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PSYCHOLOGY

AT UCLA

Highlights of Insight and Excellence
in Undergraduate Research

University of California, Los Angeles
Volume 1 – Spring 2014

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Founder's Note

As scholars in psychology at one of the most prestigious research universities in the world, we began to ask questions about the educational experience that students were receiving. What emerged based on conversations we had was that many students feel that their classroom education is mostly conceptual, theoretical, and abstract, without sufficient opportunities to apply this knowledge in meaningful, hands-on ways. Many students communicated feeling intimidated by the world of academia, mentioning that on a campus as large as UCLA, getting mentorship from some of the most renowned yet busiest professors in the field isn't always easy. It was with this information that we realized the enormous potential of a publication platform that could allow undergraduate students to publish their findings and gain considerable experience evaluating and editing articles within psychology.

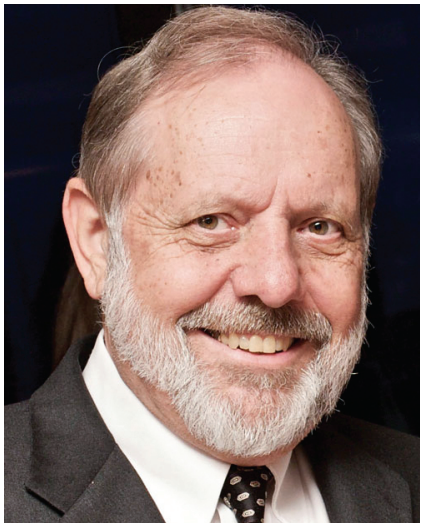
In the spring of 2013, we created The Undergraduate Research Journal of Psychology at UCLA. While undergraduate authors from across the country are provided with an outlet to disseminate their research to peers and to gain valuable feedback on their work, UCLA undergraduate editors are provided with the opportunities to develop strong foundations in APA style and learn critical skills in reading, evaluating, and producing professional research articles. Weekly training workshops provide editors with the tools to evaluate and edit journal submissions, encouraging students to question their worlds with new curiosity and promote involvement in research-related events and opportunities. We have also worked to make the journal accessible to all ages and backgrounds by broadening submission types to include "stepping stone," or lay articles for people who do not (yet) specialize in a specific area of research.This year we received over 120 submissions from nearly 50 universities worldwide. Submissions came from as close as UCLA to as far as Cambridge, and we now have nine manuscripts from 10 determined authors that exemplify the quality research being conducted by undergraduates all around the world. We would like to acknowledge each author who submitted an article, as it represents the pure ambition that undergraduates possess to step outside of the classroom and apply their knowledge.

The ultimate goal of The URJP at UCLA is to serve as a peer-reviewed collaborative platform where wonder, curiosity, and truth are the motivators, where graduate students and faculty serve as mentors passing on their expertise and passion, and where undergraduate students can be empowered, not intimidated, to join the conversation of science. In the past year, The URJP at UCLA has grown from a group of four volunteer editors engaged in research to a large and diverse community of intensely motivated young psychologists across the country. We are confident that this community will only continue to expand. Our hope is that this journal and the workshops we host will serve as models for other institutions seeking to facilitate research-oriented cooperation and conversation among the next generation of psychological theorists and researchers.

We would like to thank our dedicated team of editors, marketing and submissions members, and designers, without whom none of this would have been possible. We would also like to thank our graduate student mentors, Britt Ahlstrom, Dr. James Ashenhurst, John Danial, Kate Humphreys, Sami Klebanoff, Elizabeth Raposa, Christina Shonberg, Tawny Tsang, and Yolie Vasquez-Salgado, whose experience and expertise have been invaluable throughout the editing process, as well as Adam Blake, Alyssa Cheadle, Bita Mesri, and Chéla Wiley who have kindly written articles of advice for undergraduate students.

Chardee Galan, Anthony Osuna, Chloe Tagawa, & Lauren Wong

Preface



Robert A. Bjork
Distinguished Research Professor

I am delighted to have the opportunity to write a preface for this, the inaugural edition of The Undergraduate Research Journal of Psychology at UCLA. In this large and diverse Psychology Department, undergraduate students play an important role in research, as they do at other research universities throughout the nation.

At UCLA, the majority of the students who graduate with a BA in Psychology, a BS in Psychobiology, or a BS in Cognitive Science, become actively involved in research projects during their undergraduate careers, an involvement that is an enriching, sometimes life-changing, experience for most students. Participating in research, among its other benefits, such as being an exercise in analytical and creative thinking, can also offset one disadvantage of pursuing a popular major at a large university–namely, having very few opportunities to take small courses of the type that encourage discussion and let students and faculty get to know each other. Working with faculty and graduate students in a research laboratory can provide opportunities for the types of substantial and meaningful discussions and interactions that are often missing in large-lecture courses.

Faculty, graduate students, and postdoctoral fellows also profit in very significant ways from the involvement of undergraduate students in the research process. Projects not only get completed that would not otherwise get completed, but also often get completed more successfully, given the ideas, efforts, and questions contributed by the undergraduates involved. When everything works the way it should, there is a wonderful symbiosis between the research efforts of faculty members, postdoctoral fellows, graduate students, and undergraduate students. A major factor in drawing exceptional faculty members to the Department of Psychology at UCLA, for example, and keeping them here, is the opportunity to mentor and collaborate with extraordinary graduate students. Those graduate students, in turn, are attracted to this Department by the opportunity to mentor and work with exceptional undergraduate students, an opportunity that can not only increase their own research productivity, but also enrich their graduate careers from a social and mentoring-experience standpoint. Over my own years as a faculty member, first at the University of Michigan, then at UCLA, it has been thrilling to watch undergraduate students gain confidence and increase their aspirations based on working with, and modeling on, graduate-student collaborators, and to see how such a mentoring experience can enrich graduate students' lives as well.

I want to congratulate everyone involved in taking the initiative and expending the energy to create this new journal, which has the potential to be a key companion to this Department's yearly, and highly successful, Psychology Undergraduate Research Conference, a conference that has been copied by multiple other Departments of Psychology and is about to meet for its 23rd year. The 9 articles selected for this first issue from over 120 submissions from nearly 50 institutions worldwide, illustrate yet another very basic benefit of undergraduates becoming involved in research: Their contributions as researchers can enrich the entire field of psychological science.

Faculty Endorsements

Elizabeth Ligon Bjork, Ph. D "I am pleased to have the opportunity to write a few words of support for the inaugural edition of The Undergraduate Research Journal of Psychology at UCLA. I hope that the establishment of such a journal will encourage those undergraduates not already involved in research to explore such an opportunity and, for those already involved, I hope it will serve as a recognition of the indispensable role that you play in the research enterprise here at UCLA and at other colleges and universities both nationally and internationally. Congratulations to Editors-in-Chief Anthony Osuna and Lauren Wong, and their fellow student and faculty collaborators for this initiative, and I hope this edition will just be the first of many successful ones to follow."

Gregory A. Miller, Ph. D "The Undergraduate Research Journal of Psychology at UCLA is a student-initiated, student-run forum for some of the exciting science we do. Research participation is one of the most important elements of the education we offer. Our faculty are proud of this journal and of the students who edit it and contribute to it."

A. Janet Tomiyama, Ph. D "Peer review and publishing are the cornerstones of academic research careers. The value of The Undergraduate Research Journal of Psychology at UCLA, therefore, cannot be overstated. It's a wonderful opportunity for students at the beginning of their career in Psychological science – exactly the time when students need support the most."

Jesse Rissman, Ph. D "I commend the students in my department for having the vision and dedication to launch The Undergraduate Research Journal of Psychology at UCLA. Undergraduate students are making increasingly vital contributions to research projects around the globe, and it is wonderful to see efforts like this to provide a peer-reviewed forum for them to showcase their work to the broader community. My lab would certainly not be what it is without the many talented undergrad RAs who help design experiments, develop stimuli, run subjects, and analyze data. I plan to encourage my students to consider publishing their work in this journal."

Rena Repetti, Ph. D "The Undergraduate Research Journal of Psychology at UCLA reflects the initiative, drive, and sophistication of its editorial board and the many students who submitted their research papers. This impressive issue contains a wide range of fascinating research articles; I applaud the professionalism and hard work that went into its creation. UCLA is very proud of our many students who get involved in research activities and we are grateful to those who organized this publication outlet. The excitement of publishing and knowing that your work is available to others is one of the great rewards of science. I hope that the journal will serve as an inspiration to students far and wide who are curious and interested in psychological research."

