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Focus and Scope
The articles that appear in NSPB represent the work of students from graduate psychology departments. Published work includes theory, research, literature reviews, and commentaries on the field. NSPB considers articles from all schools of thought on all topics relevant to psychology. NSPB may be particularly attractive to authors whose work does not fit the missions of larger psychology journals, and those looking to gain exposure to academic publishing. NSPB prides itself on publishing the early work of new and budding scholars.

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The current issue of the New School Psychology Bulletin (NSPB) epitomizes the idea of a team effort. This issue would not have been possible without the myriad contributions from the larger psychology community.

We are indebted to each and every one of the individuals listed below, as well as to each and every one of our readers.

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To Nina Khoury, our newly appointed layout editor, whose creative acumen allowed us to exponentially improve our journal's aesthetic appeal;

To our peer reviewers, whose forthright opinions and expertise allowed us to select the best articles for our journal and whose insights were invaluable in shaping the content of our journal;

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To Janiera Warren, without whom our department and journal would cease to function;

And to all the authors who trusted us with their work–we thank you for your intellect, your passion, and your patience.

Finally, we would like to extend our deepest gratitude to the previous Editorial Board, Lorraine Afflitto, Zishan Jiwani, Emily Weiss, and Ali Revill, for granting us the opportunity to step into their large shoes and continue the mission of NSPB. It is a privilege and an honor to work on this editorial board.

Letter from the Editors

It is hard to believe it has been nearly two years since COVID-19 became part of our widespread vernacular. Through this challenging time, we have been amazed by the dedication and resilience of those in academia and beyond. This has been a time of unprecedented shifts and adaptations; the editorial board is no exception to this phenomenon. Our editorial staff saw the departure of Lorraine Afflitto, Emily Weiss, and Zishan Jiwani and welcomed three new members: Heleen Raes, Lexi Karas, and Danielle Bryson who joined Ali Revill in producing thoughtful and consistent content for our beloved journal.

The three articles in this issue are dynamic considerations of memory, language, and emotion. The Tee and Taylor article assesses the experimental evidence for memory storage mechanisms in the brain while Gittoes and Roeser investigate the impact of anxiety and depression on language processing. Pizziferro and colleagues dissect the specific emotion regulation skills that most impact the psychological functioning for psychotherapy clients during COVID-19.

Even amidst a time of total upheaval, our collective commitment to scholarship offers inspiration and strength. We are grateful for this journal, where the belief that diverse approaches to scholarship can enact positive change is deeply held.

Welcome to NSPB Vol. 19 Issue 1. Thank you to everyone who was involved in this publication.

Your devoted editors,

Danielle Bryson
Lexi Karas
Heleen Raes
Ali Revill

Where is Memory Information Stored in the Brain?

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Abstract

Within the scientific community, memory information in the brain is commonly believed to be stored in the synapse – a hypothesis famously attributed to psychologist Donald Hebb. However, there is a growing minority who postulate that memory is stored inside the neuron at the molecular (RNA or DNA) level – an alternative postulation known as the cell-intrinsic hypothesis, coined by psychologist Randy Gallistel. In this paper, we review a selection of key experimental evidence from both sides of the argument. We begin with Eric Kandel's studies on sea slugs, which provided the first evidence in support of the synaptic hypothesis. Next, we touch on experiments in mice by John O'Keefe (declarative memory and the hippocampus) and Joseph LeDoux (procedural fear memory and the amygdala). Then, we introduce the synapse as the basic building block of today's artificial intelligence neural networks. After that, we describe David Glanzman's study on dissociating memory storage and synaptic change in sea slugs, and Susumu Tonegawa's experiment on reactivating retrograde amnesia in mice using laser. From there, we highlight Germund Hesslow's experiment on conditioned pauses in ferrets, and Beatrice Gelber's experiment on conditioning in single-celled organisms without synapses (*Paramecium aurelia*). This is followed by a description of David Glanzman's experiment on transplanting memory between sea slugs using RNA. Finally, we provide an overview of Brian Dias and Kerry Ressler's experiment on DNA transfer of fear in mice from parents to offspring. We conclude with some potential implications for the wider field of psychology.

Keywords: Memory, information, brain, neuron, synaptic hypothesis, cell-intrinsic hypothesis

When we memorize a phone number, where is this information stored? Regardless of the type of memory (e.g., episodic, semantic, autobiographical) or the theories of memory (e.g., storehouse, reconstructive), memory information must be stored somewhere in the brain.

Psychologist Karl Lashley's lifelong search for the answer ended somewhat fruitlessly: "I sometimes feel, in reviewing the evidence on the localization of the memory trace, that the necessary conclusion is that learning just is not possible" (Lashley, 1950, pp. 477-478). His workwas continued by one of his Ph.D. students, Donald O. Hebb, well-known today for his synaptic hypothesis:

When an axon of cell A is near enough to excite cell B and repeatedly or persistentlytakes part in firing it, some growth process or metabolic change takes place in one or bothcells such that A's efficiency, as one of the cells firing B, is increased (Hebb, 1949, p.62).

This hypothesis is often summarized as "cells that fire together wire together" (Shatz, 1992). The key idea is that changes in synaptic strength and connectivity may serve as the fundamental mechanism for information storage in the brain (Trettenbrein, 2016); that is, memory information is stored in the synapse. Some researchers, however, are not convinced. Perhaps the most outspoken among this minority group is C. Randy Gallistel:

> We do not yet know in what abstract form (e.g., analog or digital) the mind stores the basic numerical quantities that give substance to the

> foundational abstractions, the information acquired from experience that specifies learned distances, directions, circadian phases, durations, and probabilities. Much less do we know the physical medium in nervous tissue that is modified in order to preserve these empirical quantities for use in later computations (Gallistel, 2016).

Eric Kandel's Experiment on Sea Slugs: Memories Stored in Synapses

The groundbreaking work on how memory is believed to be stored in the human brain was performed by the research laboratory of Eric R. Kandel on the sea slug *Aplysia* (Kupfermann et al., 1970; Pinsker et al., 1970). *Aplysia*, also known as the California brown sea hare, is a marine snail with no external shell. It is typically about 20 cm (7.87 inches) in length and 1 kg (2.2 lbs) in weight. Kandel chose *Aplysia* for his study of memory because it has a simpler model of a nervous system compared to mammals; *Aplysia* only has about 20,000 neurons, in comparison to about 86 billion neurons in the human brain (Azevedo et al., 2009).

Aplysia's simple protective reflex of protecting its gills was instrumental in Kandel's experiment (Kupfermann et al., 1970; Pinsker et al., 1970). He found that some types of stimuli resulted in the strengthening of the sea slug's protective reflex, signifying learned fear. Strengthening was due to an amplification of the synapses that connect the sensory neurons to the motor neurons that produced the protective reflex. Kandel found that weaker stimuli resulted in short-term memory (shorter duration strengthening of the protective reflex that lasted for minutes to hours). In contrast, more powerful stimuli resulted in long-term memory (longer duration strengthening that remained for weeks). He also found that long-term memory required new protein to be formed, whereas short-term memory did not. If the process of synthesizing new protein was blocked, long-term memory formation was also blocked, but not short-term memory. The essence of Kandel's discovery was that synapses grew/changed when new memories were formed; he consequently interpreted this as evidence that short-term memory and long-term memory in the sea slug were stored in the synapse. During the 1990s, he showed that the same type of long-term growth/changes in the synapses associated with a protective reflex (learned fear) in sea slugs also applied to learned fear in the amygdala of mice, and thus, by extension of the animal model, was applicable to humans as well (Kandel, 2006).

Kandel concluded that memory in the brain was stored in synapses, and changes to synapses were central to the formation of short-term and long-term memories. In other words, Kandel's Experiment provided evidence in support of Hebb's synaptic hypothesis (Hebb, 1949). Based on his discovery of the synapse as the physiological basis of memory storage, Kandel was awarded the 2000 Nobel Prize in Physiologyor Medicine (Nobel Prize, 2000).

Key Experiments on Mice: Declarative Memory and the Hippocampus

Following Kandel's work in *Aplysia*, the next key experimental findings in support of Hebb's synaptic hypothesis were discovered in mice, the most notable of which was performed by John O'Keefe (O'Keefe, 1976; O'Keefe & Dostrovsky, 1971). Using electrophysiology (the study of electrical properties of biological cells and tissues), he recorded the firing of individual neurons in the

hippocampus of mice that were awake and freely moving in a room. O'Keefe discovered that some specific hippocampus neurons were always activated when the mice were at a particular location in the room, whereas other specific hippocampus neurons were activated when the mice were at a different location in the room. Based on this observation, O'Keefe interpreted that the hippocampus contained cognitive maps of the external environment, which the mice utilized for navigation. He named these neurons *place cells*. He concluded that memoryof the environment was stored as a combination of these *place cells*. This work subsequently helped define the role of the hippocampus in declarative memory in humans. For example, neuroimaging studies have found evidence of the existence of *place cells* in humans as well (Hassabis et al., 2009), especially in patients with Alzheimer's disease where the hippocampus were frequently affected at an early stage, which resulted in these patients often losing their wayand unable to recognize the environment. For his groundbreaking work on *place cells* in the hippocampus, O'Keefe was awarded the 2014 Nobel Prize in Physiology or Medicine (Nobel Prize, 2014).

The discovery of *place cells* paved the way for the interpretation of results found in a study on rabbits by Bliss and Lomo (1973). By stimulating the hippocampus using a high-frequency train of action potentials, they found prolonged/persistent strengthening of the synapses (i.e., long-term potentiation; LTP) in all three major hippocampal pathways (perforantpathway, mossy fiber pathway, Schaffer collateral pathway). In two of these pathways (perforant and Schaffer collateral), the LTP was found to be consistent with Hebb's synaptic hypothesis, which consequently reinforced the notion that memory information was stored in the synapse (Bliss & Collingridge, 1993; Mayford et al., 2012).

Joseph LeDoux's Experiments on Mice: Procedural Fear Memory and the Amygdala

Classical conditioning experiments on mice conducted by the research laboratory of Joseph E. LeDoux at New York University found some of the strongest evidence that reinforced Hebb's synaptic hypothesis. Classical conditioning was first studied by Ivan Pavlov, who used his dogs as subjects (Nobel Prize, 1904). Typically, his dogs would salivate when food was presented, but not when a bell was rung. However, if the bell was rung before the food was presented and this sequential process was repeated, his dogs would eventually salivate even when the bell was rungwithout presentation of the food. Meaning, the bell (conditioned stimulus) resulted in the same response as the food (salivate). In LeDoux's experiments, he paired an audio tone with an electric shock to the feet of mice, which subsequently resulted in a conditioned fear response (freezing behavior) to the audio tone alone (LeDoux, 1995; Rogan et al., 1997). This form of learning, termed fear conditioning, was known to involve the amygdala, which receives auditory input and regulates autonomic fear responses. LeDoux found that this conditioned fear resulted in LTPin the auditory neurons of the amygdala, to which he concluded that the LTP constituted memory of the conditioned fear. That is, memory was stored by way of strengthening the synapses, as hypothesized by Hebb.

Synapse as the Basic Building Block for Artificial Intelligence Neural Networks

In the adjacent field of artificial intelligence (AI), the concept of synapse serves as the underlying basis for neural networks (NN). At each neuron in the AI's NN, there are multiple inputs and multiple

outputs; each of the inputs (x_i), is weighted (multiplied) by a numerical value(w_i), after which all the weighted inputs are summed (added) to produce an output (y):

$$y = \sum_i x_i w_i$$

The output (y) subsequently serves as the input to other neurons. Here, the synapse (in a brain) is conceptually analogous to the numerical value that each input to the neuron (of the AI's NN) is weighted by, also known as the synaptic weight. The sequential cascade (i.e., series interconnection) of one neuron's output serving as another neuron's input in an AI's NN is known as a layer. Recent advances in computing power/speed have enabled the use of many such layers,resulting in what is termed Deep Learning (LeCun et al., 2015). An AI's NN with 20 layers and hundreds of millions of synaptic weights have been highly effective in recognizing images and human faces, to the extent that a variant of Deep Learning called Deep Convolutional Neural Networks have been hypothesized to mimic neurons in the visual cortex of the brain (Lindsay, 2020). Deep Learning has also been successfully applied to natural language processing (understanding semantics), most notably in 2011 when IBM's Watson computer defeated two human champions (Ken Jennings and Brad Rutter) in the television quiz show *Jeopardy!* (Markoff, 2011). In 2017, Google's AlphaGo computer conquered the game Go when it defeated the world's number one Go player, Ke Jie (Mozur, 2017). The Deep Learning approach employed by AlphaGo was a variant known as reinforcement learning, a computer learning method based on the psychological concepts of operant conditioning and reinforcement that psychologist B.F. Skinner initially proposed, which has been associated with the dopamine reward system in the brain (Niv, 2009). The impressive feats accomplished by these synapse-based DeepLearning neural networks, along with the hypothesized similarities with the brain (i.e., visual cortex, semantics, dopamine reward system), indirectly supported Hebb's synaptic hypothesis.

Lingering Doubts on Synapse as the Physical Basis of Memory

Despite the conceptual similarities, synaptic weights in AI's NN have constant values thatdo not change after the training phase has been completed. In contrast, synapses in the brain areconstantly changing, in part due to the inevitable existence of noise (Faisal et al., 2008).

Furthermore, AI's NN are based on modern computers that function using registers (a type of computer memory used for addition and mathematical multiplication operations), whereas there is no evidence that such registers exist in the brain. Consequently, it would be fair to surmise thatthe brain is very unlikely to function in the same way as AI-based neural networks.

For decades, Hebb's synaptic hypothesis, along with key supportive experimental results of the synaptic mechanism of memory, held great promise for the development of new medications/ treatments for memory-related illnesses such as Alzheimer's disease (loss of explicit memory). The general idea was that since memory was stored in the synapse, addressing/resolving the synaptic pathology could help treat memory disorders (Jackson et al., 2019). However, the long-awaited breakthroughs have yet to be found, raising some doubts against Hebb's synaptic hypothesis and the subsequent associated experimental findings.

One indicative counterevidence arose from the study of motor memory in mice. Using two-photon

microscopy, it was found that learning a new motor skill (i.e., new motor memory) was indeed accompanied by the formation of new synaptic connections (Yang et al., 2009). However, unexpectedly, synaptic spines were found to be turning over (changing) at a high rate in the absence of learning, to the extent that newly formed synaptic connections (supposedly encoding new memory) would have vanished in due time, implying that motor memories far outlived their supposed constituent parts (synapses; Trettenbrein, 2016). This perplexing findingwas perhaps best summarized by Emilio Bizzi and Robert Ajemian:

> If we believe that memories are made of patterns of synaptic connections sculpted by experience, and if we know, behaviorally, that motor memories last a lifetime, then how can we explain the fact that individual synaptic spines are constantly turning over and that aggregate synaptic strengths are constantly fluctuating? How can the memories outlast their putative constitutive components? (Bizzi & Ajemian, 2015, pp. 91-92)

They further pointed out that this mystery existed beyond motor neuroscience, extending to all of systems neuroscience, given that many studies have found such constant turnover of synapses regardless of the cortical region. In other words, synapses are constantly changing throughout theentire brain: "How is the permanence of memory constructed from the evanescence of synaptic spines?" (Bizzi & Ajemian, 2015, p. 92). This is perhaps the biggest challenge against the notionof the synapse as the physical basis of memory.

