of this study is to identify any differences that may exist between various physiological responses to acute stress based on individual levels of sleep deprivation. Collectively, it was hypothesized that sleep-deprived individuals would experience a greater increase in heart rate, blood pressure, and skin conductance when exposed to an acute stressful stimulus than those who were non-sleep-deprived. Stress response was assessed by changes in heart rate, blood pressure, and skin conductance in response to an acoustic stimulus. Individuals were asked to listen to a 3-minute sound recording of soothing music, during which physiological measurements were taken. The results of this study indicated no statistically significant differences in physiological responses between those who were sleep-deprived and non-sleep-deprived, with the exception of differences in mean arterial pressure. The difference in mean arterial pressure was significant only with respect to those who indicated having obtained “significantly less” and “significantly more” sleep (sleep-deprived MAP:  $\bar{x}=4.33$  vs. non-sleep-deprived MAP:  $\bar{x}=-15.84\pm 7.30$ ) the night before the study, compared to a typical night’s sleep. We were unable to find support for our hypothesis based on these results. The lack of significant findings is likely reflective of the paucity of severely sleep-deprived participants; thus, our findings may be better understood in the context of mild to moderate sleep deprivation. We concluded that further research needs to be conducted to properly identify scientific definitions for sleep deprivation and quality of sleep.

## Introduction

Sleep deprivation on college campuses is incredibly common, with research indicating that as many as 60% of college students are regularly sleep-deprived (Lund *et al.*, 2010). The importance of a good night sleep cannot be underestimated, as sleep-deprived college students perform significantly worse on tests of cognitive functioning compared to their non-sleep-deprived peers (Pilcher & Walters, 1997). In addition to these deficits, the psychological effects of sleep deprivation are well documented (Bos & Macedo, 2019). Chronic sleep deprivation accurately predicts the onset of a wide array of psychological disorders, including anxiety and depression (Chu *et al.*, 2019). Physiological consequences of sleep deprivation are also present, ranging from immune dysfunction to cardiovascular disease (Irwin *et al.*, 1999).

Acute sleep deprivation is associated with hyperactivation of the sympathetic nervous system and hypoactivation of the parasympathetic nervous system (Zong *et al.*, 2005). This is evident in the fact that sleep deprivation is positively correlated with hyperactivation of the hypothalamic-pituitary-adrenal (HPA) axis (Meerlo *et al.*, 2002). Studies of both humans and rats have shown elevated resting levels of cortisol and adrenocorticotropic hormone in sleep-deprived subjects (Meerlo *et al.*, 2002 & Minkel *et al.*, 2014). Increased sympathetic nervous system arousal brought on by sleep deprivation has also been shown to increase next-day blood pressure levels (Tochikubo *et al.*, 1996). Finally, hyperactivity of the HPA axis can lead to increased nocturnal catecholamine levels, which is correlated with greater incidence of cardiovascular disease (Irwin *et al.*, 1999). Considering the short and long-term health deficits linked to sleep deprivation, further study is clearly warranted in this arena.

Although there is a plethora of research on chronic conditions associated with sleep deprivation, there is relatively little research examining the effects of sleep deprivation on the

severity of the acute physiological stress response (Meerlo *et al.*, 2008). One study that attempted to examine this question concluded that sleep deprivation's ability to trigger the HPA axis resulted in an increased cortisol response to a stressful task involving a public speaking activity (Minkel *et al.*, 2014). Despite the promising results of this research, we have been unable to locate a single study that directly examines the effects of human sleep deprivation on physiological reactivity to an acute stressor that is not performance or task-based.

In this study, we investigated how severity of sleep deprivation affects physiological reactivity to an acute auditory stimulus. We hypothesized that subjects who were sleep-deprived would exhibit greater increases in heart rate, blood pressure, and skin conductance upon exposure to the stressor, compared to subjects who were not sleep-deprived. Blood pressure and heart rate were chosen as physiological dependent variables because they have been shown to change in response to stimulation of the HPA axis. (Nogueira-Ferreira *et. al*, 2018). Skin conductance was chosen as the third physiological dependent variable because acute stress stimulates the autonomic nervous system, which in turn changes conductivity at the skin surface (Novak, 2019). In order to determine if sleep deprivation significantly affected the acute physiological stress response, we compared the peak-to-peak changes in skin conductance ( $\mu\text{S}$ ), heart rate (BPM), and blood pressure (mmHg) of sleep-deprived subjects, compared to non-sleep-deprived subjects, following brief exposure to an unexpected auditory stressor.

## **Materials**

In this experiment, we measured physiological changes in blood pressure (BP), heart rate (HR), and skin conductance, also known as electrodermal activity (EDA), using three different measurement devices. An Omron 10 Series+ digital, automatic BP monitor was used to measure

BP on a standard systolic/diastolic scale (Model: BP791IT, Biopac Systems, Inc. Goleta, CA). HR was measured using a pulse oximeter device to record pulse in beats per minute (Model: 9843, Biopac Systems, Inc. Goleta, CA). Biopac Student Laboratory electrodermal activity (BSL EDA) finger electrodes (Model: SS3LA, Biopac Systems, Inc. Goleta, CA) were used to measure peak-to-peak changes in EDA in microsiemens ( $\mu\text{S}$ ). Data recording and analysis were conducted using the Biopac Student Lab System (BSL 4 software, MP36), in addition to the Biopac Systems, Inc. Reference Manual (ISO 9001:2008, Biopac Systems, Inc.) for equipment setup and calibration. A short audio clip containing soothing, instrumental music paired with a stressful audio stimulus was created (MixPad by NCH Software, Clips: “Reflections on the Water”, volume -9 dB; “Ambience Forest 6”, volume -15 dB; “Buzzer 2”, volume 12 dB) and presented to each participant. The use of soothing music was presented through a Bose over-the-ear headset (Model: 315351-0010) as a distraction for the stressful audio stimulus and the three physiological measurements being collected.

## **Methods**

### *Participants*

50 Participants, ages 20-40 were recruited on a voluntary basis from the University of Wisconsin-Madison Physiology 435 class. Physiological measurements were collected at the UW-Madison Medical Sciences Center in a private, controlled environment. A consent form was signed by all participants, outlining the confidentiality of each individual’s participation, the time required for the experiment (approximately 15 minutes), the physical and emotional risks associated with participation, and contact information for any questions or concerns.

## *Procedure*

The criteria for participation in this study included the willingness and ability to consent, as well as current enrollment as an undergraduate in the University of Wisconsin-Madison's Physiology 435 class. For safety reasons, individuals with underlying cardiovascular conditions were excluded from participating in the experiment. After consent was obtained, a short survey was administered to the participants regarding demographics, sleep habits, and presence of stress-related medical conditions (Appendix A). Exclusion criteria of participants was outlined in this survey. Prior to testing, the Biopac System was prepped with the BSL EDA finger electrodes attached to channel 1 of the Student Lab System. "Electrodermal Activity" (EDA, 0-35 Hz) was selected for EDA ( $\mu\text{S}$ ). An automated blood pressure cuff was placed on the left upper arm of the participant. The pulse oximeter probe was placed on the participant's left index finger. The BSL finger electrodes were strapped to the participant's right index and middle fingers after applying electrode gel. Calibration procedures were conducted for the SLD EDA finger electrodes according to the Biopac Systems Inc. Reference Manual. Over-the-ear headphones were also placed on each participant. Experimentation was conducted in a private room to ensure consistency, as well as ensuring that there were no external visual or auditory stimuli that would interfere with the variables being tested. The participant was asked to sit with both feet flat on the floor and both hands face down on a desk with their eyes closed, facing a wall. Data collection began shortly before the start of an audio clip that the participant was asked to listen to for the duration of the experiment. The experimental timeline included one set of baseline measurements taken two minutes before the music started playing (Figure 1). Measurements were recorded again, 45 seconds after the start of the music. 1 minute and 39 seconds after the start of the audio clip, the stressful auditory stimulus (a loud car horn) was played at -16 dB

through the headphones, followed immediately by an additional set of measurements. The audio clip was played for a total duration of 3 minutes and 2 seconds. Upon completion of the experiment, participants were assisted by the investigators to remove all testing equipment.