Nim Tottenham, Ph. D "The research that one performs as an undergraduate student often forms the foundation for one's future career in academia. Having an means of publishing the findings at such an early stage is a wonderful resource."

Ted Hutman, Ph. D "Participating in research as an undergraduate is a critical way to figure out if and what you like about doing research. It's a great opportunity to sample different research domains, to hone your interests, and develop your skills. Presenting your results in posters or talks requires you to summarize your work concisely, and it allows you to receive and repsond to audience feedback. Writing up your work for publication documents the full arc from planning and implementation to interpretation and next steps. The Undergraduate Research Journal of Psychology at UCLA provides a unique forum disseminating your work, both to peers and to prospective mentors!"

Lara A. Ray, Ph. D "As a UCLA faculty member, it is my pleasure to support The Undergraduate Research Journal of Psychology at UCLA. This journal offers a unique opportunity for students to share their scientific work and to experience the publication process in ways that will foster their professional development and contribute to the science of psychology. Having over 100 peer-reviewed publications, I can say with confidence that I am very excited each and every time I publish a research article. I believe that each manuscript contributes to the field and ultimately, to the development of better treatments for addictive disorders. I am thrilled that UCLA students will have this exciting resource and I am confident that the excitement of research and its contribution to society will fuel their passion and professional growth."

Advice From A Grad

Adam B. Blake
Graduate Student
Cognitive Area, Department of Psychology, UCLA

Getting In--The Long Summer

Summer break is rapidly approaching, but if you are thinking about applying to graduate school, "break" may not be an apt description. The summer is a fantastic opportunity to develop an impressive application and decide which programs to apply to – preparing now will significantly ease the stress of applying. Good luck!

Develop a Strong Application

Many students in the pre-application stage worry that their grade-point average and Graduate Records Examination scores are too low. In actuality, these are only used as indicators of potential and are only noteworthy if they are below average or extraordinarily high. The focus of an application to a graduate program should be to express dedication to psychological research in an academic setting. This should be the driving factor: the personal statement should describe it, the curriculum vitae (CV) should showcase it, and the letters of recommendation should affirm it. Your personal statement and CV are your chance to show that you are a serious researcher with a dedication to the field. It is of utmost importance to mention anything even tangentially related to the field you are going into. Think critically about your research history and do not be discouraged if it seems paltry. Even something as simple as coding data shows a commitment to research and an understanding of how studies are conducted. Over the summer you should be writing and polishing your personal statement and CV to perfection. When you have given your absolute best effort to it, ask graduate students and professors to review your work. Be prepared: your paper will come back bleeding red ink, but don't take it personally. Any professor reading your paper has a good understanding of what admissions committees are looking for and is helping you tailor your paper to that end.The letters of recommendation are possibly the most important documents in an application. They are endorsements of work-ethic, personality, and research experience. It is not the purpose of this article to cover this in detail, but the importance of these letters cannot be understated. A well-written recommendation from a professor, who understands which qualities are valued in academia, will considerably strengthen any application. If you do not know at least three professors who can write about your potential as a graduate student, then this should be your summer goal. Take advantage of office hours, think deeply about class material, and start interesting discussions on topics of shared interest. Cultivating relationships in this manner will demonstrate your ability to think analytically. If you can convey this quality, you are well on the way to an impressive recommendation.

Picking a Program

While you are developing and refining your application, you should be on the search for suitable programs. Many students take a misguided approach and only look into "big name" schools. Instead, consider research you find interesting and then contact the people running those labs. Use an appropriate database or search engine to look for articles on topics that interest you. When you find research that piques your curiosity, write down the names of the authors on those papers. Research these people and see whether they are still doing similar research. When you have whittled your list down to only professors who have research labs, it is crucial that you contact these people. Not every lab will be accepting students every year. Most labs have an upper limit for how many students they can fund, and sometimes professors have the opportunity to take a sabbatical and will not be able to take students while they are away. Ask about their current studies, where past students have gone after graduation, and contact any current graduate students about their experiences. Remember, if you are accepted you will be in a program that will take an average of five years to complete – a huge investment – so gather as much information as you can. Finally, communication is going to help your application tremendously. Each person reviewing your application will be assessing many other files. Between two similar applications, a familiar name will have a significant edge, especially if you have conversed about your shared research interests.

Advice From A Grad

Bita Mesri, BA
Graduate Student
Clinical Area, Department of Psychology, UCLA

Letters of Recommendation

Worried about getting a good letter of recommendation? Just follow the advice below. You'll have one less thing to worry about AND one more thing that will help you land your dream job or attend your dream graduate school.

If you're looking to get a good letter of recommendation, you first want to think about who would be the best letter writer. Most people think they should simply ask the person who knows them best. While that is not a bad option, it is actually wiser to ask someone within the same field or related field to the program for which you need a letter. For example, it may be wiser to ask a professor at UCLA who knows you moderately well than the Resident Advisor that lives on your floor who knows you very well; the reason being that the professor's words will probably hold more weight. Or for another example, you wouldn't want to get a letter from a family member or friend even though they know you well. In the same line, think about to which field you will be applying and try to get someone who has been in the same field or is currently in the same field. For example, if you're looking to do social work, it may be better to get a letter from a professor in Psychology than Physics. Even more, it may be better to get a letter from a social worker for whom you have interned.

Now that you know who your target letter writer is, you need to think about how to get a good letter from them. If you are working for the individual, make sure that you are doing your best everyday. If you are looking to get a letter from a professor, you'll need to begin building a relationship with him/ her. Most students don't have much interaction with professors outside of the classroom. Asking a professor to write a letter of recommendation for you simply because you receive a good grade in his/her class will result in a generic letter, which will contribute nothing to your application. If you're looking for an exceptional letter, you really need to have a great relationship with them. An easy way to start the communication is by attending their office hours. These are times that the faculty members have set aside for their students and will be happy to see someone attend. However, you do not need to limit your questions to the material of the course. Instead, you can use this time to ask about their career path and choices. If you're trying to prepare for graduate school or get into the work place, who better to ask than someone who has done just that and successfully so? And of course, everyone loves talking about himself/ herself.

If you want to take it one step further, I would recommend volunteering your time to work in their lab. In this role, the faculty member or graduate student advisor will have a lot of interactions with you and will know you well enough to write a more in-depth letter. In turn, your experiences within their lab could inform your future career path and can open doors to work in other labs in the future.

Once you've thought about who will write your letter, sought them out and worked with them, you'll need to ask them to write your letter in an appropriate manner. Ideally, they will already know what you future goals are, but if not, you'll want to make them aware of your future plans and explain why you think they would be able to write you a good letter. You want to talk about how important they are in the field along with your role in their lab/ work and your strengths. Make sure to give them ample time to write a letter (minimum 1 month) and provide them with clear instructions on where to submit the letters. It would be great to add your CV, your transcript, a bullet point list of your responsibilities, or any other information that you think are relevant and may help jump-start their writing process. You really want to make this process as painless as possible. And of course, don't forget to send them reminder e-mails and a thank you note when the process is over.

Advice From A Grad

Alyssa C. D. Cheadle, MA, MTS
Graduate Student
Health Area, Department of Psychology, UCLA

A Day in the Life of a Grad

It is probably a cliché to say that no two days are alike for a graduate student. For me, this variety makes the hard work and long hours of graduate student life worth it. What makes up those long hours? It varies from student to student and as your program progresses. In the first few years, you spend more time in classes and doing class-directed reading and assignments. Later on, your own research projects fill your time. Depending on your program and your funding, other responsibilities will surface. You may work as a teaching assistant or on your advisor's projects. If you are a student in a clinical program, you will spend time in clinical training and start to see clients of your own. I am a third year graduate student in the Health Psychology area at UCLA. At this point in my program, I am mostly done with coursework and my time is spent mainly on my own research, on teaching assistant duties, and on projects I work on for my advisor.

Here's how I spent my time on a typical day last quarter: Wednesdays were some of my busiest days. First thing in the office, I like to review my to-do lists. Time management is a big topic for graduate students; to-do lists are a must for managing all of the steps in many projects. In the mornings, I also spend a few minutes tweeting about posts on the graduate-student run blog Psych in Action (psychologyinaction.org).