David Glanzman's Experiment on Sea Slugs: Memories Not Stored in Synapses

Doubts on the synaptic basis for memory were validated in a study conducted by the research laboratory of David L. Glanzman at the University of California, Los Angeles, which found that long-term memories could be restored after synapses were pharmacologically eliminated (Chen et al., 2014). It is worth noting that Glanzman was previously a postdoctoral researcher in Eric Kandel's lab at Columbia University. Glanzman grew *Aplysia* neurons in Petri dishes and trained/treated them with the hormone serotonin, which subsequently triggered the growth of new synapses as expected and predicted by Kandel's study. After that, the neurons were given pharmacological treatments (anisomycin and chelerythrine) that disrupted long-term memory. More significantly, they reversed the synaptic growth resulting from the serotonin, in which the synapses reverted to the way they were before being trained/treated by serotonin. In addition to the reversal, some synapses that existed prior to the serotonin training/treatment were also lost. Based on Hebb's synaptic hypothesis, the long-term memory should have been erased as well, given the reversal of the synaptic growth and loss of synapses. Surprisingly, the long-term memory remained intact. This finding suggested that, while synapses have grown during long-term memory formation, storage/recollection of the memory was not dependent on retaining/maintaining the synapses.

Thus, these results challenged Hebb's hypothesis that synapses store long-term memories. Glanzman concluded that "long-term memory storage and synaptic change can be dissociated" (Chen et al., 2014, p. 1). For people who suffer from post-traumatic stress disorders (PTSD), this result suggested that the potential use of medications

(propranolol) to disrupt the synapses will unlikely eliminate painful memories. At the same time, this result offered some hope to people who suffer from dementia or Alzheimer's disease; some parts of the memories may be recoverable despite the neurodegeneration (deterioration/loss of synapses).

Susumu Tonegawa's Experiment on Mice: Reactivating Retrograde Amnesia Using Laser

Further evidence against Hebb's synaptic hypothesis was reported by Susumu Tonegawa at the Massachusetts Institute of Technology. In an experiment conducted by Tonegawa's research lab (Ryan et al., 2015), neurons in conditioned/trained mice were injected with anisomycin, disrupting synaptic growth/consolidation (Kandel deemed necessary for memory storage). Consequently, retrograde amnesia was induced, in which the mice could not retrieve the memory via an emotional/fear trigger. However, these "lost" memories could be reactivated by shining a laser onto the corresponding memory neurons that were tagged during the conditioning/training stage. Here, laser refers to optogenetics, a biological technique that employs light to control neurons that have been genetically modified to express light-sensitive ion channels. Tonegawa's experiment on mice was, in essence, a replication of Glanzman's experiment on sea slugs; in both cases, the animals were trained/conditioned, and then, pharmacological treatments (anisomycin) were used to disrupt the growth of synapses, which, according to Hebb's synaptic hypothesis, should have erased the memory. However, in both cases, the memory remained retrievable despite the pharmacological blocking of the synapse. Tonegawa's study concluded that an increase in synaptic strength was not a crucial requisite for the storage of memory information. This further reinforced the doubts on Hebb's synaptic hypothesis cast by Bizzi and Ajemian (2015).

Germund Hesslow's Experiments on Ferrets: LTP Cannot Explain Conditioned Pauses

Pavlovian eye-blink conditioning experiments on ferrets conducted by the research laboratory of Germund Hesslow at Lund University raised further doubts on Hebb's synaptic hypothesis (Johansson et al., 2014). Typically, the eye would blink in response to the presentation of an air puff, similar to the way Pavlov's dog would salivate in response to the presentation of food. In Hesslow's study, the air puff was paired with an electrical pulse to the paw of the ferret, analogous to the bell in Pavlov's study. Prior to conditioning, the electrical pulse to the paw produced no eye blinks; after conditioning (stimulating the paw with an electrical pulse before presenting the air puff), the electrical pulse to the paw produced eye blinks even in the absence of an air puff. Hesslow measured the electrophysiological responses of Purkinje cells in the cerebellum that were associated with eye-blinks in order to examine how the cells would respond to the paired stimulus (electrical pulse to the paw). Prior to conditioning, the electrical pulse to the paw did not change the firing pattern of the Purkinje neurons. After conditioning, a 200-millisecond electrical pulse to the paw resulted in an approximately 200-millisecond pause in the Purkinje cells' neural spike activities; likewise, a 300-millisecond electrical pulse resulted in an approximately 300-millisecond pause. These findings indicated that the Purkinje cell neurons were able to remember the time duration (e.g., 200-millisecond, 300-millisecond) of the paired stimulus (electrical pulse to the paw) in a rather precise and proportionate manner. Hesslow

concluded that LTP could not account for the Purkinje cells' ability to remember the time durations: "Mere strengthening or weakening of these synapses cannot account for the time course of the conditioned pause response" (Johansson et al., 2014, p.14932). Consequently, Hesslow's experiments further added doubts to Hebb's synaptic hypothesis.

Beatrice Gelber's Experiments on Paramecium: Conditioning Without Synapses

Pavlovian conditioning experiments on *Paramecium aurelia* in the 1950s, conducted by psychologist Beatrice Gelber at Indiana University and the University of Chicago, raised further questions on Hebb's synaptic hypothesis (Gelber, 1957). *Paramecium aurelia* is a single-cell organism, typically oblong or slipper-shaped, covered in cilia(hair-like filaments). Most people would remember encountering *Paramecia* at some point in high school science classes by wayof peering through a microscope. Gelber was interested in finding out whether simple single-cell organisms such as *Paramecia* were capable of Pavlovian conditioning, which was and still is widely considered a sophisticated form of learning. One of her astonishing findings ended up being published in *Science* (Gelber, 1957). In that study, a micro drop of bacterial suspension (i.e., food) was introduced at the edge of a container that had a "hungry" culture of *Paramecia*. In the experimental group, a clean wire was simultaneously lowered into the middle of the container; after 8 minutes, the wire was removed. The control group received the food without the wire. After 30 minutes, a clean and sterile wire was introduced in each of the cultures/containers. Gelber found that *Paramecia* in the experimental group surrounded the wire significantly more than those in the control group. Based on this result, along with othervariations of experimental design, she concluded that *Paramecium aurelia* was indeed capable of Pavlovian conditioning. Despite the gravitas of this discovery, Gelber's studies were ignored and/or dismissed by her contemporaries and largely forgotten until earlier this year (January 2021), when Harvard psychologist Samuel J. Gershman brought Gelber's work back into the spotlight (Gershman et al., 2021). Barring Hesslow's studies on ferrets (Johansson et al., 2014), the prevailing theory is that Pavlovian conditioning is mediated by Hebb's synaptic hypothesis. However, single-cell organisms clearly do not have synapses; if *Paramecia* can be conditioned to remember, they must be using a non-synaptic form of memory storage. Therefore, synapses may not actually be essential for memory storage, calling Hebb's synaptic hypothesis into question

Alternatives to Hebb's Synaptic Hypothesis

The logical question to pose at this point is: if memory information is not stored in the synapse, then where is it? Glanzman suggested that memory might be stored in the nucleus of the neurons (Chen et al., 2014). On the other hand, Tonegawa proposed that memory might be stored in the connectivity pathways (circuit connections) of a network of neurons (Ryan et al., 2015). In disagreement with Tonegawa, Hesslow emphasized that memory is highly unlikely to be a network property and further posited that the memory mechanism is intrinsic to the neuron (in agreement with Glanzman; Johansson et al., 2014). Decades earlier, Gelber (1962) hypothesized that memory is "coded in macromolecules" (p. 166; inside the cell of the *Paramecia*), and she further postulated that "the biochemical and cellular physiological processes which encode new responses are continuous across the phyla" (p. 166), implying that

the memory mechanisms would be "reasonably similar for a protozoan and a mammal" (p. 166). Gershman expressed a cautious agreement with Gelber that "if the hypothesis is correct, then single cells hold more surprises in store for us" (Gershman et al., 2021, p. 11). The collective views of Glanzman, Hesslow, Gelber, and Gershman is known as the cell-intrinsic hypothesis – in which memory information is stored in information-bearing molecules inside the neuron (Gallistel, 2017).

Plausibility of the Cell-Intrinsic Hypothesis

Peter Sterling from the University of Pennsylvania and Simon Laughlin from the University of Cambridge suggested that storing memory and performing computations using molecular chemistry inside the neuron would be energetically cheaper in comparison to using neural spikes and synapses (Hebb's synaptic hypothesis; Sterling & Laughlin, 2015). Gershman further elaborated that "a synaptic memory substrate requires that computations operate via the propagation of spiking activity, incurring an energetic cost roughly 13 orders of magnitude greater than the cost incurred if the computations are implemented using intracellular molecules" (Gershman et al., 2021, p. 2). It is worth noting here that 13 orders of magnitude equate to 10^{13}, suggesting that synaptic memory would require approximately 10 trillion times more energy than molecular memory. Within the neuron, two major types of molecules are known to be capable of storing information: deoxyribonucleic acid (DNA), and ribonucleic acid (RNA; Gallistel, 2017).

Francis Crick, who was awarded the 1962 Nobel Prize in Physiology or Medicine for deciphering the helical structure of the DNA molecule (Nobel Prize, 1962), was first to suggest that "memory might be coded in alternations to particular stretches of chromosomal DNA" (Crick, 1984, p. 101). The hypothesized epigenetic (non-genetic influences on gene expression through DNA methylation or demethylation) mechanism for memory was further elaborated by molecular biologist Robin Holliday (Holliday, 1999). Recent work by researchers at Johns Hopkins University School of Medicine (Yu et al., 2015) concluded that neurons constantly rewrite their DNA: "We used to think that once a cell reaches full maturation, its DNA is totally stable" but "this research shows that some cells actually alter their DNA all the time, just to perform everyday functions" (Johns Hopkins Medicine, 2015). In a collaborative effort among researchers at the University of Alabama at Birmingham, Bates College, and Vanderbilt University, 9.2% of DNA in the hippocampus of mice were found to be altered after fear conditioning (Duke et al., 2017). Another recent work (McConnell et al., 2017) concluded that no two neurons are genetically alike: "We were taught that every cell has the same DNA, but that's not true" because "neural genes are very active" (Makin, 2017).

All single-stranded RNA in the cell is made from double-stranded DNA via a process called transcription (Alberts et al., 2002). Consequently, changes in the DNA would be passed onto the RNA. Alternatively, RNA could also potentially be altered on its own, without necessarily involving the DNA. It is worth noting here that there are many types of RNA (messenger RNA, transfer RNA, ribosomal RNA, microRNA). An RNA-based hypothesis of memory and computation has recently been proposed by Hessameddin Akhlaghpour of The Rockefeller University (Akhlaghpour, 2020).

David Glanzman's Experiment: Transplanting Memory Between Sea Slugs Using RNA

Glanzman conducted a follow-up experiment (Bedecarrats et al., 2018) to test the cell-intrinsic hypothesis – specifically, on memory information storage in RNA molecules inside theneuron. *Aplysia* sea slugs were given repeated mild electric shocks to their tails (experimental group), resulting in an enhanced defensive withdrawal reflex to protect them from potential harm. Subsequently, when those sea slugs were tapped, their defensive withdrawal response averaged 56 seconds in duration. On the other hand, sea slugs that did not previously receive electric shocks (control group) responded for only about 1 second. RNA from both groups was subsequently extracted. RNA from the experimental group was injected into one new group of naïve sea slugs (sea slugs that have never received any electric shock), whereas RNA from the control group was injected into another new group of naïve sea slugs. Glanzman found that the group of naïve sea slugs that received RNA from the control group exhibited a defensive withdrawal response of about 5 seconds. Remarkably, the group of naïve sea slugs that received RNA from the experimental group had a response of about 38 seconds. In other words, naïve seaslugs that received RNA from the experimental group responded as if they themselves had received electric shocks, displaying a response duration that was similar in length to those that actually received electric shocks (experimental group). Glanzman attributed the longer response duration to the RNA injection and concluded that "it's as though we transferred the memory" because "if memories were stored at the synapses, there is no way our experiment would have worked" (University of California, Los Angeles, 2018). Building on the findings of his previousstudy (Chen et al., 2014), he was hopeful that RNA could potentially be used to ameliorate theeffects of Alzheimer's or PTSD in the not-too-distant future.

Dias and Ressler's Experiment on Mice: DNA Transfer of Fear from Parents to Offspring

An experiment conducted by Brian G. Dias and Kerry J. Ressler at Emory University found that fear conditioning in mice could be transferred from parents to offspring (Dias & Ressler, 2014). Using Pavlovian conditioning, they trained mice to be fearful of a scent (acetophenone, which smelled like cherry blossom) by pairing it with a mild electric shock. After conditioning, the mice learnt to associate the scent with pain, startling in the presence of the scent even without an electric shock. They found that offspring of the conditioned mice were startled more in response to the scent, even though the offspring were not previously conditioned to associate the scent with pain from an electric shock. Astonishingly, the sensitivity was also observed in the second-generation mice (grandchildren). Dias and Ressler concluded that the conditioned fear associated with the scent was transferred to the offspring via DNA in the sperm or eggs of the mice, suggesting that the offspring inherited the fear from their parents. In short, traumatic memories could be inherited, at least in mice. Ressler suggested that humans may also inherit epigenetic alterations that influence behavior: "A parent's anxiety could influence later generations through epigenetic modifications to receptors for stress hormones" (Callaway, 2013). He added that "knowing how the experiences of parents influence their descendants helps us to understand psychiatric disorders that may have a transgenerational basis, and possibly to design therapeutic strategies" (Eastman, 2013).

Conclusions

After more than 70 years of research efforts by cognitive psychologists and neuroscientists, the question of where memory information is stored in the brain remains unresolved. Although the long-held synaptic hypothesis remains as the de facto and most widely accepted dogma, there is growing evidence in support of the cell-intrinsic hypothesis. As Glanzman summed up rather succinctly, "I expect a lot of astonishment and skepticism" (McFarling, 2018). In a recent interview in April 2021, Gallistel was quoted saying, "Scientists are human. Like all humans, they're prisoners of preconceptions. When a preconception takes strong hold, it becomes almost unshakable" (Join Activism, 2021). He further reiterated a famous quote by physicist Max Planck that "science progresses one funeral at a time" (Join Activism, 2021).

A synapse connects one neuron to another. Without synapses, most neurons would not be able to communicate with one another; sensory information (e.g., from the retina) would not reach the brain in the first place. Consequently, both the synapse and the cell are likely to be crucial to memory, with each serving a potentially different but inter-dependent function; while the cell might be storing the memory information, the synapse might be required for the initial formation and the subsequent retrieval of the memory (Tee & Taylor, 2021). A potentially helpful analogy here is the way a road leads to a warehouse that stores goods; while the warehouse stores the goods, the road is required for the initial delivery and subsequent pickup of the goods.Following this analogy, it would be risky to store all goods in just one warehouse (in case of fire or burglary). Furthermore, there is a finite amount of storage space/capacity in each warehouse. Therefore, it would be wise and/or inevitable to store goods across multiple warehouses that are interconnected by a network of roads. When goods are picked up from the multitude of warehouses, the complex logistical process may not always result in a perfect retrieval of the expected quantity or type of goods. Likewise, it would make sense to store memory information across multiple neurons interconnected by a network of synapses in the brain. When memories (e.g., episodic, autobiographical) are retrieved from the multitude of neurons, the complex recollection process may not always result in a perfect retrieval. Such a model could potentially account for errors of omission (forgetting information) and errors of commission (remembering the wrong information) in reconstructive memory.

Lastly, if DNA is indeed involved in the storage of long-term memory in humans, thereare profound implications beyond neuroscience and cognitive psychology. For example, could memories associated with PTSD, substance use or racial discrimination be inherited from one generation to another? If so, how would such inherited memories affect members of a community (collective memory)? These types of open research questions have far-reaching ramifications for clinical, developmental, and social psychology.

