

Brightbeat: Effortlessly influencing breathing for cultivating calmness and focus

by

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B.S., Sharif University of Technology (2014)

Submitted to the Program in Media Arts and Sciences
in partial fulfillment of the requirements for the degree of

Master of Science in Media Arts and Sciences

at the

MASSACHUSETTS INSTITUTE OF TECHNOLOGY

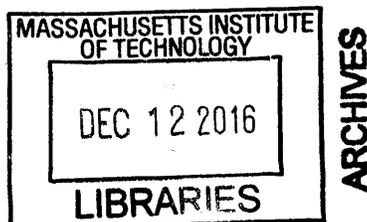
September 2016

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Abstract

There are many health-related risks associated with chronic stress. One approach for addressing this issue comes from strengthening inside self-regulation abilities rather than eliminating outside sources of stress. However, technology has not been explored to its full potential for delivering calming interventions. While many of the persuasive technologies developed for fostering behavior change focus on cognitive processes, little attention has been given to influencing behavior through automatic processes of the brain.

Considering the bidirectional relationship between psychophysiological signals and self-reported emotional states, manipulating the physiological signals that a human being has voluntary control over is promising for achieving a desired emotional state. In this work, we focus on respiration due to the fact that it is both a voluntary and involuntary response of the body. It is a good indicator of stress, but can also be manipulated to induce calmness.

This thesis introduces BrightBeat: a set of seamless visual, auditory, and tactile interventions that mimic a calming breathing oscillation with the aim of influencing physiological syncing and consequently bringing a sense of focus and calmness. These interventions are designed to run easily on commonplace personal electronic devices, respect the user's privacy, and to not require constant focus or attention in order to be effective. We have designed BrightBeat interventions iteratively and have examined both objective and subjective measures of impact through a series of studies with N=54 users in total. From an objective perspective, BrightBeat interventions significantly influenced calmer (slower) breathing and had a lasting influence. From a subjective perspective, considering the individual differences, these interventions have been shown to improve self-reported calmness and focus. Also, participants reported high preference for using them in the future.

This work has been supported by the Robert Wood Johnson Foundation and the MIT Media Lab Consortium.

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Acknowledgments

I am honored to call professor Rosalind Picard my mentor and advisor. She is an inspiration to me. In the past two years, her enthusiasm and curiosity has astonished me and her endless support, encouragement, and guidance has helped me immensely. I'm very grateful for her and for being given the chance to learn from her. Her nice and compassionate attitude has made many of my days in the past two years. I would like to express my deepest appreciations to professor Pattie Maes and Sepandar Kamvar for sharing their time and insightful comments with me and helping me see every question more thoroughly and thoughtfully.

I would like to thank all my friends and colleagues in the affective computing group whom I've learned a lot from directly and indirectly: Vincent Chen, Karthik Dinakar, Micah Eckardt, Szymon Fedor, Javier Hernandez, Natasha Jaques, Kristy Johnson, Grace Leslie, Daniel Lopez, Ehi Nosakhare, Akane Sano, Sara Taylor, Craig Ferguson, and Rashmi Karki. I would like to specially thank Asaf Azaria for helping me become a more grateful person.

I'm thankful to the media lab and to MIT for all the chances I've been given to explore my passion without limits. The abundance of resources and talent has always inspired me. I am very grateful for being surrounded by the smartest and most driven people in the world from different parts of the world.

I'm forever grateful to my family and I have no words to thank them enough for their unconditional love and support which has helped me grow as a person. I specially thank my mom for being very close no matter how many miles away we are and for teaching me to work hard for what I care about. I thank my sisters for instilling curiosity in me as a kid and for being my closest friends as an adult. I thank my brother for all the fun memories we have together that still make me laugh even when I'm down. I thank my dad for teaching me to stay calm by example rather than by words.

I would like to express my sincere gratitude to Ardavan for his endless love and support and for helping me develop a growth mindset. He has taught me to be persistent in taking baby-steps of self-improvement and growing as a person. Nothing is more comforting than his calmness, optimism, patience, and understanding.

I thank my friends in different states of the US, Iran, and Switzerland for supporting me through all my ups and downs and for being there for me at the face of difficulty. It's their love and friendship that keeps me sane. I wish there were no geographical boundaries and I could go give a hug to each one of them and express my deepest gratitude in person!

Last but not least, I would like to thank the participants of the series of studies I've run for all their valuable input that has helped improve this work.

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Chapter 1

Introduction

1.1 Motivation

Chronic stress has been shown to negatively impact health and wellbeing in several ways. For example, it affects the immune system [77], gastrointestinal [7], cardiovascular [4,86], and neuropsychiatric [67] health. It also affects cognitive functions, memory, and learning [3, 50]. This wide range of health- and wellbeing-related risks calls for minimizing unnecessary stress in daily life situations. One way to tackle this issue is eliminating external stressors. However, stress is perceived differently among different individuals and there is no consensus around modeling stress or stressors. In fact, new directions are being suggested to address the limitations of previous models [51]. This motivates the idea of developing internal techniques to gain resilience against stress rather than fighting it externally.

Scientific evidence confirms that the relationship between breathing and regulatory effects is bidirectional. Stress has physiological indicators including changes in core temperature, cardiovascular tone, respiratory patterns, and more [20]. Among all, respiration has a unique characteristic of being both voluntarily and involuntarily controlled. Also, voluntary deep and slow breathing can induce physiological, affective, and cognitive calm [31].

In the current day and age when individuals spend hours in front of screens, wearing headphones, or other wearable devices, technology can be a promising medium for delivering seamless calming interventions throughout everyday life activities. However, little research has been conducted around conveying a portion of the positive influences of a meditative experience *while* being involved in daily activities. In fact, most of the technology designed for health and wellbeing related behavior change relies solely on cognitive processes of the brain rather than delivering an intervention that doesn't require constant attention. Adams et al. refer to the latter as mindless computing [1]. The lack of scalable systems for self-regulating that respect the user's privacy and don't require his/her constant attention and instead mostly rely on the automatic processes of behavior change has motivated this thesis. Thus, this thesis ironically uses the techniques from mindless computing to promote a respiratory induced mindful state in everyday activities.

1.2 Outline

The next chapter provides a literature review about stress and its physiological manifestation and why breathing is a good candidate for self-regulation. It then reviews the existing technologies and systems that facilitate measuring or influencing breathing which directly or indirectly have addressed a meditative experience. This motivates the third chapter, which provides examples of physiological synchrony and introduces a series of interventions where commonplace technological devices mimic a calming breathing signal in order to ease self-regulation and induce calm. We call these interventions BrightBeat. Chapter 4 and 5 explain manual and automated versions of BrightBeat, providing detailed explanation of the system design, user study protocol and deployment, and finally demonstrating results both in terms of self-reported changes and physiological data. Chapter 6 concludes by summarizing the impacts of BrightBeat interventions and providing future directions.

1.3 Contributions

This thesis contributes to bringing mindfulness to every day activities through iterative design and implementation of a series of interventions, BrightBeat, that respect individual differences and privacy and validates them through structured user studies. BrightBeat users had slower relative breathing rates and were able to achieve their goal breathing rate a higher percentage of the time. Also, BrightBeat is shown to improve self-reported focus and calmness. This thesis also tests the hypothesis of physiological syncing through interaction with means of technology such as screens, headphones, and wristbands by detailed analysis of physiological data. It also replicates the results related to the relationship between voluntarily slowing breathing rate and achieving an emotional state of calmness and focus.

Chapter 2

Background

2.1 Breathing and calmness

In this section, we briefly review the physiological responses of stress with a deeper focus on respiratory patterns. Then, we discuss self-regulation in relation to voluntary modification of breathing, summarizing the scientific evidence for the effectiveness of breathing exercises on patient and healthy populations both for treatment and improvement purposes.

2.1.1 Stress and physiological response

Psychologically, stress is a complex phenomenon. It is perceived and experienced differently by different individuals, and even by the same person in different situations. Several theories have been devised to model stress in the workplace [5, 10, 27, 81, 96]. However, new directions are still being suggested to address the limitations of these models [51]. Under a broader umbrella, stress can refer to a spectrum ranging from negative (distress) to positive (eustress) [stimulation] [78].

As summarized by [9], the physiological response to stress starts with activating the hypothalamic pituitary adrenal axis (HPA) and the sympathetic nervous system (SNS).

The consequent release of several chemical mediators prepares the body for a fight-or-flight response which results in behavioral adaptations including increased arousal and alertness, improved cognition, focused attention, elevations in core temperature, suppression of digestive function, growth, reproduction, and immunity, adaptive redirection of energy, increases in cardiovascular tone, respiratory rate, and intermediate metabolism. On the other hand, the parasympathetic nervous system (PNS), can either assist or inhibit the SNS by decreasing or increasing its activity respectively [20]. Thus, increased PNS activity can counteract the SNS response and return the body to a relaxed state.

In general, the stress response is meant to be short during which the suppression of growth, production, and immune system are beneficial or at least have no adverse consequences and improve chances of the individual for survival [20]. However, chronic stress can damage the body in different ways. To name a few, chronic stress has been shown to negatively impact immune system [77], gastrointestinal [7], cardiovascular [4, 86], and neuropsychiatric [67] health. It also affects cognitive functions, memory, and learning [3, 50].

As a result, successful stress regulation plays an important role in maintaining health and wellness. In this context, Porges has developed the “polyvagal theory” which explains the evolution of the vagus nerve and its role in the PNS system and relates the autonomic function to behavior [70]. This theory articulates three phylogenetic stages of the development of the vertebrate autonomic nervous system (ANS). The most evolved ANS component or “Myelinated vagus” causes advanced behavioral functions like social communication, self-soothing and calming, and inhibiting arousal. The second component or “Sympathetic-adrenal system” causes mobilization or active avoidance. The most primitive component, “Unmyelinated vagus”, causes immobilization such as death feigning, behavioral shutdown, or passive avoidance. These three circuits have a hierarchical structure in which they respond to challenge. The less advanced circuits come into play only when the more evolved ones have failed.

Cardiac elements like heart rate variability (HRV) have been shown to be closely re-

lated to stress reactivity [32]. HRV is the physiological phenomenon of the variation of beat-to-beat intervals between consecutive peaks in an ECG signal [8]. More specifically, cardiorespiratory parameters like respiratory sinus arrhythmia (RSA) were put under the spotlight by Porges through the development of polyvagal theory [69]. RSA is heart rate variability in synchrony with respiration, where beat-to-beat intervals are shortened during inspiration and prolonged during expiration [6]. He proposed RSA as a reliable continuous measure of the vagal brake which refers to the unique characteristics of the most evolved autonomic nervous system (ANS) component or “Myelinated vagus” [70]. More researchers witnessed the importance of respiratory functions in understanding autonomic mechanisms of the body [24].

Though respiration is primarily regulated for metabolic and homeostatic purposes, it can also change in response to emotions [37]. Many scholars have studied respiratory patterns as indicators of physiological emotional responses. Bloch et. al. showed respiratory patterns can distinguish between six basic emotions: joy-laughter, sadness-crying, fear-anxiety, anger, erotic love and tenderness [11]. A review by Boiten and his colleagues have summarized that primary correlates of respiratory patterns including rate, amplitude, and volume reflect general dimensions of emotional response such as calm-excitement, relaxation-tenseness, and active-passive coping [12, 13]. Butler et. al. showed RSA can indicate emotional reactivity and regulatory efforts [17]. Sturgeon et. al. showed RSA to be a promising indicator of resilience to pain [89].

2.1.2 Self-regulation through voluntary respiratory modification

Several ancient to contemporary practices have used breathing techniques for self-regulation. For example, Pranayama (a formal practice of controlling the breath), Qigong (a Chinese system for physical exercise and breathing control), and Alexander breathing technique (a technique to help with voice and breathing) are just a few. Besides practice, scientific evidence confirms the bidirectional relationship between breathing and regulatory effects.

Not only are respiratory patterns indicators of emotional reactivity, but also voluntary modification of breathing can influence emotional reactions. For example, Philippot et. al. conducted an interesting experiment composed of two studies. In the first study, participants were asked to produce specific emotions and describe the consequent breathing patterns. In the second study, the same breathing instructions were given to new participants to investigate the impact of manipulating respiration on emotional states. The purpose of the study was hidden from the participants through a cover story. Induced respiration manipulation resulted in differentiated emotional feelings without participants' awareness of the process [66].

Deep and slow breathing has been widely used as part of relaxation techniques in patient populations to treat somatic disorders such as hypertension and pulmonary diseases [29, 31], psychiatric disorders including anxiety and depression syndromes [15, 45], post-traumatic and other stress-related disorders [33, 44, 57, 62, 72], and substance abuse [16]. Not only in patient populations, but also in healthy individuals the physical and psychological effects of deep breathing and meditation have been shown. For example, it improves cardiovascular health [74], mental function [82], and attentiveness [88], wellbeing, mood, and stress tolerance [14, 16]. Not only rate, but also the inhalation to exhalation ratio (i/e) has been shown to be related to the relaxation response [91].

Sighs are another respiratory behavior which our body uses as a healing process. Normal breathing consists of considerable correlated variability and some random variability. It is known that negative emotions reduce correlated variability and sustained attention reduces total variability. Both conditions evoke spontaneous sighing which is related to subjective relief and restores respiratory variability influenced by stress or attention [94]. It has been shown that these healing characteristics are not exclusive to spontaneous sighing. In fact, when physiologically appropriate, they follow instructed sighs as well [95].

The scientific evidence showing effectiveness of voluntary respiratory modification on emotional regulation calls for designing technologies that ease the regulatory process. In the next section, we provide a brief overview of the role that technology has played in

mediative practices that focus on breathing.

2.2 Technology and meditative experience

With the widespread use of smartphones, advent of wearable devices, and culture of spending countless hours with technological devices, meditative practices have found their way into technological devices. In this section, we review the connection between technology and meditative practices when it is combined with breathing, either by addressing it, measuring it, or influencing it. We start with less interactive examples such as mobile applications that encourage deep breathing in a guided meditation training session and move to more interactive ones introducing sensors and biofeedback examples, virtual environments, and finally responsive environments and objects.

2.2.1 Desktop and mobile applications

There have been several attempts at building mindfulness training, meditation, or simply calming interventions in the form of desktop software or mobile applications. Headspace [41], Stop, breathe, and think [87], Calm [19], Chakra Meditation [63], Breathe [47], and Insight timer [42] are only a few out of many meditation applications that come with training session videos, audio recordings, music, and text. Some of them have incorporated gamification [21] mechanisms to engage participants further and help create a habit of meditation practice. As mindful breathing has been an indispensable part of meditation, these apps, by extension, briefly touch breathing. A few apps have paid more overt attention to breathing. MyBreath Lite [40] is an interactive breathing training app which uses the smartphone's built-in microphone to capture breathing and provide real-time audio and visual feedback. Stress Doctor [39] is another app that introduces deep breathing techniques. In order to score the effectiveness of the exercise session, it also measures heart rate and heart rate variability (HRV) via smartphone's built-in camera.

Also, there have been applications for reminding users to take breaks to tackle the

problem of sedentary work style and long work sessions. CalmDown [23] is a desktop application for Mac for reminding a user to take breaks either in the form of going for a walk or deep breathing by fading in/out colors on the screen. More recently, the Let Me Relax [73] system has been developed. This system is a sedentary state recognition platform using accelerometer data from the smartphone and the smartwatch sensors. Then, it suggests simple relaxation techniques from a pool of interventions which include deep and slow breathing. A study with 22 participants showed that the system could help the intervention group reduce self-reported stress compared to the control group who didn't receive the relaxation notifications.

2.2.2 Sensors and biofeedback

Researchers and engineers have long been developing sensors for capturing physiology including respiration. FlexComp [49] is one of the early devices with high signal quality. It is a wired device (Fig. 2-2), with a control module, where several encoders can be attached. The sensors include surface electromyography (sEMG), electrocardiography (ECG), electroencephalography (EEG), skin conductance (SC), peripheral temperature, blood volume pulse (BVP), respiration amplitude, goniometer adapter, and force adapter. Later, wearable measurement devices came out in different form factors. For example, LifeShirt [98] is a garment that measures physiology and also serves as an electronic diary of symptoms and activities. LifeShirt (Fig. 2-1) comes with embedded inductive plethysmography sensors. It continuously monitors respiration, heart activity, inductive cardiography, motility, and posture. The captured data are displayed and stored on a handheld computer (Visor). After offline analysis, cardiorespiratory parameters like heart rate, RSA, tidal volume, stroke volume, pre-ejection period, apnea-hypopnea index, thoraco-abdominal coordination, and sighing can be extracted. Zephyr BioHarness [65] is a more recent device which is composed of a BioModule and an adjustable chest strap with conductive fabric sensors as shown in Fig. 2-3. It measures respiration (breathing waveform,

amplitude, rate), ECG data (heart rate, heart rate RR, heart rate variability and ECG), motion (3-axis accelerometry, activity), posture, and temperature. The recorded data can be transmitted wirelessly, or downloaded offline. The strap sensor within the chest belt measures the size differential between the expansion and contraction of the thoracic cavity and sends it to BioModule which produces the breathing waveform, estimates amplitude, and breathing rate. However, these devices were mostly designed as a tool for researchers and clinicians rather than focusing on the wearer and his/her needs and experiences.

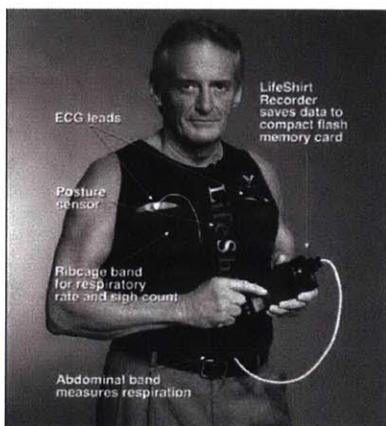


Figure 2-1: The LifeShirt



Figure 2-2: FlexComp



Figure 2-3: Zephyr BioHarness

McCraty is among the pioneers of scientifically using biofeedback for improving wellness through training, education, and self-monitoring [52]. He commercialized his research through emWave, an ear-clip sensor for measuring PPG that comes with a mobile and desktop application. Through training sessions, it guides users toward larger RSA amplitude by calculating a coherence score based on HRV.

RESPeRATE [43] is another device for guided breathing. This device composes rhythmic guiding tones in real-time, while measuring the user's respiration pattern. It is cleared by the Food and Drug Administration (FDA) and is available without a prescription. It has been verified through clinical trials as an effective method for reducing blood pressure and thus has been used for the treatment of hypertension [25, 93].

Moraveji conducted an elaborate set of studies in this area [56]. He prototyped a



Figure 2-4: Spire tracker and charger (left) and components (right)

breathing measurement device and several feedback mechanisms while focusing on the end-user. He developed Breathbelt for ambulatory continuous measurement of breath rate. He introduced peripheral paced respiration (PPR) for visual pacing in parallel to information work by a bouncing bar on the desktop screen [54]. He later introduced social features to PPR and studied how they affected the user. He identified design principles through an iterative design process [55] and later implemented Breathaware, a gamified drop-down menu in the system tray for informing users about their breathing by showing rate, estimating calm points, daily milestones, encouraging messages, and a buddy list. A next iteration was breathTray and then Breathwear in a mobile context. He created the “Calming Technology” lab at Stanford where they further studied inducing cognitive, affective, and physiological calm through technological interventions.

Moravji later created Spire [85], a wearable tracker that measures breathing patterns to encourage overall mindfulness and provide a more thorough guided meditation experience. Spire (Fig. 2-4) is worn on a belt or bra and captures breathing by a pressure sensor.

In recent years, more and more wellbeing trackers are coming to market with an increased focus on breathing. Leaf [90] is an example in the form of smart jewelry with motion sensors that can be worn as a necklace, bracelet, or snapped onto clothing. It has guided meditation sessions during which it captures respiration. Prana [71] is another tracker that is worn in the waist area. Prana evaluates breath patterns differentiating between diaphragmatic and chest breathing and posture. It is accompanied with an app



Figure 2-5: Leaf fitness tracker (left), Prana posture and respiration tracker (middle), Apple watch meditation app (right)

that provides active training sessions and gamifies breathing exercises. Apple has also announced a mindfulness app, called Breathe, for Apple Watch in WWDC 2016 [2]. Breathe coaches users through timed breathing sessions in order to help them de-stress. The app can be launched from the watch face or set up in advance with reminders and it measures heart rate throughout the session. Fig. 2-5 shows Leaf, Prana, and Breathe meditation app for Apple Watch.

2.2.3 Virtual environments

Virtual reality (VR) first came to existence through science fiction in 1935 [97]. However, it soon evolved and now several VR headsets are available off-the-shelf including Oculus Rift, HTC Vive, Samsung Gear VR, Google cardboard, Microsoft HoloLens, and many more. VR has been widely used for entertainment including games, films, media, concerts, museums, and theme parks. It has also been used in urban and architecture design and for retail purposes. Another VR usage has been in training, mostly for military applications. VR provides a low- or no-risk simulated environment for training novice personnel. Also, medical and therapeutic applications of VR have emerged recently. Surgical simulators [84], exposure therapy for post traumatic stress disorder (PTSD) patients [75],

treating anxiety and phobias [64] and acute pain [36, 58, 80] are a few examples. As VR is capable of engaging user's senses and recreating environments at low cost, it is also a good candidate for mindfulness meditation training [38].

Meditation chamber [79] is an immersive virtual environment primarily designed for meditation training and secondarily for reducing stress, anxiety, and pain. Meditation Chamber utilizes biofeedback sensors to monitor arousal. It then visually changes the virtual environment. Several visualizations have been implemented including setting sun and rising moon and mirroring actions during the progressive muscle relaxation. Results from a controlled study showed that the virtual environment has been more effective than the biofeedback alone.

Virtual Meditative Walk (VMW) [30] is another responsive virtual environment specifically designed to teach mindfulness based stress reduction (MBSR). The goal of MBSR is to enable people to reduce stress and improve their health via maintaining and improving psychological states. VMW (Fig. 2-6) allows users to walk in a virtual forest. It utilizes finger electrodes for measuring EDA and provides real-time visual and sonic feedback. The fog represents user's EDA levels and fades away when the EDA levels go down. Results from a study with chronic pain patients comparing the virtual environment to the control group were presented. The control users only practiced MBSR but did not interact with the VMW environment. The participants interacting with VMW reported reduced pain levels.

2.2.4 Responsive environments and objects

Recently, more immersive experiences have been created with the goal of cultivating mindfulness about body signals. This ranges from interactive objects to responsive architectures. Here, we mention the most prominent examples in this area.

Providing a physical responsive manifestation for the internal physiology can help interactive learning about the body states and processes and cultivating mindfulness about

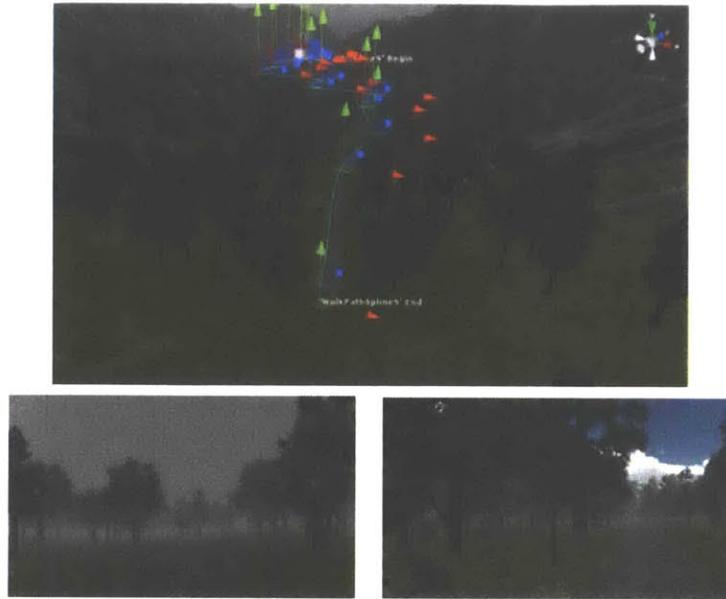


Figure 2-6: VMW virtual environment - As users reach an inferred meditative state, the fog dissipates and the sounds become more audible and spatial.



Figure 2-7: BodyVis and its evolution through three rounds of prototyping

them. BodyVis [60], an e-textile interactive shirt designed for children, is an example in this area (Fig. 2-7). BodyVis has dynamically removable organs that communicate with a Zephyr BioHarness [65] device and visualizes heart rate and breathing rate of the wearer. It also has a touchscreen stomach and visualizes eating and digestion process.

SonicCradle [92] is another example in the form of an interactive environment. It is a dark and quiet chamber with a hammock chair surrounded by four speakers (Fig. 2-



Figure 2-8: SonicCradle diagram and photograph

8). Participants' breathing is measured using Thought Technology sensors [48] attached to their abdomen and thorax. Software manipulates sound coming from different spatial directions in real-time using breathing data by changing volume, equalization, and reverb. The goal of SonicCradle is to provide a stress-relieving experience through which users gain a heightened breath awareness.

ExoBuilding [76] is another example of an adaptive architecture which takes physiological data into account. ExoBuilding (Fig. 2-9) utilizes a Mind Media NeXus10 [59] device and finger electrodes to measure electroencephalogram (EEG), electrocardiogram (ECG), respiration, and electrodermal activity (EDA). The size of the prototype is mapped to the occupant's respiration. The embedded LEDs in the fabric blink with the same frequency as heartbeat and the speaker system sonifies the heartbeat. Graphics are projected on the fabric and their visibility corresponds to the EDA data. A controlled study comparing three conditions of static, regular movement, and biofeedback with ExoBuilding suggested that the responsive environment condition successfully triggered behavioral and physiological changes such as lower respiration rates and higher respiration amplitudes compared to the other two conditions.

Another recent study applied biofeedback to ambient lighting in a home environment during which users could self-regulate their breathing patterns to improve their HRV [99]. Users wore a pulse sensor that communicated heart rate features to the environment. Light

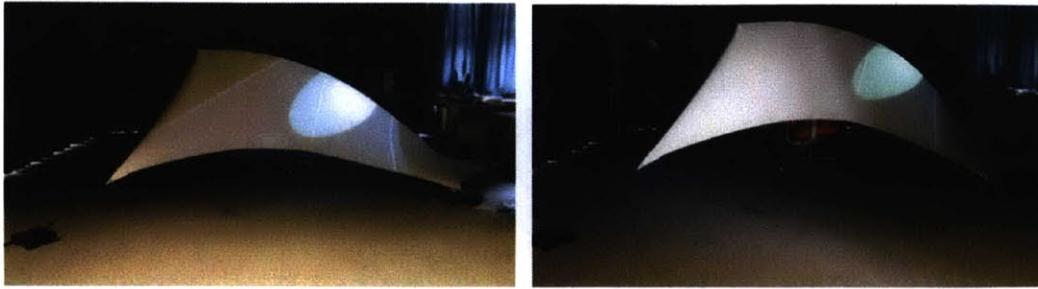


Figure 2-9: ExoBuilding adaptive architecture: contracted (left) and expanded (right)

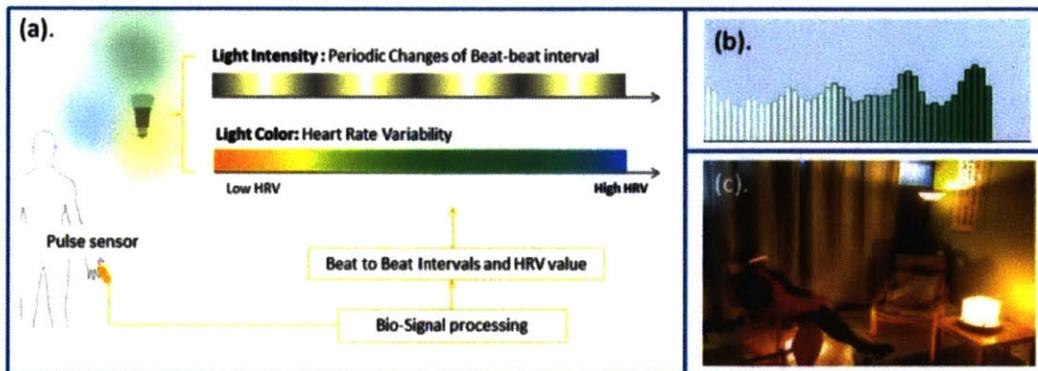


Figure 2-10: Ambient lighting biofeedback. (a) biofeedback mechanism. (b) Traditional graphical interface. (c) Responsive environment.

intensity periodically changed to resemble beat-to-beat intervals and the light color would represent HRV value. A controlled study showed the effectiveness of the environment compared to a traditional graphical interface. Fig. 2-10 shows the experimental setting.

Chapter 3

BrightBeat: A set of unobtrusive calming interventions for personal use

3.1 Physiological synchrony

In the previous sections, we mentioned the psychophysiological influences of voluntary breath modification. We also mentioned the role that technology has played in creating meditative experiences and measuring and influencing respiratory patterns. However, most of these technologies are designed to require full attention of the user and are not designed to work while the user is engaged in everyday life activities (See examples in sections 2.2.1 and 2.2.2 and 2.2.3). Also, many of them primarily rely on user's motivation and ability rather than the fast and automatic mental processes for behavioral change (which Adams et al. refer to as mindless computing [1]). More recent examples have explored peripherally informing users [56]. However, they are still designed to engage the user cognitively rather than automatically influencing him/her. Other examples include creating more immersive experiences to convey biofeedback response to the user in an ambient manner (Section 2.2.4). However, these systems rely on extra resources which make them less scalable. Also, they don't preserve the privacy of the user as they broadcast and magnify the physiological changes of the user into the physical environment.

Physiological synchrony and emotional contagion are known phenomena [34]. Levenson and Ruef have written a chapter in which they have provided several examples when ANS systems synchronize such as between patients and therapists, students and teachers, dyads and groups, mother and infant, spouses, and even in viewing oneself on videotape [46]. These examples show that the synchronization doesn't necessarily require participants to be intimately engaged in a face-to-face interaction, but can also happen across physical (in videotape) or interpersonal (between strangers) distance. This motivates further exploring the role of technology in this mirroring process.

In other contexts such as human circadian rhythm [22], technology has been shown to affect the body's biological rhythm. Several scholars have studied the effect of evening exposure to LED-backlit displays on circadian physiology which results in delayed sleep (See [18] for example). Solutions such as the f.lux application [26] have evolved to adjust screen blue light emission to address such problems. These examples show technology can be a promising medium to influence biological rhythms for improving wellbeing.

3.2 BrightBeat design

In this regard, we have designed BrightBeat. BrightBeat recreates a rhythmic oscillation similar to a calming breathing signal in the form of visual, auditory, or haptic feedback which overlays what the user sees, hears, or senses. Its goal is to rely more on automatic processes of physiological syncing rather than cognitive effort for reaching a desired state. It comes in the form of a set of unobtrusive interventions to be used on personal electronic devices to facilitate self-regulation while being engaged in other activities. These interventions are designed to require minimal cognitive attention to help self-regulate effortlessly. They utilize primary functionalities of personal electronic devices and are thus, scalable. They respect privacy of the user by providing seamless interventions on a personal level.

We have built several BrightBeat interventions in the form of visual, auditory, and

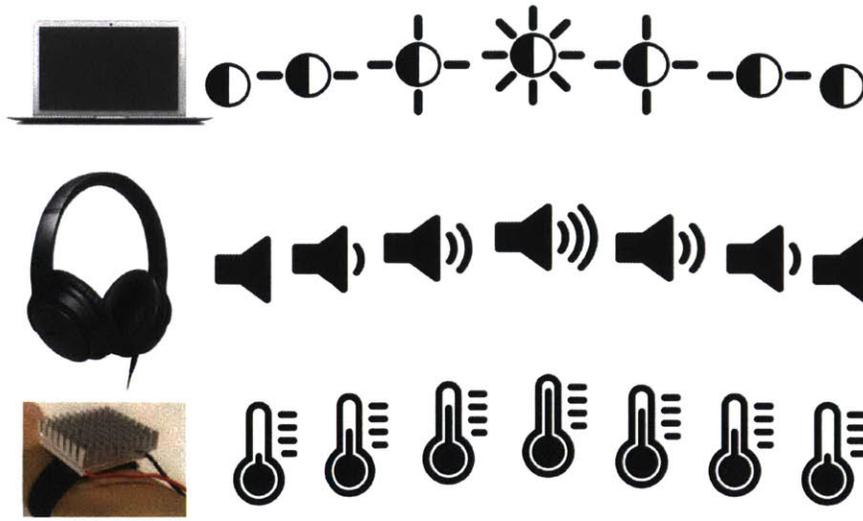


Figure 3-1: Schematics of BrightBeat systems.

thermal feedback. Each of these components are standalone, but they can also be combined. The visual BrightBeat changes the brightness of the screen with the same frequency of a calming breathing rate. The brightness changes are barely observable and may not be noticed if the user is deeply focused on another task. The audio BrightBeat oscillates the volume with the same frequency while playing white noise through headphones. We have chosen white noise in order to remove bias from playing specific type of music. However, the auditory component can also oscillate at ambient sound or music. Again, the minimum and maximum volume are barely audible. The thermal BrightBeat oscillates the temperature of a custom built wristband with the same frequency. The cool and warm temperature limits are minimally perceptible. Fig. 3-1 schematically illustrates an exaggerated version of the three mentioned modules.

In summary, we envisioned BrightBeat as a system that:

- measures breathing unobtrusively;
- on a personal level, subtly notifies the user if (s)he is breathing too fast;
- is easy to implement and scale;
- doesn't require continues conscious attention.

The next two chapters describe the iterative process of designing BrightBeat interventions and their detailed explanations through a set of user studies.

Chapter 4

Experiment 1 - Manual

In order to understand how users respond to BrightBeat and test its efficacy, we developed a prototype with which participants interacted in the context of a lab study. This chapter explains the iterative process of the design of the initial system and the respective data analysis. Note that the focus of this study was to explore the idea of BrightBeat through iterations. As a result, instead of focusing on the functionality and fully automating the process, we rather focused on the design iterations and kept a human in the loop to accelerate the implementation process. After building a proof-of-concept system and analyzing the initial user data, we found promising results. Thus, we improved the system and ran further studies which are explained in the next chapter.

4.1 Pilot study 1

As introduced earlier, we envisioned BrightBeat as a system that:

- measures breathing unobtrusively;
- on a personal level and in a calming manner, notifies the user if (s)he is breathing too fast.

Toward this end, we utilized Spire [85] for measurements. Spire is a stone-shaped wearable device that clips to clothing and measures physical movement, position, and breathing patterns using a pressure sensor. Spire has an iOS consumer app and an API. In terms of raw respiration data, the app interface shows a smoothed plot of breathing waveform. Also, through the API we have access to breathing rate in a given interval.

The first notification mechanism we came up with was mimicking inhalation and exhalation by brightening and darkening the screen. Consequently, we developed a software with an interface for receiving raw breathing waveform and rate along with two parameters, minimum (minB) and maximum brightness (MaxB) values. The software would mirror the user's breathing by altering screen brightness between the minimum and maximum brightness limit. The brightness was matched to raw breathing waveform data.

In order to test the usability of the system, we recruited 3 participants (1 male) to interact with BrightBeat. We explained how the system works and that it's there to give users breathing awareness. We also instructed them to breathe deeply and slowly to self-regulate when stressed. Participants wore a Spire device and a Zephyr BioHarness and spent approximately 3 minutes listening to a calm piano piece of music. Due to unavailability of real-time breathing data by Spire's API, we utilized a wizard-of-oz method where the experimenter looked at the breathing waveform provided by the Spire app and changed the system's response manually. Afterwards, participants read news articles of their choice on a MacBook with BrightBeat installed on it for up to 20 minutes.

All participants mentioned that mirroring exact inhalation and exhalation, which is usually irregular is stressful. They also mentioned it is hard to know if you are doing the correct thing when you are receiving feedback all the time. However, they liked the idea of being subtly informed about their breathing and preferred a more rhythmic feedback. A user mentioned: "I knew breathing can calm you down (or at least I thought so). But I haven't tried controlling it actively. I was surprised how powerful such a simple thing can be."

Based on users' feedback about the initial version of BrightBeat, we decided to upgrade to a rhythmic notification mechanism and test it with a larger group of people in a controlled lab study setting. This section explains the process in detail.

4.1.1 System design

To capture more detailed physiological data, we utilized Zephyr BioHarness [65] instead of Spire. This device is composed of a BioModule and an adjustable chest strap with conductive fabric sensors. It measures respiration (breathing waveform, amplitude, rate), ECG data (heart rate, heart rate RR, heart rate variability and ECG), motion (3-axis accelerometry, activity), posture, and temperature. The recorded data can be transmitted wirelessly, or downloaded offline via a docking cradle using the "Log downloader" windows application that comes with the device. The strap sensor within the chest belt measures the size differential between the expansion and contraction of the thoracic cavity and sends it to BioModule which produces the breathing waveform, estimates amplitude, and breathing rate. To check the quality of Zephyr data, we also used SenseView [53] which is an Android application to visualize sensor readouts in real-time. It supports Zephyr BioHarness, but has access to a limited number of measurements. For example, it has access to breathing rate but neither amplitude nor waveform.

As per pilot participants' request, we turned constant feedback to a feedback that appears only if the user is breathing too fast. In this version, the system would monitor the user during a three-minute relaxation period and set a goal breathing rate (GBR) for each participant. Since breathing rate during cognitive tasks is naturally faster than during a relaxed state, and to account for individual differences in breathing habits, we defined GBR as a linear function of the user's mean breathing rate (MBR) during the relaxation period: $GBR = MBR + 2$. Whenever the user is breathing faster than GBR, BrightBeat would appear.