### *Data Analysis*

HR, BP, and EDA were recorded three times during the experiment: at baseline, 45 seconds after the start of the soothing music, and directly following the stressful stimulus. Differences were calculated between HR, BP, and EDA for each participant at baseline, compared to those recorded directly after the stressful stimulus. These differences in physiological responses served as the primary source of data for statistical analysis. The second set of measurements, taken during the music and before the stressful stimulus, was ignored, as the purpose of this set of measurements was purely deceptive. Using the VassarStats program, four analyses for difference of mean were conducted on each of the three dependent variables, measured at baseline and directly following the stressful stimulus. For each physiological variable being tested, we ran two separate two-tailed t-tests, one 3-sample ANVOA analysis, and one 5-sample ANOVA analysis to determine if significant differences were present in the physiological variables based on levels of sleep deprivation. For the first t-test, participants were considered sleep-deprived if they indicated feeling subjectively sleep-deprived on the pre-experiment survey. For the second t-test, participants were considered sleep-deprived based on their Likert scale rating; participants who indicated receiving “significantly less than normal” or “less than normal” sleep were considered sleep-deprived, while participants who indicated receiving “same,” “more than normal,” or “significantly more than normal” sleep were not considered sleep-deprived. For the 3-sample ANOVA analysis, we divided the participants into three groups based on the percentage of sleep that they had received the previous night,

compared to a typical night. The first group received 0-79.9% of sleep compared to a typical night, while the second and third groups received 80-89.9% and 90-100+%, respectively. Finally, for the 5-sample ANOVA analysis, participants were separated into five groups, with each group comprising a different score on the Likert scale. We opted to use an ANOVA analysis because it measures the level of significant difference between the means of multiple different treatments. We used a two-tailed t-test to look for significant differences between the means of two treatment groups. The purpose of this analysis was to determine if significant differences in physiological responses were observed between more specific qualitative descriptions of sleep habits. For all analyses run, differences were considered significant at a p-value of  $< 0.05$ .

### *Positive Control*

To ensure functionality of the three pieces of equipment being used and the Biopac software program, positive control tests were conducted by the investigators. The test for BP began with a baseline resting measurement. The resting BP measurements (n=2) are found in Table 1 (Before Exercise). Each participant then performed 3 minutes of vigorous exercise that included running 10 flights of stairs. The BP measurements after exercise are also found in Table 1 (After Exercise). To more easily compare the changes in BP, the mean arterial pressure (MAP) was calculated for both BP measurements before and after exercise for each participant ( $MAP = \text{Diastolic Pressure} + 1/3 [\text{Systolic Pressure (SP)} - \text{Diastolic Pressure (DP)}]$ ). The test for HR began with a baseline resting measurement. The resting HR measurements (n=4) are found in Table 2 (Before Exercise). Each participant then performed 1.5 minutes of jumping jacks, upon which their HR was again measured. The HR values after the exercise period are also found in Table 2 (After Exercise). The changes in HR were also calculated in Table 2. The test for skin

conductance began with a baseline resting EDA measurement. These measurements (n=4) are found in Table 3 (Before Exercise). Each participant then performed 30 seconds of jumping jacks, during which their EDA was being measured (During Exercise). The EDA of each participant was then measured for a 1-minute period after exercise concluded (After Exercise) to further display that a change did in fact occur during exercise. All positive control tests indicated that the equipment being used was functional and that the measurements being tested in this experiment are attainable.

### *Pilot Studies*

To confirm that physiological variables did not significantly change over time from baseline at rest, one group member wore the blood pressure monitor, pulse oximeter, and EDA finger electrodes at the start of a 15-minute period (approximate length of experiment) without any auditory or visual stimulus. The data for BP, HR, and EDA were recorded by the BP monitor, pulse oximeter, and Biopac system, respectively. Data was again collected at the end of the 15-minute period to establish an appropriate and accurate range of baseline measurements in the absence of physiological stimuli. A baseline BP of 130/85 (MAP = 100) was observed at the beginning of the 15-minute period, and a BP of 131/86 (MAP = 100) was measured after the 15-minute period. A pulse of 78 beats/minute was observed at baseline, while a pulse of 77 beats/minute was observed after the 15-minute period. Finally, a mean EDA value of -0.2568  $\mu$ S was observed at baseline, and a value of -0.2663  $\mu$ S was measured after the 15-minute period.

## Results

There were 50 participants involved in this study, including 19 males and 31 females ranging from ages 20 to 40, with a mean age of 22. Four participants' mean arterial pressure measurements were omitted due to technical problems with equipment. The number of hours of sleep obtained the night before the study was self-reported by each participant, with the minimum being 4 hours and the mean being  $7 \pm 1.34$  hours. Hours of sleep on a typical night were also self-reported, with an average of 6.99 hours per night (Figure 2). Sleep deprivation was measured according to a Likert scale, with "significantly less than normal" and "less than normal" sleep categorized as sleep-deprived, and "same," "more than normal," and "significantly more than normal" sleep categorized as non-sleep-deprived. A second definition of sleep deprivation was used to measure subjective sleep deprivation. The subjective measure was self-reported, and those who reported feeling sleep-deprived, regardless of how many hours of sleep they obtained the night before, were categorized as such. A third definition of sleep deprivation was used based on percentage of sleep acquired the night before the study, compared to a typical night of sleep. Participants were divided into three groups: 0-79.9% of typical sleep, 80-89.9% of typical sleep, and 90-100+% of typical sleep. The most sleep-deprived individual in the study, got 71.43% of their normal amount of sleep. Most of the participants attained between 6 and 9 hours of sleep and attained between 84% to 110% of their typical sleep (Supplemental Figure 1).

### *Heart Rate*

The change in heart rate from baseline to the stressful stimulus at 1:39 was calculated for the sleep-deprived group ( $n=18$ ,  $\bar{x}=7.5$ ,  $SD=\pm 11.13$ ) and for the non-sleep-deprived group ( $n=32$ ,  $\bar{x}=8$ ,  $SD=\pm 7.85$ ), as categorized by the Likert scale. A two-tailed t-test showed that there

was not a statistically significant ( $p=0.425$ ) difference in the change in heart rate between these groups.

A 5-sample ANOVA was run on the “significantly less than normal” ( $n=1, \bar{x}=31$ ), “less than normal” ( $n=17, \bar{x}=7.42, SD=\pm 11.46$ ), “same” ( $n=23, \bar{x}=19.48, SD=\pm 8.06$ ), “more than normal” ( $n=7, \bar{x}=5.71, SD=\pm 4.07$ ), and “significantly more than normal” ( $n=2, \bar{x}=2, SD=\pm 11.31$ ) sleep groups. The ANOVA determined that there was not a statistically significant ( $p=0.102$ ) difference in the change of heart rate between these groups (Figure 3C).