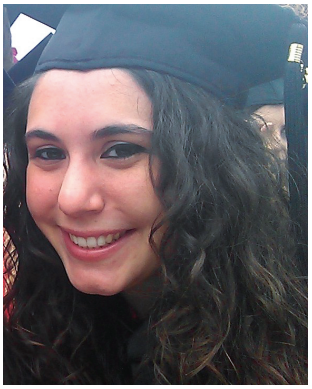
My first meeting was a lab. Every professor runs their lab a little differently. This lab does a mix of discussing journal articles and giving feedback to students on their research. Many students attend more than one Professor's lab meeting to develop breadth and get feedback from diverse perspectives. After lab, I held my office hours which were some of my favorite hours of the week because I especially enjoy one-on-one teaching with students! Midday, I went to the Health Psychology area's Brown Bag forum. Most departments or areas have a regular forum for professors, students, and visiting scholars to discuss their work. It is an opportunity to learn about research in your own department and elsewhere, and it always gives me ideas for my own research.

My first opportunity to get some of my own research done came in the afternoon. Many professors have repeatedly recommended to us that we make time for our own writing and research every day, and defend that time against interruptions. Later in the afternoon, a student doing a project in my lab would meet with me so we could discuss the next steps in her project. The last scheduled part of my Wednesday was another lab meeting–this time with my primary advisor's lab. That lab time is dedicated to giving feedback on student and visiting scholar presentations which is invaluable to improving your work.

After that lab meeting, I had an hour or so to squeeze in a little more work before heading to a running class at Drake Stadium. Balance is something that every graduate student struggles to achieve. Of course we want to do all of our work, to sleep, to spend time with friends, to exercise, to eat well...but sometimes it feels like there's just not enough time for everything! Being a Health Psychology student, I know just how important it is make time for healthy habits. My running class helps to clear my head and make sure I get a good night's sleep that night!

No two days as a graduate student are the same, and I'm lucky that I have some more relaxed days to balance out the busyness of a typical Wednesday. Schedules vary from student to student and from quarter to quarter, but setting good time management habits and balancing work with healthy habits and fun makes almost any schedule workable. And because you're working on your passion, the hours of work are [usually] a joy!

Advice From A Grad



Chéla Willey, MA
Graduate Student
Cognitive Area, Department of Psychology, UCLA

What if I Don't Get Into a Ph.D Program?

This is a question you should continually ask yourself as you are filling out all those Ph.D applications that may or may not have an impact on your near-future plans. What if I don't get into graduate school? What is my "Plan B" or my intermediary plan? How can I make the most of the next year or two before I apply again? Let's first discuss the most probable reason as to why you might not be (or were not) chosen for a Ph.D program.

If you do not get into graduate school the first time you apply, you are not alone. Graduate school admissions is becoming increasing more competitive. However, it should not be a surprise that when you apply to graduate school, you are not applying to the school, nor sometimes even the department of interest; you are applying to specific labs or advisors that conduct very specific research. Graduate committees and potential advisors are interested in finding a good fit between the lab's research and potential students. Now, this doesn't mean that you have to have been already actively involved in that particular area of research. While this could help portray your confidence as a good fit, it is more necessary to have general research experience. However, it is extremely important to convey confidence in your interest in that particular lab/advisor/area of research in addition to the set of research skills you can bring to the table. Many times applicants do not fully convey this interest, at least not to the extent that the admissions committee is seeking. Remember, the graduate program is looking for someone in which to invest five to six years' worth of time and money. They want to be absolutely confident that you are fully invested as well.

So if you did not get into graduate school, take this opportunity to find (or confirm) what interests you so much that you wish to do it for the next six years of your life. First, it does not hurt to also apply to a couple of Masters programs, preferably in the area of interest or in a general experimental program. The application deadlines for most Masters programs are a bit later (beginning of the year) than those of Ph.D programs which means if you did not originally apply to Masters programs, you still have the opportunity. Going through a Master's program does many things for your future Ph.D applications and your happiness as a Ph.D student. It first allows you to take graduate level classes in your areas of interest and allows you to explore the types of research that you would enjoy pursuing. It provides you with an opportunity to conduct graduate level research with the help of an advisor that not only culminates into a Master's thesis but also has the potential for at least one scientific publication. These invaluable research experiences and achievements can place your next year's PhD application at the top of the stack of viable applicants. Further, they can help your statement of purpose find a strong sense of direction and purpose.

However, there is the possibility that you also did not get into or did not apply to Masters programs. There are numerous other opportunities in which you can apply to stay in the game and ready for next year's round of applications. For example, if you are already a part of a research lab, find out about post-baccalaureate positions, such as a lab manager. A simple internet search of 'post-baccalaureate positions in psychology' can help find one suited for you at institutes across the globe. Paid summer research programs are also invaluable experiences for anyone interested in graduate school. More times than not, summer research programs allow you the opportunity to present the work you've completed during the program and include useful graduate school workshops and GRE prep. If you are interested in criminal justice, social, forensic or counseling psychology, government positions/internships in social services, judicial branches, or veteran hospitals may be another viable option. All of these opportunities can only strengthen your application and help you to ensure that you are making the right decisions concerning graduate school. As more students are opting to stay in school after receiving their bachelor's the more you will need to sell yourself and your accomplishments, thus make the next years productive towards your ultimate goals.

Jason Carpenter

University of California, Los Angeles

Jason Carpenter is a 4th year Double Major in Cognitive Science and Applied Mathematics at UCLA. He loves learning about the cognitive functions of the mind as they provide deep insight into how people think and the resulting human behavior. Jason believes that in order to develop a truly holistic understanding of the mind, scientists must incorporate the use of mathematics to develop algorithms which reflect individual cognitive functions. His current research in the Visual and Multisensory Perception Lab allows him to pursue this very interest, working with the algorithms underlying multisensory perception. He seeks to continue his higher education through pursuing a graduate degree in Cognitive Science. Jason developed his love for the learning process at iD Tech, a tech summer camp for kids. Working as an Instructor, teaching children how to program and make video games inspired him to want to pursue a career in education. In his promoted position as Assistant Director, Jason has developed a passion for leadership and has continued to hone his abilities as a leader amongst his peers in his role as Vice President of Sigma Pi. Combining his deep desire for performing novel research for the benefit of all and his true love of education, Jason's experiences have guided him to aspire to one day become a Professor. When he's not dreaming big, Jason loves to carve out time for running, hiking, basketball, drumming and traveling.

How do you juggle all of your responsibilities?

In order to juggle all of my responsibilities performing research, taking classes, and serving as Vice President of Sigma Pi, it is crucial that I make priorities and take care of the most important tasks first and foremost. Between my various roles, I have a lot on my plate but I make it a priority to always take some time to find healthy ways to relax and de-stress through yoga, drumming, running, playing basketball, hanging out with friends, etc.

Was there any particular experience that sparked your research interests?

I met with my TA from my Psychology 100B course to discuss how to get involved in research, only for me to discover my background in both Cognitive Science and Applied Mathematics to be ideal for the lab that he works in, performing Computational Cognitive Science. Once accepted as a research assistant in the lab, I asked to be placed on a project that would challenge me to delve deeper into studying the connection between my two majors and fortunately was given the truly unique opportunity to be at the forefront of the computational study of multisensory perception under the mentorship of graduate student Brian Odegaard, my former TA. I am forever grateful to Ladan Shams, Brian, and the Visual and Multisensory Perception Lab for this opportunity.

Who has influenced you the most?

My graduate student mentor, Brian Odegaard, has been an invaluable resource to me. He has prepared me for pursuing great opportunities such as being published in URJP and presenting my research at PURC, Powell Undergraduate Research Week, and UCLA's Science Poster Day. Under his tutelage I have been able to grow rapidly as an undergraduate student from one who had never before performed research to one who is experienced and comfortable in a lab setting, working on a complex research project. Having his guidance has given me the opportunity to perform high quality research without fear of failure and has inspired me to want to continue in this endeavor beyond graduation, powerfully influencing my career aspirations.

What is your dream job?

My dream job is that of a professor of Cognitive Science. I believe that being a professor would be the most rewarding position for me on all fronts. It would allow me to conduct novel research for the benefit of mankind. I would get the opportunity to mentor young minds, both of the undergraduate students that I would be teaching, but also of the graduate students and research assistants working in my lab. Finally, it would give me a role through which I could truly shine as a leader in the academic community. This career aspiration drives me to work hard as an undergraduate and to hold myself to a higher standard.