As changing brightness in a regular and rhythmic manner as opposed to mirroring the

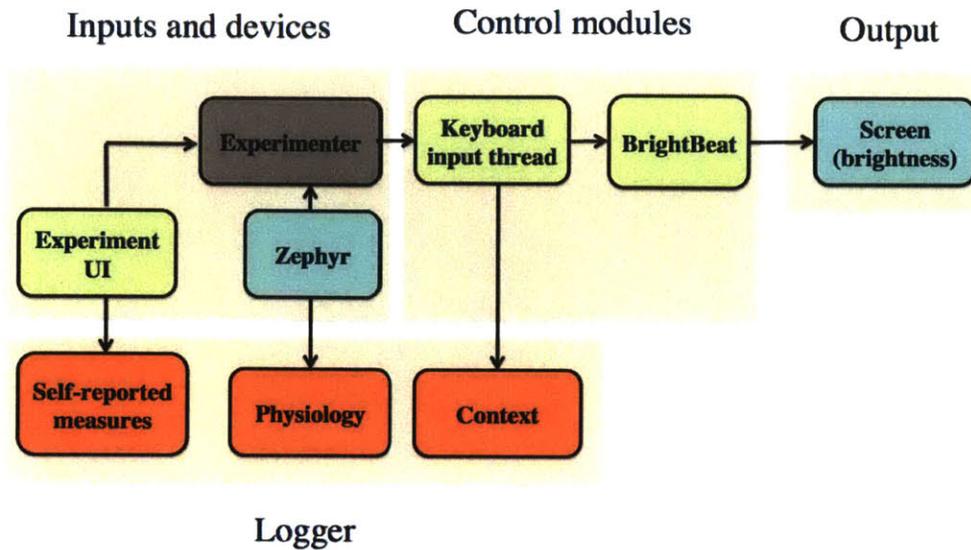


Figure 4-1: Pilot 1 - System design. Color codes: blue: hardware module, green: software module, orange: data, grey: experimenter.

exact inhalation and exhalation was more preferred, we slightly modified the BrightBeat software. In this version, BrightBeat was a regular beat starting from minB, going up linearly to maxB, and returning to minB again with the same frequency as GBR. Fig. 4-1 depicts the schematic system design.

4.1.2 Study protocol and deployment

In total, N=13 participants were recruited. Participants were graduate students from two different universities in the Boston area. To compare placebo effect versus BrightBeat effect, participants were randomly assigned to a control or intervention condition. The control group was told that they would receive an imperceptible version of the intervention where the changes were so subtle that they could not be seen consciously. The intervention group received the actual BrightBeat. They were told that BrightBeat is there to give them breathing awareness and it oscillates the screen brightness when they are breathing too fast. Due to an error in delivering the BrightBeat intervention for one user and due to an extremely high MBR in another during the relaxation task which resulted in extremely

high overall GBR, two users were dropped from the analysis. The final analysis is based on N=11 (4 female) participants consisting of 5 control and 6 intervention users.

The experiment was conducted in a lab setting and lasted between 25 to 35 minutes. It was composed of relaxation sessions, reading tasks, and quizzes. The experimental flow is shown in Fig. 4-2. First, participants started with a relaxation session and listened to a calming piano piece while sitting and breathing comfortably. Participants were instructed to proceed to the next page after they have reached a relaxed state. Consequently, the relaxation period ranged from 2 to 5 minutes for different participants. The experimenter calculated MBR from SenseView readings and set the system's parameters accordingly. Participants interacted with the system and confirmed the parameters, including minB, maxB, and GBR. Afterwards, they did a first reading task followed by a question set. Then, a second reading task was presented, followed by a set of questions. Finally, participants were told to relax while reading an article. They were informed about the fact that there was no quiz after the "relaxing and reading" task. In the end, the participants filled out a survey and rated their experience with the system along with their self-reported focus and calmness states. In order to elicit stress, which is associated with faster and irregular breathing patterns, participants were told that they would be tested on the tasks and their results would be compared to others. The participants were strictly timed during the tasks and the form would automatically proceed to the next page after the time limit. The experimenter was blind to the condition and only interacted with the participants before the reading tasks and after the session was over, to avoid affecting their breathing response to the system. The intervention group was exposed to BrightBeat during reading tasks and the final relaxation part but not during question sets.

Please refer to Appendix B for the participants' consent form and the experiment material for this round of the study.

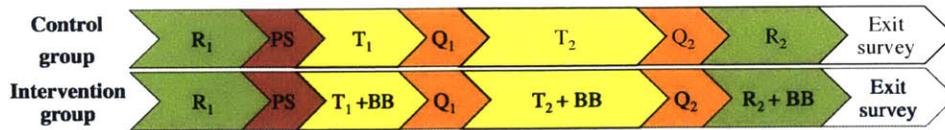


Figure 4-2: Pilot 1 - Experiment design. Pre XS: Initial survey and experience sampling. R1: Relaxation while watching video. T₁, T₂: Reading tasks. Q₁, Q₂: Quizzes. R₂: Relaxation while reading. Post XS: exit survey and experience sampling. PS: Parameter setting. BB: BrightBeat (which appears whenever the user breathes faster than his/her GBR).

4.1.3 Results

Analysis of physiological measurements

In order to show the effect of the intervention, we present a sample user from the intervention group (Fig. 4-3) and the control group (Fig. 4-4). The green overlay shows relaxation, red shows parameter setting, white shows reading, grey shows BrightBeat intervention, and yellow shows question answering.

As shown in Fig. 4-3, during the first relaxation session, the user is breathing deeply and calmly. During this phase, the breathing rate is mostly lower than GBR. It is also shown how the BrightBeat intervention influenced the user to breathe slowly and deeply. During the first reading task, the user started to breathe fast at the beginning of the task but as soon as he had received two BrightBeat interventions, he began breathing below his GBR for a few minutes. He became stressed near the end of the reading task as the time was going to be over and the BrightBeat appeared to him.

However, Fig. 4-4 shows a user in the control group. Most of the time, he is breathing faster than his GBR. At the beginning of the reading task, he is more aware of his breathing and is below his GBR. However, the effect soon wears off.

Fig. 4-5 shows the average breathing rate for all the participants during different tasks in the control and intervention groups with one standard deviation error bars. The red line shows the GBR for each user. As shown in the figure, the control group is usually above their personalized GBR unlike the intervention group. The MBR of each participant is

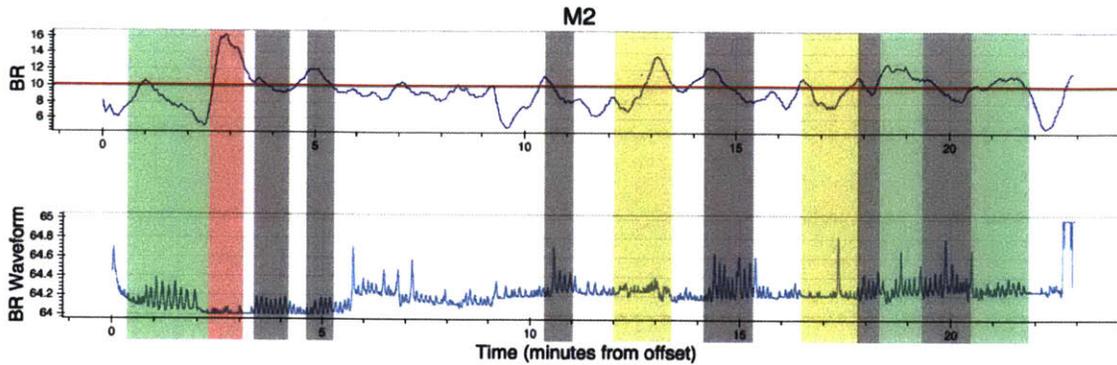


Figure 4-3: Pilot 1 - A sample user in the intervention group. The red horizontal line is the goal breathing rate (GBR) of 10 BPM. GBR is set 2 BPM above the mean breathing rate of the first relaxation session. When the user breathes faster than his GBR, BrightBeat appears. As plotted, delivering BrightBeat resulted in deep and slow breaths with some lasting influence that wore off only gradually. Color codes: grey: BrightBeat on, green: relaxation, red: parameter setting, white: reading, yellow: question answering.

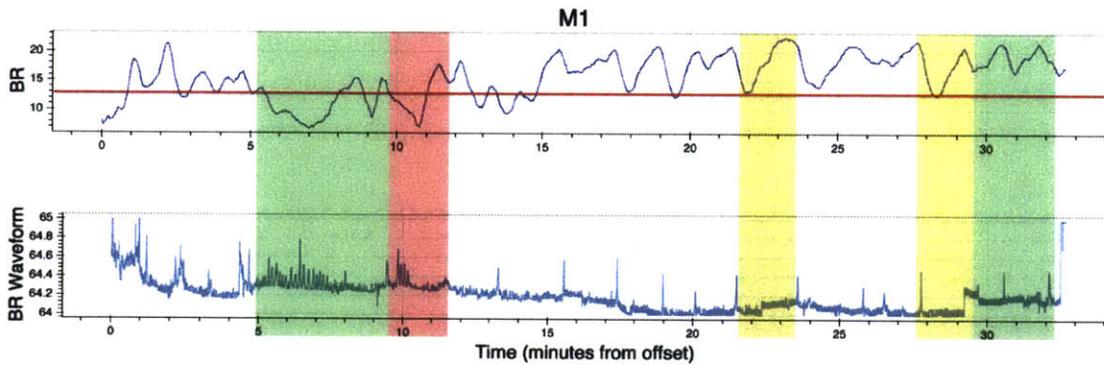


Figure 4-4: Pilot 1 - A sample user in the control group. The red horizontal line is the goal breathing rate (GBR) of 13 BPM. GBR is set 2 BPM above the mean breathing rate of the first relaxation session. This user is reaching his GBR only at the beginning of the first reading task being more aware of his breathing. Color codes: green: relaxation, red: parameter setting, white: reading, yellow: question answering.

shown above each plot. There is no significant difference between the average MBR of the two groups. Participants in the control and intervention groups are shown by blue and green color respectively.

Fig. 4-6 shows the task-by-task performance of the participants. We show the average difference between the breathing rate and the MBR for each user and each task with one

Average BR for all participants during different tasks

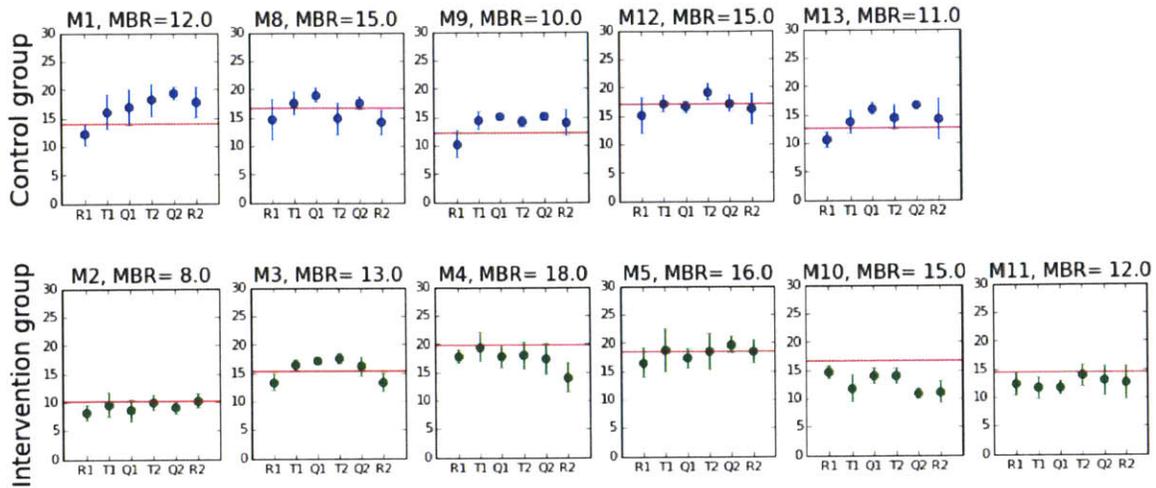


Figure 4-5: Pilot 1 - The average breathing rate of all the participants during different tasks with one standard deviation error bars. The individual MBRs are written on top of the plots and the red line shows the GBR. Intervention group users tend to reach their GBR more and during wider range of tasks.

standard deviation error bars. We have used MBR in this analysis to have a baseline of zero during the relaxation session. The red line shows zero. Blue and green refer to participants in the control and intervention group respectively. To visualize the difference more clearly, the points in each group are sorted. Overall, the intervention group is lower. This means that the intervention participants have been closer to their relaxed state of breathing compared to their control counterparts.

In order to measure how the average participant did in the control and intervention group, we calculated the percentage of the time spent below the GBR during each task for each group. Fig. 4-7 shows the results. On average, the control group reached their GBR less than 10% of the time while it was 68% for the intervention group. While answering questions, participants in the intervention group reached their GBR more than 70% of the time even though they did not receive the BrightBeat during that part of the experiment. However, the control group reached their GBR less than 1% of the time in the same condition. This hints at a lasting effect of the BrightBeat intervention. The difference

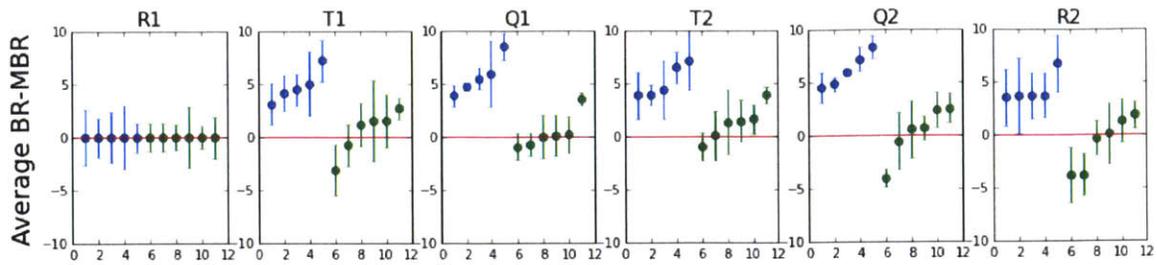


Figure 4-6: Pilot 1 - The task-by-task average difference between the breathing rate (BR) and the MBR with one standard deviation error bars. The red line shows zero. Blue and green points refer to the participants in the control and intervention group respectively and are sorted in ascending order. During the first relaxation task, the average breathing rate is the same as MBR and thus all points fall on the zero line. In the rest of the tasks, the intervention group is lower. This means that the intervention participants have been closer to their relaxed state of breathing compared to their control counterparts.

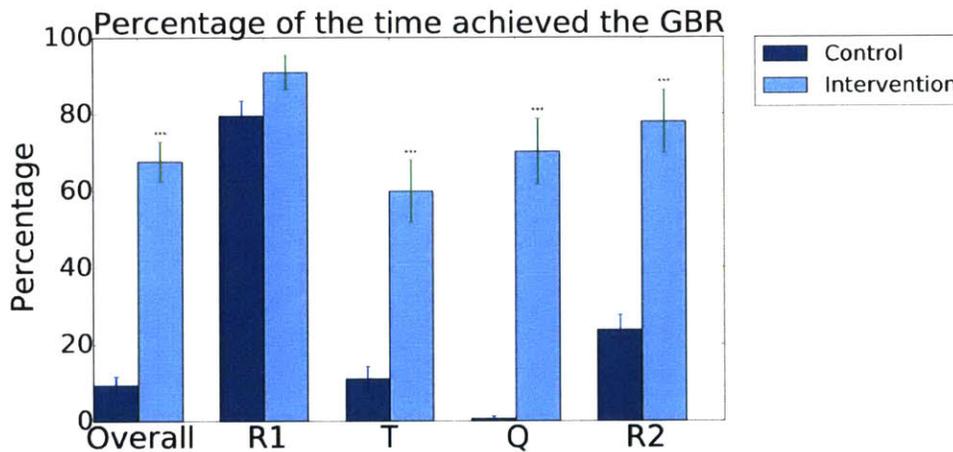


Figure 4-7: Pilot 1 - Percentage of the time achieved the GBR in each task. The intervention group has achieved its GBR more than the control group during several tasks. Overall: overall except baseline, R1: Relaxing and watching (baseline), T: reading tasks, Q: answering questions, R2: relaxing and reading. *** : $p < 0.001$.

between control and intervention in T, Q, R2, and overall is significant ($p < 0.001$).

For the detailed box-plot analysis of breathing rate data, please refer to the appendix (Fig. A-1).

Analysis of task performance

While this work focused on changing breathing rate, and did not make any hypotheses about task performance, we did want to examine whether BrightBeat, which could have cost some cognitive load or distraction, might have interfered with task performance. In order to objectively quantify the influence of BrightBeat on cognitive performance, we have looked at the quiz results. Fig. 4-8 shows the bar chart of the average user performance in each group, across different tasks with one standard deviation error bars. No significant difference was observed between the control and the intervention groups. This shows that BrightBeat did not negatively impact performance. However, it is important to mention that many factors influence task performance including the time of the day, the user’s fluency in English, and his/her prior knowledge about the task. Please refer to Appendix B for the content of the reading tasks and quizzes.

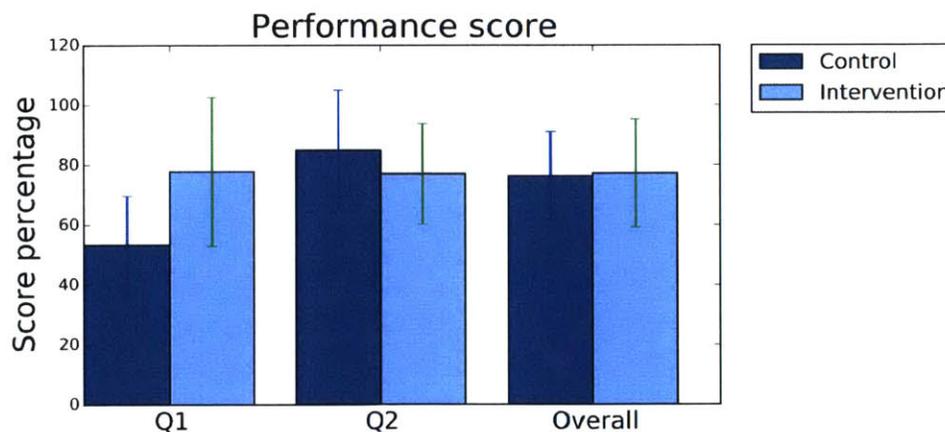


Figure 4-8: Pilot 1 - Average performance score of the control and intervention groups.

Analysis of self-reported measurements

Users’ self-reported ratings were also aggregated. Table 4.1 shows the average rating of each group for different survey items and the standard error value. The higher average

	Control	Intervention
How much did BrightBeat affect your breathing awareness?*	3.8 ± 0.5	5.8 ± 2.0
How did it affect your focus?***	4.5 ± 1.0	5.0 ± 2.1
How did it affect your stress/calmness level?***	4.5 ± 1.7	5.3 ± 1.6
Would you like to use BrightBeat in the future?*	5.8 ± 1.0	4.8 ± 1.8

Table 4.1: Pilot 1 - The average user rating (using 7-item likert scale questions). The higher average number in each row is highlighted in red. Overall, the intervention users reported higher focus and calmness compared to the control users. Extreme labels were as follows: *1: not at all, 7: very much, **1: decreased it significantly, 7: increased it significantly, ***1: stressed me out, 7: calmed me down.

number in each row is highlighted in red. Overall, participants in the intervention group reported improvements on focus and calmness more than the control group. However, more variance is observed in the intervention group. Both groups have a preference for using the system in the future. However, based on users' comments, we suspect that ratings from the control group for future usage may not be reliable. Users in the control group had difficulty defining the system. They could still self-report their experience throughout the lab study, but "using the system in the future" was vague for them. Some attributed it to a quiet place to work in, or the office environment, or even the time of the day instead of the placebo version of the BrightBeat. As a result, we have removed this question from the next rounds of the study as we believe it is not a reliable measure to compare the intervention group against. Please refer to the appendix for the detailed histogram of future usage preference (Fig. A-9).

User feedback

Users' feedback was a valuable source of information to improve the system. User M5 mentioned: "I like the way that it kind of hypnotized me! It really boosted my focus." User M13 mentioned: "I would like to try it in a real situation [...]". There were also constructive comments about increasing the flexibility of the system; M2 said: "It's cool that it reminds me to breathe calmly. But I want to limit it to a few times in an hour.

Something changing back there all the time becomes distracting." M7 mentioned: "When I want to relax, I like it. But sometimes I want to be excited rather than calm [...]" M12 said: "For me just a little signal would be enough [...] and [I] didn't need the rate to be dictated." M4 mentioned: "The settings were fine, but not the best. If they were the *best*, I would have felt much more comfortable with them." The comfortability of the wearables is very important as M6 stated: "The [Zephyr] band didn't let me breathe as easily as I normally do. It wasn't fair!"

Chapter 5

Experiment 2 - Automated

Primary results from the first experiment were promising. Thus, we made several improvements to the system and planned a larger study. In summary, we automated the system, minimized interactions between the experimenter and the user, changed parameter setting to make BrightBeat even less perceptible, updated instructions, and engaged auditory and tactile senses along with vision. This section explains the details of this process and the results of the corresponding studies.

5.1 Pilot study 2

5.1.1 System design

In this round, we highly improved the system design by automating the process and extending BrightBeat to engage other senses besides vision.

We utilized pyzephyr [61], an open source python library to connect to Zephyr Bio-Harness via bluetooth. We have used a subset of modules from pyzephyr. Through "Protocol" reading the data stream from the device and sending command messages to the device is available. "MessageFrameParser" is for parsing data messages from a serial stream. Also, "SignalCollector" supports collecting continuous and timestamped signal

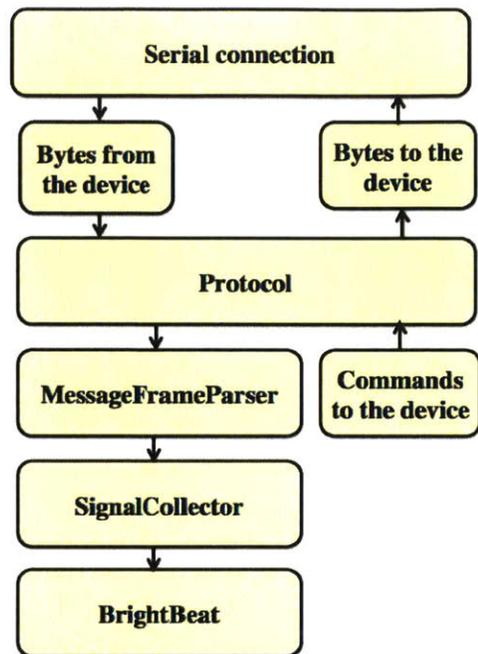


Figure 5-1: Schematic of pyzephyr and BrightBeat modules in relation to Zephyr Bio-Harness.

streams. Fig. 5-1 provides a schematic view of data flow between device and pyzephyr. Note that the relationship between the BrightBeat module and pyzephyr modules is also depicted. BrightBeat was triggered whenever the user was above his/her GBR.

Moreover, a second computer was added as the BrightBeat control terminal. This allowed the experimenter to stay away from the user, log-in to the computer running BrightBeat, and control the procedure. These automations made it easier for the experimenter, more accurate for BrightBeat delivery, and reduced bias in the data by minimizing interactions between the experimenter and the participant.

Also, BrightBeat evolved to a combination of interventions. Auditory and tactile senses were engaged as well as visual. SoundBeat was a new added module that would play white noise in the background and change the volume with the same frequency as the brightness module. The module had three input arguments, minimum volume (minV), maximum volume (maxV), and GBR. The maximum volume was barely perceptible. SoundBeat, similar to BrightBeat, was a regular beat starting from minV, going up

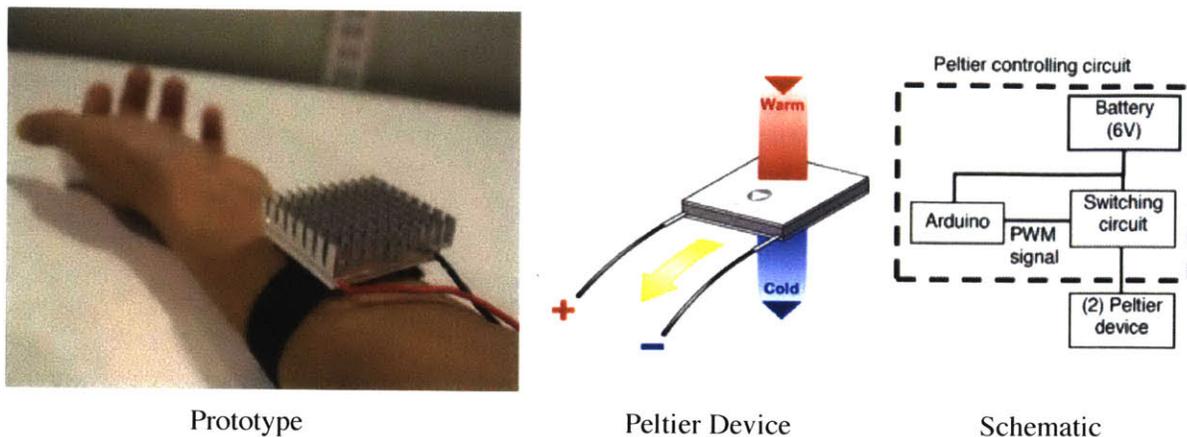


Figure 5-2: Custom wristband with oscillating temperature.

linearly to maxV, and returning to minV again with the same frequency as GBR. This oscillation would sound like an ocean wave.

Also, we developed a custom thermoelectric cooler (TEC) wristband that would alternate between cool and warm temperatures. This device works based on Peltier effect [83]: when a current is made to flow through a junction between two conductors of different materials, heat may be generated or removed at the junction. HeatBeat module was developed to control the Peltier device. HeatBeat would work similarly as the other modules. The wristband would start from the cool temperature, linearly go up to the warm temperature, then the power source would reverse voltage and it would return to the cool temperature. The power source would reverse voltage with the same frequency as GBR. The temperature changes were very subtle and the cool and warm extremes were barely perceptible. Fig. 5-2 shows the wristband prototype and circuit schematics.

Fig. 5-3 summarizes the updated version of the system.

5.1.2 Study protocol and deployment

N=6 graduate students from three different universities in the Boston area were recruited among which 4 were females and the rest were males. Participants were randomized into control (2) and intervention (4) groups.

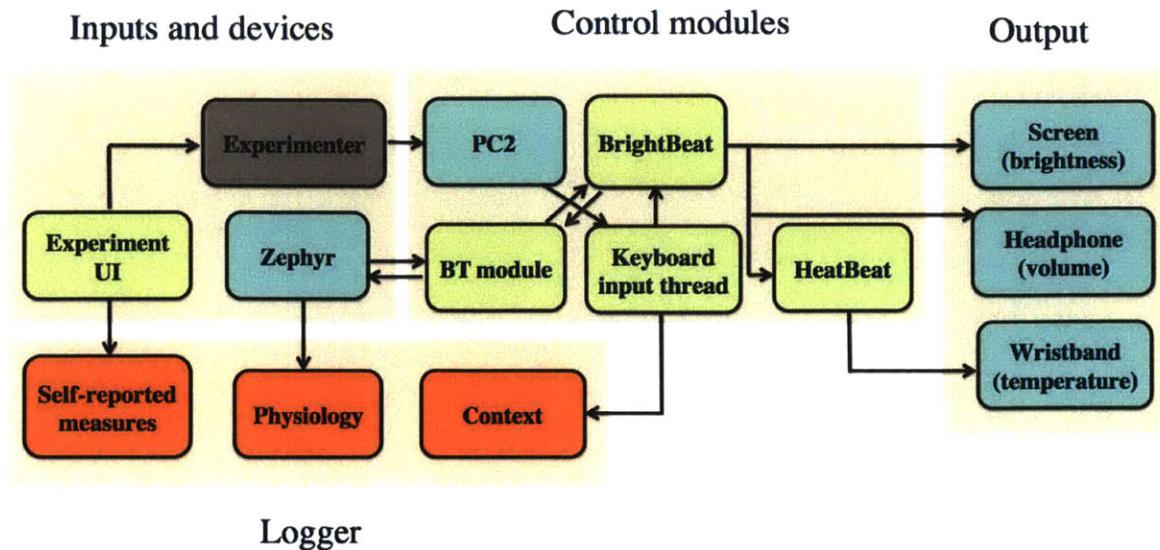


Figure 5-3: Pilot 2 - System design. Color codes: blue: hardware module, green: software module, orange: data, grey: experimenter.

In this round, we applied minor modifications to the study procedure. First, we tweaked the relaxation part. In study 1, some participants became engaged with the music video instead of becoming calmed by it. Some were more distracted, were fidgeting, or paying attention to the study environment. As a result, the physiological responses didn't represent a resting state for some of the participants. As GBR was set according to the relaxation phase, it wasn't a good approximation of resting breathing rate for some of the participants. Thus, in the second round, we included a short introduction video about meditation followed by a guided breathing exercise to make it easier for participants to reach a relaxed state. Also, we included 3 tasks with different lengths and difficulty levels. The content and questions were updated, so that the returning participants from study 1 weren't familiar with them. Also, a few questions were added in the beginning of the study for measuring self-efficacy and user's motivation in order to cancel out the effect of such social-cognitive elements on the response to the intervention. In study 1, users were asked to rate how BrightBeat affected their focus and calmness. However, we did not capture their initial levels of calmness and focus. As a result, we added pre and post ex-

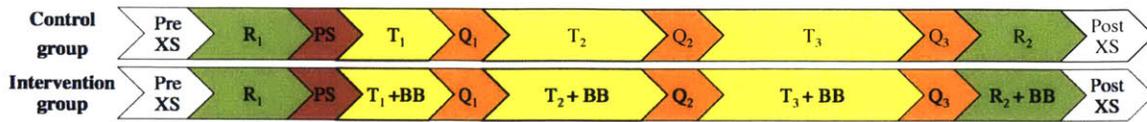


Figure 5-4: Pilot 2 - Experiment design. Pre XS: Initial survey and experience sampling. R1: Relaxation while watching video. T₁, T₂, T₃: Reading tasks. Q₁, Q₂, Q₃: Quizzes. R₂: Relaxation while reading. Post XS: exit survey and experience sampling. PS: Parameter setting. BB: BrightBeat (which appears whenever the user breathes faster than his/her GBR).

experience sampling which captured self-reported focus, calmness, and emotional valence before and after the study. This would enable us to account for the variations in initial stress levels and observe how they affected participants to respond differently to the intervention. The experimental flow is shown in Fig. 5-4. Last but not least, we changed the instructions so that the user didn't feel obliged to respond to BrightBeat. We mentioned that the goal of the study was reading tasks and answering questions, rather than focusing on BrightBeat which is only a reminder for fast breathing: "These oscillating interventions are not there to tell you exactly when to breathe. You do NOT have to inhale/exhale when it is light/dark, mute/unmute, cold/warm. Instead, you should just try to focus on your task. These interventions are only occasionally there to remind you to think about slowing your breathing or to breathe in a fuller and more relaxed way (e.g. you may decide to reposition yourself, and sit in a way where your chest can expand more fully). BrightBeat, HeatBeat, and VolumeBeat are not tasks. They are only gentle reminders to breathe in a way that you think is best to help you do your task." Given these tweaks, the experiment took 40 to 50 minutes in total.

Please refer to Appendix B for the participants' consent form and the experiment material for this round of the study.

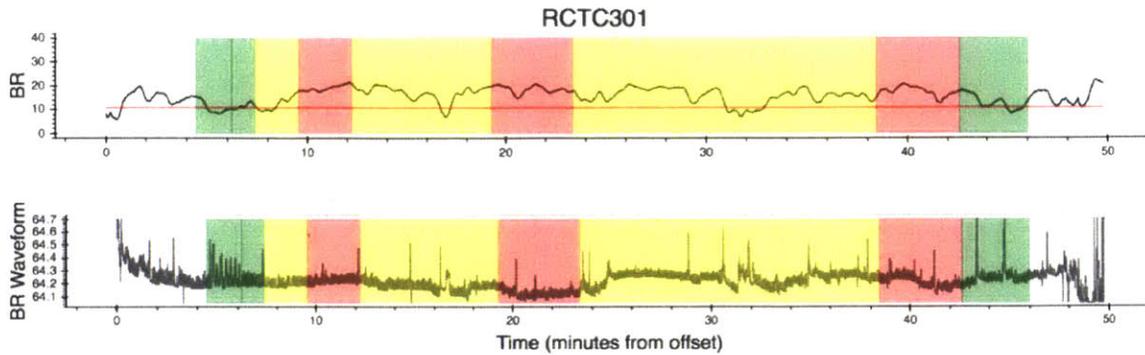


Figure 5-5: Pilot 2 - A sample user in the control group. The red horizontal line is the goal breathing rate (GBR) of 10 BPM. GBR is set according to the first relaxation session. This user is mostly breathing faster than her GBR and only occasionally slows down by taking deep breaths or sighing. Color codes: green: relaxation, yellow: reading, red: quizzes.

5.1.3 Results

Analysis of physiological measurements

Figures 5-5 and 5-6 show an example user in the control and intervention group respectively. The first observation when comparing the two plots, is that the control user is mostly breathing much faster than her GBR and only occasionally slowing down by taking deep breaths or sighing. However, the intervention user is usually below his GBR and sometimes going up during the quizzes where BrightBeat has been off. However, both users have healthy variability of breathing rate.

Fig. 5-7 shows the task-by-task performance of the participants. We show the average difference between the breathing rate and the MBR for each user and each task with one standard deviation error bars. The red line shows zero. Blue and green refer to participants in the control and intervention group respectively. To visualize the difference more clearly, the points in each group are sorted. Overall, the intervention group is lower. This means that the intervention participants have been closer to their relaxed state of breathing compared to their control counterparts.

In order to measure how the average participant did in the control and intervention

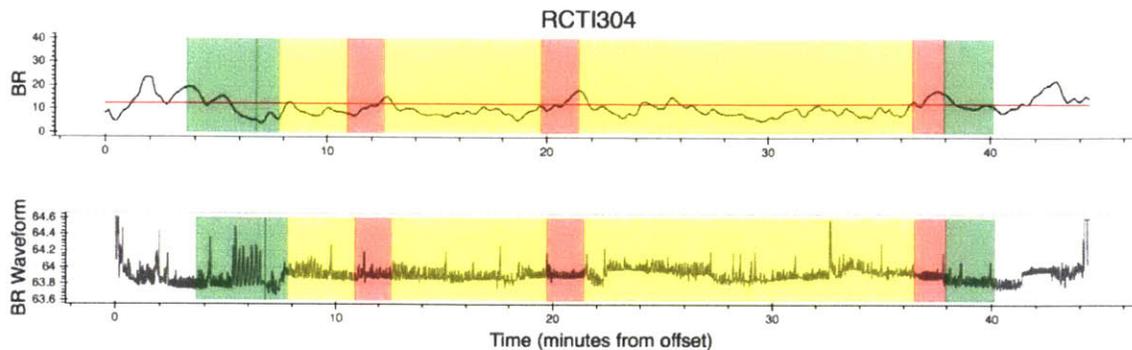


Figure 5-6: Pilot 2 - A sample user in the intervention group. The red horizontal line is the goal breathing rate (GBR) of 11 BPM. GBR is set according to the first relaxation session. When the user breathes faster than his GBR, BrightBeat appears. This user is often reaching his GBR except a few times including during the quizzes where BrightBeat has been off. Color codes: green: relaxation, yellow: reading, red: quizzes.

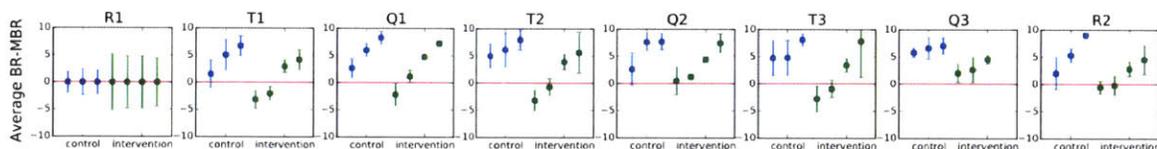


Figure 5-7: Pilot 2 - The task-by-task average difference between the breathing rate (BR) and the MBR with one standard deviation error bars. The red line shows zero. Blue and green points refer to participants in the control and intervention group respectively and are sorted in ascending order. During the first relaxation task, the average breathing rate is the same as MBR and thus all points fall on the zero line. In the rest of the tasks, the intervention group is lower. This means that the intervention participants have been closer to their relaxed state of breathing compared to their control counterparts.

group, we calculated the percentage of the time spent below the GBR during each task for each group (Fig. 5-8). An overall trend is observed which is the intervention group reached their GBR more than the control group even during the tasks where BrightBeat was not presented. However, the detailed task-by-task difference for control and intervention groups is not significant. Fig. 5-9 shows the results aggregated for different types of the tasks. The intervention group are reaching their GBR more than the control group overall ($p < 0.001$) and during the reading tasks ($p < 0.01$). The same trend is observed during quizzes and the final relaxation task though not significant.

Please refer to the appendix for more detailed analysis plots regarding average breath-

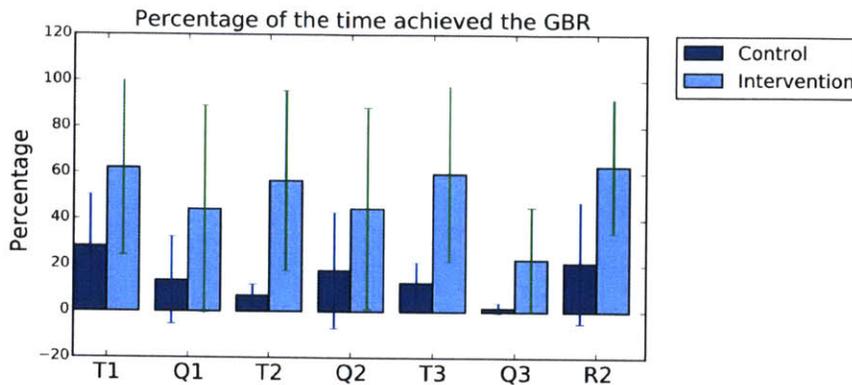


Figure 5-8: Pilot 2 - Percentage of the time achieved the GBR in each task averaged over all participants in each group. The intervention group has achieved its GBR more than the control group during several tasks. T₁, T₂, T₃: reading tasks, Q₁, Q₂, Q₃: quizzes, R2: relaxing and reading.