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Mental Health and Language: Anxiety and Depression Impact Sentence Recall Differently

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Abstract

The present study examined how two mental health disorders (anxiety and depression) impact people's ability to process language. Participants ($N = 64$) were asked to read and recall sentences. A secondary naming task was used to prompt lexical rehearsal of the second noun in the stimulus sentence that was either part of the subject (e.g., *Tania and the glass moved…*) or final phrase (e.g., *... above the glass and the donkey*). Corrections during writing and recall mistakes were modelled in generalized mixed models. In line with the hypothesis that mental health disorders impair language processing, both anxiety and depression affected sentence recall accuracy but only anxiety impacted the execution process. Understanding the impact of mental health disorders on language processing is crucial to develop targeted support for individuals who would otherwise be systematically disadvantaged in educational, social, and professional life. Future research may benefit from separating samples dependent on symptom severity and comorbidity.

Keywords: Depression, anxiety, sentence recall, mental health, language processing

Mental health conditions such as anxiety and depression affect a large part of the world population (Demyttenaere et al., 2004). Globally, anxiety is prevalent in 264 million (3.6%) people, while depression affects 322 million (4%; World Health Organization, 2017). Depression is characterized by low moods, sadness, fatigue, a lack of self-esteem, and feelings of hopelessness (Beck et al., 1998). The prevalence of depression increases every year (Skovlund et al., 2017), specifically among adolescents and young adults (Twenge et al., 2018). Likewise, anxiety is more prevalent in younger than older adults (Bandelow & Michaelis, 2015; Flint et al., 2010) with a higher prevalence among students, who often experience symptoms of restlessness, constant fear or worry, difficulty concentrating, irritability and sleep disturbances (American Psychiatric Association [APA], 2013; Stallman, 2010; Storrie et al., 2010).

Both depression and anxiety are known to impact cognitive skills. Individuals with higher levels of depression endure cognitive dysfunctions, which influence their ability to attend to (Duque & Vázquez, 2015), recall (Dillon & Pizzagalli, 2018), and process information at average speed (Beats et al., 1996; Diamond et al., 2008; Tsourtos et al., 2002). Similarly, anxiety disorders impair information-processing (Mogg & Millar, 2000; Wilson et al., 2006) and cognitive performance (Derakshan & Eysenck, 2009) due to restrictions on attentional capacity (Christopher & MacDonald, 2010; Eysenck et al., 2007). Anxiety further results in mind blanking (APA, 2013), which is a disconnect of attention from perception whereby an individual's focus may wander outside of their environment or simply disappear, with attention failing to bring stimuli into conscious awareness (Ward & Wegner, 2013). Mind blanking impairs people's memory and ability to concentrate (Derouesne et al., 1993). Yet little is known about how anxiety and depressive disorders influence language processing.

Austin and colleagues (2001) explored the idea that mood disorders such as depression may be

associated with a distinct pattern of cognitive- and language-related impairments. They reported impairments in both verbal recall (Austin et al., 1999) and verbal recognition (in automatic tasks) in subjects with depression (Brown et al., 1994). Similarly, depressed participants have been found to perform poorly on information processing tasks in terms of processing speed and flexibility (Jones et al., 2010). Gronwall (1997) found that this occurred when a motor response, in particular, was required for the Paced Auditory Serial Addition Test (PASAT) task. Participants suffering from a depressive disorder not only made more mistakes on information processing tasks but were also more likely to display non-task-related pupil dilation during a task, which suggests of difficulty in coping with a high cognitive load or information processing beyond the task at hand (Jones et al., 2010).

Researchers have sought to explain the neurocognitive deficits attributed to anxiety and depression through motivational impairments (Barch et al., 2019; Cohen et al., 1982; Porter et al., 2007). However, neuropsychological tasks conducted on depressed participants indicate problems in specific brain regions (Siegle et al., 2007), particularly difficulties in cognitive processing speed and executive function (Sheline et al., 2006; Venezia et al., 2018). Cognitive dysfunctions have been commonly identified amongst individuals with anxiety and depressive disorders, impacting various domains such as attention (Duque & Vázquez, 2015; Keller et al., 2020, memory (Dillon & Pizzagalli, 2018), problem solving (Jones et al., 2017; Remmers et al., 2015), and motor functioning (Bennabi et al., 2013; Buyukdura et al., 2011; Felger et al., 2016). For example, Rose and Ebmeier (2006) examined working-memory performance in patients with major depressive disorder and found slower reaction times and reduced recall accuracy, revealing an impairment of central-executive functions. Deficits in executive functions are well known in individuals with depression (Degl'Innocenti et al., 1998; Grant et al., 2001) and comorbid anxiety (Airaksinen et al., 2004).

All the aforementioned cognitive domains are involved in language processing. These findings suggest that cognitive functions which relate to language processing are impaired in individuals with mental health problems. Researchers have found that depression is linked to increased sentence-onset durations (De Lissynder et al., 2010), frequent pausing (Mundt et al., 2007), poor fluency (Akiyama et al., 2018), prolonged latency of response (Abas et al., 1990), and more production errors (Gohier et al., 2009; Vilgis et al., 2015), which is further exacerbated by depression chronicity (Vilgis et al., 2015). This indicates a primary link between depression and people's ability to produce language. For individuals with anxiety, researchers report a dominating impact on the language comprehension system, i.e., anxiety has been linked to a top-down processing bias (Bradley et al., 2000; Mogg & Bradley, 2005; Wilson et al., 2006) leading to shallow representations of meaning during reading. Crucially, the current review suggests that anxiety and depression may affect language processing differently.

Understanding how mental health disorders impact language processing is important because individuals with anxiety and depression may otherwise be systematically disadvantaged in various every-day contexts that require linguistic skills, e.g., exams, job interviews, academic conferences, communication, etc. For example, attrition rates and poorer outcomes in higher education are significantly greater amongst

individuals diagnosed with anxiety and/or depression (Cogburn et al., 2015; Dyrbye et al., 2006). Enhanced understanding could positively improve outcomes for such individuals, as appropriate measures could enhance the delivery of communication and tools to ensure that individuals receive the support they require. Adaptations could also be made where necessary. This would increase inclusivity and life satisfaction, and ensure a sustainable future by providing equal opportunities for everyone.

To summarize, existing research has shown that depression impacts people's ability to formulate sentences, while anxiety is related to a poorer ability to understand language. A form of language processing where language production and comprehension intersect is sentence-recall tasks, in which participants are asked to read short sentences and subsequently recall the sentence from memory. In order to recall the linguistic form of a stimulus, people need to decode the sentence in sufficient detail before reassembling its linguistic form from a conceptual memory representation (Lombardi & Potter, 1992; Potter & Lombardi, 1998; Potter, 2012; Roeser et al., 2020). Sentence recall tasks are used to understand to what extent this occurs. The method used in the present study is a sentence recall task combined with a manipulation used in language production research (Roeser et al., 2019; Martin et al., 2010; Martin et al., 2014) whereby arrays of images are used to elicit sentences that either start with a conjoined phrase and finish with a simple noun (e.g., [N1] *and the* [N2] *moved above the* [N3]) or vice versa (e.g., [N1] *moved above the* [N2] *and the* [N3]) while keeping the overall complexity of the sentence (e.g., number of content words and phrases) constant. This manipulation allows the researcher to test hypotheses about how sentences are chunked during encoding, i.e., into individual picture names or syntactic phrases. In addition, authors have used this paradigm to test how information associated with lexical items interacts with syntactic units (Griffin, 2001; Roeser et al., 2019). In particular, in this study, the ease of recalling the second noun (N2) contained in the sentence was manipulated; importantly, N2 was either part of the sentence-initial phrase (e.g., [N1] *and the* [N2] *moved*…) or the sentence-final phrase (e.g., … *above the* [N2] *and the* [N3]). This manipulation helps to determine whether recall is sensitive to syntactic and lexical factors.

To the authors' knowledge, no research at present has investigated to what extent anxiety and depression impact people's ability to recall sentences at different linguistic levels. That is, language recall in individuals with anxiety or depression could be sensitive to the syntactic form of the sentence, its lexical contents, or perhaps both. To explore which level of linguistic representation is affected by anxiety and depression, the syntactic structure and the lexical content of the target sentence was manipulated. As discussed, an understanding of how mental health disorders specifically impact language processing is crucial in supporting individuals who face mental health challenges. In particular, an identification of which linguistic aspect(s) is impacted could result in modifications and targeted treatments for such individuals.

The present study explored how anxiety and depression impact people's ability to recall sentences. It was hypothesized that both disorders would affect language processing differently. In particular, anxiety was expected to impair people's ability to comprehend sentences, and thus affect the accuracy of the recalled sentence. While the product of the recall was expected to be impacted

by anxiety, no effects on the writing execution process were predicted. In contrast, for depression, increased difficulty with language encoding and thus the execution of writing was hypothesized.

Method

Participants

The current study recruited 64 students (19 males, 45 females; median age = 20 years; range = 18-27) from Nottingham Trent University and University of Nottingham in the UK. All participants were native English speakers and reported no reading or writing impairments. Other demographics (e.g., race, ethnicity, social economic status) were not recorded.

The study was approved by the Social Sciences Research Ethics Committee of Nottingham Trent University. Participants were recruited through the university research studies platform with four research-participation credits offered as incentives for participation. All participants were able to access the information sheet online and in-person when attending to complete the study. All participants signed consent forms and were provided with debriefing sheets at the conclusion.

Design & Materials

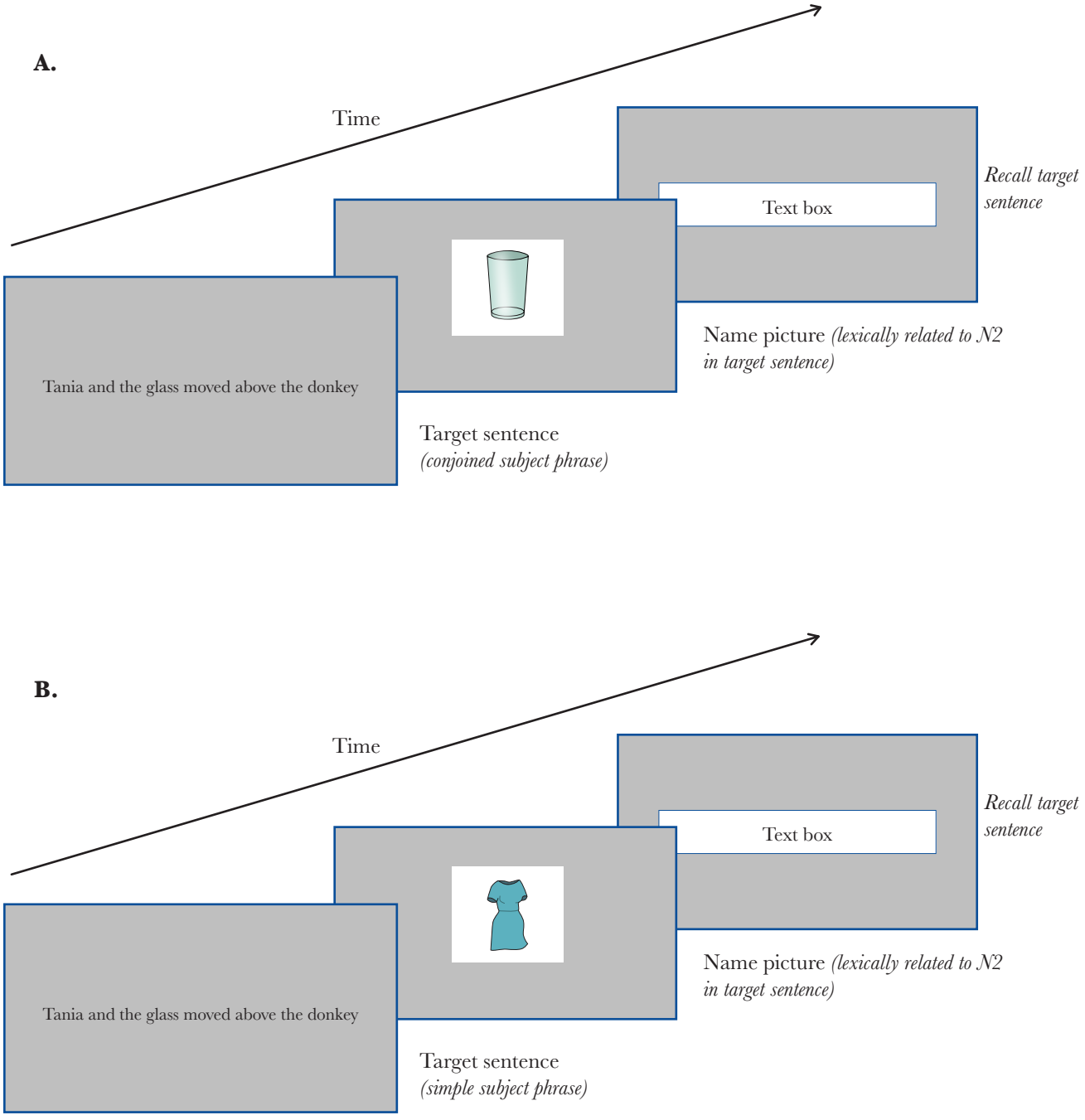
Participants took part in a sentence recall task. Participants were asked to read a sentence and subsequently recall it in writing (i.e., typing) after responding to a secondary picture-naming task. The study used a 2 x 2 factorial within-subjects design and manipulated the syntactic configuration of the target sentence and the ease of recalling one lexical item of the sentence (see Figure 1). As for the syntactic manipulation, shown in Figure 1A and 1B, the stimulus sentence either started with a simple subject noun phrase and ended with a conjoined noun phrase (e.g., *Tania moved above the glass and the donkey*) or started with a conjoined noun phrase and ended with a simple noun (*Tania and the glass moved above the donkey*). In a secondary picture-naming task, lexical recall of the second noun in each sentence (henceforth, N2) was either facilitated (Figure 1A) or not (Figure 1B): between presentation of the target sentence and recall, participants either saw and named a picture that was *related* to N2 (picture of a glass in the example) or unrelated (picture of a dress).

Note that the lexical manipulation was always part of a conjoined noun phrase but either in the sentence initial subject position or sentence final. Importantly, the overall complexity of the sentence was held constant. The dependent variables were product and process-oriented recall measures operationalized as the number of mistakes in the recalled sentence and the number of corrections during the writing process. Psychometric scales were used to assess anxiety and depression levels for every participant (see Measures). These were administered after the sentence-recall task was completed.

Twenty-four stimulus items were created. Items were counterbalanced across four Latin square lists so that every participant saw one condition of each item, but all participants saw the same number of conditions. Furthermore, items were counterbalanced for the first name used in the target sentence (e.g., *Tania* or *Peter*); names were used to avoid determiners in the first position of the sentence (see Roeser et al., 2019). A colored version of the Snodgrass (1980) picture set (as cited in Rossion & Pourtois, 2004) was used for the naming component; pictures were selected using naming norms collected for the same student population (Torrance et al., 2018). Related and unrelated N2 images were matched with regards to naming diversity, British National Corpus frequency, and

Figure 1

Schematic Overview of Experimental Paradigm



Note. The target sentence was presented first for participants to read. The next screen shows a picture that had to be named. The picture shown was either related (A) to the second noun in the target sentence or not (B). The final screen required participants to recall the target sentence by writing it into a textbox.

length of the most commonly used name used for the picture. The noun that was used in the center position of the sentence was also involved in the secondary picture naming task in the "related" condition. Naming norms were used for these pictures to support the effectiveness of the manipulation. In particular, the aim of the picture naming task was to facilitate memory rehearsal of the noun used in the target sentence. Participants would be assumed to use the anticipated name for the picture that corresponds to the noun in the target sentence. Therefore, names used in the target sentence were selected from the most commonly used name for the respective pictures as indicated by the naming norms. Because there is some variance associated with how well people remember short or long words, typically related to the corpus frequency of these words (Baddeley et al., 1975; Tehan & Tolan, 2007), both word length and frequency of the picture names were controlled.