Bayesian Causal Inference in Multisensory Perception

Jason Carpenter
University of California, Los Angeles

For a moment, imagine walking in a jungle, and suddenly, you perceive a lion entering into your view some distance away. Now, imagine in the same moment, you also hear the roar of a lion coming from the general direction of the visually perceived lion. Should you be concerned about a single lion, or are there multiple lions to be worried about? According to Körding et al. (2007), as observers, any time we encounter multisensory stimuli, our brains must determine whether these stimuli come from the same cause and should be combined, or whether they come from different causes and should be separated. This complex task that the brain must solve is an example of "causal inference" in multisensory perception and can be effectively modeled using a branch of statistics known as "Bayesian statistics" (Körding et al., 2007; Shams & Beierholm, 2010; Shams, Ma, & Beierholm, 2005; Beierholm, Körding, Shams, & Ma 2008; Wozny, Beierholm, & Shams, 2008). As applied to perception, Bayesian statistics specifies that the things we perceive are a combination not only of the information we obtain from our senses, but also prior biases we might have. These predispositions can influence our perception of where sensory events occurred, or our inferences about how many sources likely caused the things we sensed. Thus, the number of lions we perceive is not based entirely on the visual image and the roar we hear; instead, our brains help us make use of statistical regularities (such as whether multiple lions normally roam the area) and patterns (did the roar sound like the lion we saw, or another species?) to help determine what is happening in the world around us.

This "Bayesian" way of thinking about perception is well-demonstrated in a model established by Körding et al. (2007), which incorporates the two central pieces of information in this branch of statistics. One piece of information is called the "likelihood." In a Bayesian framework, the observer does not have access to the true sensory source locations, but instead only has access to rough estimates of where the source is located. For example, imagine that the lion is being clouded by fog. These sensory estimates represent probabilities of where the stimuli could be, which can be characterized by bell curves, formally deemed "Gaussian distributions." These distributions are characterized by means, which represent the most probable location for each stimulus, and variances, which characterize how reliable the signal is. On a very foggy day, the variance of the visual signal might be large (because we are not sure of where the lion is), but on a clear sunny day, the variance of the visual signal might be small (because we can see the lion clearly). Similarly, the auditory signals we receive can be characterized by means and variances. If the lion's roar was quite loud, it could be characterized by a distribution with a narrow variance, and we should hope that the mean (the lion's most probable location) is not too close to our current location! The other piece of information used in a Bayesian framework is the "prior." Through our everyday sensory experiences, we develop an estimated probability of whether two co-occurring signals come from a single source or two distinct sources. Over the course of a lifetime, humans develop a strong prior inclination to integrate simultaneous multisensory signals that are close together in space, and this effect gradually weakens as the distance between the signals increases (Wozny, Beierholm, & Shams, 2008). In the example of the roaring lion, if we perceive the roar coming from a considerable distance away from the visually perceived lion, we are likely to infer the presence of two lions, rather than a single lion. Utilizing the two essential pieces of Bayesian information, the likelihood and the prior, we can approximate perceptual information important for keeping ourselves safe from the lion(s).

These two elements, likelihood and prior, are included in a simple formula (deemed "Bayes' Rule") that specifies how we can compute the probability of estimating whether there are one or two lions. Bayes' Formula states that our final estimate of the probability of a single lion (called our "posterior" probability), is equal to the product of the likelihood and prior, divided by a normalizing constant. This can be written as the following: P(A|B), our posterior probability of inferring a single lion, given the sensory signals that occurred, is equal to the

probability of the likelihood of the visual & auditory signals we sensed, given a single lion in the world (P(B|A)) times the prior probability of a single lion being present, based on our past experience (P(A)), divided by a normalizing constant (P(B)). Thus, our inference about the number of lions is captured in the Bayes' simple equation: P(A|B) = P(B|A) * P(A) / P(B).

As shown in the example above, Bayesian models (such as the one by Körding et al., 2007) combine the likelihood distributions and the prior distributions to estimate the resulting "posterior" probability of whether there was a common cause or distinct causes for the sensory signals. Bayesian models then use this posterior probability in their final estimation of the locations of the sensory signals. Fascinatingly, these models closely simulate human behavior, meaning our brains might actually perform perceptual calculations via a Bayesian framework, and some researchers even think that neurons themselves might process information in a way very similar to Bayes' Rule (e.g., Deneve, 2008). Future studies will probe the utility of these statistical rules in accounting for how our brains process information.

The causal inference model by Körding et al. (2007) has previously been shown to be particularly adept at modeling participants' behavior in situations requiring localization of audiovisual stimuli, and effectively models certain perceptual illusions, such as the "ventriloquist effect." The ventriloquist effect is a phenomenon that has been used to study how visual and auditory signals get bound together in multisensory perception (Alais & Burr, 2004). Similar to when a ventriloquist moves a puppet's mouth, and our perception is that the puppet is talking, this effect involves the visual capture of auditory stimuli when presented at small spatial discrepancies. This effect is so strong, it is even apparent when meaningless flashes of light and bursts of sounds are played close together. When participants try to localize sounds which happen at the same time as flashes of light, they will often localize the sound close to where the light occurred, which is also known as "visual capture."

Because of the observed visual capture, the ventriloquist effect led researchers to hypothesize that the most reliable signal dominates in a winner-take-all competition, meaning that an observer's localization judgment is based exclusively on the most reliable signal (Battaglia, Jacobs, & Aslin, 2003). Contrary to this notion, the ventriloquist effect is best accounted for by Bayesian statistics rather than the winner-take-all heuristic; instead of one modality always capturing the other, the source locations are characterized by a mutual attraction in space with the influence of each modality inversely weighted by the noise of the stimuli (Alais & Burr, 2004; Magosso, Cuppini, & Ursino, 2012). Spatial locations of stimuli corrupted by greater perceptual noise in one modality are typically judged closer towards the location of the stimuli perceived by the modality with less perceptual noise. This type of attraction is well-captured by the Bayesian causal inference model proposed by Körding et al. (2007). In most scenarios, auditory signals are much noisier than visual signals, resulting in visual capture of coincident auditory stimuli, as observed in the ventriloquist effect. Conversely, Alais & Burr (2004) showed that auditory capture can occur if visual stimuli are degraded so they become noisier than auditory signals, like when the visual stimuli are extremely blurry. In these uncommon situations, the visual stimuli are localized closer to the auditory stimuli. It is important to understand these general principles underlying how the brain incorporates individual noisy perceptual signals from different sensory modalities, in order to come to a more complete understanding of how we effortlessly engage in multisensory perception of the external world.

Let us return to our example with the lions: imagine that the lion enters your far right visual field, but instead of naturally turning your head and eyes to look at the lion, you only turn your eyes to look at it. Does this have any effect on the causal interpretation of whether or not the visually perceived and the aurally perceived lion are the same lion or two distinct lions? Past research suggests that it should: audiovisual fusion areas function differently when our eyes and head are aligned, and when they are misaligned (Hartnagel, Bichot, & Roumes 2007). When eye- and head-position are in alignment, audiovisual fusion is most accurate straight-ahead. However, when they are misaligned as in the example given, audiovisual fusion areas with the greatest precision are located halfway between gaze position and straight-ahead (with respect to head position) (Hartnagel et al., 2007; Pouget, Deneve, & Duhame, 2002; Deneve, Latham, & Pouget, 2001). These partially shifting audiovisual fusion areas reflect changes in our tendency to integrate audiovisual stimuli, depending on the positions of our eyes in a given moment. Such partially shifting receptive fields reflect the dissociation between visual (eye-centered) and auditory (head-centered) reference frames at misaligned eye- and head-positions. They

are a direct consequence of a flexible neural network in the brain that can continuously perform multidirectional computations transferring information between sensory modalities (Pouget et al., 2002; Deneve et al., 2001).

Perceptual causal inference calculations are performed all the time in our daily lives at a subconscious level and are crucial to our understanding of perception as a subcomponent of human cognition. The Bayesian Causal Inference model is an effective computational algorithm that closely simulates human perception (Körding et al., 2007; Wozny, Beierholm, & Shams, 2010). While great strides have been made in understanding how eye and head position influence multisensory perception, there is still much more to be discovered. Future research should investigate how aligned and misaligned eye, head, and body positions can influence estimates about the number of sources of sensory signals in the world, and estimates about the locations of sensory signals. The Visual and Multisensory Perception Laboratory at UCLA is currently investigating the Bayesian Causal Inference model's capacity to characterize the computational changes that take place as the brain tries to reconcile the distinct reference frames for vision and audition. This research has the potential to open the door for a deeper understanding of how the human brain reconciles all multisensory information to produce a unified perceptual experience of the world.