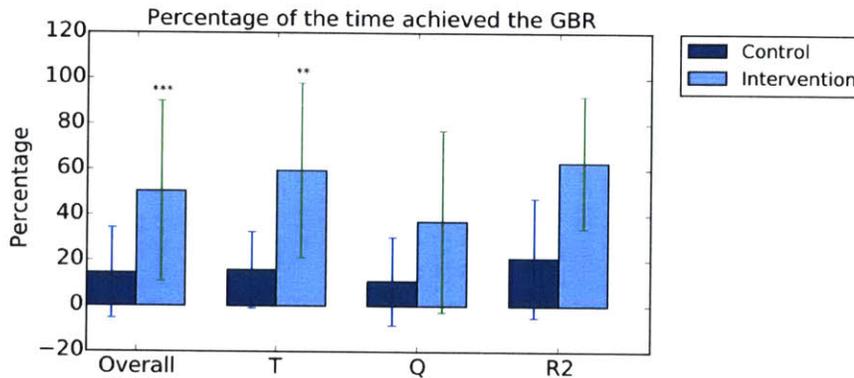


Figure 5-9: Pilot 2 - Percentage of the time achieved the GBR in each task. The intervention group has significantly achieved its GBR more than the control group during several tasks. Overall: overall except baseline, T: reading tasks, Q: quizzes, R2: relaxing and reading. *** : $p < 0.001$, ** : $p < 0.01$.

ing rate for all participants during different tasks (Fig. A-4), task-by-task box-plot analysis of breathing rate data (Fig. A-3) and the observed relation between rate and amplitude (Fig. A-2)

Analysis of task performance

In order to objectively quantify the influence of BrightBeat on cognitive performance, we have looked at the quiz results. Fig. 5-16 shows the bar chart of the average user performance in each group, across different tasks with one standard deviation error bars. The only significant difference was observed in Q1 response in which the intervention users performed better than the control users ($p < 0.01$). However, no other significant difference was observed between the two groups. This shows that BrightBeat did not negatively impact performance. However, it is important to mention that many factors influence task performance including the time of the day, the user's fluency in English, and his/her prior knowledge about the task.

Please refer to Appendix B for the content of the reading tasks and quizzes.

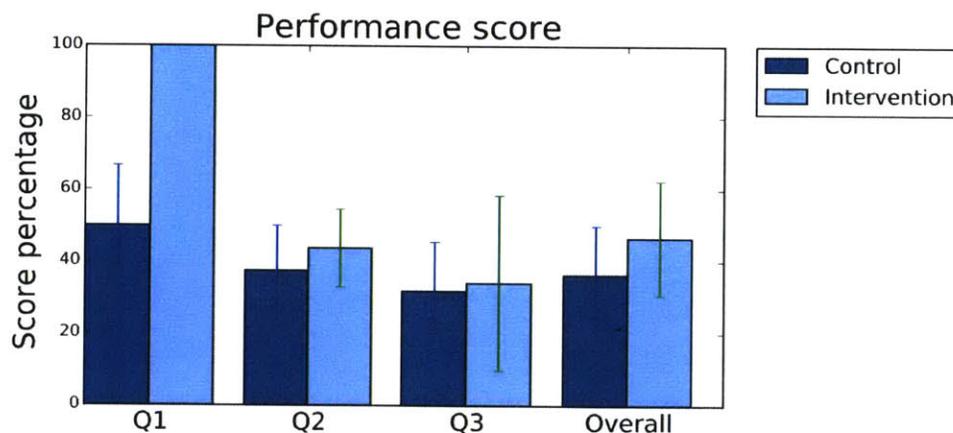


Figure 5-10: Pilot 2 - Average performance score of the control and intervention groups.

Analysis of self-reported measurements

Table 5.1 summarizes user ratings. The highest average number in each row is highlighted in red. Overall, the intervention group reported improvements in focus and calmness compared to the control group. Among different BrightBeat components, the response to the oscillating volume has been the most positive.

	Control Overall	Intervention			
		Brightness	Volume	Temperature	Overall
Awareness*	2.5 ± 1.0	2.3 ± 1.5	5.3 ± 2.2	3.3 ± 2.1	4.5 ± 1.3
Focus**	3.0 ± 0.6	2.3 ± 1.0	3.8 ± 1.5	2.3 ± 1.9	3.8 ± 1.5
Calmness***	3.0 ± 1.2	2.3 ± 1.0	4.8 ± 1.9	3.8 ± 2.2	4.3 ± 1.0
Future usage*	-	3.0 ± 2.4	4.5 ± 1.3	2.5 ± 2.4	4.5 ± 1.3

Table 5.1: Pilot 2 - The average user rating (using 7-item likert scale questions). The highest average number in each row is highlighted in red. Overall, the intervention users reported higher focus and calmness compared to the control users. Extremes labels were as follows: *1: not at all, 7: very much, **1: decreased it significantly, 7: increased it significantly, ***1: stressed me out, 7: calmed me down.

However, we realized users' responses to different intervention mechanisms were completely different. Among 4 users, 1 highly preferred the brightness module, 1 the temperature module, and 2 the volume module. In this case, averaging the numbers does not provide a good representation. Instead, clusters of users who preferred a specific type of intervention can be averaged together. We will conduct a more comprehensive analysis of user feedback in the main study. Please refer to the appendix for the detailed histogram of future usage preference (Fig. A-10).

5.2 Main study

After the pilot study, we improved the system and deployed a larger study with more participants (N=32). Modifications include removing the temperature module, updating parameter settings section, and adding experience sampling after each task. This section explains the details of system design, procedure, and data analysis of this round.

5.2.1 System design

During the pilot study, we realized that the TEC wristband could go out of sync. The main reason was that HeatBeat controlled the Arduino board through a serial connection.

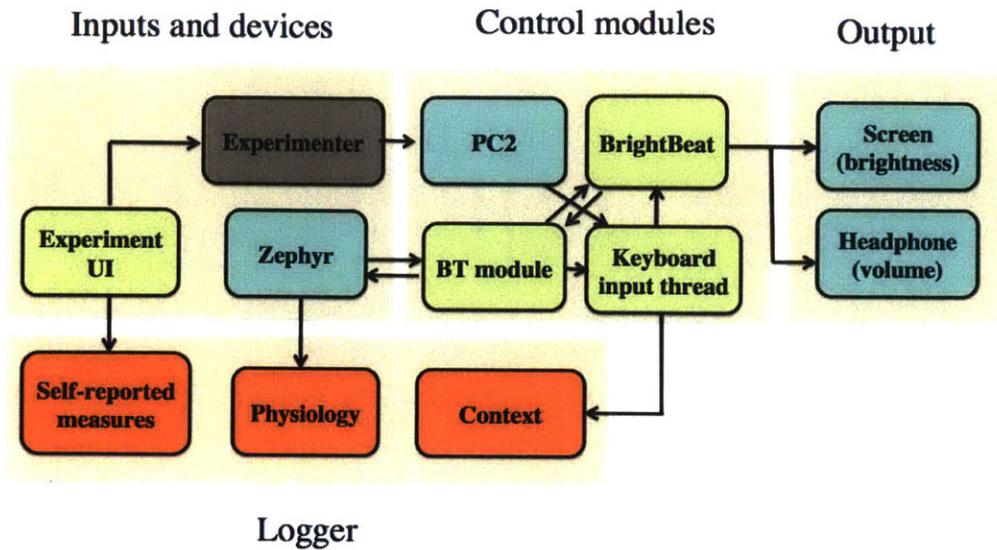


Figure 5-11: Main - System design. Color codes: blue: hardware module, green: software module, orange: data, grey: experimenter.

However, the brightness and sound modules were controlled with the same Python code in the BrightBeat main module. Thus, the brightness and volume oscillations were synced but the temperature change would face unnecessary delays frequently. Although some of the participants expressed particular interest in the temperature module, we had to remove it from the main study as it could confuse participants.

5.2.2 Study protocol and deployment

The study was advertised through printed notices over MIT campus and Media Lab mailing lists. A webpage was created for the study which informed participants about the procedure and allowed them to schedule their visit [28]. In total, N=32 participants including undergraduate (8), graduate (13), and staff (11) members of MIT were recruited among which 19 were females and the rest were males. Participants were randomized into control (13) and intervention (19) groups.

The experimental procedure was similar to the pilot study, composed of relaxation sessions, reading tasks, and quizzes. In the pilot study, we only sampled self-reported

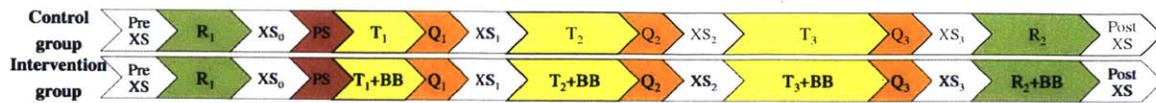


Figure 5-12: Main - Experiment design. Pre XS: Initial survey and experience sampling. R1: Relaxation while watching video. T₁, T₂, T₃: Reading tasks. Q₁, Q₂, Q₃: Quizzes. XS₀, XS₁, XS₂, XS₃: Experience sampling. R₂: Relaxation while reading. Post XS: exit survey and experience sampling. PS: Parameter setting. BB: BrightBeat (which appears whenever the user breathes faster than his/her GBR).

calmness, focus, and emotional valence before and after the study. In this round, we added experience sampling after each task, as well. First, it gave us a better and more detailed understanding of how the tasks and BrightBeat affected users' mood. Also, it allowed us to notice the participants with fluctuating mood reports vs. the ones who maintained a steady mood over the course of the experiment. This enabled us to account for resilience: someone's ability to maintain their current mood when introduced to a stressful task. The experimental flow is shown in Fig. 5-12.

In this round, a few details were changed. First, we tweaked the instructions. Besides mentioning that the goal of the study was to focus on the reading tasks and answering quizzes correctly, we stressed BrightBeat as an alternative method of notification which appears when the user is breathing fast and is probably tense. Thus, it might be helpful to respond to it either by adjusting posture or breathing more slowly and deeply.

Also, we slightly modified the parameter setting section. Instead of considering $GBR = MBR + 2$, we made GBR relative to MBR: $GBR = 120\% * MBR$. Participants could lower or heighten GBR between MBR and 140% of MBR until they found the best match for themselves. However, most of the participants continued with the original GBR. Given these tweaks, the experiment took 40 to 50 minutes in total.

Please refer to Appendix B for the participants' consent form and the experiment material for this round of the study. The detailed study protocol for the final version of the study and the COUHES approval letter are also attached to the appendix.

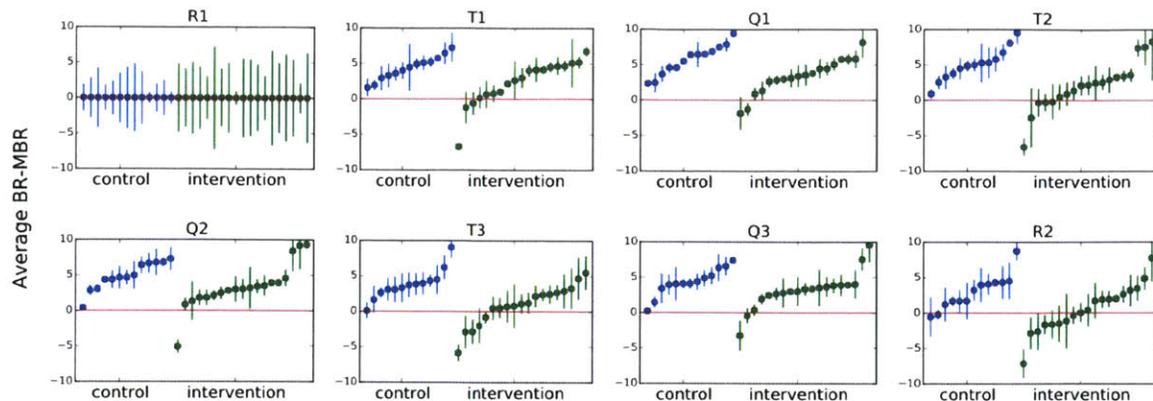


Figure 5-13: Main - The task-by-task average difference between the breathing rate (BR) and the MBR with one standard deviation error bars. The red line shows zero. Blue and green points refer to participants in the control and intervention group respectively and are sorted in ascending order. During the first relaxation task, the average breathing rate is the same as MBR and thus all points fall on the zero line. In the rest of the tasks, the intervention group is lower. This means that the intervention participants have been closer to their relaxed state of breathing compared to their control counterparts.

5.2.3 Results

Analysis of physiological measurements

Figure 5-13 shows the task-by-task performance of the participants. We show the average difference between the breathing rate and the MBR for each user and each task with one standard deviation error bars. The red line shows zero. Blue and green refer to participants in the control and intervention group respectively. To visualize the difference more clearly, the points in each group are sorted. Overall, the intervention group is lower which means they were able to hit their goal more frequently comparing to the control group.

In order to measure how the average participant did in the control and intervention group, we calculated the percentage of the time spent below the GBR during each task for each participant. Then, we averaged all participants in each group (Fig. 5-14). An overall trend is observed which is the intervention group reached their GBR more than the control group even during the tasks where BrightBeat was not presented. In other terms,

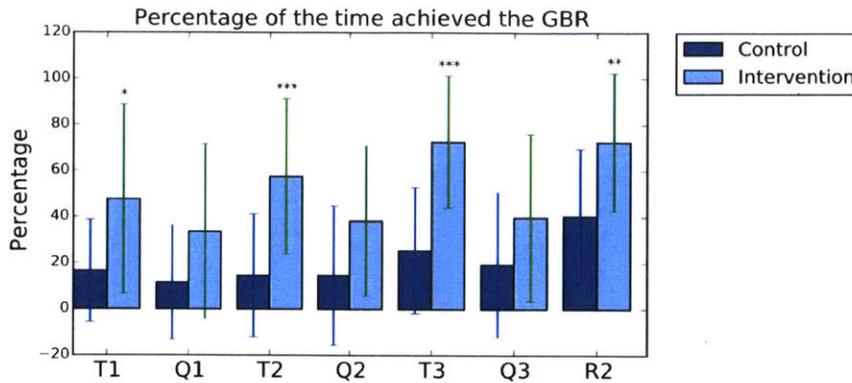


Figure 5-14: Main - Percentage of the time achieved the GBR in each task averaged over all participants in each group. The intervention group has significantly achieved its GBR more than the control group during several tasks. T₁, T₂, T₃: reading tasks, Q₁, Q₂, Q₃: quizzes, R2: relaxing and reading. * : $p < 0.05$, ** : $p < 0.01$, *** : $p < 0.001$.

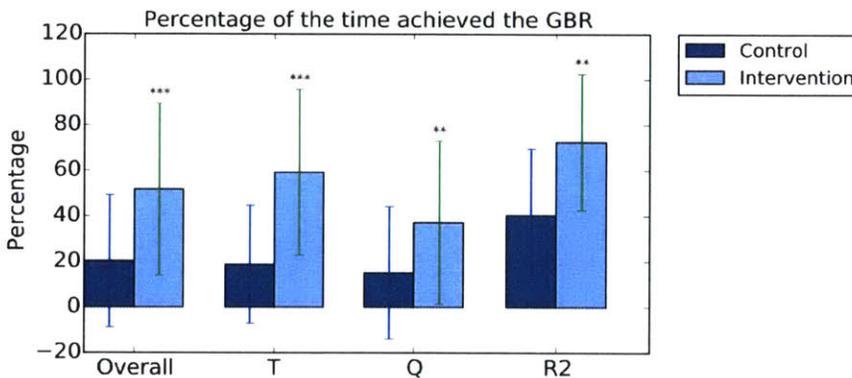


Figure 5-15: Main - Percentage of the time achieved the GBR in each task. The intervention group has significantly achieved its GBR more than the control group during all types of tasks. Overall: overall except baseline, T: reading tasks, Q: quizzes, R2: relaxing and reading. ** : $p < 0.01$, *** : $p < 0.001$.

the intervention group were breathing significantly more calmly during three different reading tasks ($p_{T1} < 0.05$, $p_{T2} < 0.001$, $p_{T3} < 0.001$) and the final relaxation session ($p_{R2} < 0.01$). We have aggregated the results for different types of tasks (Fig. 5-15). The intervention group are significantly reaching their GBR more compared to the control group overall ($p_{overall} < 0.001$), during reading tasks ($p_R < 0.001$), and quizzes ($p_Q < 0.001$).

Please refer to the appendix for more detailed analysis plots regarding average breathing rate for all participants during different tasks (Fig. A-7), task-by-task box-plot analysis of breathing rate data (Fig. A-6) and the observed relation between rate and amplitude (Fig. A-5)

Analysis of task performance

In order to objectively quantify the influence of BrightBeat on cognitive performance, we have looked at the quiz results. Fig. ?? shows the bar chart of the average user performance in each group, across different tasks with one standard deviation error bars. No significant difference was observed between the control and intervention groups. This shows that BrightBeat did not negatively impact performance. However, it is important to mention that many factors influence task performance including the time of the day, the user’s fluency in English, and his/her prior knowledge about the task.

Please refer to Appendix B for the content of the reading tasks and quizzes.

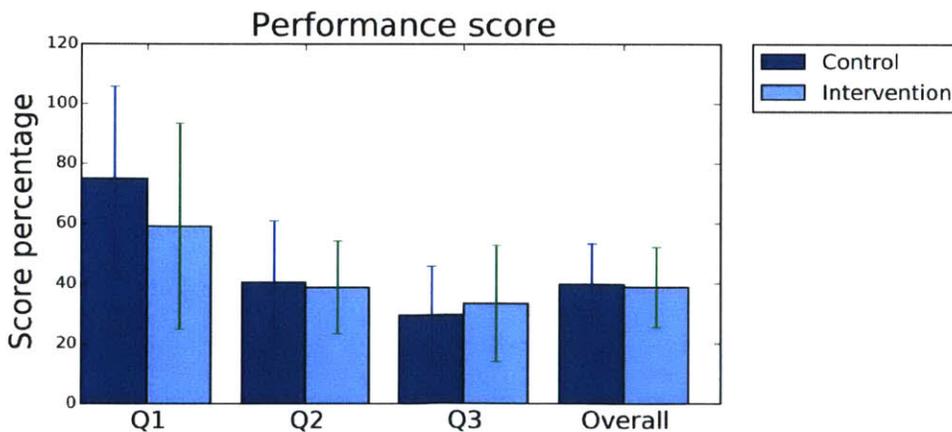


Figure 5-16: Main - Average performance score of the control and intervention groups.

	Control (N=13)	Intervention (N=19)		
	Overall	Brightness	Volume	Overall
Awareness*	4.1 ± 1.8	4.0 ± 1.8	4.2 ± 1.8	4.2 ± 1.6
Focus**	3.6 ± 1.1	4.1 ± 1.7	4.8 ± 1.3	4.5 ± 1.2
Calmness***	4.1 ± 1.0	4.3 ± 1.1	5.0 ± 1.5	4.8 ± 1.1
Future usage*	-	4.6 ± 1.8	5.4 ± 1.3	5.1 ± 1.4

Table 5.2: Main - The average user rating (using 7-item likert scale questions). The highest average number in each row is highlighted in red. Overall, the intervention users reported higher focus and calmness compared to the control users. Extremes labels were as follows: *1: not at all, 7: very much, **1: decreased it significantly, 7: increased it significantly, ***1: stressed me out, 7: calmed me down.

Analysis of self-reported measurements

Table 5.2 shows the summary of user ratings. The highest average number in each row is highlighted in red. Overall, the intervention group reported improvements in focus and calmness compared to the control group. Among different BrightBeat components, the response to the oscillating volume has been the most positive. Please refer to the appendix for the detailed histogram of future usage preference (Fig. A-11).

However, we observed different preferences among users for the visual and auditory components. Some preferred a specific intervention type for the future and some rated both the visual and the auditory elements the same. Table 5.3 averages the results for the favorite type of the intervention. In other terms, we removed the ratings from the least favorite intervention for the users in the intervention group and recalculated the results. Overall, 3 participants greatly favored the visual component. 12 greatly favored the auditory component. The remaining 4 rated both components the same. Consequently, the overall rating for the visual component is based on N=7 users (12 votes not counted) and the auditory component based on N=16 users (3 votes not counted).

Please refer to the appendix for the detailed time series of self-reported measures (Fig. A-8).

	Control (N=13)	Intervention	
		Brightness (N=7)	Volume (N=16)
Awareness*	4.1 ± 1.8	5.0 ± 1.2	4.4 ± 1.8
Focus**	3.6 ± 1.1	5.1 ± 1.4	4.8 ± 1.3
Calmness***	4.1 ± 1.0	5.0 ± 1.0	4.9 ± 1.5
Future usage*	-	5.8 ± 1.3	5.4 ± 1.3

Table 5.3: Main - The average user rating for the participants with the preferred type of intervention (using 7-item likert scale questions). Overall, the intervention users reported the preferred intervention to improve their focus and calmness compared to the control users. Extremes labels were as follows: *1: not at all, 7: very much, **1: decreased it significantly, 7: increased it significantly, ***1: stressed me out, 7: calmed me down.

User feedback

As mentioned earlier, our goal was to simulate a stressful task and analyze the effectiveness of BrightBeat in that context. Users' comments confirmed the stressful nature of the task in different ways: "The content of the articles was difficult to digest in such a short time without prior knowledge..."; "The timed reading and tests made me more stressed out."; "Very stressful, haha!"; "I was interested in the readings, but the fixed time limit stressed me out."

However, there were interesting insights about how different components of BrightBeat could help focus. Specially, users mentioned it was mostly attracting attention when they were distracted and it helped them regain focus: "The subtle changes were helpful at keeping my focus. [...] Every time my eyes started to feel tired, the screen would noticeably brighten and that helped me immensely. I would appreciate the use of this during the work day, but especially times when I'm feeling distracted." Another user mentioned: "I was only aware of subtle changes on the screen a couple of times, I think I became more aware of it the more distracted I was." Another user said: "The screen brightness was noticeable and helpful as a reminder...". Also, users mentioned the calming effect of BrightBeat, specifically the volume component. "As for the audio, I find listening to ambient sounds very relaxing. I often work with "noisli" playing in my headphones, even with music from time to time." Another user said: "The soothing sounds were great[...]".

Chapter 6

Conclusions and future work

6.1 Conclusions

We have designed and written BrightBeat software. We have built and tested entirely new kinds of interventions in the form of oscillation of screen brightness, headphone volume, and wristband temperature. We have evaluated these interventions through rigorous user testing. We have carried out a series of studies to explore the potential of bringing mindfulness to everyday activities through mindless computing and testing the hypothesis of physiological synchrony while interacting with technology.

We showed that the participants in the intervention group were closer to the breathing rate in their relaxed state and had significantly lower relative breathing rate. Also, the intervention users reported higher calmness.

We provided a successful example of behavior change intervention that does not require constant attention. The interventions were scalable, respectful of users' privacy, and did not demand attention and thus, could run alongside users' primary attentional task. In fact, they improved self-reported focus rather than distracting the user from his/her primary task. They also didn't affect the user's performance in the reading tasks and quizzes.

Our plan was not to design a one-size-fits-all solution, but instead respect users' differences and preferences. Thus, the interventions were tuned to each individual to be

the most effective. Also, we took users' preferences into account. Participants highly preferred to use their favorite interventions in the future.

6.2 Future work

We originally planned to measure breathing using personal electronic devices to make BrightBeat more scalable. For example, using the laptop camera, respiration rate can be extracted from the subtle color changes of the skin [68]. Also, using smartphone's built-in accelerometer, subtle motion changes can be captured and analyzed to provide respiration rate [35]. However, these methods are prone to motion artifacts and thus, we utilized a commercial sensor, Zephyr, to capture physiological data more reliably. In the future, a more robust method using camera or motion data analysis can replace the current sensor and highly improve the scalability of this work.

In order to measure the effectiveness of BrightBeat, we simulated a situation resembling everyday work-related stress. This allowed us to keep all the variables constant except the intervention and compare participants through the same situation of reading tasks and quizzes. However, people's response in real life situations may be different. Thus, future studies in real life settings will strengthen our analyses.

In this thesis, we have mostly focused on the breathing rate. However, the breathing pattern can also be further explored. For example, Diest et al. conducted an experiment with thirty participants who were instructed to breathe at 6 or 12 BPM, and with an i/e of 0.42 or 2.33. They showed that lower i/e ratio was associated with relaxation, stress reduction, mindfulness, and positive energy [91]. In the future, we would like to explore the role of i/e ratio in the effectiveness of the intervention. Also, more detailed analysis on respiration signal can be conducted including inter-breath intervals and frequency analysis.

Though our target was breathing rate and we provided analyses regarding the same target, we have gathered a set of physiological signals which allow us to answer more

questions and do further analysis. For example, we have gathered EDA on both wrists in the main study. Analyzing features from EDA (such as number of peaks, mean values, etc.) and asymmetry between the two wrists can be studied in control vs. intervention groups to further our understanding about the influence of BrightBeat interventions. Posture data gathered from Zephyr can be analyzed to see if there's a relation between breathing and posture and if BrightBeat indirectly affected posture. Also, given ECG from Zephyr and BVP from E4 wristbands, breathing in relation to heart functions can be studied.

Appendix A

Detailed data analysis plots

This section includes the detailed plots of data analysis for the different rounds of the study.

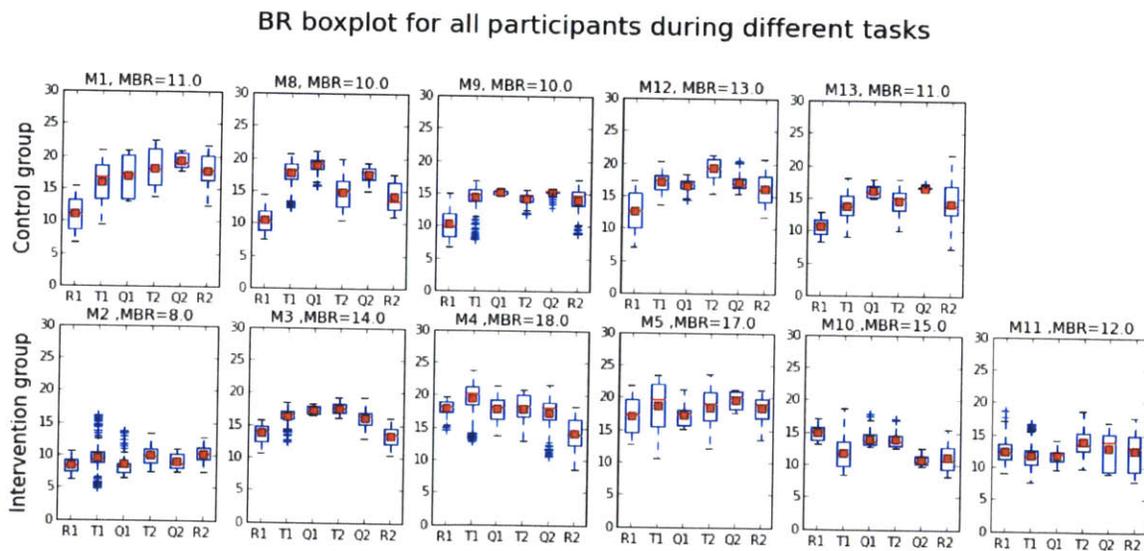


Figure A-1: Pilot 1 - Box plot of breathing rate for each individual in control and intervention groups. The individual MBRs are written on top of the plots. The red squares show the mean in each task for each user. This plot gives a more detailed view of participants breathing rates during several tasks.

Normalized amplitude versus BR for all participants

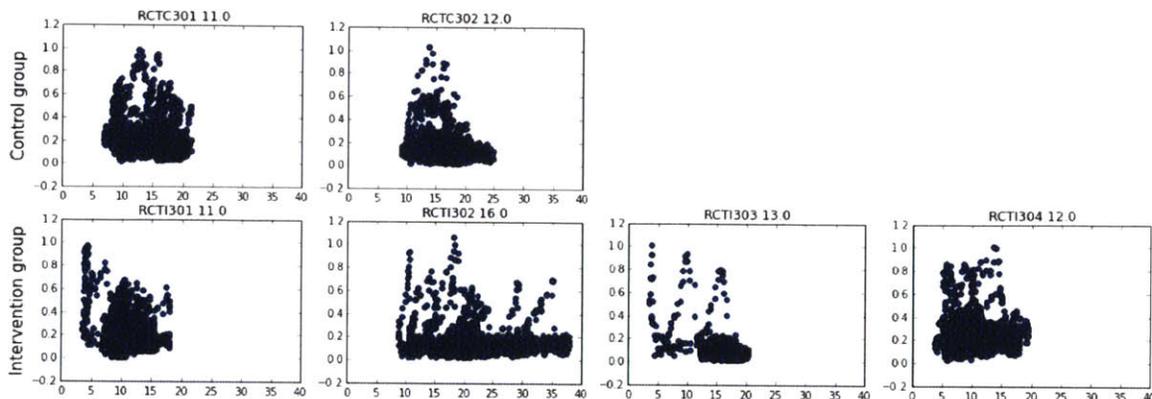


Figure A-2: Pilot 2 - Normalized amplitude versus breathing rate for each user. The individual MBRs are written on top of the plots. The intervention users have points under lower rate and higher amplitude that stand out from majority of the points. However, for the control users, higher amplitude points have higher relative rate.

BR boxplot for all participants during different tasks

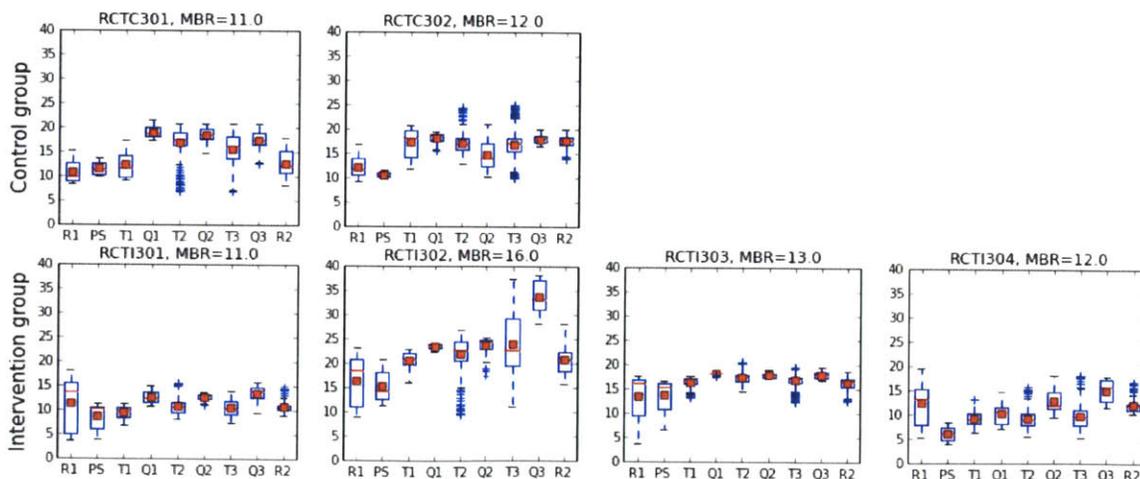


Figure A-3: Pilot 2 - Box plot of breathing rate for each individual in control and intervention groups. The individual MBRs are written on top of the plots. The red squares show the mean in each task for each user. This plot gives a more detailed view of participants breathing rates during several tasks.

Average BR for all participants during different tasks

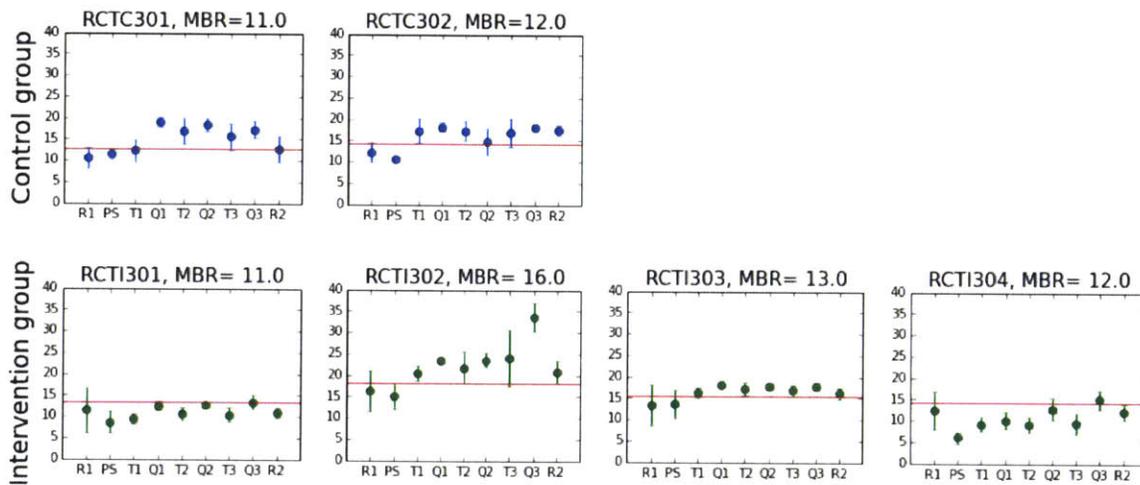


Figure A-4: Pilot 2 - The average breathing rate of all the participants during different tasks with one standard deviation error bars. The individual MBRs are written on top of the plots and the red line shows the GBR. Intervention group users tend to reach their GBR more and during wider range of tasks.

Normalized amplitude versus BR for all participants



Figure A-5: Main - Normalized amplitude versus breathing rate for each user. The individual MBRs are written on top of the plots. The intervention users have points under lower rate and higher amplitude that stand out from majority of the points. However, for the control users, higher amplitude points have higher relative rate.

BR boxplot for all participants during different tasks

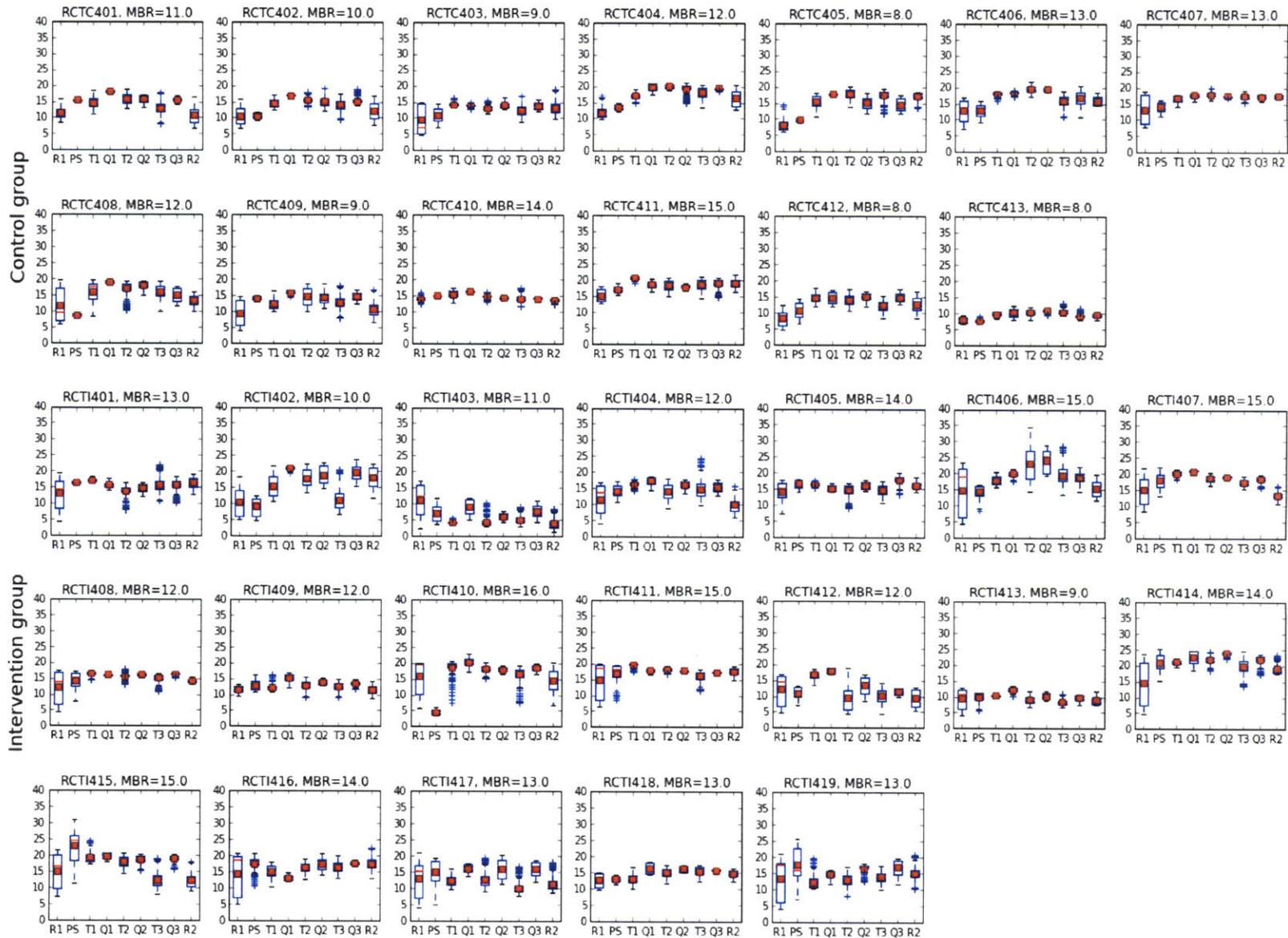


Figure A-6: Main - Box plot of breathing rate for each individual in control and intervention groups. The individual MBRs are written on top of the plots. The red squares show the mean in each task for each user. This plot gives a more detailed view of participants breathing rates during several tasks.