Twenty-four filler items were added, including sentences with structural ambiguities, taken from Van Gompel et al. (2001). These included sentences such as "Peter yelled at the protester with the loudspeaker," which can have two different interpretations (i.e., Peter used the loudspeaker, or the protester had a loudspeaker). Adding filler sentences was intended to prevent participants from adapting strategies to reproduce target sentences with structural similarities.

Measures

Anxiety

Participants' levels of anxiety were measured using the Beck Anxiety Inventory (BAI; Beck et al., 1988). The BAI consists of 21 items addressing symptoms common to anxiety (e.g., unable to relax, fear of worst happening, fear of losing control) with responses measured on a 4-point Likert scale (0 = *not at all*; 3 = *severely, it bothered me a lot*). Items prompted participants to respond to questions about the intensity of cognitive, affective, and somatic symptoms of anxiety experienced within the last month. The by-participant sum of scores can range from 0-63, with scores between 0-21 indicating low levels of anxiety, 22-35 moderate levels, and 36 and above suggesting potentially concerning levels of anxiety (Beck et al., 1988). The BAI has been reported to have high internal consistency with a Cronbach's α of .94 (Fydrich et al., 1992) and a high test-retest reliability (r = .75; Muntingh et al., 2011).

Depression

The Beck Depression Inventory (BDI; Beck et al., 1961) was used to estimate levels of depression. The BDI consists of 21 items measured on a four-point scale. Each question involves statements to measure the intensity, severity, and depth of depression on a 4-point scale (0 = *I do not feel sad*; 3 = *I am so sad and unhappy that I can't stand it*). By-participant totals range between 0-63; 0-11 is considered normal, 11-16 indicates mild mood disturbances, 17-20 borderline clinical depression, 21-30 moderate depression, 31-40 severe depression, and 40 and above extreme levels of depression. Pace (1995) reported a high internal consistency (α = 0.92) for the BDI using a sample of American undergraduate students.

Procedure

Participants were tested individually in soundproof lab cubicles on a computer screen. The experiment was created and presented in Experiment Builder and keystroke data were recorded using EyeWrite (Torrance, 2012). Participants were instructed to read a sentence at their own pace. After finishing reading the sentence, participants pressed ENTER and a picture was presented that had to be named

using a headset. Finally, after finishing the naming task, participants had to write the sentence they read before on the computer keyboard. For that purpose, a text box appeared on the screen.

The experiment started with three practice items to familiarize the participant with the task. Each participant saw 6 blocks which each contained 8 trials, rendering a total of 48 trials per participant (24 items and 24 fillers). Trials were presented in a randomized order within and across blocks. Participants were offered short optional breaks after each block. After completion of the experiment, participants were asked to complete the BAI and BDI questionnaires presented in Qualtrics (Qualtrics, Provo, UT). The study took approximately 35 minutes to complete.

Data Analysis

Two dependent variables were operationalized as indicators of sentence-recall difficulty: (1) the number of correction operations (backspaces, deletions) during typing was used as an indicator of writing-process related recall difficulty; (2) the Levenshtein distance was calculated using the *R* package stringdist (Van der Loo, 2014) as a measure of mistakes made in the final recalled sentence. The Levenshtein distance is a frequently used string metric from machine learning that measures the number of single-character edits (i.e., insertions, deletions, or substitutions) that are needed to change one string to the other: in this case, the produced sentence to the previously displayed sentence (Levenshtein, 1966). In other words, the Levenshtein distance indicates the inaccuracy or mistakes made in the recalled sentence that were not edited. The Levenshtein distances provides a gradual measure of inaccuracies with low values indicating minor mistakes, such as typographical errors, and large values indicating more severe mistakes, such as word omissions or substitutions.

Outcome variables (i.e., the number of corrections and the Levenshtein-distance) were modelled through generalized mixed-effects models using the *R* package glmmTMB (Brooks et al., 2017). Models were fitted using a zero-inflated Poisson distribution (Lee & Wagenmakers, 2014). This was important to capture properties of the distribution of both outcome variables: the outcome variables were discrete count data, followed an exponential function, and showed a relatively large number of zero observations. Model predictors were the main effects of subject noun phrase(s) (i.e., conjoined, simple), N2 (i.e., related, unrelated), BAI and BDI scores, and all two-way interactions and three-way interactions of each BDI and BAI with subject noun phrase and N2 name. Continuous predictors were standardized, and categorical predictors were centered (sum-coded) to estimate the effect magnitudes and to reduce collinearity between predictor variables. Centering predictor variables has two advantages over standard treatment contrasts (see Schad et al., 2020): (1) multicollinearity between predictors is reduced; (2) main effects can be interpreted independently of other predictors. Random intercepts were included for participants and items with by-participant and by-item random slope adjustments for subject noun phrase and N2 (Barr et al., 2013; Bates et al., 2015).

All analyses were completed in R. Both data and scripts, in R-markdown format, are available at the OSF (https://osf.io/aemcu/).

Results

Firstly, the BDI and BAI scores were tested for internal consistency. Reliability coefficients were established using McDonald's (Dunn et al., 2014). McDonald's omega is a reliability coefficient, similar to Cronbach's alpha, that takes into account

the strength of association between items (Dunn et al., 2014). High internal consistency was found for both the BAI =0.93, 95% CI [0.9–0.95]) and the BDI = 0.9, 95% CI [0.86–0.93]). By-participant sums were obtained for all items of the BDI (median = 30, *IQR* = 12.25) and the BAI (median = 33.5, *IQR* = 18). Kendall's rank correlation showed evidence for a moderate positive correlation for the BAI and BDI = 0.47, 95% CI [0.34–0.61]).

A descriptive summary of the number of correction operations during writing and the number of mistakes in the final sentence can be found in Table 1.

Table 1

Descriptive Overview of the Number of Correction Operations and Recall Mistakes (Measured as Levenshtein Distance Between Target Sentence and Recalled Sentence)

		Correction operations				Recall mistakes				
Subject phrase	N2	*M*	*SD*	*IQR*	*Max*	*M*	*SD*	*IQR*	Max	*N*
conjoined	related	3.96	3.96	5	22	2.6	5.51	2	37	383
conjoined	unrelated	4.81	4.81	5	38	4.88	8.37	6	48	384
simple	related	3.67	3.67	4	23	2.01	4.33	2	35	384
simple	unrelated	4.61	4.61	5	29	4.41	7.61	5	40	383

Note. M = mean; SD = standard deviation; IQR = interquartile range; N = number of observations

Results are summarized in Table 2. A main effect of BAI on the correction rate ($p = 0.015$) was found with a ratio smaller than 1; this effect indicates that overall, individuals with a higher anxiety score showed less text editing while recalling the target sentence. For the recall accuracy, results showed a main effect of N2 ($p = 0.006$) depicting fewer mistakes in the recalled sentence when the picture used in the secondary naming task was related to the noun in the second position of the target sentence. Further, for the correction rate, a two-way interaction was observed between subject noun phrase and N2 ($p < 0.001$). Pairwise comparisons with Tukey's correction showed a lower correction rate for nouns related to the naming task than for unrelated nouns, but only when the sentence started with a simple phrase (= 0.78, 95% CI [0.65 - 0.92], $p = 0.004$), not when the sentence started with a conjoined subject noun phrase (= 0.99, 95% CI [0.84 - 1.17], $p = 0.91$).

Importantly, three statistically significant three-way interactions were found: the factors subject noun phrase and N2 affected (1) the correction rate as a function of anxiety scores ($p = 0.006$), and both the recall mistakes as a function of (2) anxiety scores ($p < 0.001$) and (3) depression scores ($p < 0.001$).

Table 2

Results of the Generalized Linear Mixed-Effects Model Analysis for Correction Rate (Number of Correction Operations) and Recall Accuracy (Difference Between Produced Sentence and Target Sentence)

	Correction operations			Recall mistakes		
Predictors	Rate ratio($\hat{\beta}$)	95% CI	*p-value*	Rate ratio($\hat{\beta}$)	95% CI	*p-value*
Main effects						
Subject phrase	1.05	0.84 – 1.27	0.639	1.02	0.76 – 1.29	.853
N2	0.88	0.74 – 1.01	0.096	0.65	.45 – .85	**0.006**
BAI (anxiety)	0.82	0.69 – 0.95	**0.015**	1.00	0.8 – 1.21	0.994
BDI (depression)	1.05	0.88 – 1.21	0.563	1.01	0.82 – 1.21	0.885
Two-way interactions						
Phrase x N2	1.28	1.1 – 1.45	**<0.001**	0.90	0.76 – 1.03	0.164
Phrase x BAI	1.06	0.87 – 1.25	0.516	0.89	0.66 - 1.12	0.37
Phrase x BDI	0.97	0.8 – 1.14	0.741	1.12	0.85 – 1.39	.35
N2 x BAI	0.97	0.81 – 1.13	0.679	1.02	0.72 – 1.31	0.911
N2 x BDI	1.00	0.83 – 1.17	0.991	0.91	0.66 – 1.16	0.503
Three-way interactions						
Phrase x N2 x BAI	1.30	1.06 – 1.55	**0.006**	0.60	0.48 – 0.73	**<0.001**
Phrase x N2 x BDI	0.95	0.77 – 1.12	0.561	1.65	1.34 – 1.94	**<0.001**

Note. Subject phrase was dummy coded to render the additional difficulty for conjoined phrases as opposed to simple phrases; N2 was dummy coded to render the advantage for related pictures as opposed to unrelated pictures seen in the secondary task. Results are presented for all main effects and interactions. Presented values indicate the rate-ratio (i.e., change in the outcome variable) where a value of 1 indicates the absence of change, values smaller than 1 indicate a reduced change, and values larger than 1 indicate a positive change.

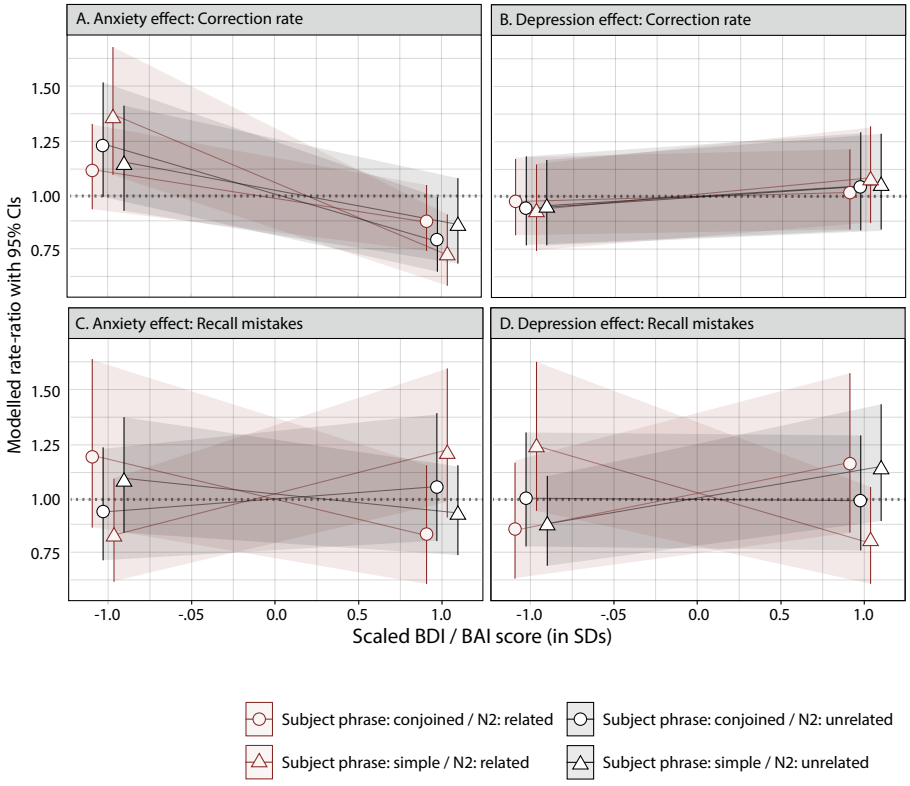
Overall, these interactions indicate that the impact of anxiety and depression on sentence recall depends on the linguistic configuration of the target sentence, in particular the ease of recalling the noun in the second position and whether it was part of the subject phrase of the sentence or not. This was found for both the recall process (editing) and the product (mistakes) in anxiety measures; however, for depression this did not relate to editing throughout the recall process but only affected the number of mistakes in the final text. These results are illustrated in Figure 2 for the modelled correction rate and the recall mistakes. Shown are the rate-ratio changes (y-axis) on BAI and BDI scores (x-axis) displayed for each condition separately. The rate-ratio indicates how many

times a score on the outcome variable is larger for individuals with higher BDI / BAI scores. For example, a rate ratio of 1 indicates no change and a rate ratio of 2 indicates that for every increase of 1 on the BDI / BAI scale, the outcome score is two times larger, indicating a positive effect. Scores lower than 1 indicate a reduced effect.

Three-way interactions were inspected in nested contrasts with Tukey's correction for multiple comparisons. Nested differences will be addressed in the order shown in Figure 2. First, as visualized in Figure 2A, participants with anxiety scores of 1 SD below sample average showed significantly more editing throughout recall than participants with anxiety scores of 1 SD above sample average, but only for target sentences that started with a simple noun and matched the picture name used in the secondary naming task (= 1.85, 95% CI [1.2 - 2.86], p = 0.01); all other conditions were non-significant (p > 0.05). In other words, participants with higher anxiety levels showed a tendency to refrain from editing during recall; this resulted in more editing when the sentence started with a conjoined phrase compared to a simple phrase in which the second noun matched the picture seen in the naming task (= 1.44, 95% CI [1.05 - 1.96], p = 0.022) with no difference for unrelated picture (p > 0.05) and less editing when the sentence started with a simple phrase and included a noun that was related to the picture name compared to an unrelated picture name (= 0.66, 95% CI [0.5 -

Figure 2

Modelled Rate-Ratio Changes for Number of Corrections and Mistake-Rate in the Recalled Sentence (Measured as Levenshtein Distance)



Note. Effects are shown for the scaled BAI (anxiety) and BDI (depression) scores. Ribbons show 95% CIs.

0.86], $p = 0.003$) and no difference for sentences that started with a conjoined phrase ($p > 0.05$). It was observed that anxiety interacted with linguistic factors, impacting the frequency of editing during the recall process. This effect was absent for depression, as shown in Figure 2B, which suggests that depression did not impact how participants recall sentences.

Anxiety levels, but not depression levels, impacted editing behavior. Higher scores on both trait scales, however, changed the number of mistakes made in the recalled sentence (i.e., mistakes that were not edited). For participants with anxiety scores of 1 SD above sample average, fewer mistakes were found for sentences in which the N2 noun related to the picture seen in the naming task compared to unrelated pictures for sentences starting with a conjoined phrase (= 0.49, 95% CI [0.31 - 0.77], $p = 0.002$); the same effect was observed for participants with an anxiety score of 1 SD below sample average for sentences starting with a simple phrase (= 0.53, 95% CI [0.34 - 0.82], $p = 0.004$). No other contrasts were statistically significant. This effect is shown in Figure 2C. Figure 2D shows that this pattern observed for anxiety was reversed for depression scores: participants with depression scores that were 1 SD below sample average showed fewer mistakes after seeing a picture related to N2 for sentences that started with a conjoined phrase (= 0.53, 95% CI [0.34 - 0.81], $p = 0.004$), while participants with depression scores of 1 SD above sample average showed the same effect when the sentence started with a simple phrase (= 0.49, 95% CI [0.32 - 0.75], $p = 0.001$). No other contrasts were statistically significant.