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Caitlin Eggleston

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Caitlin Eggleston graduated from Stanford University with a Bachelor of Arts with Honors in Psychology in 2013. She now works as full-time research assistant in the Mood and Anxiety Disorders Laboratory at Stanford University. She coordinates a research project examining the impact of a novel training paradigm on interpretation biases in depressed adolescents. She will be applying to Ph.D programs in Clinical Psychology in the fall and hopes to continue her research on depression in children and adolescents. When she is not in lab, Caitlin enjoys traveling, yoga, watching movies, and relaxing at home.

What part of research do you find the most fun/ challenging?

My favorite part of the research process is working with people. It can be difficult at times but listening to people's stories and working closely with so many different people from so many different backgrounds is very rewarding.

How do you juggle all of your responsibilities (your research, school, etc.)?

I make sure to carve out specific time for specific things, which allowed me to focus on the task at hand. When I create a schedule to balance lab, classes, and study time, it is easier to balance everything I have to get done, while still making time for extracurricular activities and friends.

Besides research what do you do for fun?

I love watching movies and spending time with my friends. I have also recently started training for a half-marathon so I am spending more time running and doing yoga. I also love to travel and have several fun trips around the US planned this year.

Where do you envision yourself in 10 years?

In 10 years, I hope to be a licensed clinical psychologist working in a University or hospital setting where I can continue to do research with clinical populations.

What is your dream job?

My dream job would be a professor working in a research institution so that I can conduct original research, teach students in the field that I love, and continue to work with individuals with depression and anxiety.

The Effects of Comorbid Anxiety on Attentional Bias in Depressed Adolescents

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The current study was designed to examine differences in attentional biases in adolescents with and without Major Depressive Disorder (MDD) and to understand the effects of comorbid anxiety symptomatology. Thirty adolescents with MDD and sixteen age- and gender-matched control (CTL) adolescents completed a positive and negative emotional faces version of the dot-probe task, a frequently used measure of attention orienting. Both CTL and MDD adolescents showed a bias away from negative emotional information, which is not consistent with the adult literature or with our hypotheses. However, controlling for severity of anxiety symptoms yielded a trend-level interaction of group and valence. These findings indicate that the inconsistencies in the adolescent depression attention bias literature may be due to comorbid symptoms of anxiety that are prevalent in adolescents with MDD.

More than 30 million adults will meet criteria for Major Depressive Disorder (MDD) during their lifetime; in addition, more than 80% of adults with MDD will experience a recurrent episode of depression (Kessler & Wang, 2009), making this disorder one of the most common of all psychiatric illnesses. MDD also affects 9% of children and adolescents in the United States (Avenevoli, Knight, Kessler, & Merikangas, 2008) and nearly 40% of these children and adolescents will experience a recurrent episode of depression within three years of their first episode (Kessler, Avenevoli, & Merikangas, 2001). Adolescent-onset depression is associated with longer and more severe depressive episodes (Lewinsohn et al., 1994) compared with those who develop depression later in life. Furthermore, adolescents who experience an episode of MDD show functional impairment in academic and occupational performance, interpersonal functioning, physical well-being, and quality of life (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003). Given the high prevalence of MDD in the general population and its enormous impact on the well-being of individuals across the lifespan, it is important to understand the mechanisms that contribute to the development of depression during late childhood and adolescence.

One possible factor contributing to the maintenance of depression is biased attention towards dysphoric, mood-congruent information in the environment. Compared to healthy controls, depressed adults are more likely to orient attention towards negative emotional (mood congruent) stimuli over neutral stimuli. Researchers have used an emotion face dot-probe task with neutral-sad facial expression pairings presented for 1000ms to document this pattern of biased attention in depressed adults (Gotlib, Krasnoperova, Yue, & Joorman, 2004b). In the dot-probe task, participants are presented with two faces (one neutral, one emotional) on a screen for 500-3000 ms, followed by a dot, which replaces either the neutral face or the emotion face. Participants are instructed to respond as quickly and accurately as possible to indicate the side of the screen on which the dot appeared. Response time to the dot probe is measured to determine where participants were allocating their attention (i.e., to which face they were attending). For instance, a bias to attend to sad faces would be reflected in shorter reaction times when the dot is behind a sad face because participants' attention is focused on the sad face. A bias away from positive emotional (mood-incongruent) faces has also been found in depressed adults and is correlated with severity of depression and anxiety symptoms (Gotlib et al., 2004a).

While this bias has been thoroughly studied in depressed adults, it is unclear whether this pattern begins in adolescence. Researchers have only recently begun to examine this bias in children and adolescents and have found mixed results depending on the type of emotional stimuli presented. Researchers have replicated Gotlib et al's (2004b) findings in

depressed 9- to 17-year-olds, demonstrating an attentional bias towards sad (mood-congruent) faces presented for 1000ms (Hankin, Gibb, Abela, & Flory, 2010). Other researchers have used an emotion word dot-probe task and found no attentional bias for negative emotional words in neutral-sad word pairings in depressed teenagers (Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 2000; Dalgleish, Taghavi, Neshat-Doost, Moradi, Canterbury & Yule, 2010).

It is common for individuals to receive concurrent diagnoses of depression and one or more anxiety disorders. Co-occurring disorders are known as comorbidities. The number of currently depressed children and adolescents with comorbid anxiety is unclear. One review found comorbid anxiety in 15-61% of depressed children (Brady & Kendall, 1992), while another review concluded that between 30-75% of depressed children meet criteria for a comorbid anxiety disorder (Kovacs, 1990). Because it is clear that anxiety often co-occurs with depression during adolescence, we also assessed severity of anxiety symptoms in this study.

The present study was conducted to examine attentional biases for negative and positive valence stimuli in depressed adolescents. We hypothesized that depressed adolescents will demonstrate an attentional bias away from positive emotion (mood incongruent) stimuli and towards negative emotion (mood congruent) stimuli, while healthy controls will show the opposite pattern of attentional biases. We also examined the effects of anxiety symptoms on the obtained depression-associated findings.

Method

Participants

Two groups of adolescents (13-18 years; mean age of 15.3 years) participated in the study: adolescents diagnosed with current depression (MDD; n = 30; 23 female) and healthy control adolescents who had never experienced any Axis I disorders (CTL; n = 16; 10 female). 43.5% of the sample identified as Caucasian, 6.5% identified as African-American, 13% identified as Hispanic, 8.8% identified as Asian, 13% identified as Biracial, 13% identified as Other, and 2.2% did not report. 2.2% of the sample had an annual family income under $25,000, 4.3% were between $25,000-$50,000, 6.5% were between $50,000-$75,000, 8.7% were between $75,000-$100,000, 56.5% were over $100,000, and 21.8% did not report.

Because all participants were under 18 years of age, an initial telephone interview was conducted with one parent of each participant to confirm that the adolescent was between the ages of 13-18 and was fluent in English and to determine whether the adolescent was likely to meet study inclusion criteria. Participants in the MDD group were recruited through the Pediatric Mood Disorders Clinic in the Department of Psychiatry and Behavioral Sciences at Stanford University and from the local community. Participants in the CTL group were recruited from the same local community. This study was conducted in compliance with a university based IRB. Parents gave written consent and adolescents gave written assent to participate in the study. All participants were part of a larger study examining the behavioral and neural correlates of attentional biases in depression. Exclusion criteria for the MDD group were 1) a history of major neurological disorder or illness; and 2) comorbid Bipolar I Disorder or Attention Deficit Hyperactive Disorder. In addition, exclusion criteria for the healthy controls included any past or current psychiatric disorder.

Trained interviewers administered the Kiddie Schedule for Affective Disorders and Schizophrenia, (KSADS-PL; Kaufman et al., 1997) to both the adolescent and parent during their first session to establish eligibility. Participants were included in the depressed group if they currently met DSM-IV criteria for MDD and in the CTL group if they did not meet criteria for any current or lifetime Axis I disorder according to DSM-IV criteria.