Average BR for all participants during different tasks

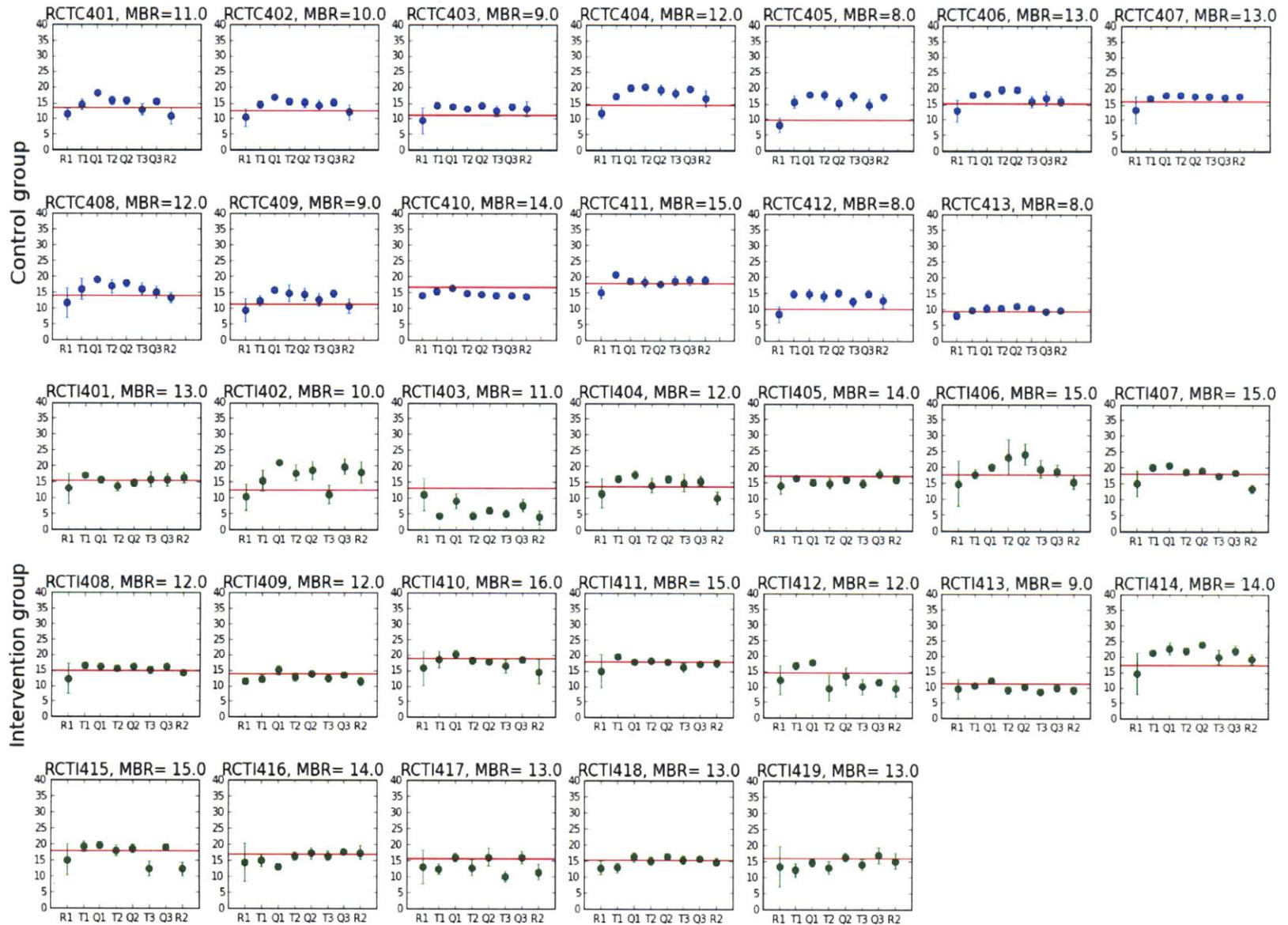


Figure A-7: Main - The average breathing rate of all the participants during different tasks with one standard deviation error bars. The individual MBRs are written on top of the plots and the red line shows the GBR. Intervention group users tend to reach their GBR more and during wider range of tasks

Experience Sampling timeline for all participants

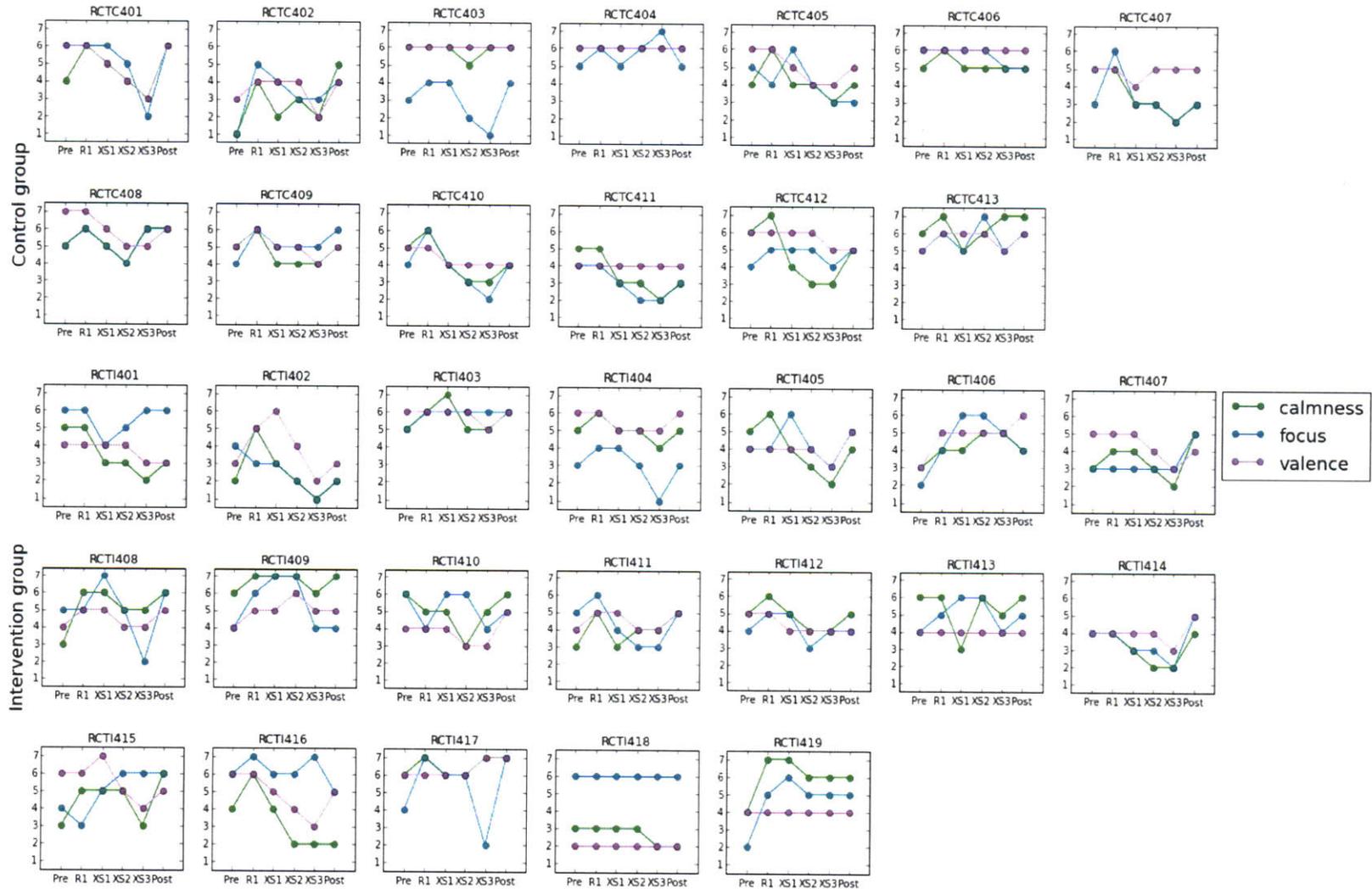


Figure A-8: Main - Timeline of self-reported calmness, focus, and valence throughout the experiment. Each point is a number between 1 (negative end) and 7 (positive end). These self-reported measures have been sampled before the session, after the original relaxation task, after each of the three reading+quizzes, and at the end of the session. The intervention group has reported higher improvements on self-reported focus and calmness.

Normalized histogram of user preference for future usage

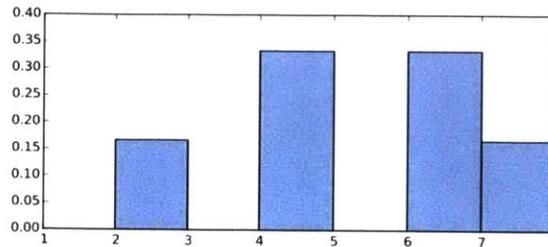


Figure A-9: Pilot 1 - Normalized histogram of future usage preference for the intervention group.

Normalized histogram of user preference for future usage

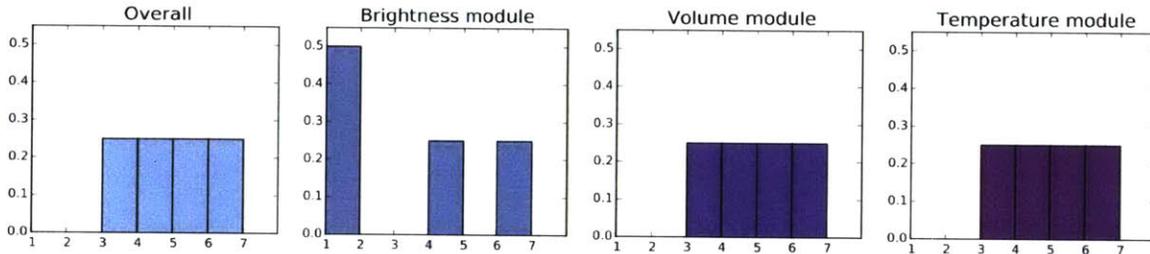


Figure A-10: Pilot 2 - Normalized histogram of future usage preference for the intervention group. Most of the users have either preferred or been neutral about future usage. Each component has a different range of user preference.

Normalized histogram of user preference for future usage

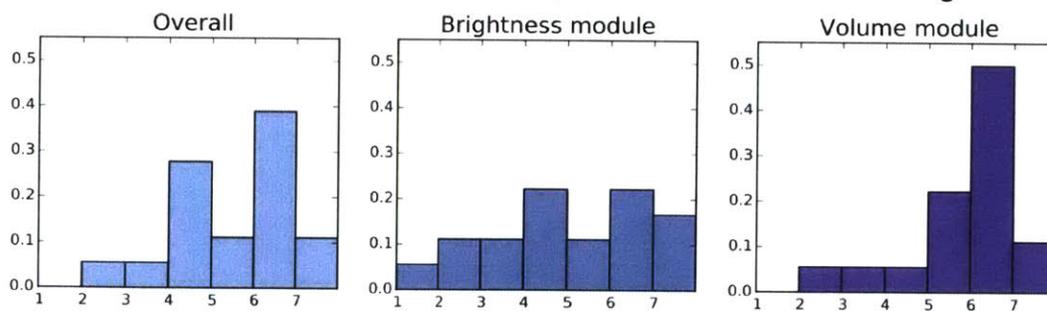


Figure A-11: Main - Normalized histogram of future usage preference for the intervention group. Most users have either preferred or been neutral about the different BrightBeat components. However, the overall response to the volume component is more positive with a peak on high preference on future usage. The response to the brightness component is more bimodal, with a peak around neutral and a peak around high preference for future usage.

Appendix B

Experiment material

This section includes the committee on the use of humans as experimental subjects (COUHES) forms and approval letter. It also includes the experiment reading tasks, quizzes, and surveys.

Final study protocol

	Massachusetts Institute of Technology Committee on the Use of Humans as Experimental Subjects	Application # (assigned by COUHES)	
		Date	

APPLICATION FOR APPROVAL TO USE HUMANS AS EXPERIMENTAL SUBJECTS (STANDARD FORM)

Please answer every question. Positive answers should be amplified with details. You must mark N/A where the question does not pertain to your application. Any incomplete application will be rejected and returned for completion. A **completed CHECKLIST FOR STANDARD APPLICATION FORM** must accompany this application.

I. BASIC INFORMATION

1. Title of Study				
Regulating breathing and posture by an unobtrusive desktop application				
2. Principal Investigator				
Name: Rosalind W. Picard		Building and Room #: E14-348A		
Title: Professor		Email: picard@media.mit.edu		
Department: Media Arts and Sciences		Phone: 617.253.0611		
3. Key Personnel				
<i>All key personnel¹ including the PI must be listed below, with a brief statement of qualifications and study role(s).</i> Important Note: all key personnel are required to complete Human Subject training before work begins on the project.				
<i>Investigators and other personnel [and institution(s)] include email address:</i>	<i>Qualifications: Describe briefly</i>	<i>Study role(s):</i>	<i>Check box if person will be obtaining consent</i>	<i>Check box if current human subject training</i>
Rosalind W. Picard (picard@media.mit.edu)	Professor	Supervisor	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>
Asma Ghandeharioun (asma_gh@mit.edu)	Graduate student	Investigator	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>

¹ MIT key personnel include all individuals who contribute in a substantive way to the execution and monitoring of the study at or on behalf of MIT or affiliated institutions. Typically, these individuals have doctoral or other professional degrees, although other individuals may be included. In particular, investigators and staff involved in obtaining informed consent are considered key personnel.

			<input type="checkbox"/>	<input type="checkbox"/>
			<input type="checkbox"/>	<input type="checkbox"/>
			<input type="checkbox"/>	<input type="checkbox"/>
4. Collaborating Institutions. <i>If you are collaborating with another institution(s) then you must obtain approval from that institution's institutional review board, and forward copies of the approval to COUHES).</i>				
No collaborating institutions				
5. Location of Research. <i>If at MIT please indicate where on campus. If you plan to use the facilities of the Clinical Research Center you will need to obtain approval of the MIT Catalyst Clinical Research Center.</i>				
MIT				
6. Funding. <i>If the research is funded by an outside sponsor, please enclose one copy of the research proposal with your application. A draft of the research proposal is acceptable. Do not leave this section blank. If your project is not funded check No Funding.</i>				
A. Sponsored Project Funding:				
<input type="checkbox"/>	Current Proposal	Proposal #	_____	
	Sponsor _____			
	Title _____			
<input type="checkbox"/>	Current Award	Account #	_____	
	Sponsor _____			
	Title _____			
B. Institutional Funding:				
<input type="checkbox"/>	Gift			
<input checked="" type="checkbox"/>	Departmental Resources			
<input type="checkbox"/>	Other (explain) _____			
<input type="checkbox"/>	No Funding			
7. Statement of Financial Interest				
Does the principal investigator or any <u>key personnel</u> involved in the study have any <u>financial interest</u> in the research?				
<input type="checkbox"/>	Yes	<input checked="" type="checkbox"/>	No	
If yes then attach a Supplement for Disclosure of Financial Interest for each individual with an interest. <i>This supplement, together with detailed guidance on this subject and definitions of the highlighted terms, is available in the COUHES site under Policies & Procedures in the Financial Conflicts of Interest section.</i>				
8. Human Subjects Training. <i>All study personnel MUST take and pass a training course on human subjects research. MIT has a web-based course that can be accessed from the main menu of the COUHES web site. COUHES may accept proof of training from some other institutions. List the names of all study personnel and indicate if they have taken a human subjects training course.</i>				
Asma Ghandeharioun is CITI human				

subjects training certified.	
9. Anticipated Dates of Research	
Start Date: 20 June, 2015	Completion Date: 20 September, 2016

II. STUDY INFORMATION

1. Purpose of Study. *Please provide a concise statement of the background, nature and reasons for the proposed study. Use non-technical language that can be understood by non-scientist members of COUHES.*

Deep breathing has been scientifically proven to affect the heart, brain, digestive system, and the immune system. It can have a profound effect on our health and physiology. Besides, research has shown that high-power postures, even for as short as two minutes, can increase testosterone and decrease cortisol hormones. This can be interpreted as higher confidence and lower stress level. Though there are many scientific clues to the profound health effects of deep breathing and high power postures, there’s a lack of conscious attention to them throughout the day. We believe designing a technology to promote this behavior has a positive effect on health and wellbeing.

Nowadays, many people are spending a significant amount of time sitting in front of screens. This makes screens great candidates for providing visual feedback. If they can promote a healthier posture and regulate breathing rate/pattern, they can highly affect wellbeing. Visual ambient feedback has long been studied and is proved to be effective. It has been explored in desktop screens, smartphones, and even external objects, ranging from ambient interactive wallpapers to pop-op notifications. However, our aim is designing an intervention that is unobtrusive and doesn’t keep people from doing their primary work. Something that doesn’t need extra attention so that users can keep it running for longer hours in order to take advantage of the positive health-related outcomes without sacrificing their attention.

In order to find the best trade-off between efficacy and obtrusiveness of the intervention, we have designed a controlled study comparing traditional superliminal visual feedback to a more sub-liminal feedback and how they will receive feedback in control, superliminal, or subliminal group. A control group is also included. All groups are informed about the details beforehand. The first includes showing breathing rate and posture on the periphery of the screen using visualization techniques that have been used in the literature for the same or similar goals. The latter includes showing similar feedback full screen, but only on a few frames out of a second. The frame rate is higher to be recognized consciously, but is proved to have subliminal influence. Visual and auditory masking has been researched and used to adopt healthy behaviors. It has been effective in several contexts including boosting academic performance and learning capacity, helping quit smoking, losing weight and healthy eating.

2. Study Protocol. *For biomedical, engineering and related research, please provide an outline of the actual experiments to be performed. Where applicable, provide a detailed description of the experimental devices or procedures to be used, detailed information on the exact dosages of drugs or chemicals to be used, total quantity of blood samples to be used, and descriptions of special diets.*

For applications in the social sciences, management and other non-biomedical disciplines please provide a detailed description of your proposed study. Where applicable, include copies of any questionnaires or standardized tests you plan to incorporate into your study. If your study involves interviews please submit an outline indicating the types of questions you will include.

You should provide sufficient information for effective review by non-scientist members of COUHES. Define all abbreviations and use simple words. Unless justification is provided for additional length, this part of the application must not exceed 5 pages.

Attaching sections of a grant application is not an acceptable substitute for a description of your study as requested here.

1. Logging screen usage and providing feedback with a desktop application –

There are 5 types of applications: (1) Control: The app will only log the screen usage. In other terms, it will record when the screen was turned on and when turned off/dimmed. (2) Superliminal: Along with logging screen usage, the app will add a peripheral visual feedback to the screen that gives feedback about healthier breathing and posture. (3) Subliminal: Along with logging screen usage, the app will pop up visual training for regulating breathing. It will also show recommended postures. The visual feedback happens only on a few frames out of many. This means it will not be recognized consciously. It also barely perceptibly oscillates the brightness of the screen, and the volume of the speaker. (4) Breathing Character: Along with logging screen usage, the app includes a character that gives feedback about user's breathing and subtle brightness change of the screen to mirror user's breathing. (5) Meditation: Along with logging screen usage, based on physiological data the application recommends short meditation exercises.

Participants will be randomly assigned to a category and will be given the respective application. The details of each version of application will be fully explained to them before starting the study.

2. Logging breathing, posture, Electrocardiogram (ECG), and electrodermal activity (EDA) through commercially available wearable devices -

Participants are asked to wear tracker sensors throughout the day and charge them during the night for the course of three weeks. They are instructed how to download data from their device. We will send daily emails to them and provide them with the link to upload their data to the portal. The sensors should be returned after the study is over. We are currently planning to use

Spire, and Zephyr, and Empatica E4 for the biosignal measurements. They are all commercially available and approved trackers. As there are several sensors coming out every day, in case we find more accurate and easier to wear devices, we may substitute them for measurement.

3. Filling in surveys at the beginning and end of the study -

We ask participants to fill out a scientifically verified wellbeing questionnaire at the beginning and end of the study. The questionnaire is attached. The exit survey will include standard questions about the usability of the app and user experience, too.

There is a short-term and a long-term version of the study. Participants will either join the former or the latter:

1. Long-term study: Participation time for the entire study is three weeks. The first week is the same for all groups and used for measurement and getting the baseline of physiology in participants. Feedback will be added to the intervention groups in the second and third weeks of the study.

2. Short-term study: Participants follow the same protocol but only under a 40-minutes lab study. Also, they will wear a thermo-electric cooler wristband that provides a barely perceptible temperature change. There's no possible risk in using thermo-electric cooler. The voltage is regulated by a heat sink. It is used in commercial products like Wristify and has proved to have no harm. (Also, we have previously used this device in similar settings. COUHES #0912003599.)

3. Drugs and Devices. *If the study involves the administration of an investigational drug that is not approved by the Food and Drug Administration (FDA) for the use outlined in the protocol, then the principal investigator (or sponsor) must obtain an Investigational New Drug (IND) number from the FDA. If the study involves the use of an approved drug in an unapproved way the investigator (or sponsor) must submit an application for an IND number. Please attach a copy of the IND approval (new drug), or application (new use).*

If the study involves the use of an investigational medical device and COUHES determines the device poses significant risk to human subjects, the investigator (or sponsor) must obtain an Investigational Device and Equipment (IDE) number from the FDA.

Will drugs or biological agents requiring an IND be used? YES NO

If yes, please provide details:

Will an investigational medical device be used? YES NO

If yes, please provide details:

4. Radiation *If the study uses radiation or radioactive materials it may also have to be approved by the Committee on Radiation Exposure to Human Subjects (COREHS). COUHES will determine if you need COREHS approval.*

<p>Will radiation or radioactive materials be used? YES <input type="checkbox"/> NO <input checked="" type="checkbox"/></p> <p><i>If yes, please provide details:</i></p> <p>Will any type of lasers be used YES <input type="checkbox"/> NO <input checked="" type="checkbox"/></p> <p><i>If yes, please provide details:</i></p>
<p>5. Diets</p> <p>Will special diets be used? YES <input type="checkbox"/> NO <input checked="" type="checkbox"/></p> <p><i>If yes, please provide details</i></p>

III. HUMAN SUBJECTS

<p>1. Subjects (that will be consented for this study)</p>	
<p>A. Maximum number: 100</p>	<p>B. Age(s): Adults (above 18 and below 65 years old)</p>
<p>C. Inclusion/exclusion criteria</p> <p>i. What are the criteria for inclusion or exclusion? All subjects will be mentally and physically healthy adults.</p> <p>ii. Are any inclusion or exclusion criteria based on age, gender, or race/ethnic origin? If so, please explain and justify No inclusion or exclusion based on age gender or race/ethnic origin</p>	
<p>D. Please explain the inclusion of any vulnerable population (e.g. children, cognitively impaired persons, non-English speakers, MIT students), and why that population is being studied. No vulnerable populations will be included. Students may be recruited from the MIT, only if they follow the above guidelines.</p>	
<p>2. Subject recruitment <i>Identification and recruitment of subjects must be ethically and legally acceptable and free of coercion. Describe below what methods will be used to identify and recruit subjects.</i></p> <p>We intend to recruit individuals from MIT students and staff. They will be approached via email and the Media Lab's internal messaging platforms.</p> <p>Please attach a copy of any advertisements/ notices and letters to potential subjects</p>	
<p>3. Subject compensation <i>Payment must be reasonable in relation to the time and trouble associated with participating in the study. It cannot constitute an undue inducement to participate.</i></p> <p>Describe all plans to pay subjects in cash or other form of payment (i.e. gift certificate): For the long-term study: Participants will receive up to \$50 Amazon giftcards. The participant compensation is proportional to their compliance to the study. Registration counts for \$3.5 and measurements for each day add \$1.5 where each source of data counts for \$0.5. The sources include two provided sensors (Empatica E4 and Zephyr) and the application. Consequently, the</p>	

APPLICATION FOR APPROVAL TO USE HUMANS AS EXPERIMENTAL SUBJECTS
(STANDARD FORM) — revised 5/31/2013

total for 3 weeks will be up to \$35. Upon finishing the study successfully, they will earn an extra \$15.

For the short-term study: Participants will receive a \$15 Amazon giftcard.

Will subjects be reimbursed for travel and expenses?

No reimbursement. The study will not demand travel nor should cause expenses to the subjects.

4. Potential risks. *A risk is a potential harm that a reasonable person would consider important in deciding whether to participate in research. Risks can be categorized as physical, psychological, sociological, economic and legal, and include pain, stress, invasion of privacy, embarrassment or exposure of sensitive or confidential data. All potential risks and discomforts must be minimized to the greatest extent possible by using e.g., appropriate monitoring, safety devices and withdrawal of a subject if there is evidence of a specific adverse event.*

What are the risks / discomforts associated with each intervention or procedure in the study?

Participants may not be used to wearing trackers throughout the day. This may be uncomfortable at first. However, participation is fully voluntarily and participants can drop any time they felt discomfort.

What procedures will be in place to prevent / minimize potential risks or discomfort?

In this case, subjects will be reminded that participation is absolutely voluntary, and that they can drop at any time throughout the study. All investigators will be available throughout the study to address any concern participants may have.

5. Potential benefits

What potential benefits may subjects receive from participating in the study?

Subjects will receive up to \$50 Amazon gift cards in the long-term study and \$15 Amazon gift cards in the short-term study.

Also, they have the opportunity to use a novel interface to regulate their breathing as well as wearing newly available fitness/wellbeing trackers, Spire, and Zephyr.

Furthermore, they may appreciate contribution to research that can help develop methods to promote healthier posture and breathing and overall wellbeing.

What potential benefits can society expect from the study?

Novel, unobtrusive, non-invasive methods for changing people's breathing pattern and posture for the better will positively affect long-term wellbeing of the society.

6. Data collection, storage, and confidentiality

How will data be collected?

Data will be recorded via three main procedures: (1) The application will record screen on/off times and record the times the intervention is running along with timestamp. (2) Spire records breathing rate and steps. Only breathing rate is used in this study. (3) Zephyr records ECG, breathing, and acceleration data.

Is there audio or videotaping? YES NO *Explain the procedures you plan to follow.*

Will data be associated with personal identifiers or will it be coded?

Personal identifiers Coded *Explain the procedures you plan to follow.*

All data gathered from a subject will be associated with a number, which will be kept separately from her/his name. No information, such as name or other private identifying information will be included in any material that will be published.

Where will the data be stored and how will it be secured?

All the data will be anonymized and accessed only by the investigators and will be cleaned from identifiers. No materials that identify the subjects will be released to publication without disguising the individuals.

What will happen to the data when the study is completed?

The data will only be available to the investigators for further reference.

Can data acquired in the study affect a subject's relationship with other individuals (e.g. employee-supervisor, patient — physician, student-teacher, family relationships)?

No. Collected data is raw ECG, breathing, and acceleration data along with app usage. The data are fully de-identified and will not affect a subject's relationship with other individuals.

7. Deception *Investigators must not exclude information from a subject that a reasonable person would want to know in deciding whether to participate in a study.*

Will information about the research purpose and design be withheld from subjects?

YES NO

If YES, explain and justify.

8. Adverse effects. *Serious or unexpected adverse reactions or injuries must be reported to COUHES within 48 hours. Other adverse events should be reported within 10 working days.*

What follow-up efforts will be made to detect any harm to subjects, and how will COUHES be kept informed?

Investigators will be available throughout the entire study to be contacted by participants and report any adverse effects directly to COUHES.

9. Informed consent. *Documented informed consent must be obtained from all participants in studies that involve human subjects. You must use the templates available on the COUHES website to prepare these forms. Draft informed consent forms must be returned with this application. Under certain **very limited** circumstances COUHES may waive the requirement for informed consent.*

Attach informed consent forms with this application.

10. Health Insurance Portability and Accountability Act ("HIPAA"). *If your study (i) involves individually identifiable health information and (ii) is sponsored by MIT Medical, an*

MIT Health Plan or another healthcare provider, then you must complete the questions below because HIPAA likely applies to your study. For more information regarding the applicability of HIPAA to human subjects research, please [click here](#).

Do you plan to obtain, use or disclose identifiable health information in connection with your research study?

YES NO

If YES, then all participants must complete an Authorization for Release of Protected Health Information Form. Please attach a copy of this draft form. You must use the [template](#) available on the COUHES website.

Alternatively, COUHES may grant a Waiver of Authorization in certain **very limited** circumstances when use of individually identifiable health information would pose only minimal risk to study participants (among other requirements). For additional information regarding whether your study might qualify for a waiver, please [click here](#).

Are you requesting a Waiver of Authorization?

YES NO

If YES, explain your rationale for concluding that: (i) use of participant health information poses no more than minimal risk; (ii) the research could not be conducted without the waiver and (iii) the research could not be conducted without the information. In addition, please explain your plan for (i) ensuring the participant health information is not improperly used or disclosed either within MIT or to any outside third parties and (ii) destroying identifiers at the earliest possible opportunity.

Will the health information you will receive for use in this study be de-identified?

YES NO

If YES, you do not need to obtain a signed Authorization for Release of Protected Health Information Form from study participants. Note, however, that if you receive identifiable participant health information that you plan to convert into de-identified information for use by other researchers, then you must obtain a signed Authorization for Release of Protected Health Information Form from each participant before receiving their identifiable health information for use in your study.

Will you be using or disclosing a limited data set?

YES NO

If YES, and you will only receive participant health information in limited data set form, then you do not need to obtain a signed Authorization for Release of Protected Health Information Form from study participants. You must complete a formal data use agreement with the party from whom you will receive the limited data set information in order for your application to be

approved.

If YES, and you will receive identifiable participant health information that you plan to convert into limited data set form for use by other researchers, then you must obtain a signed Authorization for Release of Protected Health Information Form from each participant before receiving their identifiable health information for use in your study. You must complete a formal data use agreement in order for your application to be approved..

IV. INVESTIGATOR'S ASSURANCE

I certify the information provided in this application is complete and correct.

I understand that I have ultimate responsibility for the conduct of the study, the ethical performance of the project, the protection of the rights and welfare of human subjects, and strict adherence to any stipulations imposed by COUHES

I agree to comply with all MIT policies, as well all federal, state and local laws on the protection of human subjects in research, including:

- **ensuring all study personnel satisfactorily complete human subjects training;**
- **performing the study according to the approved protocol;**
- **implementing no changes in the approved study without COUHES approval;**
- **obtaining informed consent from subjects using only the currently approved consent form;**
- **protecting identifiable health information, to the extent required by law, in accordance with HIPAA requirements; and**
- **promptly reporting significant or untoward adverse effects.**

Signature of Principal Investigator _____ **Date** _____

Print Full Name and Title _____

Signature of Department Head _____ **Date** _____

Print Full Name and Title _____

The electronic file should be sent as an attachment to an e-mail: couhes@mit.edu. In addition, two hard copies (one with original signatures) should be sent to the COUHES office: Building E25-Room 143B.

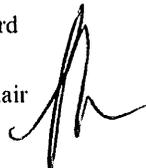
*APPLICATION FOR APPROVAL TO USE HUMANS AS EXPERIMENTAL SUBJECTS
(STANDARD FORM) — revised 5/31/2013*

Final COUHES approval letter

MIT Committee On the Use of Humans as
Experimental Subjects

MASSACHUSETTS INSTITUTE OF TECHNOLOGY
77 Massachusetts Avenue
Cambridge, Massachusetts 02139
Building E 25-143B
(617) 253-6787

To: Rosalind Picard
E14-348A

From: Leigh Finn, Chair
COUHES 

Date: 05/27/2016

Committee Action: Amendment to Approved Protocol

COUHES Protocol #: 1506127904A001

Study Title: Regulating Breathing and Posture by an Unobtrusive Desktop Application

Expiration Date: 07/15/2016

The amendment to the above-referenced protocol has been APPROVED following expedited review by the Committee on the Use of Humans as Experimental Subjects (COUHES).

This approval covers the following change(s)/modification(s):

- A short-term version of the study is being added in which participants interact with the system through a lab-study composed of pre-defined tasks (40 minutes) compared to the original long-term study (3 weeks).
- Two more sensors will be added for measurement and intervention: Empatica E4 and thermo-cooling wristband.
- Minor additions to the desktop application will be introduced including BrightBeat and VolumeBeat, which oscillate the screen brightness and volume of the computer in a barely perceptible way. HeatBeat will also be introduced using thermo-cooling wristband for providing barely perceptible temperature change on the wrist.
- With the addition of the short-term version of the study, the completion date will be extended to September 30, 2016.

If the research involves collaboration with another institution then the research cannot commence until COUHES receives written notification of approval from the collaborating institution's IRB.

It is the Principal Investigator's responsibility to obtain review and continued approval before the expiration date. Please allow sufficient time for continued approval. You may not continue any research activity beyond the expiration date without COUHES approval. Failure to receive approval for continuation before the expiration date will result in the automatic suspension of the approval of this protocol. Information collected following suspension is unapproved research and cannot be reported or published as research data. If you do not wish continued approval, please notify the Committee of the study termination.

Adverse Events: Any serious or unexpected adverse event must be reported to COUHES within 48 hours. All other adverse events should be reported in writing within 10 working days.

Amendments: Any changes to the protocol that impact human subjects, including changes in experimental design, equipment, personnel or funding, must be approved by COUHES before they can be initiated.

Prospective new study personnel must, where applicable, complete training in human subjects research and in the HIPAA Privacy Rule before participating in the study.

COUHES should be notified when your study is completed. You must maintain a research file for at least 3 years after completion of the study. This file should include all correspondence with COUHES, original signed consent forms, and study data.

Pilot 1 - Consent form

CONSENT TO PARTICIPATE IN BIOMEDICAL RESEARCH

Regulating breathing by an unobtrusive desktop application

You are asked to participate in a research study conducted by Asma Ghandeharioun and Rosalind Picard, Sc.D. from the Media Lab at the Massachusetts Institute of Technology (M.I.T.). You were selected as a possible participant in this study because of the collaborative nature of your professional work and your willingness to test new technologies. You should read the information below, and ask questions about anything you do not understand, before deciding whether or not to participate.

- **PARTICIPATION AND WITHDRAWAL**

Your participation in this study is completely voluntary and you are free to choose whether to be in it or not. If you choose to be in this study, you may subsequently withdraw from it at any time without penalty or consequences of any kind. The investigator may withdraw you from this research if circumstances arise which warrant doing so.

To participate, you should be 18 years or older and not have any ongoing medical condition (physical or mental) and also not be on any drug regimen.

- **PURPOSE OF THE STUDY**

Deep breathing and good posture have been scientifically proven to affect health and wellbeing positively. Thus, we aim at designing a technology to promote these behaviors without the need of continuous effort and conscious attention throughout the day. In this study, we will compare three conditions that are explained in the next section.

- **PROCEDURES**

If you volunteer to participate in this study, we would ask you to do the following things:

1. Engage in a series of relaxation, reading, and questions tasks – You will be given a link to a test composed of two reading tasks and questions. The readings are selected from scientific articles about research on improving wellbeing.
2. Respond to the BrightBeat intervention – BrightBeat is an intervention that appears when you are breathing faster than your goal. We ask you to sync your breathing with the BrightBeat. Try to inhale as the

screen brightens and exhale when it darkens. You will randomly be assigned to a category to either receive this intervention as it is or another version which is below the level of perception.

3. Wear the Zephyr sensor throughout the experiment -
We ask you to wear the Zephyr BioHarness chestband. The sensor should be returned after the study is over.
4. Fill in a survey at the end of the study -
We ask you to fill out an exit survey which includes questions about the usability of the app and your user experience.

The study session lasts 30-40 minutes.

- **POTENTIAL RISKS AND DISCOMFORTS**

In case you are not used to wearing chestband sensors, it might be at first uncomfortable. However, please remember that participation is absolutely voluntary, and that you can drop at any time throughout the study.

- **ANTICIPATED BENEFITS TO SUBJECTS**

You will have the opportunity to use a novel interface to regulate your breathing and posture to improve your wellbeing as well as wearing newly available fitness trackers.

Furthermore, you may appreciate that you are contributing to research that can help develop methods to promote more healthy breathing and overall wellbeing.

- **ANTICIPATED BENEFITS TO SOCIETY**

Finding novel and unobtrusive methods for changing people's breathing posture for the better will positively affect long-term wellbeing of the society.

- **PAYMENT FOR PARTICIPATION**

Participation is completely voluntary.

- **PRIVACY AND CONFIDENTIALITY**

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law.

When the results of the research are published or discussed in conferences, no information will be included that would reveal your identity. All the data gathered from you will be associated with a number, which will be kept separately from your name.

- **NEW FINDINGS**

During the course of the study, you will be informed of any significant new findings (either good or bad), such as changes in the risks or benefits resulting from participation in the research or new alternatives to participation, that might cause you to change your mind about continuing in the study. If new information is provided to you, your consent to continue participating in this study will be re-obtained.

- **EMERGENCY CARE AND COMPENSATION FOR INJURY**

If you feel you have suffered an injury, which may include emotional trauma, as a result of participating in this study, please contact the person in charge of the study as soon as possible.

In the event you suffer such an injury, M.I.T. may provide itself, or arrange for the provision of, emergency transport or medical treatment, including emergency treatment and follow-up care, as needed, or reimbursement for such medical services. M.I.T. does not provide any other form of compensation for injury. In any case, neither the offer to provide medical assistance, nor the actual provision of medical services shall be considered an admission of fault or acceptance of liability. Questions regarding this policy may be directed to MIT's Insurance Office, (617) 253-2823. Your insurance carrier may be billed for the cost of emergency transport or medical treatment, if such services are determined not to be directly related to your participation in this study.