This last result suggests that recall involved some linguistic grouping of the first phrase in the sentence and the second/last phrase; whether or not this grouping facilitated recall differed across participants with anxiety and depression. Participants with higher anxiety scores in particular benefitted from lexical match only when the facilitated noun was part of the subject (e.g., "chair" in *Tania and the chair moved...*), but not when the facilitated noun was in the last phrase of the sentence (e.g., ... *above the chair and the donkey*). This was the reverse for depression: participants with higher depression scores benefitted from lexical match when N2 was in the last phrase of the target sentence but not when it was in the first phrase.

Discussion

The present study aimed to examine the effects of anxiety and depression on people's ability to recall a sentence. It was hypothesized that both disorders would differently impact language processing. Anxiety was hypothesized to impact people's ability to comprehend sentences and thus impact the accuracy of the recalled sentence. However, no difficulty during the recall process (i.e., during writing) was anticipated. For depression, problems with the execution of writing were predicted. Specifically, this issue originates from difficulty with language encoding rather than a poorly decoded sentence.

The study found that individuals with increased levels of anxiety showed a lower correction rate but there was no evidence of an inhibited ability to recall the sentence. This, however, was observed for individuals with increased levels of depression who displayed a reduced ability to recall the stimulus correctly but conveyed no evidence of difficulty during the production process. The current findings are in line with the hypothesis that anxiety and depression differently impact language processing. However, the effect of each disorder on language processing contrasts with the existing

literature. These conflicting results will be addressed in the remainder of the discussion followed by possible explanations of how, based on the current findings, anxiety and depression impact language processing.

Results showed that depression decreased recall accuracy but did not affect the writing process. This is in contrast with existing research that predicts execution errors but not necessarily a reduced sentence recall (Abas et al., 1990; De Lissynder et al., 2010; Mundt et al., 2007). Production errors were predicted based on deficits resulting from an impairment (Austin et al., 2001) or a limitation of cognitive resources (Cohen et al., 2014). The results of the current study show that this is not the case for sentence recall. More text editing or links to the lexical and syntactic manipulation were not observed for individuals with higher levels of depression. The reduced recall accuracy observed for individuals with higher levels of depression can be attributed to a limited working-memory capacity (Rose & Ehmeier, 2006). This is supported by the lower accuracy when the secondary naming task involved the naming of a picture depicting one of the sentence items. The overlap in meaning may have caused similarity-based interference in verbal working memory (e.g., Oberauer & Lange, 2008) and thus reduced sentence-recall accuracy. In other words, when participants were asked to name a picture that shows an item used in the target sentence, the similarity of their names reduced the memory trace of the sentence item, causing difficulty during memory retrieval. As lower recall was observed only when the critical sentence item was part of the first syntactic phrase, trace decay over time may have had a combined effect with memory interference. However, it must be noted that these explanations are merely post-hoc. Explanations discussed could be considered in future research to clarify the impact of depression on sentence recall or, alternatively, memory. Memory impairments have often been reported by individuals suffering from symptoms of depression and have in fact impaired participants' recall ability in previous experiments (Schweizer et al., 2018).

There are two differences that might explain the contrast between the current results and existing research (e.g., De Lissynder et al., 2010; Mundt et al., 2007). First, participants in the present study did not have to create sentences on a semantic level but rather had to buffer meaning in memory. In other words, the task is taxing on memory because sentences had to be encoded from a conceptual representation of meaning but did not involve the generation of meaning. Second, writing execution deficits in depression might only arise in extreme levels of depression (Vilgis et al., 2015). Vilgis et al. (2015) emphasized that differences in neuropsychological functioning depend on the severity of experienced depression, although the authors also highlighted a lack of consistency within related research. The present sample did not show extreme levels of depression, which might explain the absence of effects on writing execution. An interesting avenue for future research might be to directly test whether the inhibition of the production execution process in individuals with depression is based on difficulty to create meaning in combination with memory limitations. This may provide useful insight into the effects of mental health disorders on language processing and its interaction with memory retrieval, particularly among individuals with clinical levels of depression.

There was no evidence found to suggest that individuals with anxiety show a reduced sentence comprehension ability. This would have been

reflected in a lower recall accuracy. Instead, there was an unexpected observation that higher anxiety levels resulted in less editing during sentence recall with no impact on the accuracy of the recalled sentence. Little is currently known about the effects of anxiety on language processing; therefore, any post-hoc explanation needs to be taken cautiously. At least for simple stimulus sentences, the present finding conflicts with the idea that high levels of anxiety lead to a superficial comprehension (Wilson et al., 2006). A possible explanation for this recall advantage is that anxiety disorders can result in high alertness (Pacheco-Unguetti et al., 2009). Higher levels of anxiety may therefore increase attention to the decoding of the stimulus sentence, memory rehearsal, and a more careful writing execution.

In contrast, individuals with lower levels of anxiety showed more text editing depending on the linguistic manipulation. More editing was found for sentences in which the picture naming task involved a name similar to the second item in the target sentence when that item was not part of the subject phrase. A possible explanation for this finding is the following: the recall process seems to be subject to memory interference depending on the syntactic position of the item. As text editing was highest for sentences in which the critical item was not part of the subject phrase, one explanation is that memory interference was strongest when lexical recall had to happen in parallel with writing execution rather than prior to writing onset (see Martin et al., 2014; Roeser et al., 2019; Swets et al., 2014).

An alternative explanation for the increased number of mistakes presented among participants exhibiting higher levels of depression and lower levels of anxiety could be a motivational impairment. Various research studies convey an association between depression and core deficits in motivation (Cléry-Melin et al., 2011; Scheurich et al., 2008; Moritz et al., 2017). Anxiety has been known to impair processing abilities but not necessarily performance (Eysenck, 1979). Nevertheless, if motivation was an influencing factor in the present study, then a larger number of mistakes overall would be displayed. This was not the case. Although a larger number of mistakes associated with both anxiety and depression were found, this was dependent on the syntactic configuration of the target sentence (i.e., whether the conjoined phrase was sentence-initial or final) and was reversed across individuals with higher levels of anxiety and depression. Due to these differences, the present findings cannot be explained on grounds of motivation alone.

There is one important limitation to the current results. Different effects for anxiety and depression on language processing were hypothesized, and these effects have been discussed independently. However, these two mental health disorders show stark comorbidity (Hirschfield, 2001; Moffitt et al., 2007), share overlapping symptoms (APA, 2013), and have a similar psychopathology (Zbozinek et al., 2012). Therefore, it is difficult to draw conclusions about anxiety or depression that are not influenced by the other. The results of this study support the idea that anxiety and depression have different effects on language processing and can, to some extent, be considered independently. Future research focusing on the effects of anxiety and depression on cognitive domains may want to categorize their sample into individuals with anxiety, individuals with depression, and individuals with a high extent of comorbidity. This might help to distinguish between effects that are more general in nature and effects that are

specific to either anxiety or depression. It was outside the scope of the current study to distinguish comorbidity samples.

Another possible limitation to the current study is the use of the BAI and BDI psychometric tools. Both have received criticism over the years due to the measurement of overlapping symptoms (Lovibond & Lovibond, 1995; Muntingh et al., 2011; Richter et al., 1998; Ruscio & Ruscio, 2002). However, the high levels of comorbidity between anxiety and depression, as reported in the introduction, are well established (Airaksinen et al., 2004; Hirschfield, 2001; Moffitt et al., 2007). Therefore, tools measuring anxiety and depression levels may generally experience difficulty in distinguishing symptoms that are present across both disorders, such as concentration and processing impairments (Eysenck & Fajkowska, 2018; Zbozinek et al., 2012). While this is a notable limitation to the BAI and BDI tools, this did not impact the present findings as different outcomes were discovered for anxiety and depression overall. Each disorder had distinct effects on sentence recall in the current study. Model diagnostics, particularly the variance inflation factor, also showed that neither the BAI or BDI were subject to multi-collinearity violations. Nonetheless, this study did not group participants separately according to the presence of anxiety and/or depression, as scores were alternatively considered as continuous. Future research may benefit from researching anxiety and depression as separate entities and from addressing different populations. Because the present study involved undergraduate students, the findings may not be applicable to other populations such as children or seniors. Future research will determine the cognitive mechanism that underlies these findings and is impacted by mental health factors, while contributing towards knowledge on the impact of mental health on cognitive functions and in particular, linguistic processes.

The present findings suggest that anxiety and depression impact linguistic factors involved in sentence recall in different ways. This is important because this finding has real-world implications for how individuals with mental health problems should be supported in, for example, educational and professional contexts. Particularly situations that they would otherwise be systematically disadvantaged in, such as exams, presentations, job interviews and even social fulfilment. Developing an understanding of how anxiety and depression impact linguistic processes is an important step to support these individuals in contexts where comprehending and recalling language is fundamentally important. Accordingly, modifications and adjustments could be tailored to this population to improve outcomes across various contexts, thereby enhancing life satisfaction. Enhanced awareness of the potential linguistic struggles that individuals with anxiety and depression face could be addressed using practical adjustments in order to reduce stress and improve outcomes for such individuals, e.g., extra time in exams or altering interview conditions and questions.

Conclusion

The present study explored the impact of anxiety and depression on people's ability to process language. Results show that anxiety and depression affect language processing in different ways. Higher levels of depression impaired recall accuracy, but higher anxiety levels did not. Existing literature suggests that anxiety impacts language comprehension while depression influences language production. In contrast with this view, the present findings show that production was only

impaired by lower levels of anxiety. The accuracy of the sentence recalled, however, was a function of anxiety levels, depression levels, and the linguistic properties of a sentence. The reduced recall accuracy in individuals with higher levels of depression was attributed to working-memory limitations. As for individuals with higher levels of anxiety, reduced text editing during writing may be due to increased attention to the stimulus resulting from higher levels of alertness.

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The Worse Are Well, and the Well Are Worse: Emotion Regulation Difficulties and Their Relationship with Psychological Functioning During COVID-19

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Abstract

The COVID-19 pandemic introduced a myriad of mental health consequences; however, it remains largely unknown why specific individuals may be more vulnerable to increases in psychological distress than others. Individuals' capacity for emotion regulation may indicate how one experiences distress during COVID-19. The present study aims to understand which specific emotion regulation skills most impact the psychological functioning of psychotherapy clients during COVID-19. Psychotherapy clients ($N = 33$) completed the Brief Difficulty in Emotion Regulation Scale (DERS-18), to measure six domains of emotion dysregulation, and the Outcome Questionnaire 30 (OQ-30.2) to measure psychological functioning during treatment. Multilevel Modeling (MLM) was conducted in R to measure the incremental effect of each DERS sub-scale on pre-and post-COVID-19 psychological functioning. As expected, clients with difficulties in emotional awareness and goal-directed behavior experienced worse psychological functioning during COVID-19. However, clients with difficulties in emotional self-efficacy, impulse control, and acceptance of negative emotions surprisingly reported deterioration of psychological functioning to a *lesser* degree than their counterparts with better regulation skills. Coping during a crisis is not uniform. Clinicians must understand how different emotion regulation skills play a role in navigating distress. Further, this evidence sheds light on how those who appear to be "doing worse" may have more tools to cope with circumstances out of their control.

Keywords: Emotion regulation, COVID-19, psychological distress, psychotherapy, telehealth

The COVID-19 global pandemic socially, economically, and psychologically altered many facets of our society. These changes led to global reports of higher levels of loneliness, depression, anxiety, suicide ideation, substance abuse, and overall deteriorating psychological wellness (Cao et al., 2020; Cucinotta & Vanelli, 2020; Czeisler et al., 2020; Hamm et al., 2020; Inchausti et al., 2020; Odriozola-González et al., 2020; Sood, 2020). Emerging studies showed that the continued support of psychotherapy via telehealth played a positive role in mitigating such psychological deterioration when in-person therapy was unavailable (Inchausti et al., 2020; Silver et al., 2020; Zhou et al., 2020). Similarly, individuals' strong emotion regulation skills, the process of maintaining and modifying one's emotional experience, is a predictor of wellness during peak COVID-19 changes (Panayiotou et al., 2021; Prout et al., 2020). Approach-oriented emotion regulation strategies are shown to be more effective in managing negative emotions during the pandemic than avoidance-based strategies (Restubog et al., 2020). As such, each emotion regulation skill may impact the quality of life during lockdown differently (Panayiotou et al., 2021; Prout et al., 2020). Given that overall emotion regulation mediates treatment outcomes, therapists may improve treatment during global crises by applying a detailed emotion regulation framework (Gratz et al., 2015).

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Psychological functioning broadly refers to an individual's interpersonal functioning, quality of life, and symptom severity and can be measured at several stages of treatment to evaluate its effectiveness (Beckstead et al., 2003). The impact of global health crises on individuals' psychological functioning is articulated in the literature (Bults et al., 2011; Maunder et al., 2003; Shultz et al., 2016). However, existing studies neither address the impact of specific emotion regulation skills nor analyze a treatment-seeking population who transitioned to telehealth during COVID-19. The purpose of this study is to understand which specific emotion regulation difficulties most impact psychological functioning, specifically for psychotherapy clients who transitioned to telehealth during the COVID-19 lockdown.

Mental Health and COVID-19

Adjusting to life during COVID-19 introduced uncertainty, medical risks, racial discrimination, personal losses, financial losses, and isolation—all of which undoubtedly contributed to widespread emotional distress and increased risk for psychopathology. Loneliness, domestic violence, substance abuse, depression, anxiety, and higher suicide risk increased during the first two years of the COVID-19 pandemic in several countries (Czeisler et al., 2020; Inchausti et al., 2020; Rambaran, 2020; Sood, 2020). This is apparent in the way individuals communicate to peers and family, too; overall, levels of communication about negative emotions increased, while positive emotions and life satisfaction decreased (Li et al., 2020, Wang et al., 2020). Further, those with psychiatric diagnoses may be emotionally impacted by COVID-19 to a greater extent than those without such conditions (Fernandez-Aranda et al., 2020; Yao et al., 2020). The broad range of individual outcomes points to the importance of exploring new ways to understand and mitigate psychological distress.

Individual Responses to Distress

Understanding how people cope during adverse experiences informs clinical work and may illuminate variations in psychopathology. The unprecedented nature of the COVID-19 pandemic unveiled a variety of individual coping strategies for managing anxiety around health and safety (Sood, 2020). However, research indicates that coping during a crisis is not uniform; individuals' health and lived experiences may inform their response. Observing how individuals manage during a disaster offers researchers an opportunity to understand how people may even thrive in adverse conditions. The effects of separation of families in London during World War II during the Blitz varied in different children; disparities in children's outcomes showed that some children were affected by the separation while others were not (Rao, 2020). There are implications that some people are predisposed to withstand the effects of a crisis while others may suffer.

While studies (Fullana et al., 2020; Tuason et al., 2021) include statistics on coping strategies and emotion regulation in normative populations, the current study focuses on emotion regulation in a clinical sample. For example, during the pandemic, older adults with pre-existing major depressive disorder (MDD) were coping better than expected, with no overall increase in clinical depression, anxiety, or suicidal thoughts (Hamm et al., 2020). These results indicate that not all individuals will suffer harmful effects while weathering a crisis. This is an important consideration when understanding that clinical interventions should consider variations in emotion regulation, particularly in a time of global turmoil

and uncertainty.