Procedure

Participants came to the lab for two sessions. During their first session, the adolescent and one parent separately completed the KSADS-PL with a trained interviewer. This interview was used to determine participants' eligibility and group classification. Following the interview, adolescents were assigned to the MDD group if either the parent or adolescent endorsed five or more symptoms of depression on the KSADS-PL. Adolescents who had no current or lifetime history of an Axis I disorder were assigned to the CTL group. The adolescent was then asked to complete the Children's Depression Inventory and the Multidimensional Anxiety Scale for Children (see below) in order to assess the severity of depression and anxiety symptoms, respectively. The score on the CDI was used to confirm the group classification. Adolescents assigned to the MDD group needed to have a CDI score above the clinical

Mean (SD)	*Participants*	*Age*	*CDI* **	*MASC* **	*MASC-Social* **
CTL	16 (10 F)	14.8(1.5)	3.0 (2.9)	36.0(13.8)	8.33(5.0)
MDD	30 (23 F)	15.5(1.4)	23.8 (9.6)	59.7(16.2)	17.47(6.2)

Table 1. Participant Characteristics: Depressed (MDD) and healthy control (CTL) adolescents. $^{**}p < 0.001$

cut-off score of 16 (Timbremont, Braet, & Dreessen, 2004). During their second visit to the lab, participants completed the emotion face dot-probe task.

Measures

Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS-PL). This standardized clinical interview assesses current and lifetime diagnoses for anxiety, mood, psychotic, alcohol and substance, behavioral, and eating disorders in children according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV; American Psychiatric Association, 1994). The KSADS-PL has been shown to yield valid and reliable psychiatric diagnoses (Kaufman et al., 1997).

Emotion Dot-Probe Task. A set of 35 faces expressing happy, sad, and neutral emotions was selected from the MacArthur Network Face Stimuli Set (http://www.macbrain.org/faces/index.htm), developed by The Research Network on Early Experience and Brain Development. The set of faces for the current study was selected from the larger validated set of 646 faces to include equal numbers of males and females with neutral, happy, and sad expressions, and an equal number of faces of different races (Tottenham et al., 2009). These faces were used as the stimuli in the dot-probe task, which is used to measure attention allocation and biases to positive and negative emotional information. In this task, a fixation cross is presented in the center of the screen. The cross then disappears and is replaced by a pair of faces, one on the left side of the screen and one on the right side of the screen. The pairs of faces were always composed of an emotional face (happy or sad) and a neutral face. The side of the screen on which the emotional face was presented was counterbalanced throughout the task. Both faces in each pair were of the same actor. The face pairs were presented for 1000ms followed by a dot probe that appeared in the location of one of the two faces. Using their right or left index finger corresponding with the right or left side of the screen, participants made a keyboard button-press response to the location of the probe. Their reaction time for each trial was recorded. Participants completed six practice trials without the face-pairs (i.e., just the dot probes) and four practice trials with the face-pairs before completing ninety-six trials of the emotional-neutral face pairings. The dot probe appeared at equal rates behind the neutral and the emotional faces.

Depression and anxiety symptoms. All adolescents completed the 27-item version of the Children's Depression Inventory (CDI; Kovacs, 1985), a self-report measure of depressive symptomatology in children between 8 and 17 years of age. The CDI assesses depressive symptoms during the past two weeks, and each item is scored on a three-point scale. The adolescents also completed the 39-item Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997), a self-report measure of anxiety symptoms in children between 8 and 19 years old. Studies have demonstrated good reliability and validity of both the CDI and MASC as measures of depression and anxiety respectively (Kovacs, 1985; March et al., 1997).

Statistical Analysis of Attentional Bias

Reaction times were recorded in order to compute attentional biases. Reaction times are faster when the dot-probe location is congruent with the face to which participants are attending than when the location and the attended face are on opposite sides of the screen, or incongruent; thus, we can infer to which face participants are attending based on their reaction times. Given findings that selective attentional biases in depression occur at longer stimulus durations (Joormann et al., 2007), the face stimuli were presented for 1000ms. An attentional bias score is calculated from participant reaction times during each condition by subtracting the reaction times for congruent trials, when the dot appears behind the emotional face, from the reaction times from the incongruent trials, when the dot appears behind the neutral face:

$$(1/2[(RpLe\text{-}RpRe)+(LpRe\text{-}LpLe)])$$

where R=right position, L=left position, p=probe and e=emotional stimulus (see Gotlib et al., 2004b). A negative attention bias score indicates that the participant has a bias away from the emotional face and towards the neutral face, while a positive attention bias score indicates that the participant has a bias towards the emotional face and away from the neutral face.

Results

Participant Characteristics

Demographic and clinical characteristics for the depressed and control adolescents are presented in Table 1. The two groups of participants did not differ in gender distribution, $x2(1,46) = 1.033$, $p = 0.309$. The two groups also did not differ in age, $t(44) = -1.547$, $p = 0.129$ or parent's annual salary $t(34) = 1.2659$, $p = 0.21$. As expected, the MDD group obtained significantly higher scores than did the control group on both the CDI, $t(44) = -8.437$, $p < 0.001$, and the MASC, $t(43) = -4.835$, $p < 0.001$.

Statistical Analysis of Attentional Bias between MDD and CTL

Reaction times were analyzed only for correct responses. In addition, reaction times less than 100ms and greater than 1500ms were removed to minimize the influence of outliers. Participants who had outlying reaction times that made up more than 5% of their data were excluded from analysis.

A 2 (Group: CTL, MDD) x 2 (Valence: Positive, Negative) repeated-measures analysis of variance (ANOVA) conducted on reaction times (bias scores) yielded a significant main effect of valence, $F(1,44) = 5.164$, $p < 0.05$; neither the main effect of group, $F(1,44) = 1.435$, $p = 0.237$, nor the interaction of group and valence, $F(1,44) = 0.145$, $p = 0.75$, was significant. Follow-up t-tests revealed that there was no significant difference in bias between groups for positive stimuli, $t(44) = 1.157$, $p = 0.253$, or negative stimuli, $t(44)=0.544$, $p = 0.589$. The CTL group showed a bias (B) toward positive faces ($B = 5.2285$) and away from negative faces ($B = -5.6112$) and the difference between these two valences was not significant, $F(1,44) = 2.699$, $p =0.108$, whereas the MDD group showed a bias away from positive faces ($B= -1.1176$) and a significant bias away from negative faces ($B = -8.8473$). The difference between these two valences was not significant, $F(1,44) = 2.571$, $p = 0.116$ (see Figure 1). The CTL group demonstrated the expected pattern of attention bias while the MDD group showed avoidance of both positive and neutral faces. These results are not consistent with our hypothesis. However, given that the sample has comorbid anxiety disorders, which have previously been associated with avoidance of stimuli, we used the MASC to control for symptoms of anxiety in order to examine whether the comorbid anxiety was contributing to the obtained pattern of attentional biases.

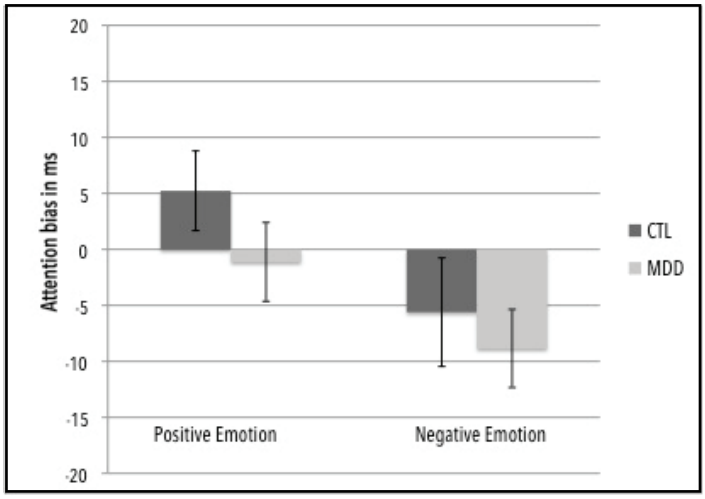

Figure 1. Attention bias in depressed (MDD) and healthy control (CTL) adolescents.