- **IDENTIFICATION OF INVESTIGATORS**

If you have any questions or concerns about the research, please feel free to contact Asma Ghandeharioun (e-mail asam_gh@mit.edu , 857.303.1203) or Rosalind W. Picard (e-mail: picard@media.mit.edu, tel: 617.253.0611).

- **RIGHTS OF RESEARCH SUBJECTS**

You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you feel you have been treated unfairly, or you have questions regarding your rights as a research subject, you may contact the Chairman of the Committee on the Use of Humans as Experimental Subjects, M.I.T., Room E25-143B, 77 Massachusetts Ave, Cambridge, MA 02139, phone 1-617-253 6787.

SIGNATURE OF RESEARCH SUBJECT OR LEGAL REPRESENTATIVE

I understand the procedures described above. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

Name of Subject

Name of Legal Representative (if applicable)

Signature of Subject or Legal Representative

Date

SIGNATURE OF INVESTIGATOR

In my judgment the subject is voluntarily and knowingly giving informed consent and possesses the legal capacity to give informed consent to participate in this research study.

Signature of Investigator

Date

Pilot 2 - Consent form

CONSENT TO PARTICIPATE IN BIOMEDICAL RESEARCH

Regulating breathing by an unobtrusive desktop application

You are asked to participate in a research study conducted by Asma Ghandeharioun and Rosalind Picard, Sc.D. from the Media Lab at the Massachusetts Institute of Technology (M.I.T.). You were selected as a possible participant in this study because of the collaborative nature of your professional work and your willingness to test new technologies. You should read the information below, and ask questions about anything you do not understand, before deciding whether or not to participate.

- **PARTICIPATION AND WITHDRAWAL**

Your participation in this study is completely voluntary and you are free to choose whether to be in it or not. If you choose to be in this study, you may subsequently withdraw from it at any time without penalty or consequences of any kind. The investigator may withdraw you from this research if circumstances arise which warrant doing so.

To participate, you should be 18 years or older and not have any ongoing medical condition (physical or mental) and also not be on any drug regimen.

- **PURPOSE OF THE STUDY**

Deep breathing has been scientifically proven to affect health and wellbeing positively. Thus, we aim at designing a technology to promote these behaviors without the need of continuous effort and conscious attention throughout the day. In this study, we will compare different conditions that are explained in the next section.

- **PROCEDURES**

If you volunteer to participate in this study, we would ask you to do the following things:

For the short-term study:

1. Engage in a series of relaxation, reading, and questions tasks – You will be given a link to a test composed of reading tasks and questions. The readings are selected from scientific articles about research on improving wellbeing.
2. Respond to the interventions –

You will be given a subset of interventions including BrightBeat, HeatBeat, and VolumeBeat that appear when you are breathing faster than your goal. BrightBeat changes your screen brightness in a barely perceptible way. VolumeBeat does the same with the volume. HeatBeat does the same on a thermo-cooling wristband you are wearing. These oscillating interventions are not there to tell you exactly when to breathe. You do NOT have to inhale/exhale when it is light/dark, mute/unmute, cold/warm. Instead, you should just try to focus on your task. These interventions are only occasionally there to remind you to think about slowing your breathing or to breathe in a fuller and more relaxed way (e.g. you may decide to reposition yourself, and sit in a way where your chest can expand more fully). BrightBeat, HeatBeat, and VolumeBeat are not tasks. They are only gentle reminders to breathe in a way that you think is best to help you do your task.

3. Wear Zephyr, Empatica E4, and a thermocooling wristband sensor throughout the experiment -

We ask you to wear the Zephyr Bioharness chestband and Empatica E4 sensor and a thermocooling wristband. The sensors should be returned after the study is over.

4. Fill in a survey at the beginign and end of the study -

We ask you to fill out an exit survey which includes questions about the usability of the app and your user experience.

The study session lasts 30-40 minutes.

For the long-term study:

If you volunteer to participate in this study, we would ask you to do the following things:

1. Install an app on your desktop –

There are 5 types of application: (1) The app will only log your screen usage. In other terms, it will record when the screen was turned on and when turned off/dimmed. (2) Along with logging screen usage, the app will add a peripheral visual feedback to your screen that gives you feedback about healthier breathing and posture. (3) Along with logging screen usage, the app will pop up visual training for regulating breathing. It will also show recommended postures. The visual feedback happens only on a few frames out of many. This means you will not recognize that conciously. (4) Along with logging screen usage, the app includes a character that gives you feedback about about your breathing/posture and there will be subtle brightness change on the screen that mirrors your breathing. (5) Along with logging screen usage, based on your physiological data the application recommends short meditation exercises.

You will be randomly assigned to a category and will be given the respective application. The details of your version of application will be fully explained to you before starting the study.

2. Wear E4 and Zephyr sensor throughout the study -
We ask you to wear these two sensors throughout the day and charge them during the night for the course of three weeks. You are instructed how to download data from your device. We will send daily emails to you and provide you with the link to upload your data to the portal. The sensors should be returned after the study is over.
3. Fill in surveys at the beginning and end of the study -
We ask you to fill out a scientifically verified wellbeing questionnaire at the beginning and end of the study. The exit survey will include questions about the usability of the app and user experience, too.

Participation time for the entire study is three weeks.

- **POTENTIAL RISKS AND DISCOMFORTS**

If you are not used to wearing chestband sensors, it might be a little uncomfortable at first. However, please remember that participation is absolutely voluntary, and that you can drop at any time throughout the study.

- **ANTICIPATED BENEFITS TO SUBJECTS**

You will have the opportunity to use a novel interface to regulate your breathing to improve your wellbeing as well as wearing newly available fitness trackers.

Furthermore, you may appreciate that you are contributing to research that can help develop methods to promote more healthy breathing and overall wellbeing.

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Finding novel and unobtrusive methods for changing people's breathing for the better will positively affect long-term wellbeing of the society.

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- **NEW FINDINGS**

During the course of the study, you will be informed of any significant new findings (either good or bad), such as changes in the risks or benefits resulting from participation in the research or new alternatives to participation, that might cause you to change your mind about continuing in the study. If new information is provided to you, your consent to continue participating in this study will be re-obtained.

- **EMERGENCY CARE AND COMPENSATION FOR INJURY**

If you feel you have suffered an injury, which may include emotional trauma, as a result of participating in this study, please contact the person in charge of the study as soon as possible.

In the event you suffer such an injury, M.I.T. may provide itself, or arrange for the provision of, emergency transport or medical treatment, including emergency treatment and follow-up care, as needed, or reimbursement for such medical services. M.I.T. does not provide any other form of compensation for injury. In any case, neither the offer to provide medical assistance, nor the actual provision of medical services shall be considered an admission of fault or acceptance of liability. Questions regarding this policy may be directed to MIT's Insurance Office, (617) 253-2823. Your insurance carrier may be billed for the cost of emergency transport or medical treatment, if such services are determined not to be directly related to your participation in this study.

- **IDENTIFICATION OF INVESTIGATORS**

If you have any questions or concerns about the research, please feel free to contact Asma Ghandeharioun (e-mail asma_gh@mit.edu , 857.303.1203) or Rosalind W. Picard (e-mail: picard@media.mit.edu, tel: 617.253.0611).

- **RIGHTS OF RESEARCH SUBJECTS**

You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you feel you have been treated unfairly, or you have questions regarding your rights as a research subject, you may contact the Chairman of the Committee on the Use of Humans as Experimental Subjects, M.I.T., Room E25-143B, 77 Massachusetts Ave, Cambridge, MA 02139, phone 1-617-253 6787.

SIGNATURE OF RESEARCH SUBJECT OR LEGAL REPRESENTATIVE

I understand the procedures described above. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

Name of Subject

Name of Legal Representative (if applicable)

Signature of Subject or Legal Representative

Date

SIGNATURE OF INVESTIGATOR

In my judgment the subject is voluntarily and knowingly giving informed consent and possesses the legal capacity to give informed consent to participate in this research study.

Signature of Investigator

Date

Main - Consent form

CONSENT TO PARTICIPATE IN BIOMEDICAL RESEARCH

Regulating breathing by an unobtrusive desktop application

You are asked to participate in a research study conducted by Asma Ghandeharioun and Rosalind Picard, Sc.D. from the Media Lab at the Massachusetts Institute of Technology (M.I.T.). You were selected as a possible participant in this study because of the collaborative nature of your professional work and your willingness to test new technologies. You should read the information below, and ask questions about anything you do not understand, before deciding whether or not to participate.

- **PARTICIPATION AND WITHDRAWAL**

Your participation in this study is completely voluntary and you are free to choose whether to be in it or not. If you choose to be in this study, you may subsequently withdraw from it at any time without penalty or consequences of any kind. The investigator may withdraw you from this research if circumstances arise which warrant doing so.

To participate, you should be 18 years or older and not have any ongoing medical condition (physical or mental) and also not be on any drug regimen.

- **PURPOSE OF THE STUDY**

Deep breathing has been scientifically proven to affect health and wellbeing positively. Thus, we aim at designing a technology to promote these behaviors without the need of continuous effort and conscious attention throughout the day. In this study, we will compare different conditions that are explained in the next section.

- **PROCEDURES**

If you volunteer to participate in this study, we would ask you to do the following things:

For the short-term study:

1. Engage in a series of relaxation, reading, and questions tasks – You will be given a link to a test composed of reading tasks and questions. The readings are selected from scientific articles about research on improving wellbeing.
2. Respond to the interventions –

You will be given a subset of interventions including BrightBeat and VolumeBeat that appear when you are breathing faster than your goal. BrightBeat changes your screen brightness in a barely perceptible way. VolumeBeat does the same with the volume. These oscillating interventions are not there to tell you exactly when to breathe. You do NOT have to inhale/exhale when it is light/dark, mute/unmute, cold/warm. Instead, you should just try to focus on your task. These interventions are only occasionally there to remind you to think about slowing your breathing or to breathe in a fuller and more relaxed way (e.g. you may decide to reposition yourself, and sit in a way where your chest can expand more fully). BrightBeat and VolumeBeat are not tasks. They are only gentle reminders to breathe in a way that you think is best to help you do your task. However, you might find it helpful to pay attention to them and let them guide you toward deep and slow breathing.

3. Wear Zephyr, Empatica E4, and a thermocooling wristband sensor throughout the experiment -
We ask you to wear the Zephyr Bioharness chestband and Empatica E4 sensor and a thermocooling wristband. The sensors should be returned after the study is over.
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We ask you to fill out an exit survey which includes questions about the usability of the app and your user experience.

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We ask you to fill out a scientifically verified wellbeing questionnaire at the beginning and end of the study. The exit survey will include questions about the usability of the app and user experience, too.

Participation time for the entire study is three weeks.

- **POTENTIAL RISKS AND DISCOMFORTS**

If you are not used to wearing chestband sensors, it might be a little uncomfortable at first. However, please remember that participation is absolutely voluntary, and that you can drop at any time throughout the study.

- **ANTICIPATED BENEFITS TO SUBJECTS**

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During the course of the study, you will be informed of any significant new findings (either good or bad), such as changes in the risks or benefits resulting from participation in the research or new alternatives to participation, that might cause you to change your mind about continuing in the study. If new information is provided to you, your consent to continue participating in this study will be re-obtained.

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In the event you suffer such an injury, M.I.T. may provide itself, or arrange for the provision of, emergency transport or medical treatment, including emergency treatment and follow-up care, as needed, or reimbursement for such medical services. M.I.T. does not provide any other form of compensation for injury. In any case, neither the offer to provide medical assistance, nor the actual provision of medical services shall be considered an admission of fault or acceptance of liability. Questions regarding this policy may be directed to MIT's Insurance Office, (617) 253-2823. Your insurance carrier may be billed for the cost of emergency transport or medical treatment, if such services are determined not to be directly related to your participation in this study.

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- **RIGHTS OF RESEARCH SUBJECTS**

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Name of Subject

Name of Legal Representative (if applicable)

Signature of Subject or Legal Representative

Date

SIGNATURE OF INVESTIGATOR

In my judgment the subject is voluntarily and knowingly giving informed consent and possesses the legal capacity to give informed consent to participate in this research study.

Signature of Investigator

Date

Experiment 1 & 2: Experience sampling

BrightBeat: MIT Media Lab study on calming technologies

* Required

Experience Sampling

How stressed/calm are you feeling right now? *

1 2 3 4 5 6 7

Very stressed Very calm

How distracted/focused are you feeling right now? *

1 2 3 4 5 6 7

Very distracted Very focused

How do you rate your emotional valence right now? *

1 2 3 4 5 6 7

Very sad Very happy

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Experiment 2 - User motivation questionnaire

BrightBeat: MIT Media Lab study on calming technologies

* Required

Behavior change traits

How confident are you about your capability of stress management? *

(e.g. doing conscious behaviors before, during, or after stressful events to self-regulate)

1 2 3 4 5 6 7

not at all very much

How willing are you to self-improve your stress-management capabilities or learn new techniques for self-regulation? *

1 2 3 4 5 6 7

not at all very much

How willing are to be hinted/notified about stress management techniques in-time? *

1 2 3 4 5 6 7

not at all very much

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Strengthening Individual Memories by Reactivating Them During Sleep

John D. Rudoy,¹ Joel L. Voss,^{1,2} Carmen E. Westerberg,¹ Ken A. Paller^{1*}

Initially fragile memories can gain stability via consolidation, but the extent to which sleep contributes to this process is unresolved (1, 2). Sleep between encoding and retrieval, relative to wakefulness, promotes memory storage in some circumstances, perhaps from internally generated memory reactivation (3, 4). Moreover, reinstating a learning context (an odor) during slow-wave sleep enhances retrieval of spatial information learned in that context (5). It remains unclear whether exposure during sleep to cues associated with newly learned information can selectively enhance the storage of individual memories.

We taught people to associate each of 50 unique object images with a location on a computer screen before a nap (Fig. 1A). Each object was paired with a characteristic sound delivered over a speaker (e.g., cat with meow and kettle with whistle). For the entirety of the nap, white noise was presented at an unobtrusive intensity (about 62 dB sound-pressure level). During non-rapid eye movement (non-REM) sleep, the sounds for 25 of the objects were presented, with white-noise intensity lowered such that overall sound levels were approximately constant (Fig. 1B).

After waking, individuals viewed all 50 objects and attempted to position each one in its original location. Absolute distance measures showed that object placements were more accurate for objects that were cued by their sounds during sleep than for those not cued [1.07 ± 0.08 cm (SE) versus 1.23 ± 0.10 cm (SE), respectively; $t_{11} = 2.6$; $P < 0.05$]. Forgetting occurred between the final stage of learning and the postnap test, with a smaller decline for cued compared with uncued objects (Fig. 1C). An advantage for cued-object locations computed in this manner was evident in 10 of the 12 participants.

Electroencephalographic (EEG) recordings provided information for determining sleep stages (6). Additionally, EEG responses to sound cues were sorted into two conditions via a median split on the difference between pre- and postnap accuracy: (i) less-forgetting accuracy was superior postnap compared with prenap [placements $0.51 \pm$

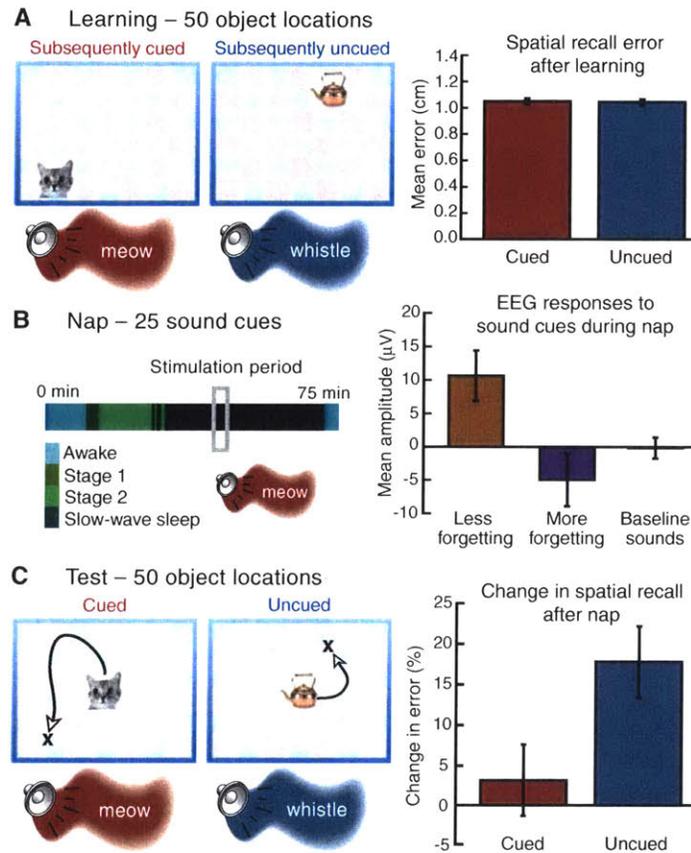


Fig. 1. (A) Individuals learned object-location associations while hearing object sounds. Accuracy at the final stage of learning was matched for objects subsequently cued or not cued by the sounds (mean \pm SE). (B) Sleep-staging data are shown for a representative participant, including the 3.5-min sequence of 25 sound cues. Vertex brain potentials differed according to level of forgetting for corresponding object locations. (C) After the nap, individuals attempted to place each object in its correct location (arrows simulate motion of objects as individuals complete the task). Better spatial-location retention for cued compared with uncued objects was reflected by a smaller change in error ($t_{11} = 3.2$, $P < 0.01$).

0.1 cm (SE) closer to correct]; (ii) more-forgetting accuracy was superior prenap compared with postnap [placements 0.60 ± 0.1 cm (SE) closer to correct]. Average EEG amplitudes measured over the interval from 600 to 1000 ms after sound onset were $15.3 \mu\text{V}$ greater when there was less forgetting ($t_{11} = 3.2$, $P < 0.01$). Thus, the degree of recall improvement or decline appeared to have been influenced by sound-induced memory processing during sleep, as indexed by brain potentials.

Participants professed no knowledge that sounds were presented during sleep. Moreover, they performed at chance when forced to guess which sounds were presented during sleep (6).

These results show that information presented during sleep can influence subsequent retrieval during waking. In an additional control experiment with 12 other participants who remained awake, sounds presented after learning did not reliably influence recall accuracy [1.15 cm from target ± 0.07 (SE) versus 1.32 cm ± 0.14 (SE) for cued versus uncued items, respectively; $t_{11} = 1.4$; $P = 0.18$].

The extent to which cues affect consolidation in waking subjects may depend on how strongly individuals attend to the cues (6). Regardless, we propose that sound cues presented during sleep prompted preferential processing of corresponding object-location associations. The hippocampus has previously been implicated in sleep-mediated consolidation (3, 5). Memory storage in our study likely depends on representations of objects, sounds, and locations in multiple cortical regions, along with hippocampal networks capable of linking these representations together (2). Although some sleep theories emphasize general plasticity mechanisms that could benefit all information learned before sleep (7), our results show that memory processing during sleep can be highly specific. Certain associations may be preferentially reactivated during sleep as a normal part of memory stabilization and consolidation.

Whereas opportunities for enhancement of memory storage may be available every time we sleep, reminders during sleep can be used to target the reactivation and strengthening of individual memories.

References and Notes

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7. G. Tononi, C. Cirelli, *Brain Res. Bull.* **62**, 143 (2003).
8. Supported by NSF under grant BCS-0818912, by the National Institute of Neurological Disorders and Stroke, and by the Alzheimer's Association.

Supporting Online Material

www.sciencemag.org/cgi/content/full/326/5956/1079/DC1
Materials and Methods
Tables S1 to S3

13 July 2009; accepted 23 September 2009
10.1126/science.1179013

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Abstract

While asleep, people heard sounds that had earlier been associated with objects at specific spatial locations. Upon waking, they recalled these locations more accurately than other locations for which no reminder cues were provided. Consolidation thus operates during sleep with high specificity and is subject to systematic influences through simple auditory stimulation.

Experiment 1 - Quiz 1

BrightBeat

Question set I

What was the main take away message of the article?

- We need to consistently keep a posture for a long time to experience its physiological, psychological, and behavioral change.
- Posing a powerful posture for a few minutes can cause a physiological change, but no psychological or behavioral change.
- Posing a powerful posture for a few minutes can cause a psychological and behavioral change, but no physiological change.
- Our posture can change our sense of power/powerlessness physically, psychologically, and behaviorally even for as short as a few minutes.

What is the neuroendocrine profile of power?

- High testosterone, high cortisol
- High testosterone, low cortisol
- Low testosterone, high cortisol
- Low testosterone, low cortisol

What were the participants told before the experiment?

The goal of the study is to ...

- measure the effect of different postures on the sense of power
- measure the effect of placement of electrocardiography electrodes on data collection
- measure the effect of sense of power on body expressions
- measure the comfortability of a set of postures

Which measures were used in the experiment to study the effect of posing different postures ?

	Used	Not used
EEG signal	<input type="radio"/>	<input type="radio"/>
Saliva samples	<input type="radio"/>	<input type="radio"/>
Electrodermal activity	<input type="radio"/>	<input type="radio"/>
Self-reports	<input type="radio"/>	<input type="radio"/>
Behavior in a gambling task	<input type="radio"/>	<input type="radio"/>

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Power Posing: Brief Nonverbal Displays Affect Neuroendocrine Levels and Risk Tolerance

Psychological Science
21(10) 1363–1368
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sagepub.com/journalsPermissions.nav
DOI: 10.1177/0956797610383437
<http://pss.sagepub.com>


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Abstract

Humans and other animals express power through open, expansive postures, and they express powerlessness through closed, contractive postures. But can these postures actually cause power? The results of this study confirmed our prediction that posing in high-power nonverbal displays (as opposed to low-power nonverbal displays) would cause neuroendocrine and behavioral changes for both male and female participants: High-power posers experienced elevations in testosterone, decreases in cortisol, and increased feelings of power and tolerance for risk; low-power posers exhibited the opposite pattern. In short, posing in displays of power caused advantaged and adaptive psychological, physiological, and behavioral changes, and these findings suggest that embodiment extends beyond mere thinking and feeling, to physiology and subsequent behavioral choices. That a person can, by assuming two simple 1-min poses, embody power and instantly become more powerful has real-world, actionable implications.

Keywords

cortisol, embodiment, hormones, neuroendocrinology, nonverbal behavior, power, risk taking, testosterone

Received 1/20/10; Revision accepted 4/8/10

The proud peacock fans his tail feathers in pursuit of a mate. By galloping sideways, the cat manipulates an intruder's perception of her size. The chimpanzee, asserting his hierarchical rank, holds his breath until his chest bulges. The executive in the boardroom crests the table with his feet, fingers interlaced behind his neck, elbows pointing outward. Humans and other animals display power and dominance through expansive nonverbal displays, and these power poses are deeply intertwined with the evolutionary selection of what is "alpha" (Darwin, 1872/2009; de Waal, 1998).

But is power embodied? What happens when displays of power are posed? Can posed displays cause a person to feel more powerful? Do people's mental and physiological systems prepare them to be more powerful? The goal of our research was to test whether high-power poses (as opposed to low-power poses) actually produce power. To perform this test, we looked at the effects of high-power and low-power poses on some fundamental features of having power: feelings of power, elevation of the dominance hormone testosterone, lowering of the stress hormone cortisol, and an increased tolerance for risk.

Power determines greater access to resources (de Waal, 1998; Keltner, Gruenfeld, & Anderson, 2003); higher levels of

agency and control over a person's own body, mind, and positive feelings (Keltner et al., 2003); and enhanced cognitive function (Smith, Jostmann, Galinsky, & van Dijk, 2008). Powerful individuals (compared with powerless individuals) demonstrate greater willingness to engage in action (Galinsky, Gruenfeld, & Magee, 2003; Keltner et al., 2003) and often show increased risk-taking behavior¹ (e.g., Anderson & Galinsky, 2006).

The neuroendocrine profiles of the powerful differentiate them from the powerless, on two key hormones—testosterone and cortisol. In humans and other animals, testosterone levels both reflect and reinforce dispositional and situational status and dominance; internal and external cues cause testosterone to rise, increasing dominant behaviors, and these behaviors can elevate testosterone even further (Archer, 2006; Mazur &

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Booth, 1998). For example, testosterone rises in anticipation of a competition and as a result of a win, but drops following a defeat (e.g., Booth, Shelley, Mazur, Tharp, & Kittok, 1989), and these changes predict the desire to compete again (Mehta & Josephs, 2006). In short, testosterone levels, by reflecting and reinforcing dominance, are closely linked to adaptive responses to challenges.

Power is also linked to the stress hormone cortisol: Power holders show lower basal cortisol levels and lower cortisol reactivity to stressors than powerless people do, and cortisol drops as power is achieved (Abbott et al., 2003; Coe, Mendoza, & Levine, 1979; Sapolsky, Alberts, & Altmann, 1997). Although short-term and acute cortisol elevation is part of an adaptive response to challenges large (e.g., a predator) and small (e.g., waking up), the chronically elevated cortisol levels seen in low-power individuals are associated with negative health consequences, such as impaired immune functioning, hypertension, and memory loss (Sapolsky et al., 1997; Segerstrom & Miller, 2004). Low-power social groups have a higher incidence of stress-related illnesses than high-power social groups do, and this is partially attributable to chronically elevated cortisol (Cohen et al., 2006). Thus, the power holder's typical neuroendocrine profile of high testosterone coupled with low cortisol—a profile linked to such outcomes as disease resistance (Sapolsky, 2005) and leadership abilities (Mehta & Josephs, 2010)—appears to be optimally adaptive.

It is unequivocal that power is expressed through highly specific, evolved nonverbal displays. Expansive, open postures (widespread limbs and enlargement of occupied space by spreading out) project high power, whereas contractive, closed postures (limbs touching the torso and minimization of occupied space by collapsing the body inward) project low power. All of these patterns have been identified in research on actual and attributed power and its nonverbal correlates (Carney, Hall, & Smith LeBeau, 2005; Darwin, 1872/2009; de Waal, 1998; Hall, Coats, & Smith LeBeau, 2005). Although researchers know that power generates these displays, no research has investigated whether these displays generate power. Will posing these displays of power actually cause individuals to feel more powerful, focus on reward as opposed to risk, and experience increases in testosterone and decreases in cortisol?

In research on embodied cognition, some evidence suggests that bodily movements, such as facial displays, can affect emotional states. For example, unobtrusive contraction of the "smile muscle" (i.e., the zygomaticus major) increases enjoyment (Strack, Martin, Stepper, 1988), the head tilting upward induces pride (Stepper & Strack, 1993), and hunched postures (as opposed to upright postures) elicit more depressed feelings (Riskind & Gotay, 1982). Approach-oriented behaviors, such as touching, pulling, or nodding "yes," increase preference for objects, people, and persuasive messages (e.g., Briñol & Petty, 2003; Chen & Bargh, 1999; Wegner, Lane, & Dimitri, 1994), and fist clenching increases men's self-ratings on power-related

traits (Schubert & Koole, 2009). However, no research has tested whether expansive power poses, in comparison with contractive power poses, cause mental, physiological, and behavioral change in a manner consistent with the effects of power. We hypothesized that high-power poses (compared with low-power poses) would cause individuals to experience elevated testosterone, decreased cortisol, increased feelings of power, and higher risk tolerance. Such findings would suggest that embodiment goes beyond cognition and emotion and could have immediate and actionable effects on physiology and behavior.

Method

Participants and overview of procedure

Forty-two participants (26 females and 16 males) were randomly assigned to the high-power-pose or low-power-pose condition. Participants believed that the study was about the science of physiological recordings and was focused on how placement of electrocardiography electrodes above and below the heart could influence data collection. Participants' bodies were posed by an experimenter into high-power or low-power poses. Each participant held two poses for 1 min each. Participants' risk taking was measured with a gambling task; feelings of power were measured with self-reports. Saliva samples, which were used to test cortisol and testosterone levels, were taken before and approximately 17 min after the power-pose manipulation.

Power poses

Poses were harvested from the nonverbal literature (e.g., Carney et al., 2005; Hall et al., 2005) and varied on the two nonverbal dimensions universally linked to power: expansiveness (i.e., taking up more space or less space) and openness (i.e., keeping limbs open or closed). The two high-power poses into which participants were configured are depicted in Figure 1, and the two low-power poses are depicted in Figure 2. To be sure that the poses chosen conveyed power appropriately, we asked 95 pretest participants to rate each pose from 1 (*very low power*) to 7 (*very high power*). High-power poses ($M = 5.39$, $SD = 0.99$) were indeed rated significantly higher on power than were low-power poses ($M = 2.41$, $SD = 0.93$), $t(94) = 21.03$, $p < .001$; $r = .99$.

To be sure that changes in neuroendocrine levels, powerful feelings, or behavior could be attributed only to the high-power or low-power attributes of the poses, we had 19 pretest participants rate the comfort, difficulty, and pain of the poses. Participants made all four poses (while wearing electrocardiography leads) and completed questionnaires after each pose. There were no differences between high-power and low-power poses on comfort, $t(16) = 0.24$, $p > .80$; difficulty, $t(16) = 0.77$, $p > .45$; or painfulness, $t(16) = -0.82$, $p > .42$.



Fig. 1. The two high-power poses used in the study. Participants in the high-power-pose condition were posed in expansive positions with open limbs.

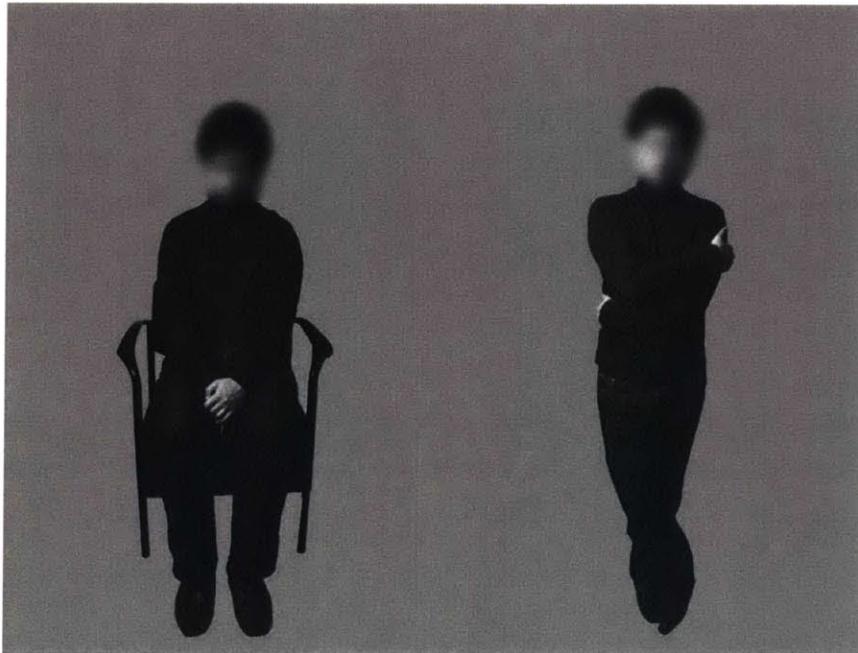


Fig. 2. The two low-power poses used in the study. Participants in the low-power-pose condition were posed in contractive positions with closed limbs.

To configure the test participants into the poses, the experimenter placed an electrocardiography lead on the back of each participant's calf and underbelly of the left arm and explained, "To test accuracy of physiological responses as a function of

sensor placement relative to your heart, you are being put into a certain physical position." The experimenter then manually configured participants' bodies by lightly touching their arms and legs. As needed, the experimenter provided verbal

instructions (e.g., “Keep your feet above heart level by putting them on the desk in front of you”). After manually configuring participants’ bodies into the two poses, the experimenter left the room. Participants were videotaped; all participants correctly made and held either two high-power or two low-power poses for 1 min each. While making and holding the poses, participants completed a filler task that consisted of viewing and forming impressions of nine faces.

Measure of risk taking and powerful feelings

After they finished posing, participants were presented with the gambling task. They were endowed with \$2 and told they could keep the money—the safe bet—or roll a die and risk losing the \$2 for a payoff of \$4 (a risky but rational bet; odds of winning were 50/50). Participants indicated how “powerful” and “in charge” they felt on a scale from 1 (*not at all*) to 4 (*a lot*).

Saliva collection and analysis

Testing was scheduled in the afternoon (12:00 p.m.–6:00 p.m.) to control for diurnal rhythms in hormones. Saliva samples were taken before the power-pose manipulation (approximately 10 min after arrival; Time 1) and again 17 min after the power-pose manipulation ($M = 17.28$ min, $SD = 4.31$; Time 2).

Standard salivary-hormone collection procedures were used (Dickerson & Kemeny, 2004; Schultheiss & Stanton, 2009). Before providing saliva samples, participants did not eat, drink, or brush their teeth for at least 1 hr. Participants rinsed their mouths with water and chewed a piece of sugar-free Trident Original Flavor gum for 3 min to stimulate salivation (this procedure yields the least bias compared with passive drool procedures; Dabbs, 1991). Participants provided approximately 1.5 ml of saliva through a straw into a sterile polypropylene microtube. Samples were immediately frozen to avoid hormone degradation and to precipitate mucins. Within 2 weeks, samples were packed in dry ice and shipped for analysis to Salimetrics (State College, PA), where they were assayed in duplicate for salivary cortisol and salivary testosterone using a highly sensitive enzyme immunoassay.

For cortisol, the intra-assay coefficient of variation (CV) was 5.40% for Time 1 and 4.40% for Time 2. The average interassay CV across high and low controls for both time points was 2.74%. Cortisol levels were in the normal range at both Time 1 ($M = 0.16$ $\mu\text{g/dl}$, $SD = 0.19$) and Time 2 ($M = 0.12$ $\mu\text{g/dl}$, $SD = 0.08$). For testosterone, the intra-assay CV was 4.30% for Time 1 and 3.80% for Time 2. The average interassay CV across high and low controls for both time points was 3.80%. Testosterone levels were in the normal range at both Time 1 ($M = 60.30$ pg/ml , $SD = 49.58$) and Time 2 ($M = 57.40$ pg/ml , $SD = 43.25$). As would be suggested by appropriately taken and assayed samples (Schultheiss & Stanton, 2009), men were higher than women on testosterone at both

Time 1, $F(1, 41) = 17.40$, $p < .001$, $r = .55$, and Time 2, $F(1, 41) = 22.55$, $p < .001$, $r = .60$. To control for sex differences in testosterone, we used participant’s sex as a covariate in all analyses. All hormone analyses examined changes in hormones observed at Time 2, controlling for Time 1. Analyses with cortisol controlled for testosterone, and vice versa.²

Results

One-way analyses of variance examined the effect of power pose on postmanipulation hormones (Time 2), controlling for baseline hormones (Time 1). As hypothesized, high-power poses caused an increase in testosterone compared with low-power poses, which caused a decrease in testosterone, $F(1, 39) = 4.29$, $p < .05$; $r = .34$ (Fig. 3). Also as hypothesized, high-power poses caused a decrease in cortisol compared with low-power poses, which caused an increase in cortisol, $F(1, 38) = 7.45$, $p < .02$; $r = .43$ (Fig. 4).

Also consistent with predictions, high-power posers were more likely than low-power posers to focus on rewards—86.36% took the gambling risk (only 13.63% were risk averse). In contrast, only 60% of the low-power posers took the risk (and 40% were risk averse), $\chi^2(1, N = 42) = 3.86$, $p < .05$; $\Phi = .30$. Finally, high-power posers reported feeling significantly more “powerful” and “in charge” ($M = 2.57$, $SD = 0.81$) than low-power posers did ($M = 1.83$, $SD = 0.81$), $F(1, 41) = 9.53$, $p < .01$; $r = .44$. Thus, a simple 2-min power-pose manipulation was enough to significantly alter the physiological, mental, and feeling states of our participants. The implications of these results for everyday life are substantial.

Discussion

Our results show that posing in high-power displays (as opposed to low-power displays) causes physiological, psychological, and behavioral changes consistent with the literature on the effects of power on power holders—elevation of the dominance hormone testosterone, reduction of the stress hormone cortisol, and increases in behaviorally demonstrated risk tolerance and feelings of power.

These findings advance current understanding of embodied cognition in two important ways. First, they suggest that the effects of embodiment extend beyond emotion and cognition, to physiology and subsequent behavioral choice. For example, as described earlier, nodding the head “yes” leads a person to be more easily persuaded when listening to a persuasive appeal, and smiling increases humor responses. We suggest that these simple behaviors, a head nod or a smile, might also cause physiological changes that activate an entire trajectory of psychological, physiological, and behavioral shifts—essentially altering the course of a person’s day. Second, these results suggest that any psychological construct, such as power, with a signature pattern of nonverbal correlates may be embodied.