Mixed Impact of Telehealth

The pandemic posed tremendous challenges to the mental health field with an abrupt shift to telehealth methods, forcing many therapists to adapt to a new modality of work to provide psychological relief to those in need (Bekes & Doorn, 2020; Miu et al., 2020; Silver et al., 2020). This introduced various challenges, including privacy and space concerns, technology issues, data security, and accessibility (Bierbooms et al., 2020; Jurcik et al., 2020).

Nonetheless, the field demonstrated flexibility, creativity, and responsiveness, presenting novel opportunities beyond in-person therapy, especially for clients who may find in-person therapy inhibiting or anxiety-provoking (Silver et al., 2020). Finally, some therapists report higher engagement and utility of psychotherapy during times of increased social isolation because of patients' desire to maintain a connection (Miu et al., 2020; Silver et al., 2020).

Given the importance of telehealth, mixed reports of effectiveness necessitate additional research to understand how psychotherapy clients are faring during COVID-19 and whether telehealth ameliorates psychological distress. Similar to responses in distress overall, there may be personal reasons that some adapt better to telehealth while others do not; these nuances must be examined. Emotion regulation and dysregulation and its relationship to psychotherapy treatment outcomes may predict a client's psychological functioning during COVID-19 and, inherently, their response to telehealth (Gross & Munoz, 1995; Gratz et al., 2015; Keltner & Kring, 1998).

Emotion Regulation and Distress

The constructs of emotion regulation and dysregulation are increasingly used to explain many forms of psychological disorders and maladaptive behaviors (Gross & Munoz, 1995; Keltner & Kring, 1998). William James (1884) defines emotions as adaptive behavioral and physiological response tendencies that surface in evolutionarily significant situations. Emotion *regulation* describes the processes by which an individual influences which emotions they have, when they have them, and how they experience and express these emotions (Gross, 1998). Gross (1998) also takes a process model view of emotion regulation, indicating that emotion can be regulated at five points in one's emotional process: (a) selection of a situation, (b) modification of the situation, (c) deployment of attention, (d) change of cognitions, and (e) modulation of responses. Psychoanalytic frameworks (Freud, 1959) and stress and coping traditions (Selye, 1956) emphasize the urge to minimize anxiety and negative emotion, and thus both traditions inform contemporary theories of emotion regulation.

Despite its clinical significance, the field has not entirely reached a consensus on the best way to define and measure emotion dysregulation, and our understanding of it continues to evolve (Gratz et al., 2015). Historically, measures of emotion dysregulation focused exclusively on a single population (e.g., examining adolescents solely) or a single aspect of emotion dysregulation (Weinberg & Klonsky, 2009). Some definitions of emotion regulation focus on the control of emotions and expressions and the reduction of emotional arousal (Garner & Spears, 2000; Kopp, 1989), while others focus on the functional nature of emotions or lack thereof (Cole et al., 1994). To account for this, Gratz and Roemer (2004) developed and validated the first measure of *several* clinically relevant difficulties in emotion regulation, the Difficulties in

Emotion Regulation Scale (DERS). DERS builds on theoretical work in emotion dysregulation in Borderline Personality Disorder (BPD; Linehan, 1993) and prior existing measures for emotion dysregulation such as the Negative Mood Regulation Scale (Catanzaro & Mearns, 1990). However, it also expands the phenomena, recognizing that emotion regulation extends beyond the minimization of negative emotion and includes aspects of awareness, acceptance, and goal-directed behavior capacity, for example (Gratz & Roemer, 2004). Since then, a shortened version of this scale (DERS-18) was created to improve its efficiency while still measuring the original six domains of emotion dysregulation (Victor & Klonsky, 2016). This measure was selected for use in the present study for its brevity, given that it was administered in conjunction with other measures at each clients' intake.

Emotion Dysregulation and Psychotherapy

Given that the ability to experience, label, and regulate emotions is crucial for psychological functioning (Kubzansky et al., 2011), emotion regulation is a key focus area during psychotherapy treatment and managing distress (Grecucci et al., 2017). As such, several emotion-focused approaches to psychotherapy emerged in recent years to treat a broad range of psychopathologies, including, but not limited to, Dialectical Behavior Therapy (DBT; Linehan, 1993), Emotion Regulation Therapy (ERT; Renna et al., 2017), Schema Therapy (Fassbinder et al., 2016), Skills Training in Affect and Interpersonal Regulation (STAIR), and Acceptance and Commitment Therapy (ACT; Gloster et al., 2020). Even treatments that do not specifically target emotion regulation may still positively impact emotion regulation (Gratz et al., 2015). Furthermore, improvements in emotion regulation skills and diminishing difficulties in emotion regulation mediate symptom improvement in eating disorders, substance use disorders, BPD, deliberate self-harm, and depression (Gratz et al., 2015). However, little research connects intervention outcomes with changes in specific dimensions of emotion regulation (Gratz et al., 2015).

Emotion Dysregulation and COVID-19

While the sweeping negative mental health consequences of COVID-19 are undisputed, our understanding of why psychological responses to COVID-19 are not uniform remains nascent. Many studies looked at the differences in responses between demographics, exploring dynamics between extroverts and introverts, males and females, and older and younger participants (Sonderskov et al., 2020). Studies also examine an individual's circumstances, revealing that those with a more significant duration of confinement, difficulty securing medical care, and greatest financial losses experienced the greatest psychological impact (Brooks et al., 2020; Pfefferbaum & North, 2020). However, few studies examine a skills-based model of conceptualizing the distress and the potential factors that protect against such despair.

An individual's emotional competence may predict distress levels during COVID-19 and shed light on opportunities for psychotherapy interventions (Park et al., 2021; Restubog et al., 2020). Velotti and colleagues (2020) found that emotion overall emotion dysregulation partially mediated the longitudinal relationship between loneliness and depression in response to COVID-19. This points to the therapeutic demand to support the regulation of negative emotional states and understand emotion regulation in a more nuanced way. Regarding specific emotion regulation skills and

positive life outcomes, one recent study on college students found that the ability to describe one's emotions and access emotion regulation strategies was the most significant predictor of quality of life maintenance during COVID-19 (Panayiotou et al., 2020). Paradoxically, the same study also found that difficulty identifying and describing emotions (alexithymia) was related to a better quality of life during COVID-19, likely because it may prevent complete encoding of the negative experience.

That said, there are several discrete domains of emotion regulation difficulties (e.g., low emotional awareness), and no known study to date addresses each of the specific domains of emotion dysregulation to understand the impact of COVID-19. Further, no study to date explores the relationship between emotion regulation and COVID-19 quality of life within a psychotherapy treatment-seeking population.

The Present Study

The purpose of the present study is to examine which specific emotion regulation skills most impact the psychological functioning of psychotherapy clients during COVID-19. Psychological functioning and emotion dysregulation were measured at several psychotherapies stages at the Safran Center for Psychological Services at The New School. Each client in the present sample was classified as having as high or levels of each emotion regulation difficulty; there are a total of six. Using a multi-level linear model, psychological functioning was compared pre-and post-COVID-19 lockdown between the high and low groups.

The authors hypothesized that 1) psychological functioning will worsen to a significantly greater extent for clients with less overall emotion regulation difficulties than for those with more emotion regulation difficulties, and 2) each individual emotion regulation will have a different impact on psychological functioning in response to the pandemic; difficulties in emotional awareness, goal-directed behaviors, and self-efficacy will be the most significant of psychological worsening. The implications of this study will illuminate ways that clinicians may hone into specific emotion regulation skills in helping clients navigate distress caused by global crisis and individual's situational traumas.

Methods

Study Design

An exploratory post-hoc analysis of psychotherapy clients was conducted using data collected from psychological intervention provided at The Safran Center for Psychological Services. The Safran Center offers low-fee brief psychotherapy intervention for various presenting problems using both psychodynamic and cognitive-behavioral approaches and a social justice framework. Therapists in the center are first-year clinical psychology doctoral students, and all services are provided in the context of clinical training. An exploratory posthoc analysis of psychotherapy clients was conducted using data collected from psychological intervention provided at The Safran Center for Psychological Services.

Participants

Participants (N = 33; female = 22, male = 10, gender non-conforming = 1) include individuals who began seeking psychological treatment at The Safran Center for Psychological Services. Participants were recruited for psychological services via word of mouth and social media. Ages ranged from 20 to 48 (M = 30.2, SD = 6.41). 64% of the sample identified as Caucasian/White, 18% as Asian or Pacific Islander, and 6% as black or

African American; the remainder declined to identify their race and ethnicity. 66% of clients identified as straight/ heterosexual, 12% as bisexual, 12% as gay/homosexual, and 10% as generally queer. Additional demographic information of client and therapist is listed in Tables 1 and 2 in the Appendix.

Procedures

Data was extracted from the Safran Center Data repository, where all clients obtained informed consent to participate in the data repository and approved research studies. All clients underwent a thorough intake process including the completion of a demographic form and multiple assessment measures, including the Brief Difficulties in Emotion Regulation Scale (DERS-18). Each client then filled out the Outcomes Questionnaire (OQ-30.2) online before therapy sessions to assess their psychological functioning level to generate a report for the clinician, providing information regarding their client's progress relative to previous sessions. After the March 2020 stay-at-home orders in New York, therapy sessions were transitioned to a HIPPA-compliant zoom online. Clients continued to fill out the OQ-30.2 questionnaire before virtual therapy sessions.

Measures

Brief Difficulty in Emotion Regulation Scale

The (DERS-18; Victor & Klonsky, 2016), an 18-item self-report measure developed to facilitate understanding of how emotion dysregulation is associated with psychiatric symptoms, other emotion-related constructs, and treatment progress. This scale is a shortened modification of the original Difficulties with Emotion Regulation Scale (DERS; Gratz and Roemer, 2004). It consisted of 36 questions and was adapted to increase its utility and reduce the participant burden. The DERS-18 demonstrates excellent reliability and validity despite half the questions and performs similarly to the original DERS measure. DERS also demonstrates validity across wide-ranging cultural contexts. The self-report questionnaire measures six emotion regulation difficulties: unwillingness to accept one's distress or negative emotional responses (Nonacceptance), difficulty engaging in goal-directed behavior and tasks when experiencing negative emotions (Goals), lack of control of one's behavior when experiencing negative emotions (Impulse), lack of awareness and acknowledgment of emotions (Awareness), limited access or awareness of strategies to help regulate emotions effectively (Strategies), and difficulty knowing and labeling what emotions are being experienced (Clarity).

Outcome Questionnaire 30.2

(OQ-30.2; Ellsworth et al., 2006), a shortened version of the OQ-45.2 (Outcome Questionnaire; Lambert et al., 2004), which is widely used to measure patient progress during therapy interventions and is designed to be repeatedly administered during treatment and termination. Patient progress is measured within three core components of the patient's life: 1) subjective discomfort (intrapsychic functioning), 2) interpersonal relationships, and 3) social role performance. Each of these areas of functioning is measured along a continuum and captures how the patient feels inside, how well they get along with significant others, and how they manage important life tasks (e.g., work, school). Each item is scored on a five-point scale (0 to 4), with the total score yielding a range of possible scores of 0 to 120; changes in scores more significant than ten are estimated to be reliable. High scores are indicative of high symptom severity. The OQ-30.2 is,

however, not intended for patient diagnoses. The shortened version is sensitive to change over short periods and is designed to be brief while simultaneously maintaining high levels of reliability and validity. The results of the OQ-30.2 are used to measure patient functioning against their baseline functioning, as well as general population functioning.

Analysis Plan

Multilevel modeling (MLM) was conducted in R (base; version 4.02) using the "lme4" package (Bates et al., 2015) as the data had a clear hierarchical structure (Tabachnick & Fidell, 2013). The "lmerTest" package (Kuznetsova et al., 2017) was used to calculate confidence intervals and significance values via Satterthwaite's method (Giesbrech & Burns, 1985). Two-level linear mixed-effects models were built with assessment time-points (Session; Level 1[*ij*]), nested within participants (Level 2 [*i*]). Restricted maximum likelihood (REML) estimation was used over complete information maximum likelihood (FIML) estimation as the number of Level 2 clusters was relatively small such that FIML estimations would be more susceptible to error bias (Hox & McNeish, 2020). Participants were not nested within therapist as cluster sizes were too small (one to two patients assigned to each therapist). Seven models were built to test each hypothesis. Session (coded 0 to 33) and pre-/post-Covid-19 (C19; coded as -0.5 and 0.5, respectively) were included in the model as Level 1 predictors, while subscale scores (grand mean-centered) were included as a Level 2 predictor. A cross-level interaction between C19 and subscale score was also included in each model. Additional Level 2 predictors (e.g., age, gender, or therapist) could not be included in the models due to a lack of statistical power resulting from a small sample size. The analysis is focused on the difference in psychological functioning changes (OQ-30.2) between the two high and low groups, not the extent of the change for any given individual. Model assumptions and the influence of outliers were assessed by examining plots of residuals at each level. The models were specified as follows:

$$H_1: \quad OQ \sim Total_j + Session_{ij} + C19_{ij} + Total.C19_{ij}$$

$$H_2: \quad OQ \sim Awareness_j + Session_{ij} + C19_{ij} + Awareness.C19_{ij}$$

$$H_3: \quad OQ \sim Clarity_j + Session_{ij} + C19_{ij} + Clarity.C19_{ij}$$

$$H_4: \quad OQ \sim Goals_j + Session_{ij} + C19_{ij} + Goals.C19_{ij}$$

$$H_5: \quad OQ \sim Impulse_j + Session_{ij} + C19_{ij} + Impulse.C19_{ij}$$

$$H_6: \quad OQ \sim Non\text{-}acceptance_j + Session_{ij} + C19_{ij} + Non\text{-}acceptance.C19_{ij}$$

$$H_7: \quad OQ \sim Strategies_j + Session_{ij} + C19_{ij} + Strategies.C19_{ij}$$

Significant interactions were probed using simple slopes analyses (Preacher, Curran, & Bauer, 2006), with high and low DERS total and subscale values being specified at one standard deviation above and below the mean.

Results

Missing Data Analysis

Little's Missing Completely at Random (Little, 1988) test was conducted on all outcome and covariate variables and found to be non-significant, indicating the data were missing completely at random, $\chi^2(1370, N = 33) = 493.172, p = 1.000$.

Main Analyses

Descriptive statistics for each DERS subscale are set out in Table 3. The results of each model are set out in Table 4. There was a significant effect of session (b range = -0.43 to -0.45, $p < .001$) and C19 (b range = 4.74 to 5.34, $p < .001$) in each model, indicating that the severity of patients' psychological symptoms generally reduced as treatment progressed, but that patients' progress diminished after C19 began (see Figure 1). The effect of C19 on symptom severity varied depending on patients' total difficulties regulating emotion. However, contrary to hypotheses, simple slopes analysis revealed that symptom severity for patients with less difficulties in emotion regulation increased following C19, $b = 7.15$, $SE = 1.46$, $p < .001$, while patients who had more significant difficulties in emotion regulation were not significantly affected by C19, $b = 3.00$, $SE = 1.60$, $p = .060$ (see Figure 2).

In terms of the individual DERS subscales, the extent of the effect of C19 on symptom severity did not depend on patients' 'clarity' in identifying emotions (see Figure 3) but did depend on the other five DERS subscales (awareness, goals, strategies, non-acceptance, and impulse). As expected, patients with lower levels of emotional awareness experienced an increase in symptom severity $b = 6.86$, $SE = 1.37$, $p < .001$, on average, when compared to those with higher levels $b = 3.81$, $SE = 1.26$, $p = .002$; see Figure 4. The OQ scores of patients with high emotional awareness increased by almost 4 points after C19, while patients low in emotional awareness increased by almost 7 points. Also as expected, symptom severity for patients who engaged in less goal-directed behavior increased following C19, $b = 6.99$, $SE = 1.35$, $p = < .001$, to a greater extent than for patients reporting greater engagement in goal-directed behaviors, $b = 3.16$, $SE = 1.36$, $p = .020$ (see Figure 5).