Statistical Analysis of Attentional Bias between MDD and CTL Controlling for Anxiety

A 2 (Group: MDD, CTL) x 2 (Valence: Positive, Negative) repeated-measures analysis of covariance (ANCOVA), covarying participants' scores on the MASC-Social subscale, was conducted to examine the effects of social anxiety on the processing of emotional and neutral faces. This analysis yielded a marginally significant interaction of group and valence, $F(1,43) = 3.739$, $p = 0.06$; neither the main effect of valence, $F(1,43) = 1.926$, $p = 0.172$, nor of group, $F(1,43) = 0.321$, $p = 0.574$, was significant. Post-hoc t-tests revealed a significant group difference for positive valence, $t(44) = 2.002$, $p = 0.05$, with the CTLs showing a bias towards positive stimuli ($B = 9.054$) and the MDDs showing a bias away from positive stimuli ($B = -3.158$). There was no significant group difference for negative valence, $t(44) = 1.009$, $p = 0.318$; both the CTL and MDD groups showed a bias away from negative faces (CTL: $B= -11.934$; MDD: $B = -5.471$; see Figure 2). After controlling for anxiety, the MDD group showed the expected bias away from positive faces and, inconsistent with the literature, also a bias away from negative faces.

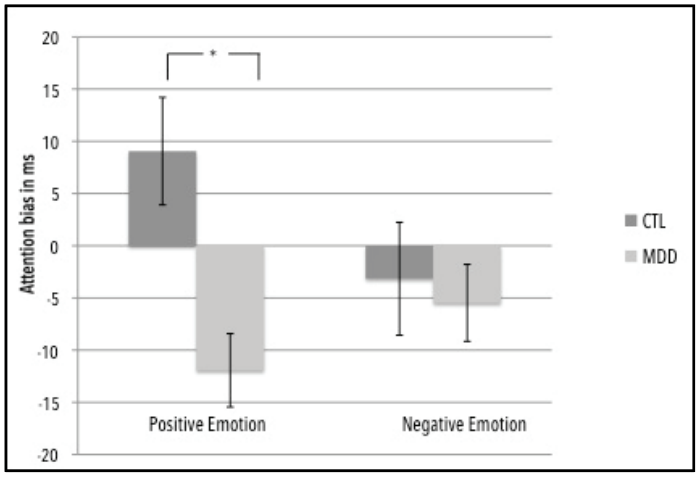

Figure 2. Attention Bias in depressed (MDD) and healthy control (CTL) adolescents when controlling for anxiety using MASC Social Scale. *$p = 0.05$

Discussion

Investigators have found that depressed adults show a biased pattern of attention towards negative emotional stimuli and away from positive emotional stimuli; however, the literature about depressed adolescents is not as consistent. This study was conducted to examine attentional biases in depressed adolescents.

Our initial analyses revealed a pattern of attention away from both positive and negative emotion faces for depressed participants. While the bias away from positive faces was expected, the bias away from negative faces was counter to our hypothesis. Discrepancies in attention allocation may be due to immature development of cognitive control, which manifests as increased attention to irrelevant information and difficulties in the processing of emotional information, which can impair attention allocation to relevant stimuli (Hare & Casey, 2005; Ladouceur et al., 2005). While neurological development is one possible explanation for our findings, another possible explanation is the interaction of depression and anxiety.

Given the differences in presentation of symptoms and overall symptom severity for comorbid individuals, it is possible that those experiencing depression and anxiety will have different cognitive biases than would be expected for individuals with only depression or only anxiety. In our sample, the MDD group scored significantly higher on the MASC than did the CTL group. We posited that anxiety symptomatology in the MDD group may interact with depression to produce the bias away from negative emotion faces.

When the MASC-Social subscale was used as a covariate in the analysis, there was a trend-level interaction of group and valence that was not present in the initial analysis, indicating that anxiety plays a role in the pattern of attention away from positive and negative emotion faces. When controlling for anxiety, the bias away from positive faces was also strengthened, further supporting the hypothesis that depressed adolescents will avoid mood-incongruent information, a pattern of attention that will be even more pronounced when anxiety is controlled.

It is clear that anxiety is playing a role in the mixed findings found in the depression literature, especially in the adolescent literature. One possible reason for these findings involves the biased processing of neutral faces in individuals with social anxiety. Even though studies in the adult literature have paired emotion and neutral faces, neutral faces may not be emotionally neutral for individuals with social anxiety. Thus, it is possible that the neutral faces are capturing attention, creating an apparent bias towards neutral faces and away from negative emotional faces in neutral-sad face pairs because they are interpreted as threatening by individuals with social anxiety (Cooney et al., 2006; Yoon & Zinbarg, 2008). Another possibility is that the adolescents with comorbid depression and anxiety find emotion faces threatening and are avoiding emotion faces altogether, as has been demonstrated in adults with social anxiety (Chen, Ehlers, Clark & Mansell, 2002). The interpretation of the neutral face by individuals with social anxiety and depression should be explored further to ensure that neutral faces are not confounding attentional allocation to emotion faces.

This study has several limitations. First, because this study was designed to assess attentional biases in depression, we used sad faces; we did not include a threat. Most of the literature on attention in currently anxious adults and children has focused on attention to angry/threatening faces. Given the current findings, it is important to consider the types of stimuli when measuring attention allocation in a comorbid sample. Previous studies of anxiety disorders have included pairings of threatening-neutral faces in addition to sad-neutral and happy-neutral faces, which could have helped to elucidate the nuances of the anxiety-depression comorbidity. Previous studies that have included angry or fearful faces, however, have not consistently found a bias towards these threatening stimuli

in depressed adults and children. Therefore, while a neutral-threat face pairing could be used to attempt to isolate attention allocation due to anxiety, researchers have not consistently found a bias towards angry or fearful faces in anxious samples, especially in socially anxious individuals (Mogg, Philippot & Bradley, 2004; Sterling, Eley, & Clark, 2006).

A second limitation of this study is that the duration of the stimuli in the dot-probe tasks permits attention shifts during stimulus presentation. In the present study, the faces were displayed on the screen for 1,000 ms. Previous studies found adults with social phobia showed no bias in attention for angry, neutral, or happy faces in an emotion face dot-probe task with neutral-emotional pairings for 1250ms durations (supraliminal presentation), while a bias towards angry faces was found when the stimulus presentation was shortened to 500ms duration (subliminal presentation) (Mogg, Philippot, & Bradley, 2004). One study found that children ages eight to eleven with social phobia avoid fearful and angry faces in an emotion face dot-probe task with faces presented for 1000ms durations (Sterling, Eley, & Clark, 2006). Participants could be changing their allocation of attention between the two faces before the presentation of the dot because of the longer, supraliminal presentation of faces in the current version of the task.

Given the limited literature on attention bias in adolescents, this study is one of the first to consider anxiety comorbidity when evaluating attention bias in a depressed sample. Unexpectedly, this study found that depressed adolescents exhibit an unexpected bias away from negative emotion faces in addition to the expected bias away from positive faces. One possible explanation for this involves the presence of comorbid anxiety disorders. While there are specific patterns of attentional bias expected in depression and anxiety found in adults, adolescents currently experiencing depression with underlying sub-clinical or clinical levels of anxiety present a different and unexpected pattern of attention, which could be the result of biased processing of sad and neutral faces.

Although anxiety and depression are separate disorders, there is considerable overlap between the two, and it is often difficult to differentiate depression from anxiety in clinical samples. This interaction may lead to discrepancies in findings across studies. Moving forward, researcher need to acknowledge the overlap of the two disorders and attempt to understand cognitive biases from both perspectives. Future research should examine biases to specific negative emotion faces (sad, angry, fearful) in anxious and depressed individuals by including a threat condition to the emotion dot probe task, in addition to the sad and happy conditions. In addition, using both subliminal and supraliminal presentations will help disentangle possible attention shifts. Researchers also need to be aware when designing and recruiting for studies that comorbid anxiety can alter the presentation of depression so it is important to take steps to control and account for anxiety when studying depression. These methodological and conceptual changes moving forward can help us understand why some studies find a bias that is lacking in other studies. The current findings emphasize the importance of considering comorbid diagnoses to better understand how the interaction of symptoms contributes to the biased processing of information in adolescents experiencing depression and comorbid anxiety.

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Rhae Ana Gamber graduated from Drexel University in March 2014 with a Bachelors of Science in Psychology. She has worked as an undergraduate research assistant in Dr. Brian Daly's Pediatric and Child/Adolescent Psychology lab, focusing mainly on childhood trauma and school mental health research. She also works as a Research Coordinator at Nemours Children's Clinic in Philadelphia, PA. Her experiences there are in pediatric endocrinology and include cognitive testing in genetically disordered populations, as well as the clinical trials and the incorporation of new treatment devices in diabetic populations. She aspires to pursue a graduate education in child psychology.

What interests you the most about psychology?

I love how psychology is part of every field, and it's basic principles can be applied in any conversation. Psychological research is the perfect tool to better understand ourselves as human beings, which I believe is the basis of all disciplines.