The change in heart rate was calculated for the subjectively sleep-deprived group ( $n=15, \bar{x}=6.2, SD=\pm 7.76$ ) and for the subjectively non-sleep-deprived group ( $n=35, \bar{x}=8.51, SD=\pm 9.58$ ). A two-tailed t-test showed that there was not a statistically significant ( $p=0.205$ ) difference in the change in heart rate between these groups (Figure 3A).

A 3-sample ANOVA was run on participants who got 0-79.9% of their typical sleep ( $n=3, \bar{x}=2, SD=\pm 6.56$ ), 80-89.99% of their typical sleep ( $n=8, \bar{x}=7.75, SD=\pm 14.95$ ), and 90-100+% of their typical sleep ( $n=39, \bar{x}=8.28, SD=\pm 7.70$ ). The ANOVA showed that there was not a statistically significant ( $p=0.522$ ) difference in the change of heart rate between these groups (Figure 3B).

### *Mean Arterial Pressure*

The change in the mean arterial pressure from baseline to the stressful stimulus at 1:39 was calculated for the sleep-deprived group ( $n=16, \bar{x}=-3.90, SD=\pm 7.65$ ) and for the non-sleep-deprived group ( $n=30, \bar{x}=-3.29, SD=\pm 5.97$ ), as defined by the Likert scale. A two-tailed t-test showed that there was not a statistically significant ( $p=0.383$ ) difference in the change in the mean arterial pressure between these groups.

An ANOVA was run on the “significantly less than normal” ( $n=1$ ,  $\bar{x}=4.33$ ), “less than normal” ( $n=15$ ,  $\bar{x}=-4.45$ ,  $SD=\pm 7.59$ ), “same” ( $n=22$ ,  $\bar{x}=-2.73$ ,  $SD=\pm 1.96$ ), “more than normal” ( $n=6$ ,  $\bar{x}=-3.06$ ,  $SD=\pm 4.11$ ), and “significantly more than normal” ( $n=2$ ,  $\bar{x}=-15.84$ ,  $SD=\pm 7.30$ ) sleep groups. The ANOVA revealed that there was a statistically significant ( $p=0.041$ ) difference in the change of mean arterial pressure between the “significantly less than normal” and the “significantly more than normal” sleep treatment groups (Figure 4C).

The change in mean arterial pressure was calculated for the subjectively sleep-deprived group ( $n=13$ ,  $\bar{x}=-2.95$ ,  $SD=\pm 6.57$ ) and for the subjectively non-sleep-deprived group ( $n=33$ ,  $\bar{x}=-3.72$ ,  $SD=\pm 6.60$ ). A two-tailed t-test showed that there was not a statistically significant ( $p=0.360$ ) difference in the change in mean arterial pressure between these groups (Figure 4A).

A 3-sample ANOVA was run on participants who got 0-79.9% of their typical sleep ( $n=3$ ,  $\bar{x}=-1.89$ ,  $SD=\pm 3.15$ ), 80-89.99% of their typical sleep ( $n=8$ ,  $\bar{x}=-2.58$ ,  $SD=\pm 3.06$ ), and 90-100+% of their typical sleep ( $n=35$ ,  $\bar{x}=-1.60$ ,  $SD=\pm 2.18$ ). The ANOVA determined that there was not a statistically significant ( $p=0.581$ ) difference in the change of mean arterial pressure between the groups (Figure 4B).

### *Electrodermal Activity (EDA)*

The change in EDA from baseline to the stressful stimulus at 1:39 was calculated based on the Likert scale rating and calculated for the the sleep-deprived group ( $n=16$ ,  $\bar{x}=0.67$ ,  $SD=\pm 0.97$ ) and for the non-sleep-deprived group ( $n=31$ ,  $\bar{x}=0.423$ ,  $SD=\pm 0.50$ ). A two-tailed t-test revealed that there was not a statistically significant ( $p=0.169$ ) difference in the change in EDA between these groups.

A 5-sample ANOVA was run on the change in EDA from baseline to the stressful stimulus. It was run on the “significantly less than normal” ( $n=1$ ,  $\bar{x}=-0.13$ ), “less than normal” ( $n=18$ ,  $\bar{x}=0.43$ ,  $SD=\pm 0.46$ ), “same” ( $n=19$ ,  $\bar{x}=0.87$ ,  $SD=\pm 1.13$ ), “more than normal” ( $n=7$ ,  $\bar{x}=0.32$ ,  $SD=\pm 0.60$ ), and “significantly more than normal” ( $n=2$ ,  $\bar{x}=0.72$ ,  $SD=\pm 0.96$ ) sleep treatment groups. There was not a statistically significant ( $p=0.383$ ) difference between these groups (Figure 5C).

The change in EDA was calculated based on the subjective sleep deprivation rating and calculated for the sleep-deprived group ( $n=15$ ,  $\bar{x}=0.72$ ,  $SD=\pm 1.35$ ) and for the non-sleep-deprived group ( $n=32$ ,  $\bar{x}=0.52$ ,  $SD=\pm 0.47$ ). A two-tailed t-test showed that there was not a statistically significant ( $p=0.229$ ) difference in EDA between these groups (Figure 5A).

A 3-sample ANOVA was run on participants who got 0-79.9% of their typical sleep ( $n=3$ ,  $\bar{x}=0.44$ ,  $SD=\pm 0.696$ ), 80-89.99% of their typical sleep ( $n=7$ ,  $\bar{x}=0.435$ ,  $SD=\pm 0.696$ ), and 90-100+% of their typical sleep ( $n=37$ ,  $\bar{x}=0.629$ ,  $SD=\pm 0.91$ ). The ANOVA showed that there was not a statistically significant ( $p=0.828$ ) difference in the change of heart rate between the groups (Figure 5B).

## **Discussion**

In this study, we attempted to determine the relationship between sleep deprivation and physiological reactivity to acute stress. We expected sleep-deprived subjects to exhibit greater increases in heart rate, blood pressure, and electrodermal activity in response to an acute stressor, when compared to individuals who were not sleep-deprived. Regardless of the method that was used to group participants, only one significant difference was found between sleep-deprived and

non-sleep-deprived participants: MAP differences between participants who indicated receiving “significantly less than normal” and “significantly more than normal” sleep on the Likert scale.

Those who obtained “significantly more than normal” sleep had a more drastic change in MAP when compared to those who had “significantly less than normal” sleep. Despite the mathematically significant difference between these groups, there were only 3 participants who fit into those categories. Therefore, we believe it would be misleading to use this as evidence to support our hypothesis. Beyond this, there were no significant differences between groups in the change of HR, BP, or EDA.

Due to the paucity of severely sleep-deprived participants, this study was unable to make any definitive conclusions regarding the physiological effects of sleep deprivation on acute stress reactivity. In Leger et. al, 2014, acute sleep deprivation was “defined as 25%-50% of a normal 8 hours night sleep,” which would correspond to a maximum of four hours of sleep. Out of the 50 participants in our study, only one reported getting four hours of sleep or less, which was not 50% of that participant’s normal sleep. Because of this restriction, our study attempted to define sleep deprivation of participants in three ways (Results).

Only 15 participants reported feeling subjectively sleep-deprived. Similarly, only 18 subjects reported getting “less than normal” or “significantly less than normal” sleep on the Likert scale. Finally, the lowest proportion of sleep a participant had obtained the previous night, compared to a typical night, was around 71%. Because of this, the results of our study may be more accurately characterized in the context of mild to moderate sleep deprivation. In this context, we conclude that mild to moderate sleep deprivation does not significantly impact physiological reactivity to an acute stressor.