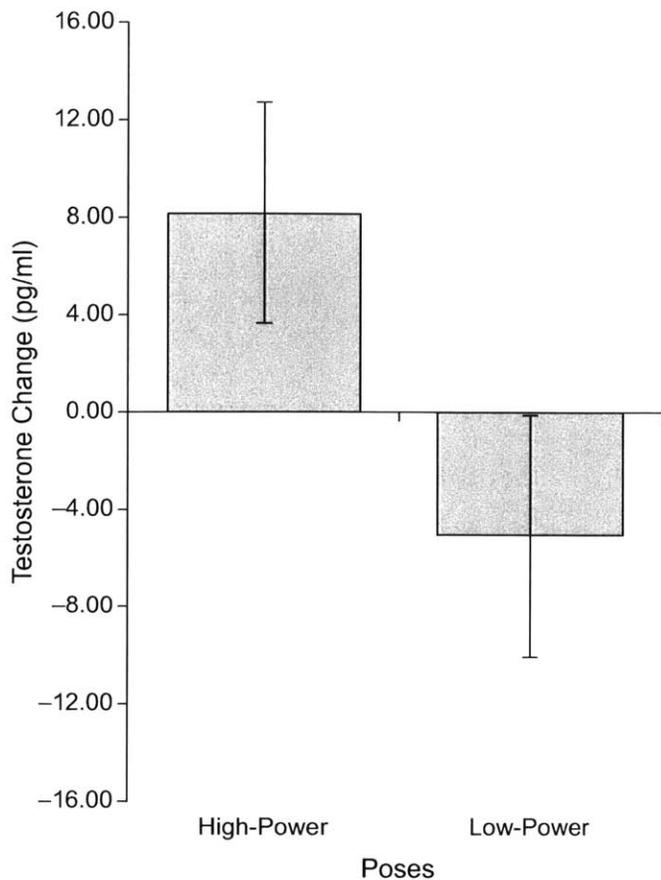


Fig. 3. Mean changes in the dominance hormone testosterone following high-power and low-power poses. Changes are depicted as difference scores (Time 2 – Time 1). Error bars represent standard errors of the mean.

These results also offer a methodological advance in research on power. Many reported effects of power are limited by the methodological necessity of manipulating power in a laboratory setting (e.g., complex role assignments). The simple, elegant power-pose manipulation we employed can be taken directly into the field and used to investigate ordinary people in everyday contexts.

Is it possible that our findings are limited to the specific poses utilized in this experiment? Although the power-infusing attribute of expansiveness and the poses that capture it require further investigation, findings from an additional study ($N = 49$) suggest that the effects reported here are not idiosyncratic to these specific poses. In addition to the poses used in the current report, an additional three high-power poses and an additional three low-power poses produced the same effects on feelings of power, $F(1, 48) = 4.38, p < .05, r = .30$, and risk taking, $\chi^2(1, N = 49) = 4.84, p < .03, \Phi = .31$.

By simply changing physical posture, an individual prepares his or her mental and physiological systems to endure difficult and stressful situations, and perhaps to actually improve confidence and performance in situations such as interviewing for jobs, speaking in public, disagreeing with a

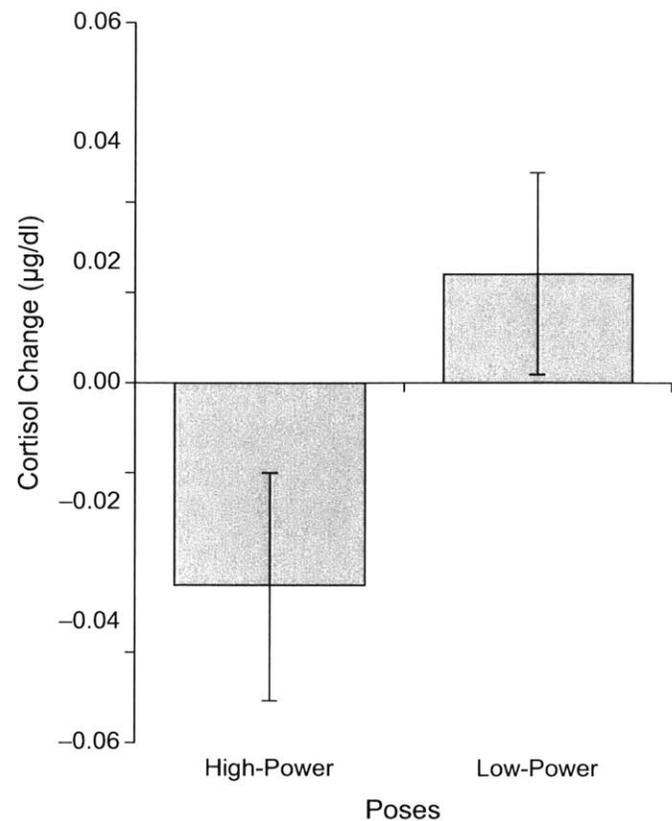


Fig. 4. Mean changes in the stress hormone cortisol following high-power and low-power poses. Changes are depicted as difference scores (Time 2 – Time 1). Error bars represent standard errors of the mean.

boss, or taking potentially profitable risks. These findings suggest that, in some situations requiring power, people have the ability to “fake it ’til they make it.” Over time and in aggregate, these minimal postural changes and their outcomes potentially could improve a person’s general health and well-being. This potential benefit is particularly important when considering people who are or who feel chronically powerless because of lack of resources, low hierarchical rank in an organization, or membership in a low-power social group.

Acknowledgments

We are gratefully indebted to the following individuals for their insight, support, and assistance with this research: Daniel Ames, Max Bazerman, Joe Ferrero, Alan Fiske and lab, Adam Galinsky, Deborah Gruenfeld, Lucia Guillory, Brian Hall, Bob Josephs, Brian Lucas, Malia Mason, Pranj Mehta, Michael Morris, Joe Navarro, Michael Norton, Thomas Schubert, Steve Stroessner, and Bill von Hippel.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Notes

1. The effect of power on risk taking is moderated by factors such as prenatal exposure to testosterone (Ronay & von Hippel, in press).
2. Cortisol scores at both time points were sufficiently normally distributed, except for two outliers that were more than 3 standard deviations above the mean and were excluded; testosterone scores at both time points were sufficiently normally distributed, except for one outlier that was more than 3 standard deviations above the mean and was excluded.

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Experiment 1 - Quiz 2

BrightBeat

Question set II

What was the main take away message of the article?

- Taking a nap after a learning task boosts memory.
- Taking a nap accompanied by a sound cue after a learning task boosts memory.
- Taking a nap accompanied by an odor cue after a learning task boosts memory.
- None of the above

Presenting a cue and testing if it reliably influences recall accuracy was performed during ...

- awake state
- slow-wave sleep
- testing phase
- awake and slow-wave sleep

Please check all the statements that are true about the experiment.

- Participants were told before taking a nap that they will be cued.
- Participants professed no knowledge that the cue that was presented to them during napping.
- Participants performed better than chance to guess which cue was presented to them during napping.

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Experiment 1 - Relaxing and reading task

How to make stress your friend

Kelly McGonigal

I have a confession to make, but first, I want you to make a little confession to me. In the past year, I want you to just raise your hand

if you've experienced relatively little stress. Anyone?

How about a moderate amount of stress?

Who has experienced a lot of stress? Yeah. Me too.

But that is not my confession. My confession is this: I am a health psychologist, and my mission is to help people be happier and healthier. But I fear that something I've been teaching for the last 10 years is doing more harm than good, and it has to do with stress. For years I've been telling people, stress makes you sick. It increases the risk of everything from the common cold to cardiovascular disease. Basically, I've turned stress into the enemy. But I have changed my mind about stress, and today, I want to change yours.

Let me start with the study that made me rethink my whole approach to stress. This study tracked 30,000 adults in the United States for eight years, and they started by asking people, "How much stress have you experienced in the last year?" They also asked, "Do you believe that stress is harmful for your health?" And then they used public death records to find out who died.

Okay. Some bad news first. People who experienced a lot of stress in the previous year had a 43 percent increased risk of dying. But that was only true for the people who also believed that stress is harmful for your health. (Laughter) People who experienced a lot of stress but did not view stress as harmful were no more likely to die. In fact, they had the lowest risk of dying of anyone in the study, including people who had relatively little stress.

Now the researchers estimated that over the eight years they were tracking deaths, 182,000 Americans died prematurely, not from stress, but from the belief that stress is bad for you. That is over 20,000 deaths a year. Now, if that estimate is correct, that would make believing stress is bad for you the 15th largest cause of death in the United States last year, killing more people than skin cancer, HIV/AIDS and homicide.

You can see why this study freaked me out. Here I've been spending so much energy telling people stress is bad for your health.

So this study got me wondering: Can changing how you think about stress make you healthier? And here the science says yes. When you change your mind about stress, you can change your body's response to stress.

Now to explain how this works, I want you all to pretend that you are participants in a study

designed to stress you out. It's called the social stress test. You come into the laboratory, and you're told you have to give a five-minute impromptu speech on your personal weaknesses to a panel of expert evaluators sitting right in front of you, and to make sure you feel the pressure, there are bright lights and a camera in your face, kind of like this. And the evaluators have been trained to give you discouraging, non-verbal feedback like this.

Now that you're sufficiently demoralized, time for part two: a math test. And unbeknownst to you, the experimenter has been trained to harass you during it. Now we're going to all do this together. It's going to be fun. For me.

Okay. I want you all to count backwards from 996 in increments of seven. You're going to do this out loud as fast as you can, starting with 996. Go! Audience: (Counting) Go faster. Faster please. You're going too slow. Stop. Stop, stop, stop. That guy made a mistake. We are going to have to start all over again. (Laughter) You're not very good at this, are you? Okay, so you get the idea. Now, if you were actually in this study, you'd probably be a little stressed out. Your heart might be pounding, you might be breathing faster, maybe breaking out into a sweat. And normally, we interpret these physical changes as anxiety or signs that we aren't coping very well with the pressure.

But what if you viewed them instead as signs that your body was energized, was preparing you to meet this challenge? Now that is exactly what participants were told in a study conducted at Harvard University. Before they went through the social stress test, they were taught to rethink their stress response as helpful. That pounding heart is preparing you for action. If you're breathing faster, it's no problem. It's getting more oxygen to your brain. And participants who learned to view the stress response as helpful for their performance, well, they were less stressed out, less anxious, more confident, but the most fascinating finding to me was how their physical stress response changed. Now, in a typical stress response, your heart rate goes up, and your blood vessels constrict like this. And this is one of the reasons that chronic stress is sometimes associated with cardiovascular disease. It's not really healthy to be in this state all the time. But in the study, when participants viewed their stress response as helpful, their blood vessels stayed relaxed like this. Their heart was still pounding, but this is a much healthier cardiovascular profile. It actually looks a lot like what happens in moments of joy and courage. Over a lifetime of stressful experiences, this one biological change could be the difference between a stress-induced heart attack at age 50 and living well into your 90s. And this is really what the new science of stress reveals, that how you think about stress matters.

So my goal as a health psychologist has changed. I no longer want to get rid of your stress. I want to make you better at stress. And we just did a little intervention. If you raised your hand and said you'd had a lot of stress in the last year, we could have saved your life, because hopefully the next time your heart is pounding from stress, you're going to remember this talk and you're going to think to yourself, this is my body helping me rise to this challenge. And when you view stress in that way, your body believes you, and your stress response becomes healthier.

Now I said I have over a decade of demonizing stress to redeem myself from, so we are going to do one more intervention. I want to tell you about one of the most under-appreciated aspects of the stress response, and the idea is this: Stress makes you social.

To understand this side of stress, we need to talk about a hormone, oxytocin, and I know oxytocin has already gotten as much hype as a hormone can get. It even has its own cute nickname, the cuddle hormone, because it's released when you hug someone. But this is a very small part of what oxytocin is involved in. Oxytocin is a neuro-hormone. It fine-tunes your brain's social instincts. It primes you to do things that strengthen close relationships. Oxytocin makes you crave physical contact with your friends and family. It enhances your empathy. It even makes you more willing to help and support the people you care about. Some people have even suggested we should snort oxytocin to become more compassionate and caring. But here's what most people don't understand about oxytocin. It's a stress hormone. Your pituitary gland pumps this stuff out as part of the stress response. It's as much a part of your stress response as the adrenaline that makes your heart pound. And when oxytocin is released in the stress response, it is motivating you to seek support. Your biological stress response is nudging you to tell someone how you feel instead of bottling it up. Your stress response wants to make sure you notice when someone else in your life is struggling so that you can support each other. When life is difficult, your stress response wants you to be surrounded by people who care about you.

Okay, so how is knowing this side of stress going to make you healthier? Well, oxytocin doesn't only act on your brain. It also acts on your body, and one of its main roles in your body is to protect your cardiovascular system from the effects of stress. It's a natural anti-inflammatory. It also helps your blood vessels stay relaxed during stress. But my favorite effect on the body is actually on the heart. Your heart has receptors for this hormone, and oxytocin helps heart cells regenerate and heal from any stress-induced damage. This stress hormone strengthens your heart, and the cool thing is that all of these physical benefits of oxytocin are enhanced by social contact and social support, so when you reach out to others under stress, either to seek support or to help someone else, you release more of this hormone, your stress response becomes healthier, and you actually recover faster from stress. I find this amazing, that your stress response has a built-in mechanism for stress resilience, and that mechanism is human connection.

I want to finish by telling you about one more study. And listen up, because this study could also save a life. This study tracked about 1,000 adults in the United States, and they ranged in age from 34 to 93, and they started the study by asking, "How much stress have you experienced in the last year?" They also asked, "How much time have you spent helping out friends, neighbors, people in your community?" And then they used public records for the next five years to find out who died.

Okay, so the bad news first: For every major stressful life experience, like financial difficulties or family crisis, that increased the risk of dying by 30 percent. But -- and I hope you are expecting a but by now -- but that wasn't true for everyone. People who spent time caring for others showed absolutely no stress-related increase in dying. Zero. Caring created resilience. And so we see once

again that the harmful effects of stress on your health are not inevitable. How you think and how you act can transform your experience of stress. When you choose to view your stress response as helpful, you create the biology of courage. And when you choose to connect with others under stress, you can create resilience. Now I wouldn't necessarily ask for more stressful experiences in my life, but this science has given me a whole new appreciation for stress. Stress gives us access to our hearts. The compassionate heart that finds joy and meaning in connecting with others, and yes, your pounding physical heart, working so hard to give you strength and energy, and when you choose to view stress in this way, you're not just getting better at stress, you're actually making a pretty profound statement. You're saying that you can trust yourself to handle life's challenges, and you're remembering that you don't have to face them alone.

Thank you.

(Applause)

Chris Anderson: This is kind of amazing, what you're telling us. It seems amazing to me that a belief about stress can make so much difference to someone's life expectancy. How would that extend to advice, like, if someone is making a lifestyle choice between, say, a stressful job and a non-stressful job, does it matter which way they go? It's equally wise to go for the stressful job so long as you believe that you can handle it, in some sense?

Kelly McGonigal: Yeah, and one thing we know for certain is that chasing meaning is better for your health than trying to avoid discomfort. And so I would say that's really the best way to make decisions, is go after what it is that creates meaning in your life and then trust yourself to handle the stress that follows.

CA: Thank you so much, Kelly. It's pretty cool. KM: Thank you.

Experiment 2 - Reading task 1

The Truth about Relativity

Why Everything Is Relative—Even When It Shouldn't Be

One day while browsing the World Wide Web (obviously for work—not just wasting time), I stumbled on the following ad, on the Web site of a magazine, the *Economist*.

Economist.com	
OPINION	SUBSCRIPTIONS Welcome to The Economist Subscription Centre Pick the type of subscription you want to buy or renew. <input type="checkbox"/> Economist.com subscription - US \$59.00 One-year subscription to Economist.com. Includes online access to all articles from <i>The Economist</i> since 1997. <input type="checkbox"/> Print subscription - US \$125.00 One-year subscription to the print edition of <i>The Economist</i> . <input type="checkbox"/> Print & web subscription - US \$125.00 One-year subscription to the print edition of <i>The Economist</i> and online access to all articles from <i>The Economist</i> since 1997.
WORLD	
BUSINESS	
FINANCE & ECONOMICS	
SCIENCE & TECHNOLOGY	
PEOPLE	
BOOKS & ARTS	
MARKETS & DATA	
DIVERSIONS	

I read these offers one at a time. The first offer—the Internet subscription for \$59—seemed reasonable. The second option—the \$125 print subscription—seemed a bit expensive, but still reasonable.

But then I read the third option: a print *and* Internet subscription for \$125. I read it twice before my eye ran back to the previous options. Who would want to buy the print option alone, I wondered, when both the Internet and the print subscriptions were offered for the same price? Now, the print-only option may have been a typographical error, but I suspect that the clever people at the *Economist's* London offices (and they are clever—and quite mischievous in a British sort of way) were actually manipulating me. I am pretty certain that they wanted me to skip the Internet-only option (which they assumed would be my choice, since I was reading the advertisement on the Web) and jump to the more expensive option: Internet and print.

But how could they manipulate me? I suspect it's because the *Economist's* marketing wizards (and I could just picture them in their school ties and blazers) knew something important about human behavior: humans rarely choose things in absolute terms. We don't have an internal value meter that tells us how much things are worth. Rather, we focus on the relative advantage of one thing over another, and estimate value accordingly. (For instance, we don't know how much a six-cylinder car is worth, but we can assume it's more expensive than the four-cylinder model.)

In the case of the *Economist*, I may not have known whether the Internet-only subscription at \$59 was a better deal than the print-only option at \$125. But I certainly knew that the print- and-Internet option for \$125 was better than the print-only option at \$125. In fact, you could reasonably deduce that in the combination package, the Internet subscription is free! "It's a bloody steal— go for it, governor! " I could almost hear them shout from the riverbanks of the Thames. And I have to admit. If I had been inclined to subscribe I probably would have taken the package deal myself. (Later, when I tested the offer on a large number of participants, the vast majority preferred the Internet-and-print deal.)

So what was going on here? Let me start with a fundamental observation: most people don't know what they want unless they see it in context. We don't know what kind of racing bike we want—until we see a champ in the Tour de France ratcheting the gears on a particular model. We don't know what kind of speaker system we like—until we hear a set of speakers that sounds better than the previous one. We don't even know what we want to do with our lives—until we find a relative or a friend who is doing just what we think we should be doing. Everything is relative, and that's the point. Like an airplane pilot landing in the dark, we want runway lights on either side of us, guiding us to the place where we can touch down our wheels.

In the case of the *Economist*, the decision between the Internet- only and print-only options would take a bit of thinking. Thinking is difficult and sometimes unpleasant. So the *Economist's* marketers offered us a no-brainer: relative to the print-only option, the print-and-Internet option looks clearly superior.

So LET'S RUN through the Economist's slow motion.

As you recall, the choices were:

1. Internet-only subscription for \$59.
2. Print-only subscription for \$125.
3. Print-and-Internet subscription for \$125.

When I gave these options to 100 students at MIT's Sloan School of Management, they opted as follows:

1. Internet-only subscription for \$59— 16 students
2. Print-only subscription for \$125— zero students
3. Print-and-Internet subscription for \$125— 84 students

So far these Sloan MBAs are smart cookies. They all saw the advantage in the print-and-Internet offer over the print-only offer. But were they influenced by the mere presence of the print-only option (which I will henceforth, and for good reason, call the "decoy"). In other words, suppose that I removed the decoy so that the choices would be the ones seen in the figure below:

Economist.com		SUBSCRIPTIONS	
OPINION		Welcome to	
WORLD		The Economist Subscription Centre	
BUSINESS		Pick the type of subscription you want to buy or renew.	
FINANCE & ECONOMICS		<input type="checkbox"/> Economist.com subscription - US \$59.00	
SCIENCE & TECHNOLOGY		One-year subscription to Economist.com.	
PEOPLE		Includes online access to all articles from <i>The Economist</i> since 1997.	
BOOKS & ARTS		<input type="checkbox"/> Print & web subscription - US \$125.00	
MARKETS & DATA		One-year subscription to the print edition of <i>The Economist</i> and online access to all articles from <i>The Economist</i> since 1997.	
DIVERSIONS			

Would the students respond as before (16 for the Internet only and 84 for the combination)?

Certainly they would react the same way, wouldn't they? After all, the option I took out was one that no one selected, so it should make no difference. Right?

Au contraire! This time, 68 of the students chose the Internet-only option for \$59, up from 16 before. And only 32 chose the combination subscription for \$125, down from 84 before.

Economist.com		SUBSCRIPTIONS	
OPINION		Welcome to	
WORLD		The Economist Subscription Centre	
BUSINESS		Pick the type of subscription you want to buy or renew.	
FINANCE & ECONOMICS		<input type="checkbox"/> Economist.com subscription - US \$59.00	
SCIENCE & TECHNOLOGY		One-year subscription to Economist.com.	
PEOPLE		Includes online access to all articles from <i>The Economist</i> since 1997. (16)	
BOOKS & ARTS		<input type="checkbox"/> Print subscription - US \$125.00	
MARKETS & DATA		One-year subscription to the print edition of <i>The Economist</i> . (0)	
DIVERSIONS		<input type="checkbox"/> Print & web subscription - US \$125.00	
		One-year subscription to the print edition of <i>The Economist</i> and online access to all articles from <i>The Economist</i> since 1997. (84)	

Economist.com		SUBSCRIPTIONS	
OPINION		Welcome to	
WORLD		The Economist Subscription Centre	
BUSINESS		Pick the type of subscription you want to buy or renew.	
FINANCE & ECONOMICS		<input type="checkbox"/> Economist.com subscription - US \$59.00	
SCIENCE & TECHNOLOGY		One-year subscription to Economist.com.	
PEOPLE		Includes online access to all articles from <i>The Economist</i> since 1997. (68)	
BOOKS & ARTS		<input type="checkbox"/> Print & web subscription - US \$125.00	
MARKETS & DATA		One-year subscription to the print edition of <i>The Economist</i> and online access to all articles from <i>The Economist</i> since 1997. (32)	
DIVERSIONS			

What could have possibly changed their minds? Nothing rational, I assure you. It was the mere presence of the decoy that sent 84 of them to the print-and-Internet option (and 16 to the Internet-only option). And the absence of the decoy had them choosing differently, with 32 for print-and-Internet and 68 for Internet-only.

This is not only irrational but predictably irrational as well.

Experiment 2 - Quiz 1

BrightBeat: MIT Media Lab study on calming technologies

Question set I

Economist.com	
OPINION	SUBSCRIPTIONS Welcome to The Economist Subscription Centre Pick the type of subscription you want to buy or renew. <input type="checkbox"/> Economist.com subscription - US \$59.00 One-year subscription to Economist.com. Includes online access to all articles from <i>The Economist</i> since 1997. <input type="checkbox"/> Print subscription - US \$125.00 One-year subscription to the print edition of <i>The Economist</i> . <input type="checkbox"/> Print & web subscription - US \$125.00 One-year subscription to the print edition of <i>The Economist</i> and online access to all articles from <i>The Economist</i> since 1997.
WORLD	
BUSINESS	
FINANCE & ECONOMICS	
SCIENCE & TECHNOLOGY	
PEOPLE	
BOOKS & ARTS	
MARKETS & DATA	
DIVERSIONS	
Study A	

Economist.com	
OPINION	SUBSCRIPTIONS Welcome to The Economist Subscription Centre Pick the type of subscription you want to buy or renew. <input type="checkbox"/> Economist.com subscription - US \$59.00 One-year subscription to Economist.com. Includes online access to all articles from <i>The Economist</i> since 1997. <input type="checkbox"/> Print & web subscription - US \$125.00 One-year subscription to the print edition of <i>The Economist</i> and online access to all articles from <i>The Economist</i> since 1997.
WORLD	
BUSINESS	
FINANCE & ECONOMICS	
SCIENCE & TECHNOLOGY	
PEOPLE	
BOOKS & ARTS	
MARKETS & DATA	
DIVERSIONS	
Study B	

In the MIT study A, approximately what percentage of participants chose the print-only option?

- 0
- 16
- 32
- 68

In the MIT studies A and B, which option was more preferred?

- email, in both studies
- print and email, in both studies
- email in study A, print and email in study B
- print and email in study A, email in study B

In the MIT studies A and B, how did the percentage of email subscription change after removing the print-only option?

- increased
- decreased
- didn't change
- wasn't reported

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Facebook Use Predicts Declines in Subjective Well-Being in Young Adults

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Abstract

Over 500 million people interact daily with Facebook. Yet, whether Facebook use influences subjective well-being over time is unknown. We addressed this issue using experience-sampling, the most reliable method for measuring in-vivo behavior and psychological experience. We text-messaged people five times per day for two-weeks to examine how Facebook use influences the two components of subjective well-being: how people feel moment-to-moment and how satisfied they are with their lives. Our results indicate that Facebook use predicts negative shifts on both of these variables over time. The more people used Facebook at one time point, the worse they felt the next time we text-messaged them; the more they used Facebook over two-weeks, the more their life satisfaction levels declined over time. Interacting with other people “directly” did not predict these negative outcomes. They were also not moderated by the size of people’s Facebook networks, their perceived supportiveness, motivation for using Facebook, gender, loneliness, self-esteem, or depression. On the surface, Facebook provides an invaluable resource for fulfilling the basic human need for social connection. Rather than enhancing well-being, however, these findings suggest that Facebook may undermine it.

Citation: Kross E, Verduyn P, Demiralp E, Park J, Lee DS, et al. (2013) Facebook Use Predicts Declines in Subjective Well-Being in Young Adults. PLoS ONE 8(8): e69841. doi:10.1371/journal.pone.0069841

Editor: Cédric Sueur, Institut Pluridisciplinaire Hubert Curien, France

Received: January 31, 2013; **Accepted:** June 12, 2013; **Published:** August 14, 2013

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Funding: The authors have no support or funding to report.

Competing Interests: The authors have declared that no competing interests exist.

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Introduction

Online social networks are rapidly changing the way human beings interact. Over a billion people belong to Facebook, the world’s largest online social network, and over half of them log in daily [1]. Yet, no research has examined how interacting with Facebook influences subjective well-being over time. Indeed, a recent article that examined every peer-reviewed publication and conference proceeding on Facebook between 1/2005 and 1/2012 (412 in total) did not reveal a single study that examined how using this technology influences subjective well-being over time (Text S1) [2].

Subjective well-being is one of the most highly studied variables in the behavioral sciences. Although significant in its own right, it also predicts a range of consequential benefits including enhanced health and longevity [3–5]. Given the frequency of Facebook usage, identifying how interacting with this technology influences subjective well-being represents a basic research challenge that has important practical implications.

This issue is particularly vexing because prior research provides mixed clues about how Facebook use should influence subjective well-being. Whereas some cross-sectional research reveals positive associations between online social network use (in particular Facebook) and well-being [6], other work reveals the opposite [7,8]. Still other work suggests that the relationship between Facebook use and well-being may be more nuanced and potentially influenced by multiple factors including number of Facebook friends, perceived supportiveness of one’s online

network, depressive symptomatology, loneliness, and self-esteem [9,10,11].

So, how does Facebook usage influence subjective well-being over time? The cross-sectional approach used in previous studies makes it impossible to know. We addressed this issue by using experience-sampling, the most reliable method for measuring in-vivo behavior and psychological experience over time [12]. We text-messaged participants five times per day for 14-days. Each text-message contained a link to an online survey, which participants completed using their smartphones. We performed lagged analyses on participants’ responses, as well as their answers to the Satisfaction With Life Questionnaire (SWLS) [13], which they completed before and immediately following the 14-day experience-sampling period, to examine how interacting with Facebook influences the two components of subjective well-being: how people feel (“affective” well-being) and how satisfied they are with their lives (“cognitive” well-being) [14,15]. This approach allowed us to take advantage of the relative timing of participants’ natural Facebook behavior and psychological states to draw inferences about their likely causal sequence [16–19].

Methods

Participants

Eighty-two people ($M_{age} = 19.52$, $SD_{age} = 2.17$; 53 females; 60.5% European American, 28.4% Asian, 6.2% African American, and 4.9% other) were recruited for a study on Facebook through flyers posted around Ann Arbor, Michigan. Participants needed a Facebook account and a touch-screen smartphone to

qualify for the study. They received \$20 and were entered into a raffle to receive an iPad2 for participating.

Ethics Statement

The University of Michigan Institutional Review Board approved this study. Informed written consent was obtained from all participants prior to participation.

Materials and Procedure

Phase 1. Participants completed a set of questionnaires, which included the SWLS ($M = 4.96$, $SD = 1.17$), Beck Depression Inventory [20] ($M = 9.02$, $SD = 7.20$), the Rosenberg Self-Esteem Scale [21] ($M = 30.40$, $SD = 4.96$), and the Social Provision Scale [22] ($M = 3.55$, $SD = .34$), which we modified to assess perceptions of Facebook support. We also assessed participants' motivation for using Facebook by asking them to indicate whether they use Facebook "to keep in touch with friends (98% answered yes)," "to find new friends (23% answered yes)," "to share good things with friends (78% answered yes)," "to share bad things with friends (36% answered yes)," "to obtain new information (62% answered yes)," or "other: please explain (17% answered yes)." Examples of other reasons included chatting with others, keeping in touch with family, and facilitating schoolwork and business.

Phase 2. Participants were text-messaged 5 times per day between 10am and midnight over 14-days. Text-messages occurred at random times within 168-minute windows per day. Each text-message contained a link to an online survey, which asked participants to answer five questions using a slider scale: (1) How do you feel right now? (*very positive* [0] to *very negative* [100]; $M = 37.47$, $SD = 25.88$); (2) How worried are you right now? (*not at all* [0] to *a lot* [100]; $M = 44.04$, $SD = 30.42$); (3) How lonely do you feel right now? (*not at all* [0] to *a lot* [100]; $M = 27.61$, $SD = 26.13$); (4) How much have you used Facebook since the last time we asked? (*not at all* [0] to *a lot* [100]; $M = 33.90$, $SD = 30.48$); (5) How much have you interacted with other people "directly" since the last time we asked? (*not at all* [0] to *a lot* [100]; $M = 64.26$, $SD = 31.11$). When the protocol for answering these questions was explained, interacting with other people "directly" was defined as face-to-face or phone interactions. An experimenter carefully walked participants through this protocol to ensure that they understood how to answer each question and fulfill the study requirements.

Participants always answered the affect question first. Next the worry and loneliness questions were presented in random order. The Facebook use and direct social interaction questions were always administered last, again in random order. Our analyses focused primarily on affect (rather than worry and loneliness) because this affect question is the way "affective well-being" is typically operationalized.

Phase 3. Participants returned to the laboratory following Phase 2 to complete another set of questionnaires, which included the SWLS ($M = 5.13$, $SD = 1.26$) and the Revised UCLA Loneliness Scale [23] ($M = 1.69$, $SD = .46$). Participants' number of Facebook friends ($M = 664.25$, $SD = 383.64$) was also recorded during this session from participants' Facebook accounts (Text S2).

Results

Attrition and compliance

Three participants did not complete the study. As the methods section notes, participants received a text message directing them to complete a block of five questions once every 168 minutes on average (the text message was delivered randomly within this 168-minute window). A response to any question within a block was

considered "compliant" if it was answered *before* participants received a subsequent text-message directing them to complete the next block of questions. Participants responded to an average of 83.6% of text-messages (range: 18.6%–100%). Following prior research [24], we pruned the data by excluding all of the data from two participants who responded to <33% of the texts, resulting in 4,589 total observations. The results did not change substantively when additional cutoff rates were used.

Analyses overview

We examined the relationship between Facebook use and affect using multilevel analyses to account for the nested data structure. Specifically, we examined whether T_2 affect (i.e., How do you feel *right now*?) was predicted by T_1 Facebook use (i.e., How much have you used Facebook *since the last time we asked*?), controlling for T_1 affect at level-1 of the model (between-day lags were excluded). Note that although this analysis assesses Facebook use at T_2 , the question refers to usage between T_1 and T_2 (hence the notation T_1 – T_2). This analysis allowed us to explore whether Facebook use during the time period separating T_1 and T_2 predicted changes in affect over this time span.

When non-compliant cases were observed, we used participants' responses to the last text message they answered to examine the lagged effect of Facebook use on well-being to maximize power. So, if we were interested in examining whether T_2 Facebook use predicted T_3 Affect controlling for T_2 Affect, but did not have data on T_2 Affect, then we used T_1 Affect instead. Excluding trials in which participants did not respond to the previous texts (rather than following the aforementioned analytical scheme) did not substantively alter any of the results we report.

Significance testing of fixed effects was performed using chi-squared distributed ($df = 1$) Wald-tests. All level-1 predictors were group-mean centered, and intercepts and slopes were allowed to vary randomly across participants (see Table 1 for zero-order correlations). We tested for moderation by examining whether each moderator variable was related to the slope of T_1 – T_2 Facebook use when predicting T_2 affect, controlling for T_1 affect.

Data from one person who scored 4SDs above the sample mean on the BDI were excluded from the BDI moderation analyses; data from one person who scored 4SDs above the sample mean on number of Facebook friends were excluded from the moderation analyses based on Facebook friends.

The relationship between mean Facebook use and life satisfaction was assessed using OLS regressions because these data were not nested. Both unstandardized (B) and standardized (β) OLS regression coefficients are reported (see Text S3).

Facebook use and well-being

Affective well-being. We examined whether people's tendency to interact with Facebook during the time period separating two text messages influenced how they felt at T_2 , controlling for how they felt at T_1 . Nested time-lag analyses indicated that the more people used Facebook the worse they subsequently felt, $B = .08$, $\chi^2 = 28.90$, $p < .0001$, (see Figure 1, top). The reverse pathway (T_1 Affect predicting T_1 – T_2 Facebook use, controlling for T_0 – T_1 Facebook use) was not significant, $B = -.005$, $\chi^2 = .05$, $p = .82$, indicating that people do not use Facebook more or less depending on how they feel (see Text S4, S5).

Cognitive well-being. To examine how Facebook use influenced "cognitive well-being," we analyzed whether people's average Facebook use over the 14-day period predicted their life satisfaction at the end of the study, controlling for baseline life satisfaction and average emotion levels over the 14-day period. The more participants used Facebook, the more their life

Table 1. Within-person and between-person zero-order correlations.

	Experience-sampled variables				Pre/post experience sampling		
	Affect	Worry	Loneliness	Facebook use	Direct contact	Pre life satisfaction	Post life satisfaction
Affect	-	.53***	.50***	.14***	-.29***	-	-
Worry	.77***	-	.37***	.17***	-.23***	-	-
Loneliness	.68***	.66***	-	.22***	-.40***	-	-
Facebook Use	.07	.13	.22*	-	-.24***	-	-
Direct Contact	-.28*	-.09	-.39***	.26*	-	-	-
Pre Life Satisfaction	-.55***	-.41***	-.40***	-.05	.29**	-	-
Post Life Satisfaction	-.66***	-.51***	-.48***	-.18	.23*	.86***	-

Note. Correlations above the dashed diagonal line represent within-person correlations obtained from multi-level analyses. Correlations below the dashed diagonal line represent between-person correlations.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

doi:10.1371/journal.pone.0069841.t001

satisfaction levels declined over time, $B = -.012$, $\beta = -.124$, $t(73) = -2.39$, $p = .02$, (see Figure 1, bottom).

Alternative explanations. An alternative explanation for these results is that any form of social interaction undermines well-being. Because we also asked people to indicate how frequently they interacted with other people “directly” since the last time we text messaged them, we were able to test this idea. Specifically, we repeated each of the aforementioned analyses substituting “direct”

social interaction for Facebook use. In contrast to Facebook use, “direct” social interaction did not predict changes in cognitive well-being, $B = -.006$, $\beta = -.059$, $t(73) = 1.04$, $p = .30$, and predicted *increases* (not decreases) in affective well-being, $B = -.15$, $\chi^2 = 65.30$, $p < .0001$. Controlling for direct social interaction did not substantively alter the significant relationship between Facebook use and affective well-being, $B = .05$, $\chi^2 = 10.78$, $p < .01$.

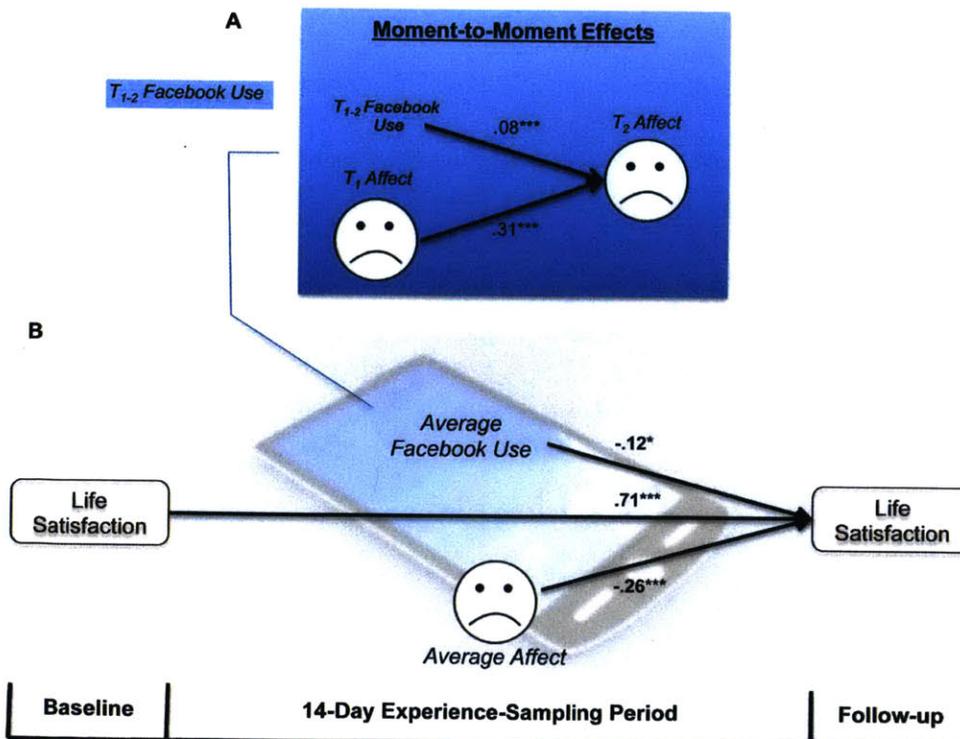


Figure 1. Facebook use predicts declines in affect and life satisfaction over time. Interacting with Facebook during one time period (T_{1-2}) leads people to feel worse later on during the same day (T_2) controlling for how they felt initially (T_1); values are regression weights from multilevel analyses (Panel A). Average Facebook use over the course of the 14-day experience-sampling period predicts decreases in life satisfaction over time; values are standardized regression weights from OLS regression analysis (Panel B). * $p < .05$, ** $p < .01$, *** $p < .001$. doi:10.1371/journal.pone.0069841.g001

Another alternative explanation for these results is that people use Facebook when they feel bad (i.e., when they are bored lonely, worried or otherwise distressed), and feeling bad leads to declines in well-being rather than Facebook use per se. The analyses we reported earlier partially address this issue by demonstrating that affect does not predict changes in Facebook use over time and Facebook use continues to significantly predict declines in life satisfaction over time when controlling for affect. However, because participants also rated how lonely and worried they felt each time we text messaged them, we were able to test this proposal further.