In contrast, clients who reported less control over their behaviors actually experienced a lower magnitude of symptom worsening, $b = 3.03$, $SE = 1.37$, $p = .027$), than clients who reported greater control over their behavior, $b = 7.12$, $SE = 1.39$, $p < .001$ (see Figure 6). Also contrary to predictions, patients who were less accepting of negative emotional experiences did not experience a significant worsening in symptom severity after C19, $b = 2.75$, $SE = 1.43$, $p = .055$, while patients reporting a greater willingness to accept negative emotions experienced a significant worsening of symptom severity after C19, $b = 6.99$, $SE = 1.31$, $p < .001$ (see Figure 7). Also unexpectedly, patients reporting lower use of strategies to regulate emotions did not experience a significant worsening in symptom severity after C19, $b = 0.89$, $SE = 1.59$, $p = .576$, while patients with greater use of emotion regulation strategies experienced a significant worsening of symptom severity after C19, $b = 8.59$, $SE = 1.40$, $p < .001$ (see Figure 8).

Discussion

The present research study explores an important gap in the literature on specific emotion regulation

difficulties and how they relate to psychological distress during a global crisis. While the focus of the present paper is COVID-19, future iterations of this work may explore other external problems, including political or economic historical markers. Overall, our preliminary results indicate that COVID-19 had a more significant adverse effect on our sample of patients who generally have *fewer* emotion regulation difficulties. While patients with less emotion dysregulation experienced symptom deterioration equivalent to a 7-point increase in OQ-30.2 score, patients with more dysregulation did not appear to worsen in symptom severity as a result of COVID-19 significantly.

The individual aspects of emotion regulation did not have a uniform or predictable impact on the effect of COVID-19. Results for two of the six emotion regulation domains (goals, awareness) aligned with the authors' hypothesis about awareness and goal-directed behavior protecting against the adverse impact of COVID-19. However, contrary to predictions, four of the six emotion regulation domains (impulse, strategies, non-acceptance, and clarity) align with the earlier finding that more significant emotion regulation difficulties don't necessarily lead to greater distress during COVID-19. For example, patients reporting higher levels of self-efficacy experienced a worsening in their symptom severity, increasing in OQ-30.2 scores by over 8.5 points on average as a result of COVID-19; patients with low self-efficacy did not experience a worsening of symptoms as a result of COVID-19. This specific emotion regulation skill demonstrated the most significant difference between low and high emotion regulation difficulty groups. This may illuminate how higher functioning individuals may experience more substantial disruption by a global health crisis, mainly because it is out of their control. Also, contrary to predictions, patients who were more willing to accept their emotional responses experienced a more significant increase in distress following COVID-19 than patients who were not accepting of their negative emotions. This finding, in particular, aligns with the literature on avoidance strategies protecting one's psychological well-being during times of crisis (Restubog et al., 2020). Finally, patients reporting lower levels of impulsivity experienced a worsening of symptom severity because of COVID-19, more than double that of patients with high levels of impulsivity. Perhaps those who exhibit greater urges to control their behaviors and environments are bound to experience more significant distress under largely uncontrollable circumstances.

So why did some unexpectedly seem to cope better than others, and how do we make sense of this phenomenon? Lei and colleagues (2014) demonstrated that individuals with major depressive disorder (MDD) and overall greater maladaptive emotion regulation skills indicate one strategy more effectively than healthy controls: *acceptance*. During a crisis, pre-existing conditions such as anxiety and depression may fortify a portion of the population by "normalizing" what they experience daily. While in other realms, individuals who experience a sense (or illusion) of control and order find their world distorted and disorganized and struggle to cope with the rapid change.

It, therefore, cannot be assumed that an affected population will exhibit maladaptive behaviors during a catastrophe. A study conducted shortly after the September 11 terrorist attacks captured not only the pervasive depression that ensued, but also the emergence of positive emotions such as gratitude, interest, and love (Fredrickson et al., 2003). Positive emotions were also associated with

resilience in those who thrived after such a tragic event. Individuals may experience gratitude in simple pleasures that may otherwise be taken for granted to regulate affect.

Emotional *flexibility* may become more critical in coping with uncertainty than simply a lack of emotion regulation *difficulty*. Psychological flexibility refers to a person's ability to consciously engage in the present moment and the capacity to adapt or adhere to behavior that promotes chosen values (Bond et al., 2006). Acceptance and Commitment Therapy (ACT) is an evidence-based intervention that incorporates acceptance strategies to increase psychological flexibility. Rather than avoid circumstances that lead to discomfort and potentially exacerbate symptoms of psychopathology, the ACT approach proposes that psychological health involves accepting both positive and negative emotions instead of working against negative experiences, which may lead to maladaptive behavior and worsening symptoms (Blackledge & Hayes, 2001). While we realize that incorporating negative emotions may be difficult for some, ACT is a process that may help an individual accept that adversity is a facet of lived experience and facing distress, rather than avoiding it, may lead to increased mental and physical health. This provides vast new territory for exploration for many clinicians working with patients struggling during COVID-19.

The timing of the study and switch from in-person psychotherapy to telehealth similarly required many clients (and therapists) flexibility. It's possible that this transition exacerbated existing pandemic-related anxieties for those unable to demonstrate such flexibility and provided ease for others. While the shift was beneficial by providing uninterrupted support, many clients still do not prefer telehealth. Qualitative inquiry is required to examine the subjective experience of the transition to telehealth. A recent qualitative study showed that only 3 of 20 participants would elect to receive telehealth for psychological services in the future (Venville et al., 2021). Additionally, each therapist may have navigated this transition differently, further impacting the present study results for each client. Further analysis may examine additional details around the transition to telehealth, including treatment modality, level of client engagement, and therapeutic alliance throughout the peak of the crisis.

Limitations and Future Research

While this study explored the emotion sub-skills and their relationship to distress during COVID-19, future research must address the adversity faced by psychotherapy clients in the context of other crises and global issues. Future research may also further explore sub-samples of psychotherapy clients. Importantly, this study examined a small sample of clients and would benefit from a larger, more diverse sample with more males and individuals of an ethnic and sexual minority. A more significant number of participants would also enable future studies to control for demographic covariates (e.g., age and gender) and whether participants had a diagnosable mental health disorder. The *post hoc* exploratory design of the present study prevented the collection and analysis of data relating to other potential mediators or moderators of the effect of COVID-19 on patient symptom progression. These modifications would allow the current findings to translate across more demographically balanced groups and periods.

Separately, given that much of the findings tie back to a theme of control, future researchers have much opportunity to explore this concept further using

instruments and measures that capture the degree to which the illusion of control indeed plays a role in psychological functioning during the crisis. Further, developing a detailed understanding of individual disorders in which emotion regulation is particularly impacted may also fruit future findings. Additionally, comparing the client population by type of psychotherapy (e.g., psychodynamic, CBT, etc.) may be insightful to assess the effectiveness of such therapies on emotion regulation and psychological functioning.

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Appendix

Table 1

Sociodemographic Characteristics of Clients

	n	*%*
Gender		
Female	22	66.7%
Male	10	30.3%
Gender Non-Conforming	1	3.0%
Sexual Orientation		
Straight/Heterosexual	22	66.7%
Gay	4	12.1%
Bisexual	4	12.1%
Queer	3	9.1%
Ethnicity		
Caucasian/White	21	63.6%
Asian or Pacific Islander	6	18.2%
Decline to Answer	3	9.1%
Black and African American	2	6.1%
Caucasian/White, Asian, or Pacific Islander	1	3.0%
Income		
Less than $10,000	7	21.2%
$10,000-$14,999	3	9.1%
$15,000-$24,999	7	21.2%
$25,000-$34,999	2	6.1%
$35,000-$49,999	6	18.2%
$50,000-$74,999	5	15.6%
More than $100,000	2	6.1%
Missing	1	3%
Age		
20–29	16	48.5%
30–39	14	42.2%
40–49	3	9.3%
Employment Status		
Full-time Employed	15	45.5%
Part-time employed	8	24.2%
Full-time student	4	12.1%
Not employed for pay	2	6.1%

Other	4	12.1%

	n	*%*
Education		
Associate's degree, academic	2	6.1%
Associate's degree, occupational/trade school	2	6.1%
Bachelor's degree	18	54.5%
Education (Highest Level Earned):	1	3.0%
High School Equivalent/GED	1	3.0%
High school graduate	1	3.0%
Master's degree	8	24.2%
Some universities/college, no degree	1	3.0%

Table 2

Sociodemographic Characteristics of Therapist

	n	*%*
Therapist Gender		
Female	11	91.7%
Male	1	8.3%
Sexual Orientation		
Straight/ Heterosexual	10	83.3%
Gay	1	8.3%
Bisexual	1	8.3%
Racial and Ethnic Groups		
Caucasian/White	8	66.7%
African American,	2	16.7%
Asian or Pacific Islander	1	8.3%
Decline to Answer	1	8.3%
Income		
Less than $10,000	3	27.3%
$10,000-$24,999	2	18.2%
$35,000-$49,999	1	9.1%
$50,000-$74,999	2	18.2%
$75,000-$99,999	2	18.2%
More than $100,000	1	9.1%

Table 3

Descriptive statistics for DERS scale and subscales

DERS Subscale	Min	Max	*M*	*SD*
Total	23	66	42.65	10.01
Awareness	3	12	6.18	2.17
Clarity	3	15	7.76	2.82
Goals	4	12	6.82	2.08
Impulse	3	15	8.06	2.99
Non-acceptance	3	15	6.52	2.72
Strategies	3	14	7.32	2.73

Table 4

Results of multilevel analyses (N=33)

Model (Scale)		Intercept b_{0j}	$Scale_i$	$Session_{ij}$	$C19_{ij}$	$Scale_i$: $C19_{ij}$	$s_{2e}^{\ 1}$	$s_{2u0}^{\ 2}$	REML Fit Criterion
Model 1									
(Total)	*b*	*45.59*	**0.60**	**-0.44**	**5.08**	**-0.21**	*54.89*	*147.15*	*3,953.20*
	SE	*2.33*	*0.23*	*0.07*	*1.16*	*0.10*			
	z		*2.64*	*-6.10*	*4.39*	*-2.08*			
	p		*.013*	*<.001*	*<.001*	*.038*			
	95%LL		*0.159*	*-0.587*	*2.796*	*-0.409*			
	95%UL		*1.052*	*-0.302*	*7.332*	*-0.013*			
Model 2									
(Awareness)	*b*	*45.98*	*0.87*	**-0.44**	**5.34**	**0.71**	*53.29*	*183.60*	*4,219.60*
	SE	*2.49*	*1.12*	*0.07*	*1.11*	*0.33*			
	z		*0.78*	*-6.43*	*4.83*	*2.13*			
	p		*.442*	*<.001*	*<.001*	*0.033*			
	95%LL		*-1.312*	*-0.567*	*3.157*	*0.052*			
	95%UL		*3.068*	*-0.302*	*7.490*	*1.363*			

Model 3									
(Clarity)	*b*	*45.82*	**2.46**	**-0.43**	**5.02**	*-0.58*	*53.49*	*128.92*	*4,211.70*
	SE	*2.13*	*0.74*	*0.07*	*1.10*	*0.31*			
	z		*3.35*	*-6.29*	*4.57*	*-1.86*			
	p		*.002*	*<.001*	*<.001*	*0.063*			
	95%LL		*1.027*	*-0.558*	*2.945*	*-1.186*			
	95%UL		*3.904*	*-0.293*	*7.155*	*0.028*			
Model 4									
(Goals)	*b*	*45.82*	*0.65*	**-0.44**	**5.08**	**0.93**	*53.24*	*181.51*	*4,218.30*
	SE	*2.48*	*1.16*	*0.07*	*1.10*	*0.39*			
	z		*0.56*	*-6.44*	*4.62*	*2.42*			
	p		*.579*	*<.001*	*<.001*	*0.016*			
	95%LL		*-1.611*	*-0.568*	*2.909*	*0.177*			
	95%UL		*2.924*	*-0.303*	*7.229*	*1.689*			
Model 5									
(Impulse)	*b*	*45.89*	**2.03**	**-0.43**	**5.08**	**-0.70**	*53.26*	*139.70*	*4,212.00*
	SE	*2.21*	*0.72*	*0.07*	*1.10*	*0.29*			
	z		*2.84*	*-6.39*	*4.62*	*-2.44*			
	p		*.008*	*<.001*	*<.001*	*.015*			
	95%LL		*0.637*	*-0.563*	*2.905*	*-1.255*			
	95%UL		*3.434*	*-0.298*	*7.210*	*-0.138*			
Model 6									
(Non-acceptance)	*b*	*45.70*	*0.62*	**-0.43**	**4.87**	**-0.79**	*53.19*	*176.24*	*4,217.90*
	SE	*2.45*	*0.87*	*0.07*	*1.10*	*0.30*			
	z		*0.71*	*-6.39*	*4.41*	*-2.59*			
	p		*.485*	*<.001*	*<.001*	*.010*			
	95%LL		*-1.092*	*-0.564*	*2.691*	*-1.384*			
	95%UL		*2.329*	*-0.299*	*7.012*	*-0.191*			
Model 7									
(Strategies)	*b*	*45.32*	*1.40*	**-0.45**	**4.74**	**-1.44**	*53.69*	*167.38*	*3,940.00*
	SE	*2.47*	*0.89*	*0.07*	*1.15*	*0.36*			
	z		*1.58*	*-6.20*	*4.12*	*-4.03*			
	p		*0.126*	*<.001*	*<.001*	*<.001*			
	95%LL		*-0.331*	*-0.588*	*2.475*	*-2.135*			
	95%UL		*3.137*	*-0.305*	*6.986*	*-0.740*			

Note: Bolded values represent the primary values compared in the study that were statistically significant.

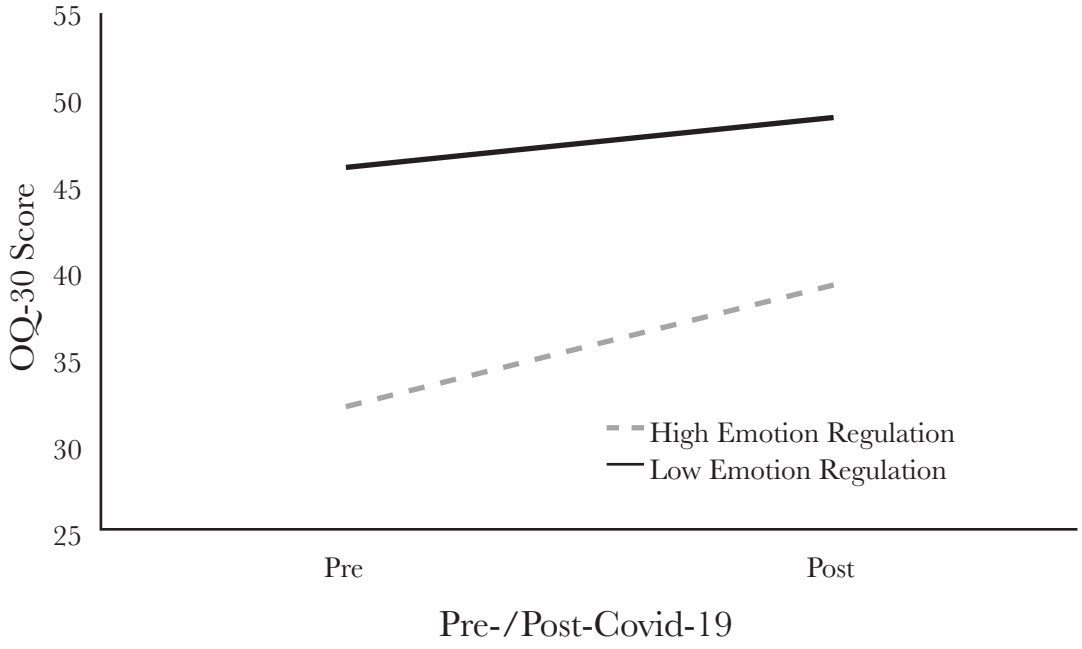

Figure 1.
Changes in symptom severity (OQ-30.2 score) as psychotherapy sessions progressed both pre-and-post-C19 controlling for DERS Total scores (models including all DERS subscales were substantially similar).