Beyond a lack of severely sleep-deprived participants, there was discordance between the different ways of measuring sleep deprivation. Participants who reported feeling subjectively sleep-deprived did not show a significant difference ( $p = 0.166$ ) in the percentage of typical sleep attained when compared to participants who were not subjectively sleep-deprived. However, when participants were grouped by their answers to the Likert scale question, there was a significant difference ( $p < 0.0001$ ) between groups in the percentage of typical sleep attained. This would make it seem like the Likert scale answer would be a more accurate way to define sleep-deprivation; however, out of the 15 participants who reported feeling subjectively sleep-deprived, 8 of them reported having gotten the “same” or “more than normal” amount of sleep, which would not make them truly sleep-deprived.

We believe that future studies in this arena should use alternative methods of defining sleep deprivation to ensure more accurate results. Additionally, future research would benefit from experimentally inducing sleep-deprivation in participants, rather than measuring coincidental incidences of sleep deprivation. Due to the ethical and financial limitations of this study, we were not able to intentionally induce sleep deprivation in subjects.

Another area of possible research in this domain would be to include survey questions about the quality of sleep that participants attained the previous night. Sleep quality has been found to have a more profound effect on health than sleep quantity (Pilcher, Ginter, and Sadowsky, 1997). Combining information on sleep quality and sleep quantity will lead to a more inclusive picture of human health. Additionally, beyond lack of sleep, oversleeping can lead to sleepiness in the same way as sleep deprivation (Leger *et. al*, 2014). However, not all chronic diseases associated with sleep-deprivation are also associated with oversleeping (Leger *et. al*,

2014). This implies that the physiological effects of oversleeping and sleep-deprivation are not the same, but our study was too limited to clarify these differences.

This experiment was also limited in that it aimed only to determine the physiological effects of a single acute stressor; the effects of multiple different stressors will almost certainly have different results. In addition, our study did not include other factors that activate the HPA Axis and the sympathetic nervous system. There is a plethora of interesting research questions that could be answered with more refined study systems, the results of which would have meaningful impacts on everyday life.

While we were unable to make definitive conclusions about the effects of sleep deprivation on acute stress reactivity, we did observe drastically varied stress reactions in response to the acute stressor (Supplemental Figure 4). This points to the complexity of the stress response and the innate heterogeneity of the human condition. Because individuals' tolerance levels for sleep deprivation and reactions to acute stress are heterogenous in nature, we believe it will be difficult for future research to isolate a single effect of sleep deprivation on acute stress reactivity.

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## Tables

*Table 1: A table showing measurements (n=2) of baseline resting BP (Before Exercise) and its corresponding mean arterial pressure (MAP) value. Also included are measurements of BP after 3 minutes of vigorous exercise (After Exercise) and its corresponding MAP value. BP values were measured on a standard (Systolic/Diastolic) scale using an automated blood pressure cuff, and the corresponding MAP value was calculated using the following equation:  $MAP = DP + 1/3 [SP-DP]$ . The averages  $\pm$  standard deviations for the corresponding MAP values are also listed. This table demonstrates a positive control for BP, as is evident in the increase in MAP values for each investigator from before exercise to after exercise.*

<b>Group Member</b>	<b>Baseline</b>		<b>Exercise</b>	
	<b>BP</b>	<b>MAP</b>	<b>BP</b>	<b>MAP</b>
<i>Marcus</i>	117/79	91.67	128/81	96.67
<i>Michael</i>	139/97	111	170/103	125.33
	<b>Average</b>	$101.34 \pm 13.67$	<b>Average</b>	$111 \pm 20.27$

*Table 2: A table showing measurements (n=4) of baseline resting HR (Before Exercise) and after 1.5 minutes of jumping jacks (After Exercise). Also included is a column indicating the change in HR from before exercise to after exercise. HR values were measured in Beats/Minute, using a pulse oximeter. This table demonstrates a positive control for HR, as is evident in the increase in HR for each investigator from before exercise to after exercise.*

<b>Group Member</b>	<b>Before Exercise</b>	<b>After Exercise</b>	<b>Change</b>
<i>Marcus</i>	76	145	69
<i>Savannah</i>	82	145	63
<i>Alex</i>	89	145	56
<i>Bailey</i>	82	161	79

Table 3: A table showing measurements (n=4) of baseline, resting skin conductance (Before Exercise), during a 30 second period of jumping jacks (During Exercise), and during a 1-minute resting period (After Exercise). Skin conductance values were measured in mean change of microsiemens ( $\mu S$ ), using BSL EDA finger electrodes. This table demonstrates a positive control for skin conductance, as seen in the increase of skin conductance during exercise, and a decrease after exercise.

Group Member	Before Exercise ( $\mu S$ )	During Exercise ( $\mu S$ )	After Exercise ( $\mu S$ )
Michael	-0.21721	0.76541	-0.24371
Bailey	0.02465	0.75539	0.33060
Savannah	0.023051	0.34212	0.55007
Alex	0.02782	0.19421	0.10227

Table 4: A table showing measurements 2 measurements (n=1), at the beginning and end of a 15-minute period, for BP, HR, and EDA. Measurement 1 for each measure indicates the data before the 15-minute period, and measurement 2 indicates the data after the 15-minute period. This table demonstrates that resting baseline physiological measurements do not significantly change over time for any of the three physiological variables in the absence of external stimuli.

Group Member	BP Measurement 1	BP Measurement 2	HR Measurement 1	HR Measurement 2	EDA Measurement 1	EDA Measurement 2
Michael	130/85	131/86	78	77	-0.2568	-0.2663

## Figures

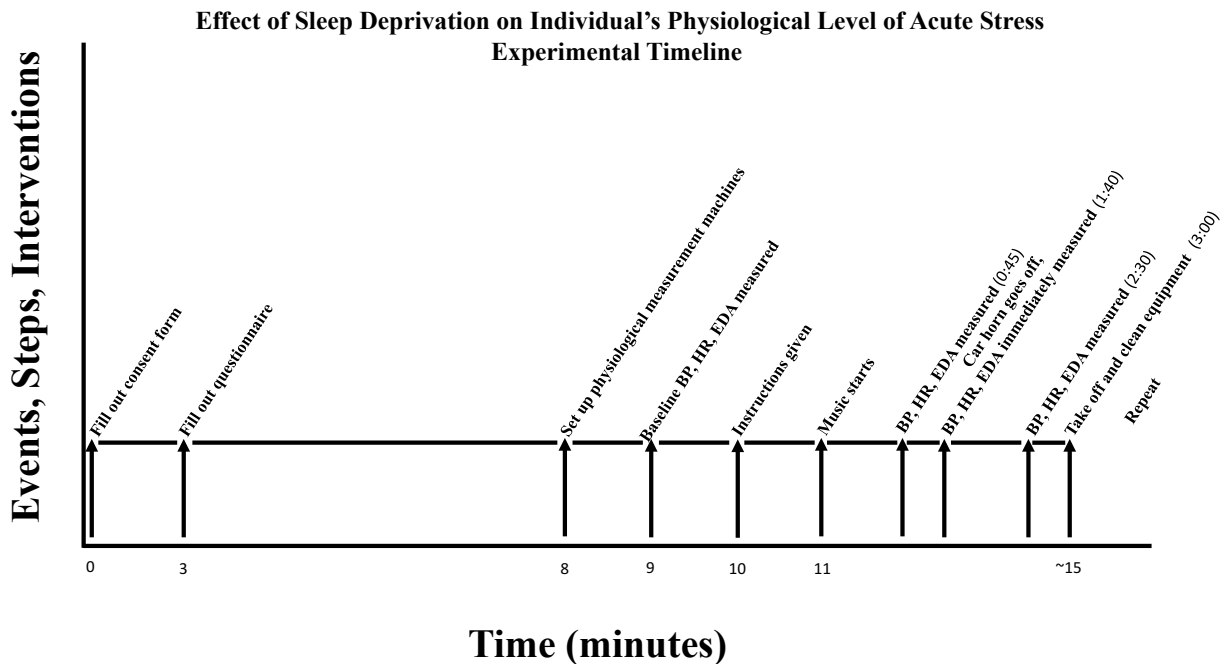


Figure 1: Timeline indicating all of the necessary steps and approximate periods of time for the duration of physiological experimentation.