We first examined whether worry or loneliness predicted changes in Facebook use over time (i.e., T_1 worry [or T_1 loneliness] predicting $T_{1,2}$ Facebook use, controlling for $T_{0,1}$ Facebook use). Worry did not predict changes in Facebook use, $B = .04$, $\chi^2 = 2.37$, $p = .12$, but loneliness did, $B = .07$, $\chi^2 = 8.54$, $p < .01$. The more lonely people felt at one time point, the more people used Facebook over time. Given this significant relationship, we next examined whether controlling for loneliness renders the relationship between Facebook use and changes in affective and cognitive well-being non-significant—what one would predict if Facebook use is a proxy for loneliness. This was not the case. Facebook use continued to predict declines in affective well-being, $B = .08$, $\chi^2 = 27.87$, $p < .0001$, and cognitive well-being, $B = -.012$, $\beta = -.126$, $t(72) = 2.34$, $p = .02$, when loneliness was controlled for in each analysis. Neither worry nor loneliness interacted significantly with Facebook use to predict changes in affective or cognitive well-being ($ps > .44$).

Moderation. Next, we examined whether a number of theoretically relevant individual-difference variables including participants' number of Facebook Friends, their perceptions of their Facebook network support, depressive symptoms, loneliness, gender, self-esteem, time of study participation, and motivation for using Facebook (e.g., to find new friends, to share good or bad things, to obtain new information) interacted with Facebook use to predict changes in affective or cognitive well-being (Text S6). In no case did we observe any significant interactions ($ps > .16$).

Exploratory analyses. Although we did not have *a priori* predictions about whether Facebook use and direct social contact would interact to predict changes in affective and cognitive well-being, we nevertheless explored this issue in our final set of analyses. The results of these analyses indicated that Facebook use and direct social contact interacted significantly to predict changes in affective well-being, $B = .002$, $\chi^2 = 19.55$, $p < .0001$, but not changes in cognitive well-being, $B = .000$, $\beta = .129$, $t(71) = .39$, $p = .70$. To understand the meaning of the former interaction, we performed simple slope analyses. These analyses indicated that the relationship between Facebook use and declines in affective well-being increased linearly with direct social contact. Specifically, whereas Facebook use did not predict significant declines in affective well-being when participants experienced low levels of direct social contact (i.e., 1 standard deviation below the sample mean for direct social contact; $B = .00$, $\chi^2 = .04$, $p = .84$), it did predict significant declines in well-being when participants experienced moderate levels of direct social contact (i.e., at the sample mean for direct social contact; $B = .05$, $\chi^2 = 11.21$, $p < .001$) and high levels of direct social contact (i.e., 1 standard deviation above the sample mean for direct social contact; $B = .10$, $\chi^2 = 28.82$, $p < .0001$).

Discussion

Within a relatively short timespan, Facebook has revolutionized the way people interact. Yet, whether using Facebook predicts

changes in subjective well-being over time is unknown. We addressed this issue by performing lagged analyses on experience sampled data, an approach that allowed us to take advantage of the relative timing of participants' naturally occurring behaviors and psychological states to draw inferences about their likely causal sequence [17,18]. These analyses indicated that Facebook use predicts declines in the two components of subjective well-being: how people feel moment to moment and how satisfied they are with their lives.

Critically, we found no evidence to support two plausible alternative interpretations of these results. First, interacting with other people "directly" did not predict declines in well-being. In fact, direct social network interactions led people to feel *better* over time. This suggests that Facebook use may constitute a unique form of social network interaction that predicts impoverished well-being. Second, multiple types of evidence indicated that it was not the case that Facebook use led to declines in well-being because people are more likely to use Facebook when they feel bad—neither affect nor worry predicted Facebook use and Facebook use continued to predict significant declines in well-being when controlling for loneliness (which did predict increases in Facebook use and reductions in emotional well-being).

Would engaging in any solitary activity similarly predict declines in well-being? We suspect that they would not because people often derive pleasure from engaging in some solitary activities (e.g., exercising, reading). Supporting this view, a number of recent studies indicate that people's *perceptions* of social isolation (i.e., how lonely they feel)—a variable that we assessed in this study, which did not influence our results—are a more powerful determinant of well-being than *objective* social isolation [25]. A related question concerns whether engaging in any Internet activity (e.g., email, web surfing) would likewise predict well-being declines. Here too prior research suggests that it would not. A number of studies indicate that whether interacting with the Internet predicts changes in well-being depends on how you use it (i.e., what sites you visit) and who you interact with [26].

Future research

Although these findings raise numerous future research questions, four stand out as most pressing. First, do these findings generalize? We concentrated on young adults in this study because they represent a core Facebook user demographic. However, examining whether these findings generalize to additional age groups is important. Future research should also examine whether these findings generalize to other online social networks. As a recent review of the Facebook literature indicated [2] "[different online social networks] have varied histories and are associated with different patterns of use, user characteristics, and social functions (p. 205)." Therefore, it is possible that the current findings may not neatly generalize to other online social networks.

Second, what mechanisms underlie the deleterious effects of Facebook usage on well-being? Some researchers have speculated that online social networking may interfere with physical activity, which has cognitive and emotional replenishing effects [27] or trigger damaging social comparisons [8,28]. The latter idea is particularly interesting in light of the significant interaction we observed between direct social contact and Facebook use in this study—i.e., the more people interacted with other people directly, the more strongly Facebook use predicted declines in their affective well-being. If harmful social comparisons explain how Facebook use predicts declines in affective well-being, it is possible that interacting with other people directly either enhances the frequency of such comparisons or magnifies their emotional impact. Examining whether these or other mechanisms explain

the relationship between Facebook usage and well-being is important both from a basic science and practical perspective.

Finally, although the analytic approach we used in this study is useful for drawing inferences about the likely causal ordering of associations between naturally occurring variables, experiments that manipulate Facebook use in daily life are needed to corroborate these findings and establish definitive causal relations. Though potentially challenging to perform—Facebook use prevalence, its centrality to young adult daily social interactions, and addictive properties may make it a difficult intervention target—such studies are important for extending this work and informing future interventions.

Caveats

Two caveats are in order before concluding. First, although we observed statistically significant associations between Facebook usage and well-being, the sizes of these effects were relatively “small.” This should not, however, undermine their practical significance [29]. Subjective well-being is a multiply determined outcome—it is unrealistic to expect any single factor to powerfully influence it. Moreover, in addition to being consequential in its own right, subjective well-being predicts an array of mental and physical health consequences. Therefore, identifying any factor that systematically influences it is important, especially when that factor is likely to accumulate over time among large numbers of people. Facebook usage would seem to fit both of these criteria.

Second, some research suggests that asking people to indicate how good or bad they feel using a single bipolar scale, as we did in this study, can obscure interesting differences regarding whether a variable leads people to feel less positive, more negative or both less positive and more negative. Future research should administer two unipolar affect questions to assess positive and negative affect separately to address this issue.

Concluding Comment

The human need for social connection is well established, as are the benefits that people derive from such connections [30–34]. On

the surface, Facebook provides an invaluable resource for fulfilling such needs by allowing people to instantly connect. Rather than enhancing well-being, as frequent interactions with supportive “offline” social networks powerfully do, the current findings demonstrate that interacting with Facebook may predict the opposite result for young adults—it may undermine it.

Supporting Information

Text S1
(DOCX)

Text S2
(DOCX)

Text S3
(DOCX)

Text S4
(DOCX)

Text S5
(DOCX)

Text S6.
(DOCX)

Acknowledgments

We thank Emily Kean for her assistance running the study and Ozlem Ayduk and Phoebe Ellsworth for their feedback.

Author Contributions

Conceived and designed the experiments: EK ED JP DSL NL JJ OY. Performed the experiments: HS NL. Analyzed the data: PV ED. Wrote the paper: EK ED PV JJ OY. Discussed the results and commented on the manuscript: EK PV ED JP DSL NL HS JJ OY.

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Experiment 2 - Quiz 2

BrightBeat: MIT Media Lab study on calming technologies

Question set II

Approximately how many participants were in the study?

- 1,000,000
- 10,000
- 500
- 80

How long was the study?

- 2 years
- 2 months
- 2 weeks
- 2 days

What was the cut-off criteria for including users in the analysis?

- If answering at least 83% of messages
- If answering at least 33% of messages
- If answering at least 33% of messages before receiving the next message
- if answering at least 33% of messages within a day

Among the items below, select the ones that were measured in experience sampling.

- perceived supportiveness
- self-esteem
- cognitive wellbeing
- loneliness

Select all the true statements about the relationship between Facebook usage and affective wellbeing.

- More facebook usage influenced affective wellbeing negatively
- More facebook usage influenced affective wellbeing positively
- Negative affective wellbeing influenced facebook usage negatively
- Negative affective wellbeing influenced facebook usage positively

Select all the true statements about the relationship between direct social interaction and wellbeing.

- Direct social interaction influenced affective wellbeing negatively
- Direct social interaction influenced affective wellbeing positively
- Direct social interaction influenced cognitive wellbeing negatively
- Direct social interaction influenced cognitive wellbeing positively

Which factor was a predictor of Facebook usage?

- negative affect
- worry
- loneliness
- none of the above

Which elements were mentioned as the caveats of the study? Select all that apply.

- small effect size
- short length of the study
- single-scale wellbeing measurement
- Low compliance rate

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Stress-related synaptic plasticity in the hypothalamus

Jaideep S. Bains¹, Jaclyn I. Wamsteeker Cusulin^{1,2} and Wataru Inoue^{1,3}

Abstract | Stress necessitates an immediate engagement of multiple neural and endocrine systems. However, exposure to a single stressor causes adaptive changes that modify responses to subsequent stressors. Recent studies examining synapses onto neuroendocrine cells in the paraventricular nucleus of the hypothalamus demonstrate that stressful experiences leave indelible marks that alter the ability of these synapses to undergo plasticity. These adaptations include a unique form of metaplasticity at glutamatergic synapses, bidirectional changes in endocannabinoid signalling and bidirectional changes in strength at GABAergic synapses that rely on distinct temporal windows following stress. This rich repertoire of plasticity is likely to represent an important building block for dynamic, experience-dependent modulation of neuroendocrine stress adaptation.

A stressor is a real or imagined threat that is interpreted by an organism as requiring immediate, adaptive responses. In animals, these include behavioural (fear and aggression) and visceral (autonomic and neuroendocrine) actions (FIG. 1). Stress has also been implicated in the emergence of numerous pathologies, including depression and anxiety disorders, and as a behavioural modifier that affects fundamental processes, such as decision making^{1–4}. In addition to these direct effects of stress on other systems, it is clear that exposure to one stressor often results in altered responses to subsequent stressors^{1–3,5–12}. This implies that the neural circuits that launch the response to stress are capable of learning and remembering. More precisely, these circuits can extract information about an individual stress event, store this information and then use it to modify subsequent responses. What are the mechanisms involved in storing this information? Where is this information stored? How does the brain make causal links between stressful experiences and embed these links within the neurons and synapses that drive stress responses?

In an attempt to address these questions, investigators have examined synapses and plasticity in several key brain structures that gate stress responsiveness, including the hippocampus, prefrontal cortex and amygdala^{13–16}. More recently, focus has turned towards understanding synaptic function and plasticity in the paraventricular nucleus of the hypothalamus (PVN). Neurons in the PVN, and more precisely those that synthesize corticotropin-releasing hormone (CRH),

integrate stress-relevant signals from multiple brain regions and launch the neuroendocrine response to stress^{17–20}. CRH-producing neurons are embedded within a population of parvocellular neuroendocrine cells (PNCs) in the PVN that release their contents into the hypophyseal portal circulation. CRH, along with other co-released secretagogues, stimulates the release of adrenocorticotropic hormone (ACTH) from the pituitary corticotropes, which in turn drives the release of corticosteroids (such as corticosterone (CORT) in rats and mice) from the adrenal cortex (FIG. 1). The activity of PNCs is controlled by synaptic inputs that convey both interoceptive and exteroceptive stressor-related information (FIG. 1). Consequently, these synapses are ideally positioned to not only integrate different stress signals but also extract information from one stressful event and use it to modify the responsiveness of the hypothalamic–pituitary–adrenal (HPA) axis.

This Review focuses largely on recent studies that have examined changes at glutamatergic and GABAergic synapses onto PNCs. These studies have revealed that several unique forms of short- and long-term plasticity occur at these synapses. To place these findings within the appropriate circuit framework, we first briefly describe the source of glutamatergic and GABAergic synaptic inputs to the PVN (FIG. 2) (for a more extensive review of the anatomical inputs to the PVN, see REF. 21) and then we describe studies that have provided the first set of rules that define the underlying principles of transmission at synapses on PNCs. Building on this

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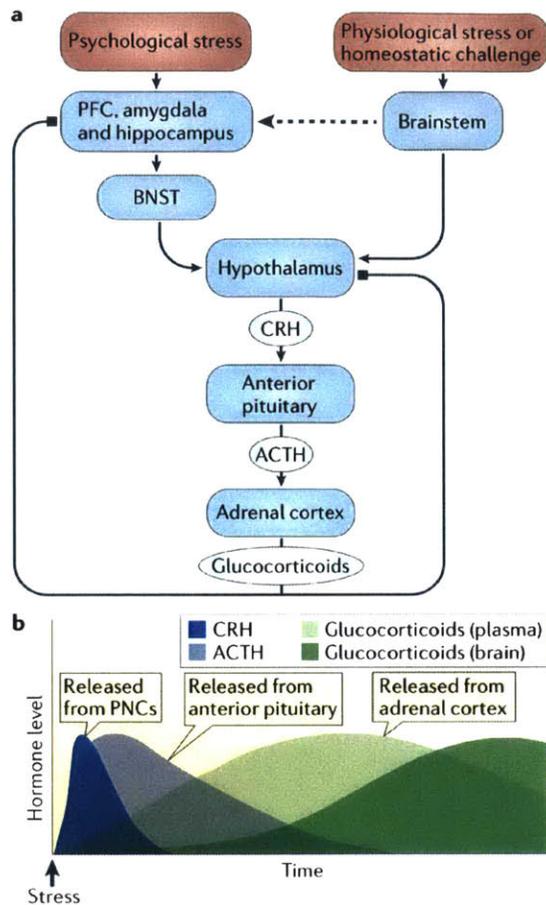


Figure 1 | Stress and functional connectivity. **a** | Psychological and physiological threats to survival are relayed via specific brain structures to the major stress-integrative brain centre, located in the paraventricular nucleus of the hypothalamus (PVN). Corticotropin-releasing hormone (CRH)-producing neurons in the PVN serve as primary coordinators of the endocrine stress response, which culminates with the release of corticosteroids ('glucocorticoids' in the figure) from the adrenal cortex. Corticosteroids have multiple effects on peripheral systems but also feed back to help quench the stress response through actions in the hippocampus (and other brain regions) and modify subsequent responses to stress through actions in the PVN. The square denotes that glucocorticoids are responsible for negative feedback and can promote more-complex adaptive changes. The dashed arrow denotes that brainstem neurons signal via the amygdala in response to some homeostatic challenges. **b** | Cascade of events illustrating recruitment of the neuroendocrine response to stress. Parvocellular neuroendocrine cells (PNCs; putative CRH-synthesizing neurons) in the PVN are activated and release CRH from axon endings located at the median eminence, part of pituitary portal circulation. CRH stimulates release of adrenocorticotropic hormone (ACTH) from pituitary corticotrophs in the anterior pituitary, which in turn act on the adrenal cortex to increase glucocorticoid release into the general circulation. Glucocorticoids act peripherally and slowly cross the blood–brain barrier to elicit diverse CNS effects, including negative feedback on the hypothalamic–pituitary–adrenal (HPA) axis. BNST, bed nucleus of the stria terminalis; PFC, prefrontal cortex.

foundation, we examine recent findings that have demonstrated embedded forms of plasticity, or metaplasticity, that are only evident after an animal has been subjected to stress. Finally, we discuss findings relating to a type of metaplasticity following stress that incorporates a time domain, which we call kairoplasticity.

Synapses of the PVN

CRH-synthesizing PNCs are innervated by glutamatergic and GABAergic fibres that, along with monoaminergic and peptidergic inputs, are crucial for regulating HPA-axis output²¹. Although reports supported the idea of glutamate and GABA regulation of the output of the PVN^{22,23}, it was the seminal findings by van den Pol and colleagues that provided the first demonstration of fast synaptic transmission mediated by glutamate and GABA in the PVN^{24–27}. At the ultrastructural level, nearly all presynaptic terminals onto PVN neurons contain numerous small, clear, round vesicles that closely appose synaptic specializations (which are indicative of glutamate or GABA packaging)^{24,26–28}. These small vesicles are often accompanied by dense-core vesicles (which usually contain peptides and amines)^{25,26,29}. Based on numerous electrophysiological studies, it is now accepted that virtually all PVN neurons receive fast glutamatergic and GABAergic synaptic inputs^{30–35}. Moreover, recent optogenetic studies have shown that genetically identified peptidergic cell populations rely

on fast glutamate or GABA signals^{36,37} to communicate with PVN neurons. These findings also support the idea that peptidergic synapses can use either glutamate or GABA as a co-transmitter³⁶. The potential interactions between slow-acting neuromodulatory systems and fast ionotropic signals are discussed in detail below.

As in other brain regions, both asymmetrical (glutamatergic) and symmetrical (GABAergic) synapses are present in the PVN^{25,29,38,39}. Quantitative immunoelectron microscopy studies indicate that approximately half of all synapses onto CRH-synthesizing neurons are GABAergic and that the others are non-GABAergic, asymmetrical and presumably glutamatergic²⁹. The proportion of GABAergic synapses in the PVN is substantially higher than that in other brain regions, such as the CA1 in the hippocampus (3% GABA) and the visual cortex (6% GABA)⁴⁰. Consequently, the PVN may be ideally suited for studying mechanisms of plasticity at GABAergic synapses, which have proven difficult to investigate in other systems. Approximately 30% of all synapses onto PVN neurons are also adrenergic or noradrenergic. These synapses have an asymmetric morphology⁴¹ but it is unclear whether they, like noradrenergic synapses in other brain areas, also release glutamate^{42,43}.

Both glutamatergic and GABAergic synapses make axosomatic as well as axodendritic contacts onto PNCs^{25,27,29,44}. They are intermingled synaptic contacts

Metaplasticity

A change in synapses that alters their ability to express plasticity.

Kairoplasticity

Derived from the Greek 'kairos', meaning the opportune or correct moment, it refers to observations that different forms of metaplasticity following stress that can be induced only during specific and distinct temporal windows.

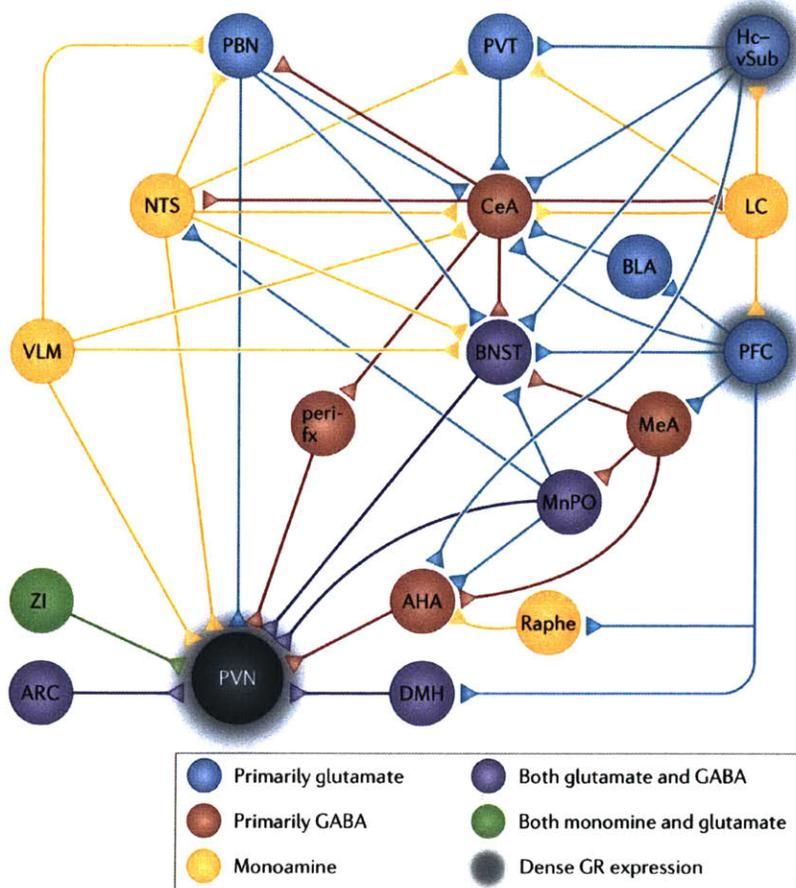


Figure 2 | Major direct and indirect CNS connections to the PVN. Compiled from multiple anatomical studies, this topological map shows key regions implicated in the brain response to stress, specifically the major glutamate, GABA and monoamine projections to the paraventricular nucleus of the hypothalamus (PVN). The halos represent areas of the brain in which high levels of glucocorticoid receptors (GRs) have been described. Particular attention is paid here to limbic regions of the brain implicated in regulation of hypothalamic–pituitary–adrenal (HPA)-axis responses to psychogenic stressors and glucocorticoid feedback. AHA, anterior hypothalamus; ARC, arcuate nucleus; BLA, basolateral amygdala; BNST, bed nucleus of the stria terminalis; CeA, central amygdala; DMH, dorsomedial hypothalamus; Hc, hippocampus; LC, locus coeruleus; MeA, medial amygdala; MnPO, medial preoptic nucleus; NTS, nucleus tractus solitarius; PBN, parabrachial nucleus; PVT, paraventricular thalamus; peri-fx, perifornical area; PFC, prefrontal cortex; Raphe, Raphe nuclei; VLM, ventrolateral medulla; vSub, ventral subiculum; ZI, zona incerta.

in close proximity to one another⁴⁴. Furthermore, PNCs (as well as other PVN neurons) have a relatively simple dendritic architecture, with a smooth shaft and low spine density⁴⁵. As a point of comparison, the spine density in neurons in other stress-responsive regions, such as the basolateral amygdala or the prefrontal cortex, is one order of magnitude higher than that observed in PNCs^{46,47}. This physical architecture, featuring a dearth of spines combined with the interspersed nature of glutamate and GABA contacts, may contribute to neurotransmitter spillover, heterosynaptic modulation and intracellular signalling crosstalk between glutamate and GABAergic synapses.

Both glutamate and GABA have key roles in regulating the activity of PNCs. Various glutamate receptor (AMPA receptor (AMPA), kainite receptor, NMDA

receptor (NMDAR) and metabotropic glutamate receptor (mGluR)) and GABA receptor (GABA_A receptor (GABA_AR) and GABA_BR) subunits have been detected in the PVN^{48–52}. Moreover, microinjection of glutamate into the PVN stimulates HPA-axis output, whereas administration of an ionotropic glutamate receptor antagonist before exposure to stress blunts the neuroendocrine stress response^{53–55}. By contrast, GABAergic synaptic transmission restrains baseline HPA-axis activity. For example, microinjections of GABA_AR antagonists in the absence of a stressor robustly activate PVN neurons and the HPA axis⁵⁴, suggesting that the release from the tonic inhibitory GABA tone (a phenomenon known as disinhibition) is an important mechanism for PVN activation. Such *in vivo* experiments in rodents are largely consistent with *ex vivo* electrophysiological recordings showing that GABA_AR blockade increases PNC firing⁵⁶. However, the activation of PNCs is probably regulated by the balance between opposing drives from glutamate and GABA inputs, as disinhibition (that is, GABA_AR blockade)-induced activation of these cells is blunted in the presence of a glutamate receptor antagonist⁵⁷.

Anatomy

Information from the internal and external environment is processed in distinct brain regions and eventually reaches CRH-producing neurons in the PVN to initiate HPA-axis activation. There are several excellent and detailed reviews on the neuroanatomical architecture of PVN afferents^{21,58} and so we will only briefly describe this architecture here (see also FIG. 2).

The direct, monosynaptic inputs to the PVN primarily arise from deep brain structures, including hypothalamic, extrahypothalamic and brainstem nuclei that are classically implicated in instinctive behaviour and physiological functions. By contrast, limbic forebrain structures, such as the hippocampus, amygdala and prefrontal cortex, which are critical for relaying information about psychological stressors, send few direct projections to the PVN. Instead, they direct monosynaptic projections to many of the aforementioned deep brain structures, which in turn relay information to the PVN.

The hierarchical organization of the stress neurocircuitry led Herman and colleagues to propose that stressor-related information is integrated in deep brain structures projecting to the PVN^{21,58}. These include non-overlapping populations of glutamatergic and GABAergic neurons⁵³ in the arcuate nucleus, the dorsomedial hypothalamus and the suprachiasmatic nucleus²¹. The inputs to the PVN from the median preoptic nucleus and the posterior bed nucleus of the stria terminalis (BNST) are primarily GABAergic. In addition, there is substantial GABAergic innervation from a ‘cloud’ or ‘halo’ of glutamate decarboxylase-expressing neurons that surround the PVN^{30,39}; this potentially originates from neurons in the anterior hypothalamus (AHA) and the perifornical region of the lateral hypothalamus. Given the extensive innervation of these hypothalamic territories by all major limbic structures, understanding the functional circuit formed by these inputs will be an important task for future investigation.

Spillover

When a neurotransmitter from one synapse acts at a neighbouring synapse; for example, when glutamate escapes the synaptic cleft and acts on nearby synapses.

Heterosynaptic modulation

When one transmitter system (for example, glutamate) affects a neighbouring but different system (for example, GABA).

A recent study using optogenetic methods revealed a functional septum–AHA–PVN circuit that regulates neuroendocrine output following a single stressor⁶⁰. Similar approaches will be critical for revealing the necessity and sufficiency of different cell populations for mediating responses to distinct stressors.

The BNST is a major extrahypothalamic source of GABAergic inputs. Activity mapping (that is, examining FOS expression) and inactivation and lesion studies have demonstrated an important role for BNST substructures in the regulation of the HPA axis^{61,62}, but a detailed dissection of the BNST–PNC circuit architecture is still needed to better understand the specific contributions of defined inputs during various stressful situations. The brainstem, especially the A2 and C2 cell groups of the nucleus tractus solitarius and the A1 and C1 cell groups of the ventrolateral medulla, provides the main monoaminergic inputs to PVN. The expression of vesicular glutamate transporter 2 (VGLUT2; also known as SLC17A6) in subpopulations of noradrenergic neurons in the brainstem is consistent with a glutamate phenotype⁶³, and recent work indicates that a subpopulation of glucagon-like peptide 1 neurons in the caudal nucleus tractus solitarius sends glutamatergic fibres to the PVN⁶⁴.

Glutamatergic synapses

Glutamatergic synapses under basal conditions. PNCs express a range of ionotropic glutamate receptor subunits that can form AMPARs, kainate receptors and NMDARs⁴⁹. Electrophysiological recordings from PNCs in acute brain slices *ex vivo* show both spontaneous and evoked synaptic currents that are sensitive to NMDAR and non-NMDAR antagonists^{31,34,35}. During repeated synaptic recruitment at rates greater than 2 Hz, the release of glutamate from the presynaptic terminals onto PNCs shows frequency-dependent short-term depression. This means that transmission is more faithful (that is, it shows a higher fidelity) at lower rates of synaptic activity³⁴. One potential consequence of this is that lowering the release probability at these synapses could allow for more faithful transmission at higher frequencies, thereby altering the filtering capacity of these synapses.

In addition to synaptic glutamate currents, extrasynaptic NMDAR-mediated signalling has been described in PNCs⁶⁵. Histological studies have shown that the parvocellular subdivision of the PVN expresses high levels of mRNAs for the kainate-preferring receptor subunits glutamate receptor ionotropic, kainate 1 (GLUK1; also known as GRIK1 and GLUR5) and GLUK5 (also known as GRIK5 and KA2)⁴⁹, which are upregulated following stress⁶⁶. Although the synaptic transmission to PNCs that is mediated by these kainate receptors remains to be characterized, GLUK1 expression is highly enriched in CRH-releasing axon terminals at the median eminence, and has been implicated in the release of the hormone during stress⁶⁷. Histological studies have also reported low to moderate expression of group I mGluRs (mGluR1 and mGluR5) in PNCs^{68,69}. Although the contributions of mGluR1 and mGluR5 to PNC excitability are largely unknown, recent studies demonstrate that the activation

of these receptors during stress initiates intracellular signalling necessary for the induction of plasticity at GABAergic synapses^{70,71} (see below). Glutamatergic synapses are a key target for several modulators that regulate HPA-axis output. For example, noradrenaline, which potently activates the HPA axis during stress^{72,73}, acts on α 1-adrenergic receptors to robustly increase the frequency of spontaneous glutamatergic synaptic currents in PNCs⁷⁴. Moreover, *in vivo* demonstrations that increases in circulating ACTH and CORT — induced by electrical stimulation of noradrenergic afferents to the PVN or by intra-PVN administration of an α 1-adrenergic receptor agonist — are blocked by intra-PVN microinjections of glutamate receptor antagonists (both NMDAR and non-NMDAR antagonists) provide additional evidence for a pivotal role of glutamate in noradrenalin-induced HPA-axis activation⁷⁵.

Endocannabinoids (eCBs) have been implicated repeatedly in the negative regulation of HPA-axis responses^{76–78}. At the level of the synapse, eCBs are released from postsynaptic cells and act on CB1-type receptors that are located in presynaptic terminals. eCBs thus signal retrogradely to inhibit the release of neurotransmitters, including glutamate and GABA. PNCs produce eCBs in an activity-dependent fashion⁷⁹. The release of these molecules at glutamatergic synapses produces depolarization-induced suppression of excitation (DSE)⁷⁹ that exhibits characteristics that are consistent with well-characterized DSE at other synapses (specifically, the DSE is CB1-mediated and is dependent on Ca^{2+} influx into the postsynaptic cell through L-type calcium channels) (FIG. 3). Although the contribution of this eCB-mediated, activity-dependent modulation of glutamate transmission to HPA-axis activity remains to be determined, one possibility is that it curtails excitatory synaptic input during periods of high PNC activity. In hypothalamic slices, application of CORT or its synthetic analogue, dexamethasone^{79–81}, stimulates the rapid synthesis of eCBs and inhibits glutamate release from presynaptic terminals. This requires rapid actions through a non-canonical, putative membrane receptor that signals through a G protein and adenylate cyclase activation pathway^{5–12,80}.

Glutamatergic synapses following acute stress. A single acute stress alters signalling at glutamatergic synapses on PNCs. This manifests as an increase in the ratio of AMPAR- to NMDAR-mediated transmission³⁵. Surprisingly, this is not due to an increase in signalling via AMPARs, but rather to a long-lasting decrease in NMDAR signalling³⁵ that results from the downregulation of postsynaptic NMDARs by local release of CRH during stress. It is unclear whether CRH is released in an autocrine fashion or originates from extra-PVN sources^{82,83}. This CRH-mediated NMDAR downregulation is an important priming step that allows these synapses to exhibit a subsequent form of activity-dependent plasticity, or metaplasticity³⁵. This metaplasticity is induced by bursts of high-frequency presynaptic stimulation and manifests as a robust short-term potentiation (STP) of excitatory transmission³⁵ (FIG. 3). This dormant

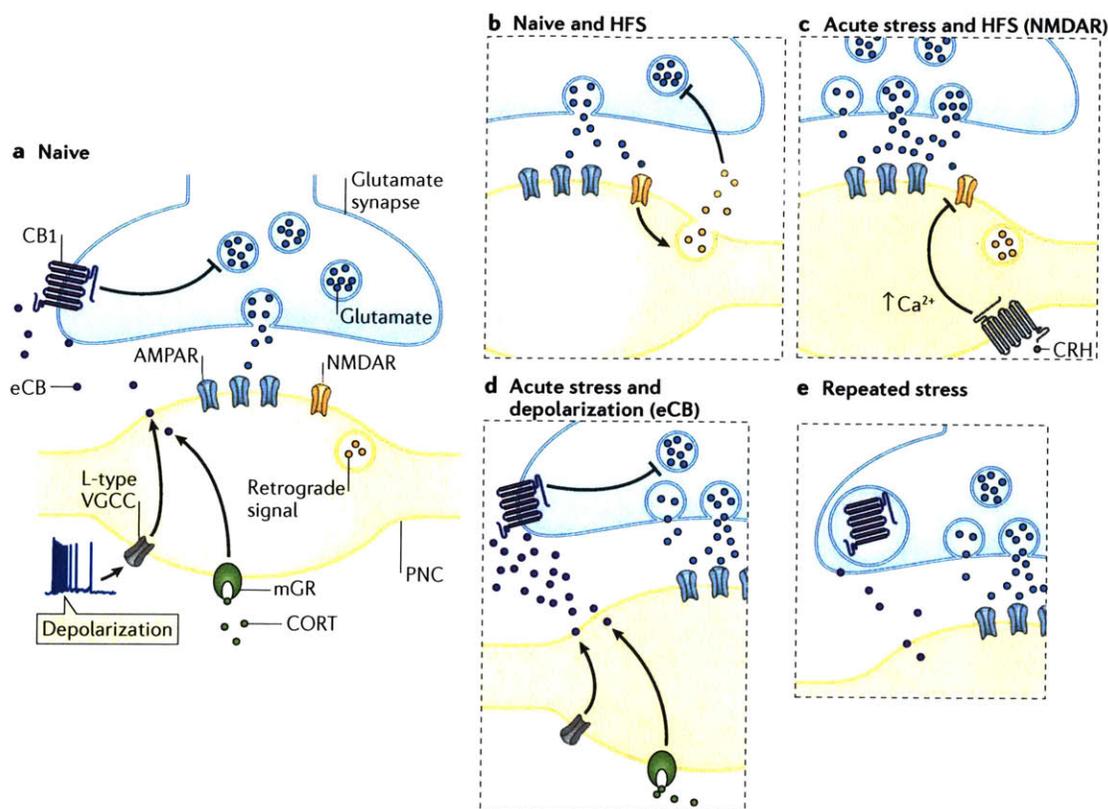


Figure 3 | Glutamatergic synapses — function and plasticity. **a** | Diagram depicts a glutamatergic synapse onto a parvocellular neuroendocrine cell (PNC) from a naive (unstressed) animal. Fast transmission is mediated by AMPA receptors (AMPA) and NMDA receptors (NMDARs)^{34,35}. Synapses also exhibit endocannabinoid (eCB)-mediated depression. eCBs, recruited either by the postsynaptic depolarization and subsequent Ca²⁺ influx through L-type voltage-gated Ca²⁺ channels (VGCCs)⁷⁹ or by the rapid actions of glucocorticoids (such as corticosterone (CORT))⁸¹, act at presynaptic CB1 to decrease glutamate release. **b** | During bursts of afferent activity, NMDAR activation liberates a retrograde signal that curtails release from glutamate terminals. **c** | Acute stress causes activation of postsynaptic corticotropin-releasing hormone (CRH) receptors, resulting in a decrease in NMDAR signalling. This eliminates the retrograde signal. As a result, glutamatergic synapses, when recruited with high activity rates, can release multiple vesicles of transmitter. This results in a short-term potentiation that persists for a few minutes after the synaptic burst. **d** | Acute stress also amplifies retrograde eCB signalling at these synapses. **e** | Repeated homotypic stress causes functional downregulation of CB1. This manifests as a progressive loss of depolarization-induced suppression of excitation (DSE). CORT is also ineffective in recruiting eCB signalling under these conditions. HFS, high-frequency stimulation; mGR, membrane glucocorticoid receptor.

STP is not due to an increase in postsynaptic glutamate signalling or an increase in release probability; instead, it is due to a switch in the mode of release at glutamatergic synapses from univesicular (that is, a presynaptic action potential releases, at most, a single vesicle of glutamate) to multivesicular (that is, a presynaptic action potential can release more than one vesicle). Importantly, STP can also be unmasked in naive slices (slices from animals not exposed to stress)³⁵ by postsynaptic blockade of NMDAR, Ca²⁺ signalling and SNARE (soluble *N*-ethylmaleimide-sensitive factor attachment protein receptor)-dependent exocytosis (by including pharmacological inhibitors in the recording pipette). These observations suggest that CRH-mediated decreases in NMDAR-dependent Ca²⁺ entry during stress prevent the release of a retrograde messenger that normally blocks the multivesicular release of glutamate following high levels of presynaptic activity (FIG. 3). There are various dendritically released factors that could be responsible for suppressing glutamate

release⁸⁴, but experiments have ruled out eCBs acting at CB1, opioids or adenosine³⁵. Future studies are needed to determine the identity of the retrograde messenger.

More information is also needed to better understand how this stress-induced metaplasticity at glutamatergic synapses may contribute to the adaptation of HPA-axis responses to repeated stress. Pre-injection of the NMDAR antagonists MK-801 (also known as dizocilpine) or memantine increase *Fos* and *Crh* mRNA expression in the PVN and increase ACTH and CORT levels in the blood following two homotypic stressors^{5,85–87}. This is consistent with the hypothesis that NMDAR downregulation plays a key part in amplifying responses to subsequent stressors. Interestingly, glutamatergic synapses in brain slices continue to exhibit a capacity for multivesicular release for more than 72 hours after a single acute restraint stress³⁵. This time course is consistent with that of stress-induced HPA-axis sensitization, as reported elsewhere⁸⁸.