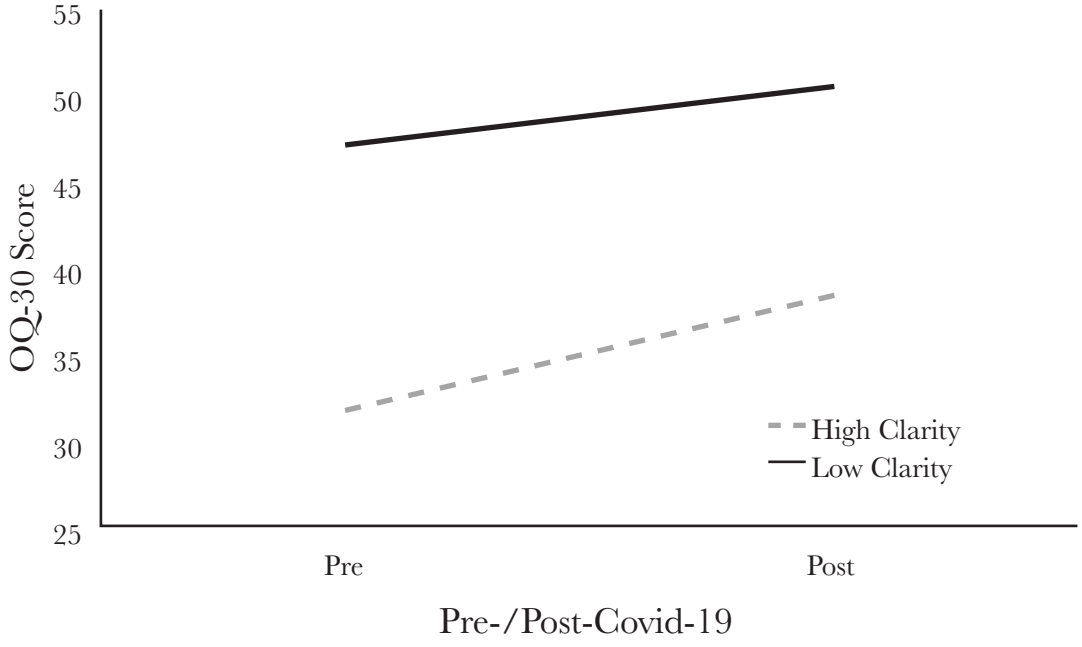

Figure 2.
Changes in symptom severity (OQ-30.2 score) from Pre- to Post-COVID-19 for patients with high and low levels of emotion regulation, controlling for the effect of treatment

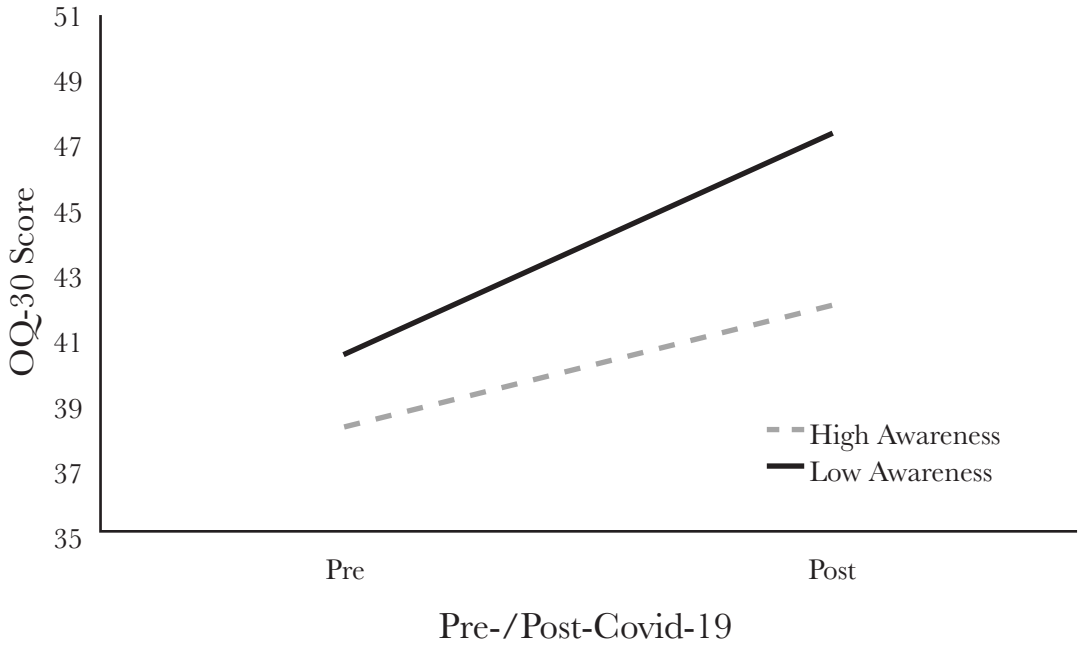

Figure 3.
Changes in symptom severity (OQ-30.2 score) from Pre- to Post-COVID-19 for patients with low and high rates of clearly identifying emotions, controlling for the effect of treatment.

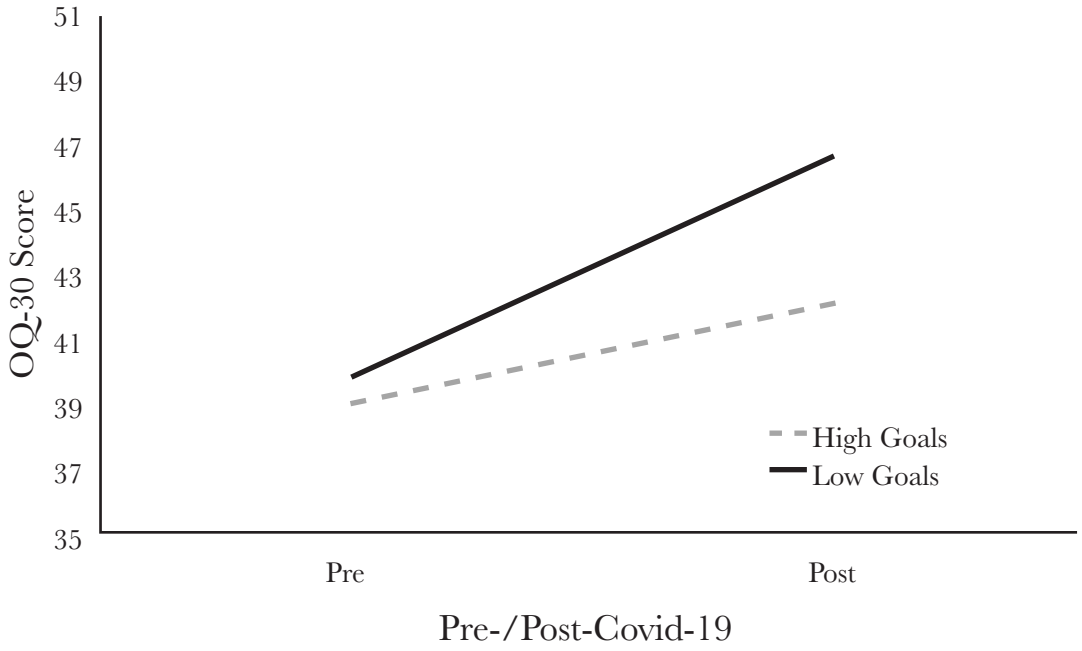

Figure 4.
Changes in symptom severity (OQ-30.2 score) from Pre- to Post-COVID-19 for patients with low and high awareness of their emotional experience, controlling for the effect of treatment.

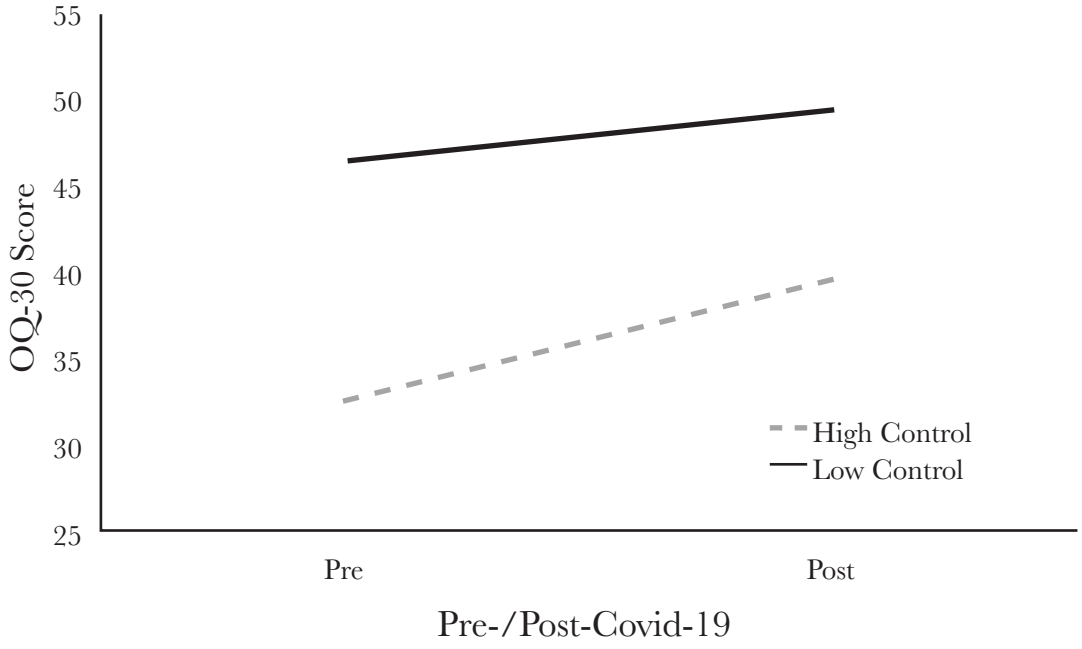

Figure 5.
Changes in symptom severity (OQ-30.2 score) from Pre- to Post-COVID-19 for patients with low and high goal-directed behavior, controlling for the effect of treatment.

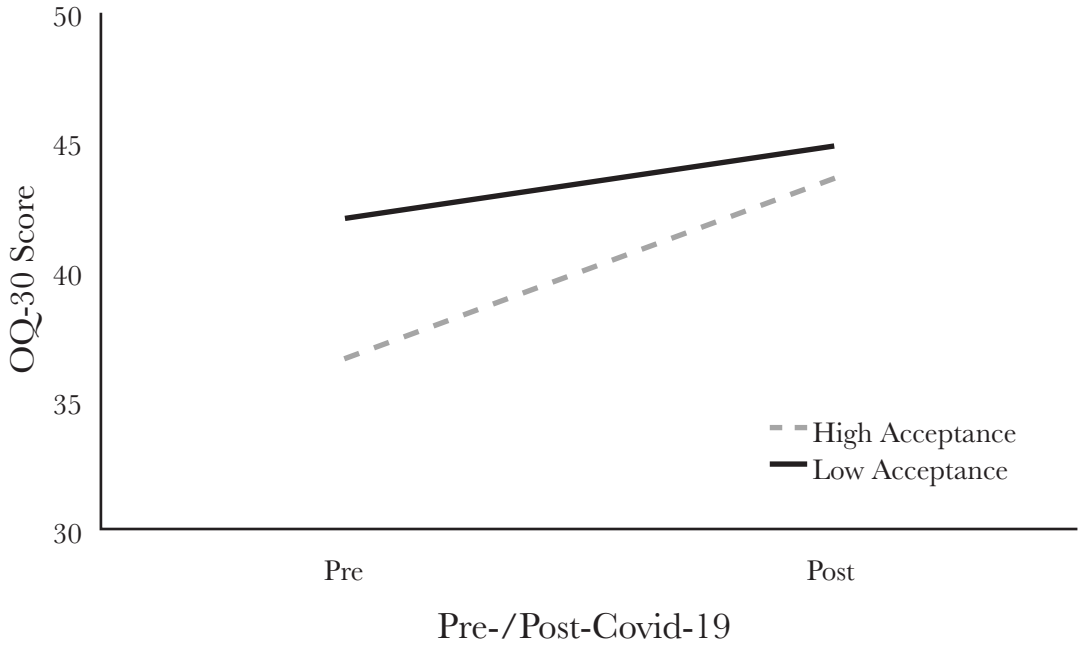

Figure 6.
Changes in symptom severity (OQ-30.2 score) from Pre- to Post-COVID-19 for patients with low and high control over behavior, controlling for the effect of treatment

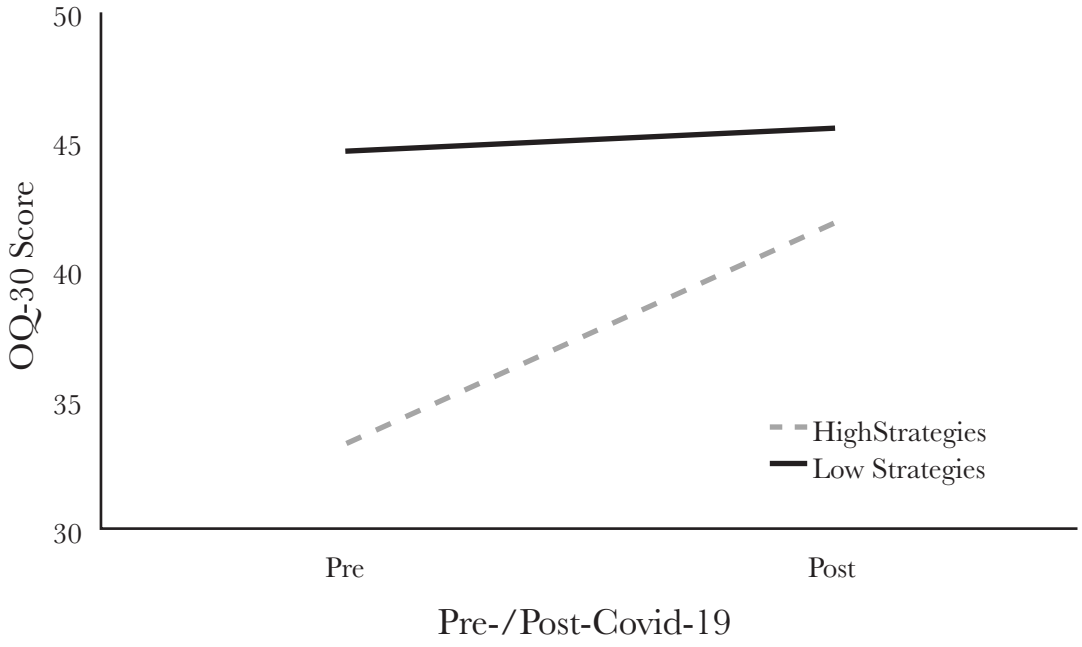

Figure 7.
Changes in symptom severity (OQ-30.2 score) from Pre- to Post-COVID-19 for patients with low and high acceptance of negative emotional experiences, controlling for the effect of treatment.