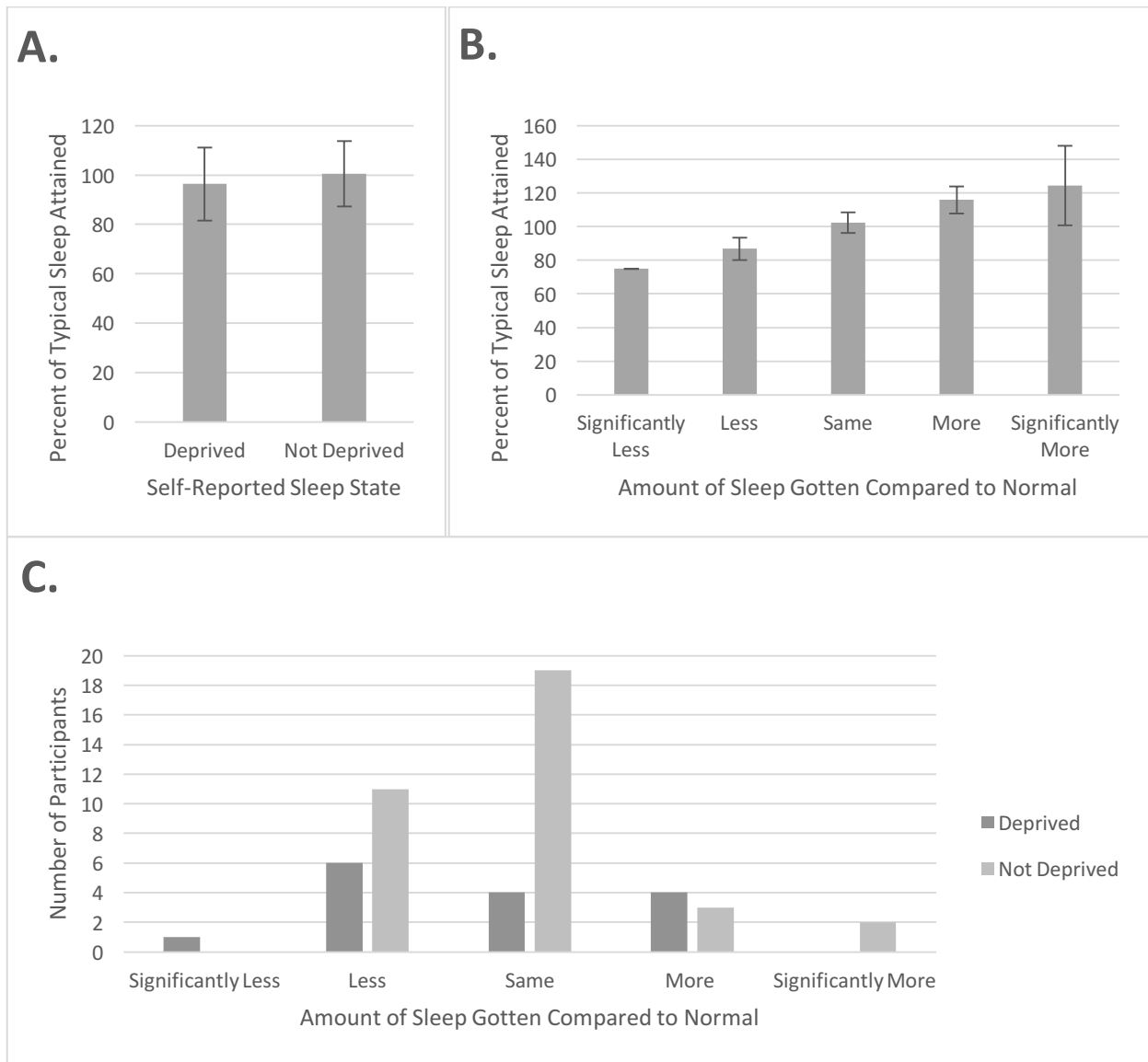


Figure 2: A comparison of the different ways to group participants. A – The percent of typical sleep attained the previous night by participants grouped by self-reported sleep state categories. B – The percent of typical sleep attained the previous night by participants grouped by their answer to a Likert scale question regarding how much sleep they had attained the previous night compared to their normal amount of sleep. C – The number of participants grouped by their answers to the Likert scale question and separated into categories based on their self-reported sleep state.