In addition to CRH, several other stress mediators act both independently and in concert to drive complex forms of metaplasticity at PNC synapses. For example, repeated exposure to the same stressor gradually diminishes CB1 signalling and this results in the loss of eCB-mediated inhibition of neurotransmitter release, including activity-dependent DSE (and depolarization-induced suppression of inhibition)⁷⁹ (FIG. 3). This functional downregulation of CB1 requires the activation of cytosolic glucocorticoid receptors. As discussed below, the actions of noradrenaline and CORT during stress functionally upregulate mGluR1 and mGluR5, respectively, at PNC synapses. This stress-induced modulation of mGluRs primes PNC synapses for subsequent activity-dependent plasticity of GABAergic synapses^{70,71}, thus in a way resembling CRH-mediated metaplasticity at glutamatergic synapses. Clearly, a state-dependent change in synaptic plasticity is an emerging idea that needs further investigation.

Long-term changes in glutamate transmission after stress. Exposure to chronic stress, or even to a single stressor, can cause long-term changes in HPA-axis activity and responsiveness, resulting in, for example, baseline hyperactivity and hypersensitivity to a novel stressor^{88–90}. Like other types of experience-dependent learning, such changes in the sensitivity and/or gain of HPA-axis responses probably involve activity-dependent, long-term synaptic changes. Neuroanatomical and electrophysiological evidence collectively suggests that experience (stress)-dependent plasticity occurs at PNC glutamatergic synapses. Only a few examples of this type of plasticity have been described to date, and most of these involve changes that are consistent with an increase in the number of glutamatergic synapses. For example, repeated variable stress in male rats for 1 week increases the number of boutons that are immunopositive for VGLUT2 on identified CRH neurons⁹¹. Repeated variable stress over 3 weeks causes approximately a twofold increase in the number of synaptic contacts terminating on CRH-expressing PNCs⁴⁴; this study reported increases in both GABA and non-GABA (that is, putative glutamatergic) terminals. Functionally, repeated homotypic stress (restraint) for 3 days in rats increases the frequency of spontaneous excitatory postsynaptic currents (sEPSCs)⁹². Likewise, early life stress (due to poor maternal care) increases the frequency of sEPSCs and miniature EPSCs (mEPSCs), which is paralleled by an increase in VGLUT2 expression in the PVN in animals examined after weaning⁶⁵. These findings collectively showing an increase in excitatory glutamate inputs to PNCs begin to provide a foundation for earlier studies indicating that these stress exposures cause a lasting sensitization and/or baseline hyperactivity of the HPA axis.

In addition to inducing the changes in phasic (synaptic) transmission described above, early-life stress results in impairment of astrocytic glutamate transporter 1 (also known as excitatory amino acid transporter 2) function, which may contribute to the unmasking of a tonic (extrasynaptic) conductance that is primarily mediated

by NMDARs⁶⁵. These findings highlight a potentially important, but under-investigated, role for glial cells in stress-induced glutamatergic synapse plasticity in the PVN. The effects of chronic stress on PNC glutamatergic synapses raise the question of whether reducing stress levels may have the opposite effect. This was assessed in a study⁹³ in which rats were exposed to an enriched early life environment (augmented maternal care), which is known to persistently attenuate neuroendocrine stress responses^{94–96}. Under these conditions, there is a decrease in mEPSC frequency in PNCs that is accompanied by a decrease in the number of glutamatergic synapses and reduced VGLUT2 expression⁹³. This reduction in the number of glutamatergic synapses is transient; that is, it is evident in neonates immediately after early life experiences, but resolves by adolescence, even though other changes relevant to HPA-axis responses (for example, transcriptional repression of *Crh*) persist until adulthood. The authors proposed that synaptic changes in early life initiate lifelong epigenetic changes that affect subsequent transcription events in PNCs.

GABAergic synapses

GABAergic synapses under basal conditions. GABA transmission mediated by anion-permeable, pentameric GABA_A receptors (GABA_ARs) consists of distinct phasic and tonic currents. PVN neurons express $\alpha 1-2$, $\beta 1-3$, and $\gamma 1-2$ GABA_AR subunits and exhibit phasic GABA_AR-gated synaptic currents⁹⁷. In addition, tonic (extrasynaptic) inhibition mediated by δ -subunits has also been demonstrated⁹⁸. The specific localization and function of other GABA_AR subunits remains unexplored. GABA release at synaptic contacts can occur spontaneously — that is, in the absence of action potentials (observed as miniature inhibitory PSCs) — and following electrical stimulation to drive action potential-dependent release events (for example, paired-pulse depression, which is observed at over 80% of synapses)^{79,99}. In cell-attached recordings in which anion gradients are unperturbed, GABA_AR blockade increases the activity of PNC⁵⁶. These data are largely consistent with *in vivo* studies showing that ongoing GABA transmission in the absence of a stressor constrains PNC activity and, thereby, HPA-axis output⁵⁴.

GABAergic synapses following acute stress. Given the evidence for tonic constraint of HPA-axis activity by GABA, it is worth considering possible mechanisms through which disinhibition may occur at the onset of a stressor. One possibility is that GABA neurons that directly synapse onto PNCs may become less active, although currently there is no direct evidence in support of this idea. This may be due to a lack of definitive *in vivo* recording data and the limitations of immediate early gene mapping. For example, FOS mapping studies are instructive, but fail to reflect short-lived increases in activity or decreases in activity, for example, in nuclei that provide GABA inputs to PVN⁵⁹. A second possible mechanism for disinhibition is that GABAergic synapses become less effective, either through a decrease in the probability of GABA release

Homotypic stress

Repeated administration of the same stressor to an animal.

or owing to a decrease in the number or function of postsynaptic GABA_ARs. Observations that acute stress decreases the probability of GABA release are consistent with this idea¹⁰⁰ (but also see REF. 71). An alternative explanation is that GABA_AR-mediated inhibition is reduced at the onset of acute stress through alterations in chloride homeostasis in the postsynaptic cell^{56,98}. This is based on electrophysiological recordings in slices prepared immediately after a single 30-minute restraint stress, which reported a depolarizing shift in chloride reversal potential in PNCs^{56,98}. This alteration in chloride homeostasis requires a decrease in the functional capacity of K-Cl co-transporter 2 (KCC2; also known as SLC12A5)⁵⁶, which is probably due to decreased membrane expression of the transporter⁹⁸. One consequence of this reduction in chloride extrusion capacity is that engagement of GABA afferents has a nonlinear effect on PNC firing¹⁰¹: at low levels of GABA input the decrease in KCC2 function results in a weakening of inhibition, whereas at higher rates of synaptic transmission and/or after enhancement of tonic GABA_AR currents there is a paradoxical GABA_AR-dependent excitation that increases action potential firing^{56,98}. Consistent with this model, genetic deletion of GABA_AR δ-subunits¹⁰² or pharmacological blockade of positive allosteric modulation of extrasynaptic GABA_AR δ-subunits blunts HPA-axis responses⁹⁸.

Kairoplasticity at GABAergic synapses. Recent experiments offer new insights into the rules and expression mechanisms for synaptic plasticity at GABAergic synapses on PNCs. These findings (discussed in detail below) describe a form of metaplasticity but are unique from classical metaplasticity in that the timing of the induction after the initial exposure to stress is critical. In naive (not exposed to stress) animals, GABAergic synapses on PNCs fail to exhibit long-term plasticity^{70,71}. However, after exposure to either a physical or a psychological stressor, both long-term potentiation (LTP)⁷⁰ and long-term depression (LTD)⁷¹ can be induced at GABAergic synapses (LTP_{GABA} and LTD_{GABA}, respectively). Importantly (and surprisingly), whether synapses exhibit LTP or LTD is not a consequence of the induction protocol used; rather, it is a function of when slices were prepared after stress. Specifically, LTP can be induced only in slices prepared immediately after a stress, whereas LTD can be induced only in slices prepared 90 minutes after stress. These temporal windows for opposing forms of plasticity arise because two specific neuromodulators, noradrenaline and CORT, exhibit discrete time domains for actions in the PVN¹³. More precisely, noradrenaline, which is released at the onset of stress, is the essential associative signal for LTP_{GABA}. That is, activation of β-adrenergic receptors and the downstream activation of protein kinase A cause a functional upregulation of (otherwise dormant) mGluR1 on PNCs. This noradrenaline-induced priming of mGluR1 creates a time window in which subsequent bursts of glutamate afferent activity effectively activates mGluR1. An mGluR1-dependent increase in intracellular Ca²⁺ and the ensuing phosphorylation of calcium/calmodulin-dependent protein

kinase II drive the insertion of GABA_A receptors into the PNC (that is, the postsynaptic) membrane⁷⁰ (FIG. 4). By contrast, LTD_{GABA} requires CORT acting on cytosolic glucocorticoid receptors in PNCs⁷¹. The activation of glucocorticoid receptors inhibits an intracellular regulator of G protein signalling (regulator of G protein signalling 4), which amplifies mGluR5 signalling and culminates in the liberation of the opioid peptide enkephalin from somatodendritic vesicles⁷¹ (FIG. 4). Enkephalin acts as a retrograde signal and inhibits the release of GABA from presynaptic terminals.

Changes in GABA transmission following chronic stress.

Chronic or repeated stress can also cause several alterations in PNC GABAergic synapses. These may include changes in the expression of GABA_AR subunits^{103,104} and a reduction in retrograde eCB signalling⁷⁹. Perhaps the most striking observations are those of putative GABAergic synaptogenesis and subcellular synapse redistribution. Under non-stress conditions, GABAergic synapses are uniformly distributed between somatic and dendritic compartments of PNCs⁴⁴. Chronic stress causes two distinct changes. First, there is a dramatic increase in the total number of GABAergic synaptic contacts; second, there is a change in relative distribution of these contacts, such that dendritic inputs become more highly represented⁴⁴. The functional consequences of such structural plasticity warrant further investigation but, interestingly, chronic stress results in a paradoxical decrease in the number of functional GABAergic synapses in electrophysiological recordings¹⁰⁴. One possible explanation for this is that chronic stress may lead to the formation of (functionally) silent synapses that are subjected to dynamic recruitment by subsequent synaptic activities in a mechanism that involves an LTP_{GABA}-like mechanism⁷⁰. Furthermore, the effect of this synaptic rearrangement on HPA-axis function remains unknown. In many neuronal circuits, the location of GABAergic synaptic input onto a principal or projection neuron reflects innervation by functionally distinct GABA subpopulations, which modify neuronal firing and/or integration of excitatory input in a very specific fashion. A rearrangement of GABA inputs onto PNCs may alter the relative contributions of specific nuclei. Such compartmentalization of GABA input also determines the efficacy of inhibition through activity-dependent shifts in the chloride driving force. A collapse of inhibition under high rates of synaptic activity occurs preferentially at dendritic GABA inputs, where chloride extrusion mechanisms and diffusional capacity are volume-limited¹⁰⁵. Further investigation will be necessary to address how changes at specific GABAergic synapses contribute to chronic stress-induced HPA-axis adaptations and the behavioural and physiological pathologies that accompany them.

Functional implications

It is increasingly clear that synapses onto PNCs are dynamic, can be modified by experience and need to be an important part of any discussion that invokes plasticity of responses to stress. The next challenge is to understand how these mechanistically discrete forms of

K-Cl co-transporter 2 (KCC2). A transmembrane potassium–chloride co-transporter that extrudes chloride and maintains the driving force for chloride influx into cells upon the opening of GABA_A receptors.

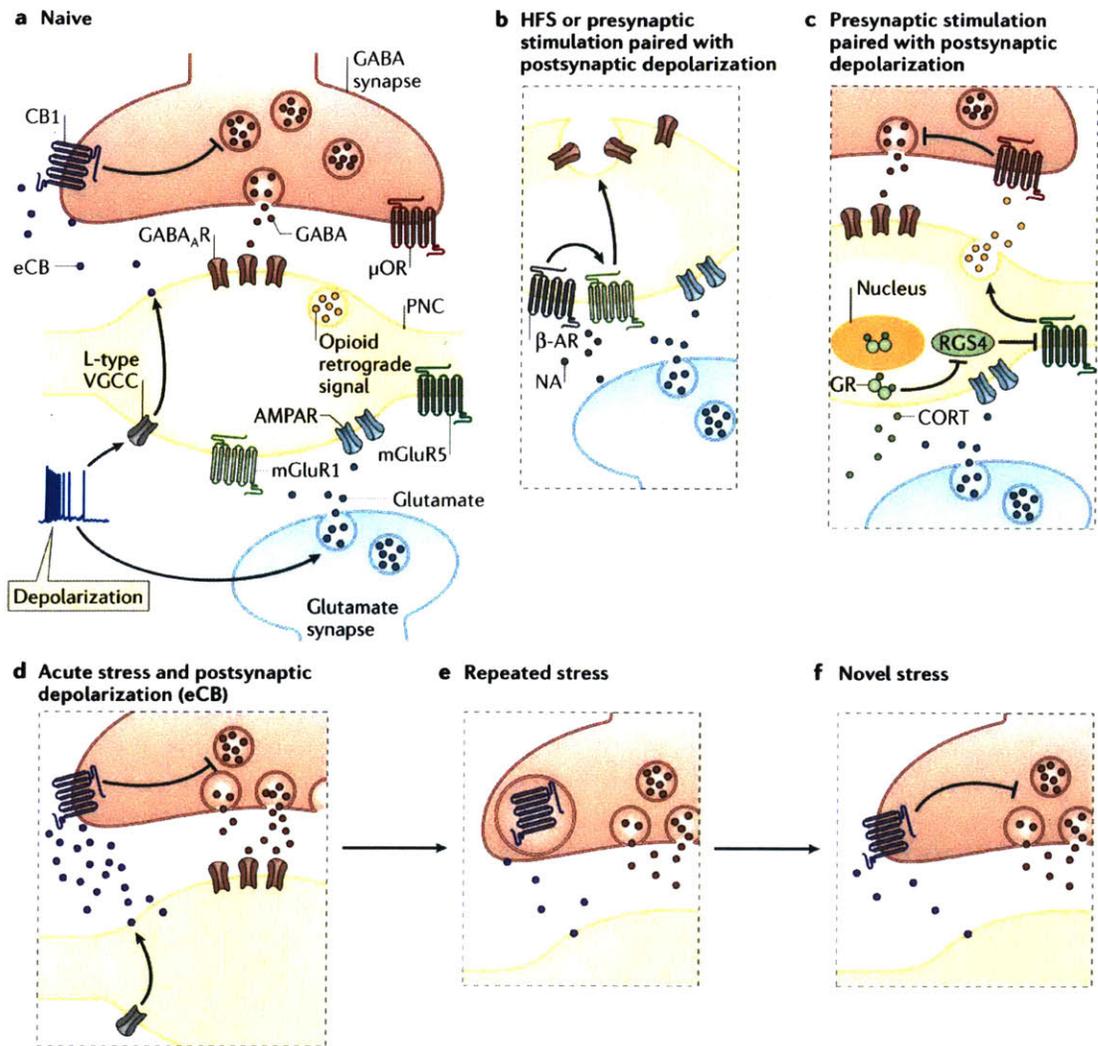


Figure 4 | GABAergic synapses — function and plasticity. **a** | Diagram depicts a GABAergic synapse on a parvocellular neuroendocrine cell (PNC). Fast transmission is mediated by GABA_A receptors (GABA_ARs). Synapses exhibit endocannabinoid (eCB)-mediated depression. eCBs, recruited by postsynaptic depolarization and subsequent Ca²⁺ influx through L-type voltage-gated Ca²⁺ channels (VGCCs)⁷⁹, act at presynaptic CB1 to decrease GABA release. **b** | An acute stress causes local release of noradrenaline (NA) and activation of β-adrenoceptors (β-ARs). This primes group I metabotropic glutamate receptors (mGluRs) in PNCs through a mechanism that is unresolved⁷⁰. Once primed, mGluR activation increases intracellular Ca²⁺, which drives phosphorylation of calcium/calmodulin-dependent protein kinase II (CAMKII) and the insertion of GABA_ARs at previously silent GABAergic synapses (not shown). **c** | If a longer time (90 minutes) is allowed for the animal to recover after stress, a different form of plasticity is observed. The longer recovery period allows circulating corticosterone (CORT) to affect biochemical processes in PNCs. Specifically, CORT amplifies signalling through mGluR5, by inhibiting the regulator of G protein signalling 4 (RGS4) in PNCs⁷¹. This removes the ‘brake’ from mGluR5 downstream signalling and allows synaptically released glutamate to liberate an opioid retrograde signal. The opioid, which is probably enkephalin, binds to presynaptic μ-opioid receptors (μOR) and decreases GABA release probability. **d** | Acute stress also amplifies retrograde eCB signalling at GABAergic synapses. **e** | Repeated homotypic stress causes a downregulation of CB1 and consequent loss of eCB signalling. **f** | Exposure to a novel stressor after repeated homotypic stress causes an immediate reinstatement of eCB signalling. This is due to the recovery of CB1Rs at presynaptic terminals. The effects of novel stressors can be mimicked either by globally increasing neural activity using electroconvulsive seizures or by increasing synaptic activity *in situ*. AMPAR, AMPA receptor; GR, glucocorticoid receptor; HFS, high-frequency stimulation.

plasticity contribute to dynamic, experience-dependent modulation of CRH neuron excitability and the resultant HPA-axis activity. At GABAergic synapses, synaptic changes must be considered within a theoretical framework that takes into account the shifts in chloride homeostasis that accompany both acute^{56,98} and

prolonged stress⁶⁵. This has recently been reviewed in detail elsewhere¹⁰¹. In brief, LTP_{GABA}, which increases the efficacy of chloride flow across the membrane, enhances the robustness of the GABA response. That is, in a simplistic scenario, LTP_{GABA} causes more-effective inhibition when the chloride reversal potential is below

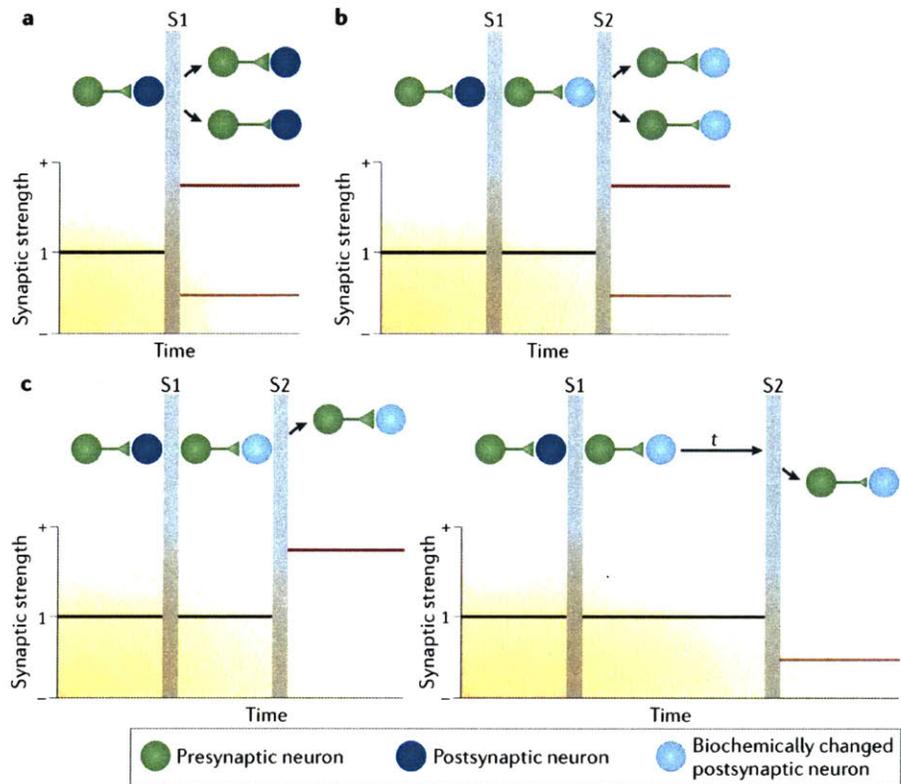
Box 1 | Plasticity, metaplasticity and kairoplasticity

In the classic interpretation of synaptic plasticity (see the figure, part **a**), presentation of a stimulus (S1) that engages two synaptically coupled neurons (shown in green and blue) modifies the efficacy of the synapse between them. Depending on the stimulus, the original synaptic response (shown by the medium size of the synapse) may be potentiated (shown as a larger synapse; synaptic strength >1) or depressed (shown as a smaller synapse; synaptic strength <1).

In synaptic metaplasticity between synaptically coupled neurons (see the figure, part **b**), presentation of S1 causes embedded biochemical changes (shown as a change in the colour of the postsynaptic cell) but does not change synaptic strength. Presentation of a subsequent stimulus (S2) may either potentiate (synaptic strength >1) or depress (synaptic strength <1) the synapse. The direction of the plasticity depends on the nature of S2.

In kairoplasticity (see the figure, part **c**), the presentation of S1 causes biochemical changes but, as in metaplasticity, does not change synaptic strength. Again, presentation of S2 may either potentiate (synaptic strength >1) or depress (synaptic strength <1) the synapse. However, the direction of the plasticity depends on the temporal window after S1 rather than on the nature of S2. For example, if S2 is presented within a narrow temporal window of S1, the synapse is potentiated (see the figure, part **c**, left panel). If the temporal window is longer (as represented by the variable *t*), S2 will cause a synaptic depression despite being the same stimulus (see the figure, part **c**, right panel).

Specific neuromodulators may be important for determining the temporal windows in kairoplasticity.



the resting membrane potential (that is, in the naive, unstressed state) and more excitation when the chloride reversal potential is above the resting membrane potential (that is, after stress). However, it is important to consider that chloride homeostasis is dynamic and is directly influenced by the chloride flux through GABA_ARs. Thus, during a period of postsynaptic depolarization (that is, during persistent spiking activity) that generates a large driving force for chloride influx, LTP_{GABA} augments the rate of chloride influx (chloride loading)^{56,106}. When combined with the impaired chloride extrusion mechanism (for example, through downregulation of KCC2) during stress, LTP_{GABA} favours the development of subsequent depolarizing GABA response and can, paradoxically, contribute to the stress-induced sensitization of PNCs. By contrast, LTD_{GABA} can have the opposite effect. In this model, the role of LTP_{GABA} and LTD_{GABA} in shaping HPA-axis responses fits with existing evidence from *in vivo* studies suggesting that noradrenaline contributes to sensitization of HPA-axis output^{107,108} and that CORT provides negative feedback to prevent overactivation of the HPA axis¹⁰⁹. The discovery of LTP_{GABA} and LTD_{GABA} in PNCs^{70,71} provides an important foundation to test this model and to tease apart the mechanistic underpinnings of neuroendocrine adaptations during repeated, and subsequently chronic, stress experiences.

Conclusions and future perspectives

The descriptions of multiple forms of plasticity at synapses onto PNCs indicate that there are multiple mechanisms by which the brain adapts to stressors. Importantly, they provide a framework in which to ask more precise questions about how organisms use information from one exposure to stress to make adjustments to ensuing stressful experiences. Here, we have described multiple embedded biochemical changes following an acute stress that can bias synapses towards plasticity when these synapses are subsequently recruited in an activity-dependent fashion. This is consistent with the definition of metaplasticity¹¹⁰. We have also noted that the nature of the plasticity is dependent on how soon after stress the synapses are interrogated. In other words, there seem to be opportune moments for the induction of distinct forms of plasticity following stress. Temporal windows for bidirectional plasticity following stress have also been described in the amygdala¹¹¹, suggesting that it may be important to incorporate time as a key variable when discussing plasticity or metaplasticity. Consequently, we propose a new descriptor for the lexicon of plasticity, kairoplasticity, which incorporates distinct temporal windows for bidirectional changes in synaptic strength (BOX 1).

Derived from the Greek word '*kairos*', meaning the opportune or correct moment, kairoplasticity refers to observations that specific and distinct temporal

windows after stress are instructive for different forms of metaplasticity. One plausible consequence of such a mechanism is that it contributes to the versatility of the system, enabling context-specific adjustment of the stress response. These observations also raise new questions: for example, how are these distinct modes of plasticity encoded by one stressor affected by subsequent experiences? Glutamatergic synapses onto PNCs, for example, are primed for multivesicular release for more than 3 days after a single stress challenge³⁵; how subsequent stressors are integrated during this time window remains unclear. Moreover, it is possible that mechanisms exist to reverse these synaptic changes. For example, living in an ‘enriched’ environment could be one way in which stress-embedded synaptic changes are erased. Recent work demonstrating that stress-induced loss of eCB signalling at PNC synapses can be restored following exposure to novel stimuli¹¹² is consistent with the idea that the salience of an experience may be a critical element in returning HPA-axis activity to baseline¹¹². Although recent studies have increased our understanding of synaptic function and plasticity in the PVN, there are still

many important and unresolved issues. The adoption of new tools that enable specific activation of identified cell populations will be extremely beneficial for gaining a better understanding of the interactions between co-transmitters in the PVN. In this vein, recent studies have described new mouse models that allow for both the identification and the genetic targeting of specific subsets of PNCs^{99,113,114}. One important limitation that must be addressed is the dearth of information about the activity of PNCs and CRH-producing neurons *in vivo*, particularly in response to stress exposure; developing and implementing approaches that allow for the use of electrophysiology and/or imaging techniques in awake, behaving animals will be a considerable advance in helping to make causal links between changes in synaptic function or plasticity and the output of stress circuitry. By filling these knowledge gaps, we will begin to have a better understanding of how specific inputs shape outputs from the PVN and we will also gain insights into how we might exploit the capacity of these inputs to be modified to help reset dysfunctional neural circuits in individuals with stress-related conditions, such as depression.

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Acknowledgements

Research by the authors is supported by the Canadian Institutes for Health Research.

Competing interests statement

The authors declare no competing interests.

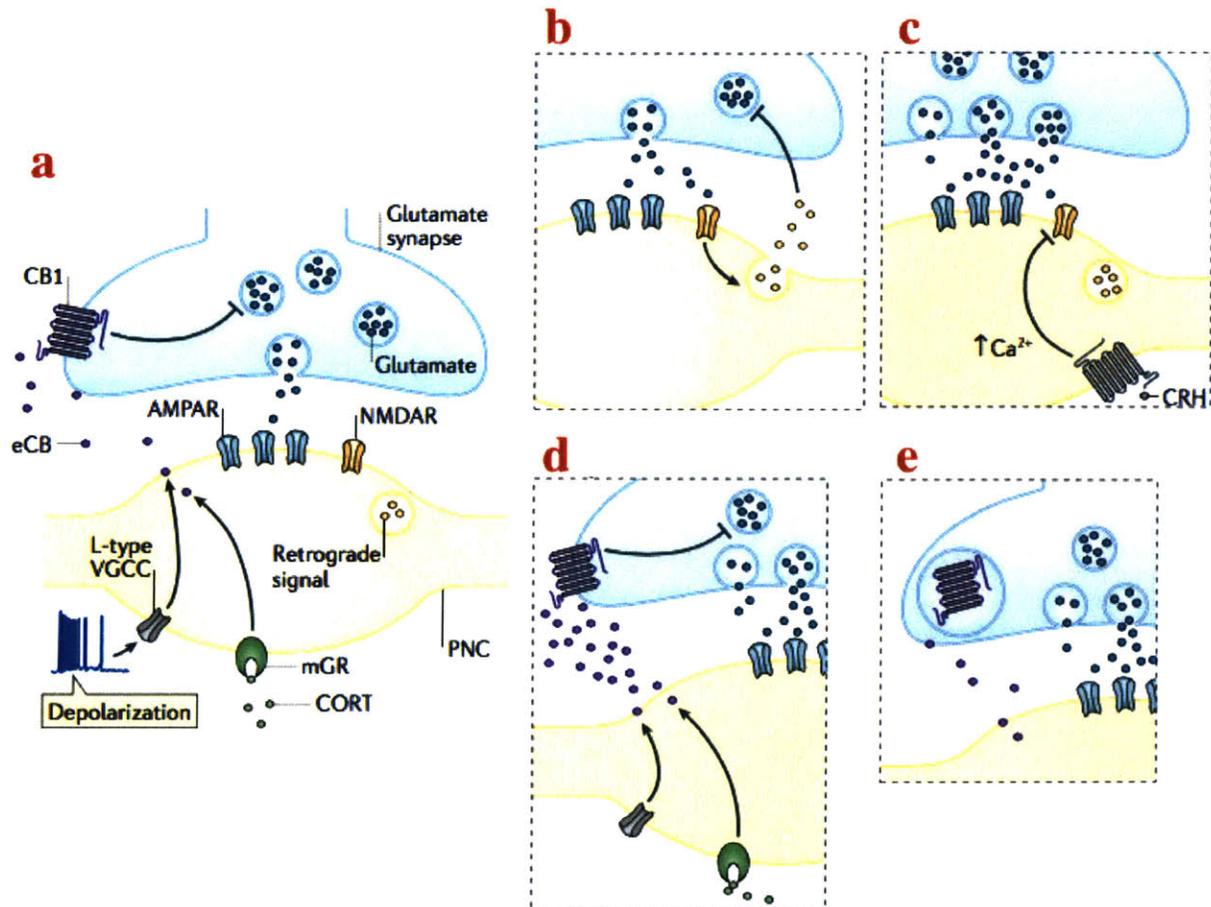
Experiment 2 - Quiz 3

BrightBeat: MIT Media Lab study on calming technologies

Question set III

Where is the major stress-integrative center of the brain located?

- hypothalamus
- hippocampus
- amygdala
- prefrontal cortex



Given the figure above about Glutamatergic synapses, match each row with its correct column.

	a	b	c	d	e
Repeated stress	<input type="radio"/>				

Acute stress and HFS (NMDAR)	<input type="radio"/>				
HFS	<input type="radio"/>				
Naive	<input type="radio"/>				
Acute stress and depolarization (eCB)	<input type="radio"/>				

S1: Presentation of a stimulus that engages two synaptically coupled neurons. S2: presentation of a subsequent stimulus.

Match each row with its correct column(s).

	plasticity	metaplasticity	kairoplasticity	metaplasticity and kairoplasticity
S1 modifies the efficacy of the synapse	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
S1 doesn't modify the efficacy of the synapse	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
S1 causes embedded biochemical changes	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
the change in synaptic strength depends of the nature of S2	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
the change in synaptic strength depends of the timing of S2	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

« Back Continue »

Experiment 2 - Relaxing and reading task

A Simple Way to Combat Chronic Stress

- [Alexander Caillet](#)
- [Jeremy Hirshberg](#)
- [Stefano Petti](#)

For most leaders today, frequent stress is inevitable. But with awareness and a little skill, its negative impacts are not.

Intense negative experiences of stress are all too common. Consider Stefano, coauthor of this article. In 1998, Stefano began a career abroad while simultaneously completing an MBA. He worked and studied 14 hours a day, seven days a week, fueled by a constant flow of stress hormones. By the end of that year, he suffered from fatigue, headaches, impatience, and irritability, yet he ignored his symptoms and kept going. Soon those symptoms escalated into full-blown burnout: dizziness, heart palpitations, inability to concentrate, panic attacks, apathy, insomnia, and depression. He eventually decided that he needed to take a full six months to rebuild his mental and physical well-being before he could return to work.

Chronic stress impacts people in different ways. In a recent global survey we conducted of 740 leaders, 84% reported experiencing stress on a regular basis (for more, read [“How Your State of Mind Affects Your Performance”](#)). As you might expect, more than half of the 84% said stress had a negative impact on their effectiveness, interactions, or business results. However, the remaining leaders — around 45% — told a different story. In their experience, stress either had no impact on their leadership or had a positive effect. More than 25% said stress actually improved their effectiveness.

What accounts for these results? And what are those people doing that Stefano did not? The leaders we’ve worked with over several decades have given us a wide variety of answers. In this article, we focus on two: tipping point awareness and stress shifting.

Tipping point awareness

Like many other leadership capacities, stress management requires self-awareness. Each of us has our personal “tipping point,” the critical edge where moderate, tolerable stress transitions to chronic stress — and a constant flux of stress hormones drive us to the point of a breakdown. Leaders who manage stress well are able to recognize signs that they’re approaching that point and consciously, deliberately step back from the edge.

Signs of chronic stress fall into three main categories: physical, mental/emotional, and behavioral. The symptoms listed below are some of the most common complaints that leaders have told us they experience only, or to a greater degree, under chronic stress.

Symptoms Reported by Leaders Under Chronic Stress

PHYSICAL

- Aches and pains
- Lightheadedness/Dizziness
- Gastrointestinal issues
- Skin issues
- Headaches
- Heart palpitations
- High blood pressure
- Shortness of breath
- Trembling/Twitching
- Jaw clenching and grinding

MENTAL/EMOTIONAL

- Anxiety/Bouts of worry
- Apathy/Lethargy
- Confusion/Disorientation
- Low or depressed mood
- Fatigue
- Forgetfulness/Memory lapses
- Lack of focus
- Panic attacks
- Difficulty making decisions

BEHAVIORAL

- Deterioration of hygiene or appearance
- Increased alcohol consumption or smoking
- Disruptions in sleep patterns
- Restlessness and fidgeting
- Irritability and aggression
- Tearfulness
- Appetite changes
- Sleep pattern disruptions

NOTE: THESE SYMPTOMS HAVE MANY POSSIBLE CAUSES ASIDE FROM CHRONIC STRESS. OFTEN, CHRONIC STRESS EXACERBATES PREEXISTING TENDENCIES OR MEDICAL CONDITIONS.

SOURCE AMERICAN INSTITUTE OF STRESS; MAYO CLINIC

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While each of these can adversely impact leadership, they all either directly or indirectly stem from instinctive reactions we evolved to protect ourselves. When we're faced with a threat, whether real or imagined, our body mobilizes to prepare us for one of three survival responses: fight, flight, or freeze. For instance, a racing heart sends a rush of blood to the major muscles used to hit, kick, or run away. As primitive, unconscious reactions kick in, higher cognitive functions suffer. Stress hormones can dramatically impair concentration, planning, and decision making, all of which happen within the prefrontal cortex.

These inborn survival mechanisms are ill-matched with present-day realities. Success in our professional and personal lives requires flexible intellectual, emotional, and social responses rather than instinctive physical reactions. And the challenges we face often persist for long periods of time, leading to chronic activation of a survival system that evolved to function only in emergencies.

Eventually, that chronic activation can push any of us to our tipping point. We all have our limits, and when we stretch them too far we experience some combination of physical, mental/emotional, and behavioral symptoms that intensify as our stress levels rise. We need the awareness to notice them, as well as the courage to make tough choices to bring our stress levels down. As one leader put it, "In a world that constantly invites us to go beyond our limits, the most courageous response is to be aware of our limits and resist the mermaids' chant that invites us to keep going." Seventeen years ago, if Stefano had heeded his own physical and emotional warning signs, he could have prevented them from escalating, avoiding a great deal of suffering.

Stress shifting

Of course, committing to reducing stress is only useful if you have some idea of how to do that. Much has been written about specific stress management strategies, from

cognitive reframing, emotional labeling, and mind/body practices to time management, fitness programs, and nutritional changes. We encourage leaders to use whatever techniques work for them. But there's one practice we consistently recommend that can enable and support all the others: intentional breathing.

Breathing is both involuntary and voluntary. We don't need to plan how and when to take each breath (thank goodness!), but whenever we decide to consciously change our breathing, we can. This gives us the power to interrupt our involuntary stress responses and establish greater balance in our autonomic nervous system.

The autonomic nervous system has two branches: the sympathetic (our natural accelerator) and parasympathetic (our natural decelerator). When these two branches are alternately activated in a consistent pattern, we can enter into a state called *coherence*. Coherence is characterized by emotional stability and increased access to the prefrontal cortex, which promotes mental clarity, focus, and concentration — just what we need to tackle leadership challenges more effectively. Stress, in contrast, is characterized by strong sympathetic activation with less parasympathetic activation, so we are constantly accelerating.

And this is where breathing comes in: On the inhale our heart rate accelerates, and on the exhale it slows down. This heart rate information is sent directly to the brain, which plays a part in regulating the autonomic nervous system. Therefore, when we engage in a regular pattern of inhalation and exhalation, we help to establish a balance between sympathetic and parasympathetic activation.

We recommend using a three-step approach to engage intentional breathing:

1. **Remember to breathe.** When stress hits, it's helpful to have a cue — like a simple sign saying “Breathe” — that reminds you to take a breath.
2. **Begin breathing intentionally.** Start with a couple of strong, long, and deep breaths. Try to notice the physical sensations that accompany these breaths.
3. **Engage in resonant breathing.** After a few of these initial breaths, move to a technique called *resonant breathing*, where the total time spent on each inhalation and exhalation together is 10 seconds, for a total of six breaths per minute. Resonant breathing is particularly helpful in accessing coherence. You may find it helpful to learn how to do this while walking; the pace of your steps can provide a regular tempo for your breath. Eventually the rhythm will continue on its own, and you can stop timing. Continue until your state of mind shifts and you feel a sense of control over your own reactions.

Establishing coherence is useful in almost any context, not just high-stress situations. The more you practice the three steps, the easier it will be to engage in intentional breathing when you need it most.

Intentional breathing played a critical role in Stefano's recuperation. It helped to regulate his nervous system, which in turn made it easier for him to sustain other healthy

practices, such as proper nutrition, better sleep, yoga, physical exercise, and cognitive/emotional exercises. In time, Stefano gained more energy, his heart palpitations ceased, and his heart rhythms became more coherent. At the same time, his depression lifted and his impatience and irritability disappeared, leaving him calm and relaxed. Today, Stefano is in excellent health. Like all of us, he sometimes faces stressors that tempt him to push beyond his limits. But he now has the awareness, knowledge, and skill to bring himself back into balance.



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This article is about [STRESS](#)

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