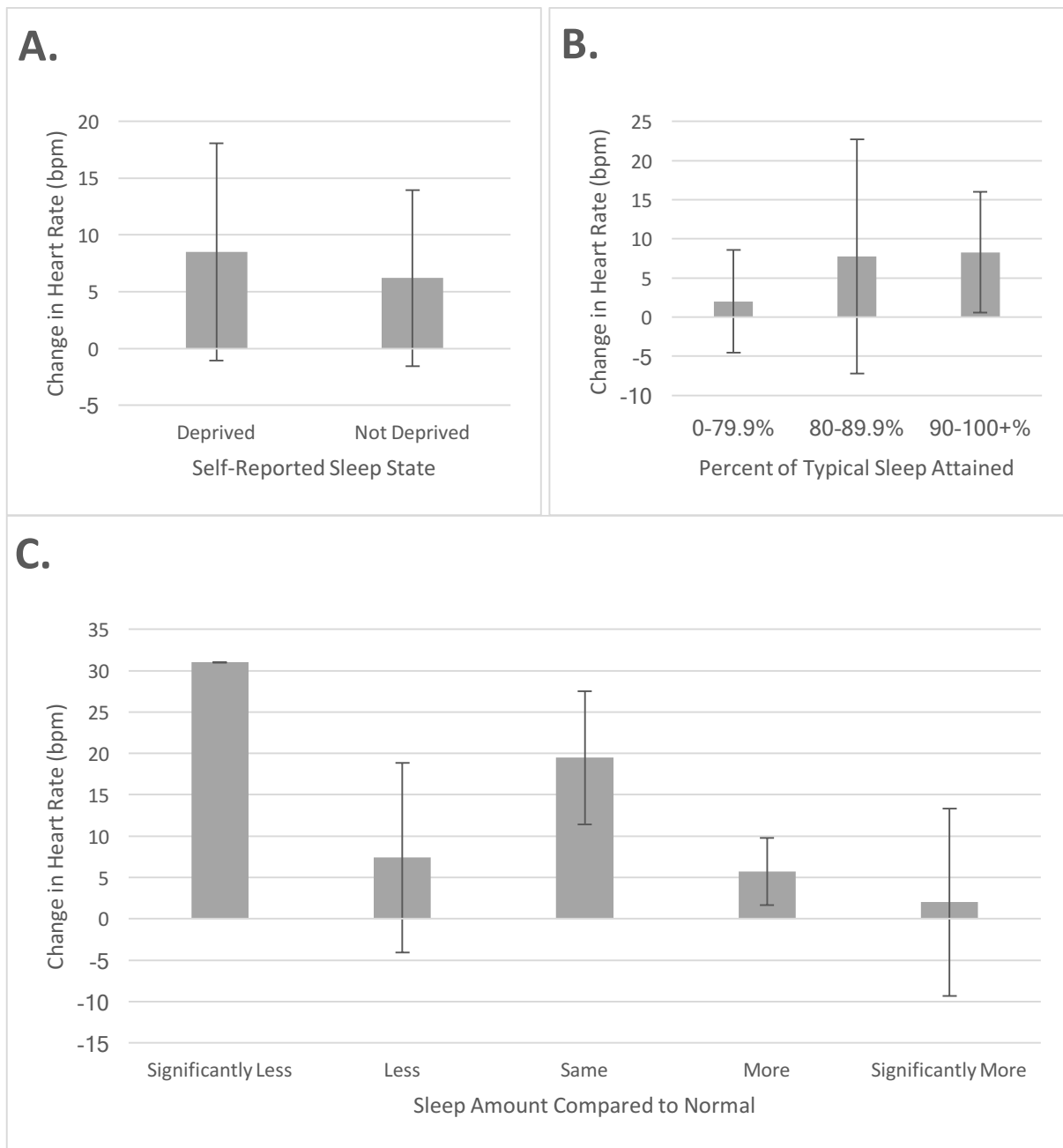


Figure 3: The mean change in heart rate, caused by a stress inducing stimulus, observed in participants grouped by A – self reported sleep state, B – percent of typical sleep attained the previous night, and C – their answer to a Likert scale question about how much sleep they had gotten compared to normal.

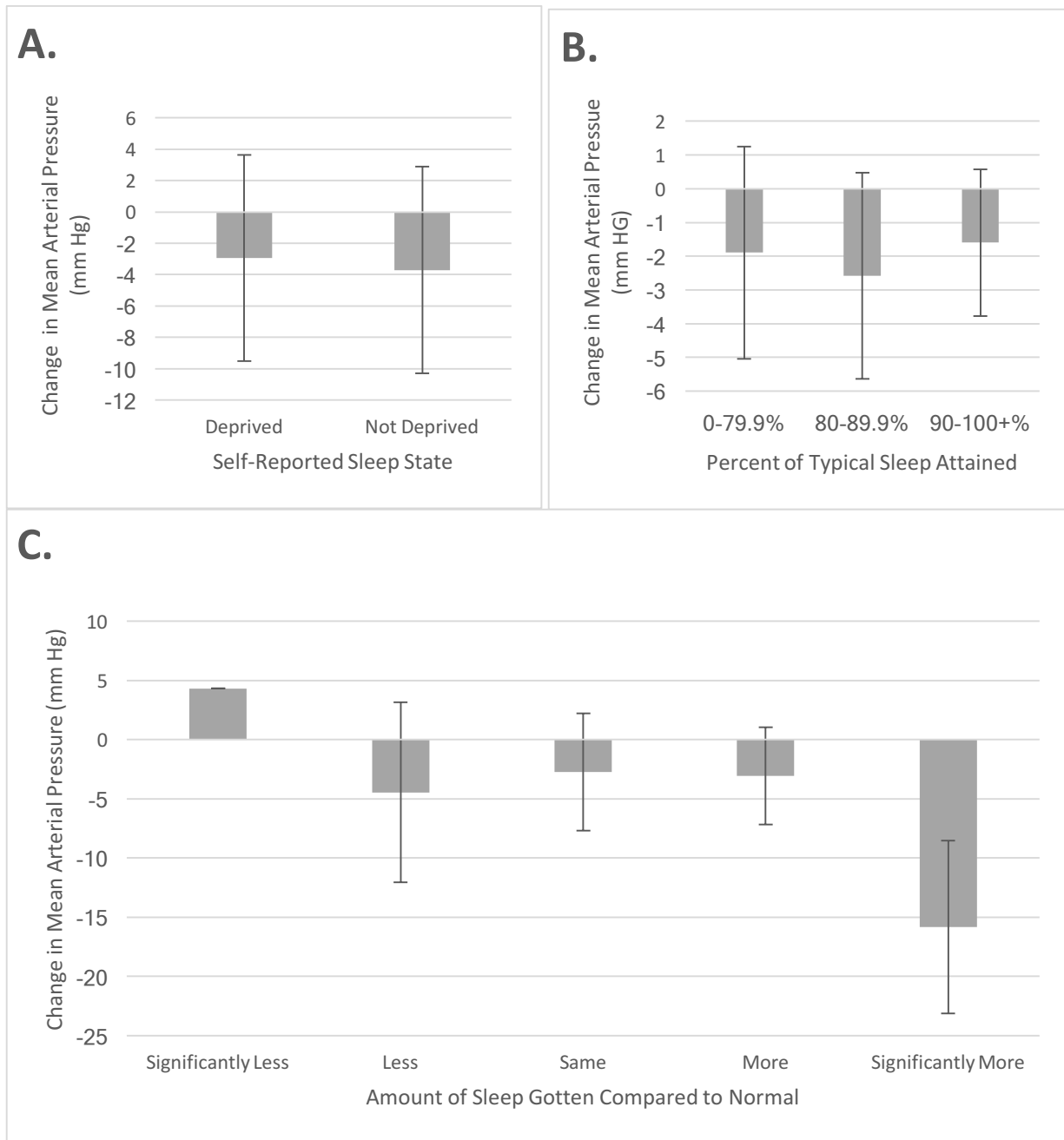


Figure 4: The mean change in mean arterial pressure, caused by a stress inducing stimulus, observed in participants grouped by A – self reported sleep state, B – percent of typical sleep attained the previous night, and C – their answer to a Likert scale question about how much sleep they had gotten compared to normal.

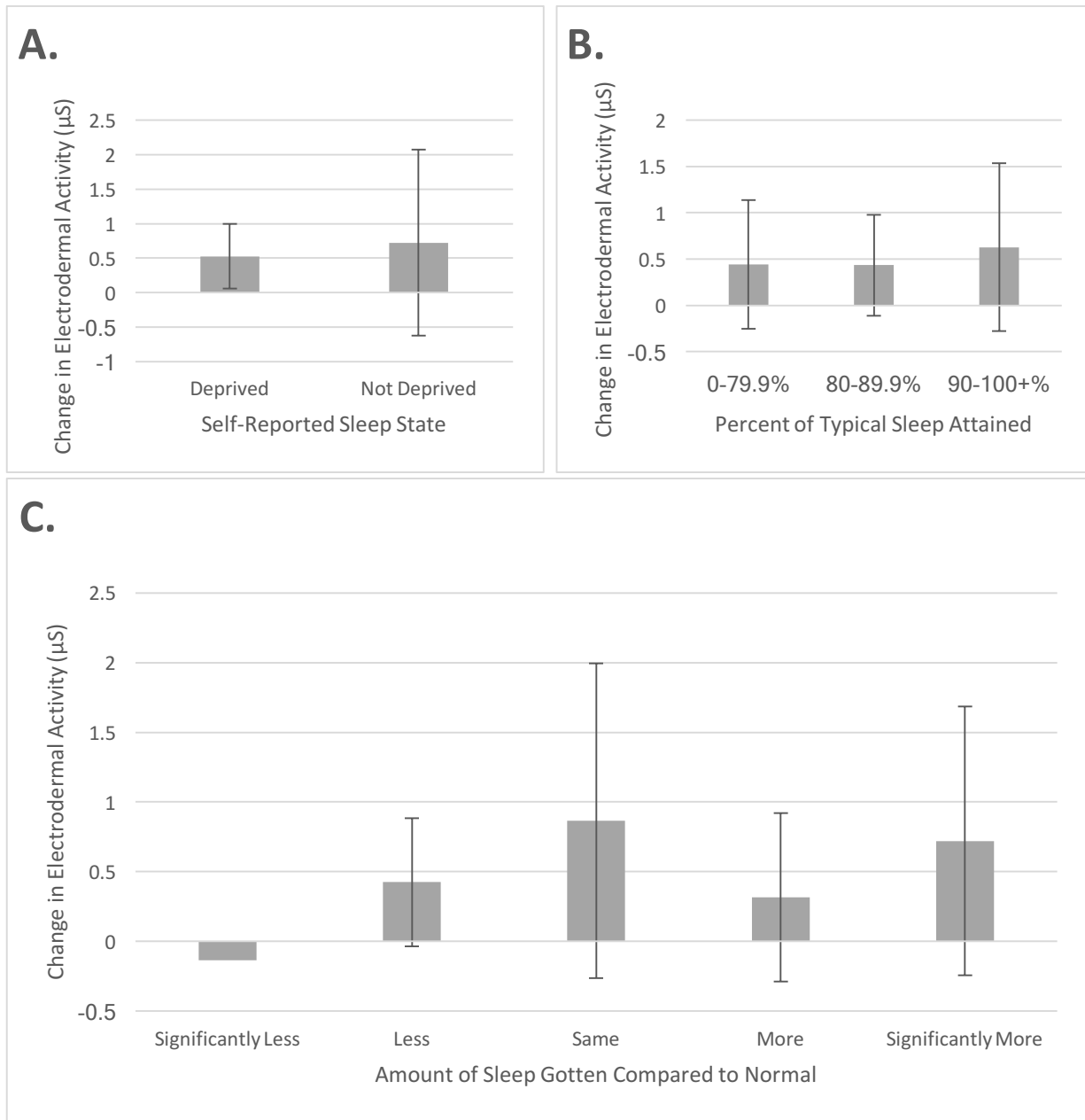


Figure 5: The mean peak-to-peak change in electrodermal activity, caused by a stress inducing stimulus, observed in participants grouped by A – self reported sleep state, B – percent of typical sleep attained the previous night, and C – their answer to a Likert scale question about how much sleep they had gotten compared to normal.

## Appendix A

1. Age: \_\_\_\_\_

2. Gender Identity (circle one):

Male

Female

Non-binary

3. On a typical night, how many hours of sleep do you get? (round to the nearest half hour)

4. Approximately how many hours of sleep did you get last night? (round to the nearest half hour)

5. Compared to a typical night, how much sleep did you get last night? (Circle one)

6. Do you feel sleep-deprived? (skip if you aren't sure)

Significantly Less	Less	Same	More	Significantly More
			Yes	No

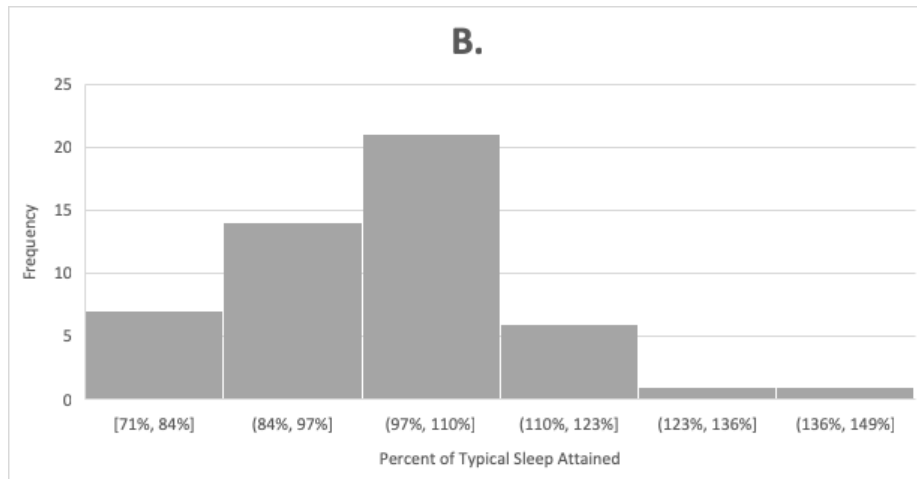
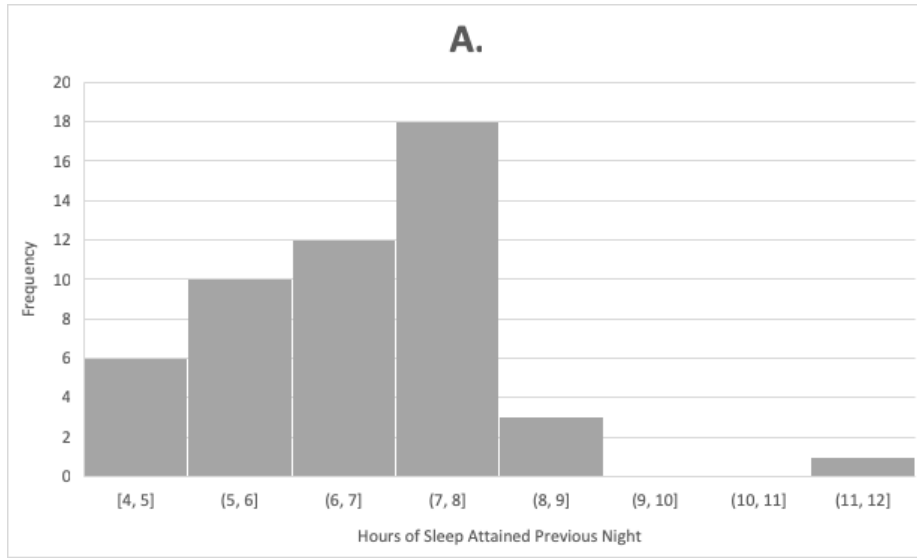
7. Do you have any medical or psychological conditions that require you to actively manage your stress (and are willing to share)?

If so, what are they? \_\_\_\_\_

8. Please indicate if you have any of the following conditions:

- Cardiovascular Conditions
- Post-Traumatic Stress Disorder
- Generalized Anxiety Disorders
- Personality/mood disorders
- Narcolepsy
- Insomnia/Hypersomnia
- Other sleep related conditions
- Other not specified: \_\_\_\_\_

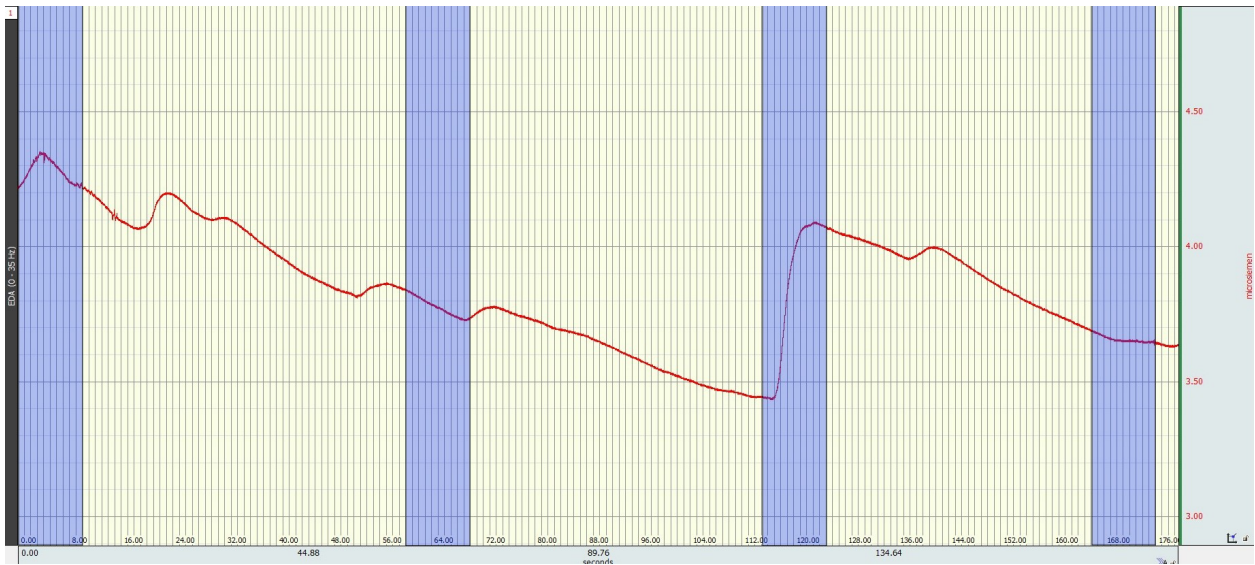
## Appendix B



*Supplemental Figure 1: Frequency histograms showing the distribution of participants over A – the number of hours of sleep they had attained the previous night and B – the percent of typical sleep that they had attained the previous night.*

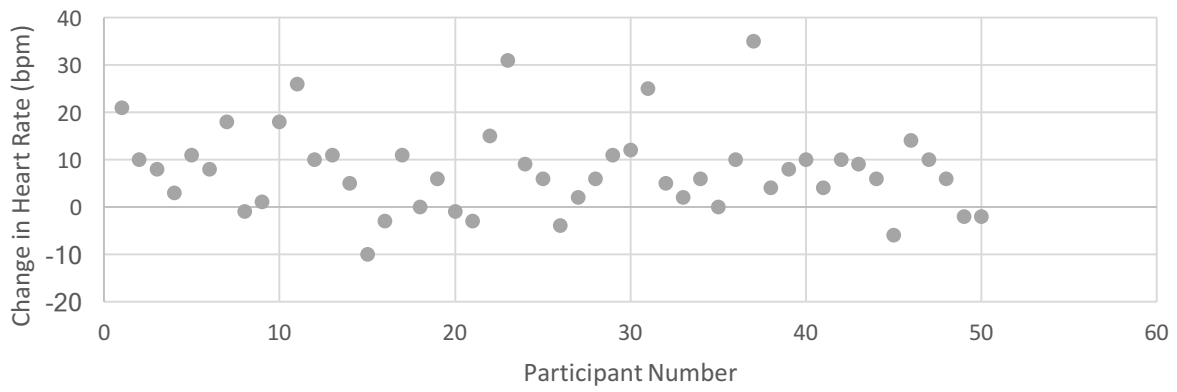


*Supplemental Figure 2: Representative EDA data collected from a sleep-deprived participant. Measurements were taken continuously over the time period. The blue highlighted regions are 10-second intervals where data was collected. The first highlighted region represents the baseline measurement. The second highlighted section begins 45 seconds into the sound recording. The third highlighted region begins at the stressful stimulus 1:39 into the sound recording. The fourth highlighted section begins 2:30 into the sound recording.*

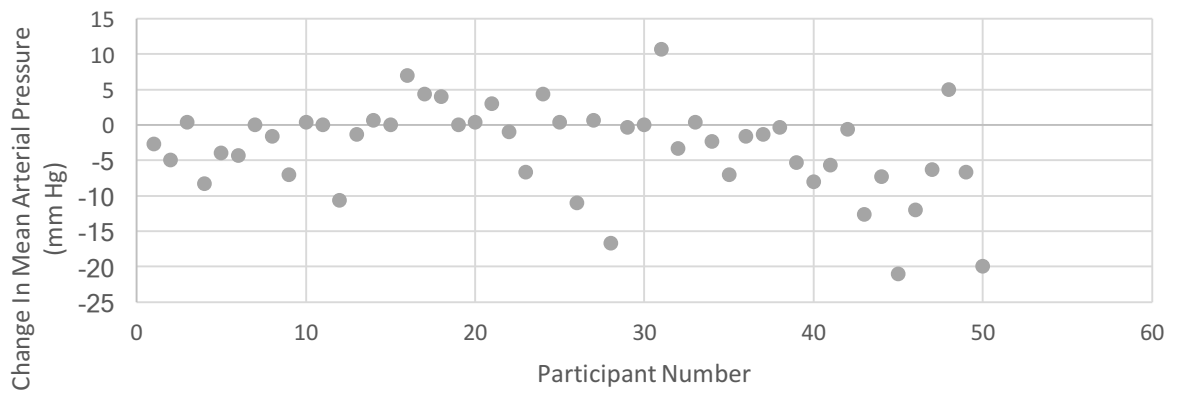


*Supplemental Figure 3: Representative EDA data collected from a non-sleep-deprived participant. Measurements were taken continuously over the time period. The blue highlighted regions are 10-second intervals where data was collected. The first highlighted region represents the baseline measurement. The second highlighted section begins 45 seconds into the sound recording. The third highlighted region begins at the stressful stimulus 1:39 into the sound recording. The fourth highlighted section begins 2:30 into the sound recording.*

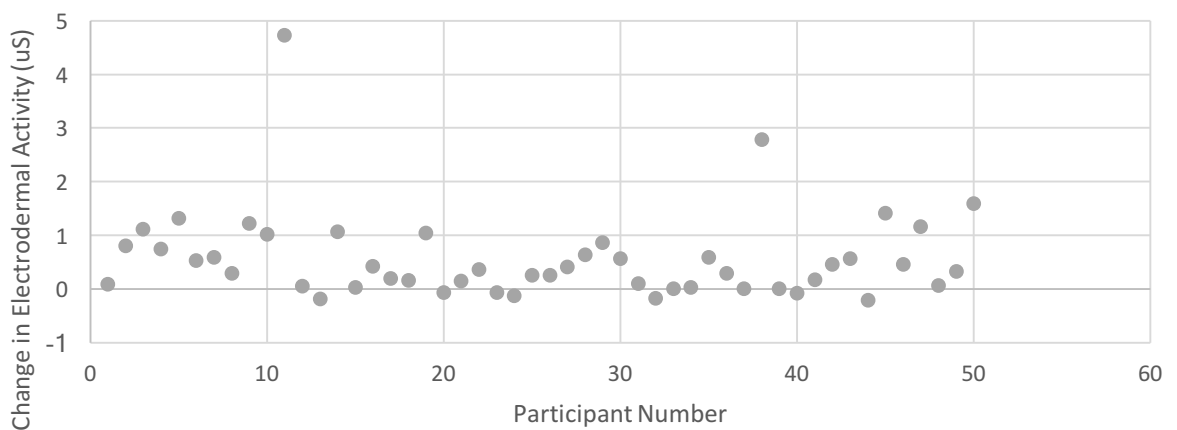
**A.**



**B.**



**C.**



*Supplemental Figure 4: Individual changes in A – heart rate, B – mean arterial pressure and C – electrodermal activity caused by stress inducing stimulus.*