Explain the process of putting your manuscript together.

Outlines and timelines! The first step of writing my manuscript was sitting down with my faculty adviser, Dr. Brian Daly, and outlining the entire paper. This allowed me to focus on individual sections without ever feeling overwhelmed by the process. We also planned my progress, dedicated the first term to literature review, the second to methods and continued data collection, and my final term to data analysis and discussion.

Besides research, what do you do for fun?

I love spending time with friends and family, exploring the food scene in Philadelphia , working out, and traveling any chance I get.

Was there any particular experience that sparked your research interests?

Working with children has always been my main interest, and actually preceded my interest in psychology and research. Many of my family members are teachers, and hearing stories about some of their students made me realize how much more we need to learn about child development, family structure, and pathology. I found psychological research to be the ideal platform to tackle these questions.

How do you juggle all of your responsibilities?

Time management! It may sound cliche, but it's true! I make a schedule, and I follow it. I avoid stress by taking projects on piece by piece, and also by setting aside time for myself.

The Differential Impact of Childhood Trauma Types on Executive Functioning in Young Adults

Rhae Ana Gamber
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Childhood trauma is associated with a variety of physical, psychosocial, and cognitive sequelae, including decrements in higher-order cognitive processes such as executive functions (EF). However, prior research has not yet investigated the relationship between specific types of childhood trauma (e.g., physical, emotional, and sexual abuse, physical and emotional neglect) and EF in young adults. A more specific understanding of potential relationships between trauma types and EF may allow for tailored identification and treatment of this population. Additionally, both objective and subjective measures of EF yield discordant findings that warrant further investigation. This study utilized self-report and psychometric measure of EF (D-KEFS and BRIEF-A), a self-report trauma questionnaire (CTQ), and psychometric intelligence testing (WASI). The sample was composed of college students (N = 125) between the ages of 18 and 23 years. Independent samples t-tests revealed that participants with history of trauma had significantly lower scores on a test of inhibition and switching ability (Color-Word Interference), [t(123) = 2.50, p = 0.01] and significantly greater self-reported EF problems (BRIEF-A GEC), [t(116) = -3.92, p < 0.01]. Linear regression analyses show physical neglect to significantly predict poorer switching ability when controlling for all other trauma types by the D-KEFS Trail Making Test [b = 0.21, t(123) = 1.99, p = 0.05] and the D-KEFS Color Word Interference test [b = 0.27, t(123) = 2.58, p = 0.01]. Additionally, linear regression analyses show emotional abuse to significantly predict self reported executive function deficit as measure by the BRIEF-A Global Executive Composite [b = -0.44, t(123) = -3.192, p = 0.002]. Psychological treatment of college students with history of emotional abuse or physical neglect may be supplemented with assessment and treatment strategies around potential EF deficits. Future studies should seek to examine whether these EF deficits persist into later adulthood.

Childhood trauma is defined by instances of and exposure to early life stress or child maltreatment, often classified as sexual, physical, and emotional abuse, as well as neglect (Majer, Nater, Capuron, & Reeves, 2010). Traumatic experiences in childhood have been linked to a variety of both short and long term negative cognitive and psychosocial consequences, with recent interest in their impact on executive functioning skills (Majer et al., 2010). Childhood trauma is a broad term, and therefore the investigation of more specific types of trauma is important to better understand potential relationships with executive function. Findings related to the various behavioral, psychological, and psychosocial outcomes of individual trauma types may support the treatment decisions of clinicians and psychologists. However, as the investigation of executive function decrements is a fairly new one, little attention has been given to the individual outcomes per trauma type. Thus, potential research findings may be able to inform more individualized treatment, and ultimately produce more successful outcomes for young adults exposed to trauma during childhood.

Types of Childhood Trauma

Emotional abuse

Of all trauma types, childhood emotional abuse is perhaps the most difficult to define. While it undoubtedly constitutes exclusive sequelae with unique consequences, there is less clarity when distinguishing emotional abuse from different parenting styles and behaviors. This subjectivity poses a particular challenge to research and the study of emotional abuse (Riggs, 2010). The extant literature generally defines this type of trauma as psychological maltreatment and non-physical aggression (Spertus, Yehuda, Wong, Halligan, &

Seremetis, 2003). Specific behaviors that fit these descriptions include extreme and relentless rejection and/or intrusiveness that are frightening to the child and lacks reconciliation initiated by the parent. Examples of rejection include antipathy and degradation, and intrusiveness includes behaviors such as controlling, guilting, and role-reversing (Riggs, 2010). While previous research on emotional abuse is largely initiated and supported by attachment and developmental theorists focusing on parental behaviors (Riggs, 2010), it is important to note that parents may not be the only perpetrators of emotional abuse. Other family members, stepparents, parent's dating partners, siblings, peers, and other adults could be involved in such dysfunction (Dong et al., 2004).

Previous research findings support a wide variety of emotions and behaviors associated with an emotionally abused child. For instance, low self-esteem, increased levels of depression, suicidality, and decreased mental and physical health are all considered potential indicators of emotional abuse (Spertus et al., 2003). However, research efforts have struggled to examine the effects of emotional abuse in the absence of other types of abuse, as it is common for multiple types of maltreatment to occur within similar timeframes. Studies that were able to investigate emotional abuse alone found that these children experienced long-term consequences that included poor body image, sexual dysfunction, anxiety, depression, and interpersonal sensitivity (Spertus et al., 2003). Other studies have found disorders of emotional regulation and impulse control, such that children and adolescents have a difficult time appropriately identifying and expressing their own emotions (Riggs, 2010). Furthermore, findings that support impulse control problems as a long-term consequence in children who have experienced emotional trauma underscore the potential for decrements in executive functioning.

Physical abuse

A general definition of physical abuse is the presence of a non-accidental injury resulting from overt physical violence or excessive punishment by an adult (Malinosky-Rummell & Hansen, 1993). Because these acts typically occur in discrete and low frequency episodes (Malinosky-Rummell & Hansen, 1993), physical abuse may not be readily detectable despite its clear characterization. Still though, prevalence rates vary among reports and studies due to discrepancies in definitions and methods for data collection, i.e. parent versus child report (Fisher, 2012). Additionally, prevalence rates are based on reported cases, so it is fair to assume known rates represent an underestimate of the true prevalence (Malinosky-Rummell & Hansen, 1993). One report that defined physical abuse as physical maltreatment involving intentional use of physical force that could or did cause injury, including beating, punching, strangling, burning, and poisoning, and excluding smacking on the bottom with an open hand, stated a prevalence rate of 1-5% in the past year, and 5-19% within the past 17 years (Fisher, 2012).

Not surprisingly, a variety of short and long-term effects of physical abuse have been identified in the literature. Aside from obvious marks and bruises, short-term effects can serve as indicators for these violent occurrences. Examples include internalized psychological problems such as hopelessness, depression, and low self-worth, increased aggression towards adults and peers, perceptual-motor deficits, and decreased general intellectual functioning. In adolescence, higher rates of noncompliance, nonaggressive conduct disorders, and delinquency occur in physically abused children as compared to non-abused peers. A wide range of emotional problems including anxiety, phobia, paranoid-ideation, psychoticism, and suicide ideation are also indicators of physical abuse. Finally, several studies support academic and intellectual delays and significantly lower IQ scores than non-abused children (Malinosky-Rummell & Hansen, 1993).

Unfortunately, these acute negative effects often progress into long-term consequences. Research findings indicate that victims of childhood physical abuse demonstrate higher rates of violence during adolescence and adulthood, toward non-familial persons, children, and romantic partners. In fact, one study cites that one-third of physically abused children go on to abuse their own children. Additionally, a stronger tendency to abuse drugs and alcohol has been linked to the resulting feelings and cognitions of childhood abuse, as 15%-35% of adult substance abusers report being physical abused as children. Finally, these adults also tend to have negative feelings about interpersonal relationships as well as interpersonal sensitivity, defined as shyness, self-consciousness, and feeling disliked or misunderstood (Malinosky-Rummell & Hansen, 1993).

Sexual abuse

Sexual abuse is defined such that it includes a continuum of severity and incidents, but is principally characterized by

sexual experiences within the first 18 years of life with an adult or someone at least five years older than the child (Dube et al., 2005). The following instances are each considered childhood sexual abuse: a child that was touched or fondled in a sexual way, a child that was encouraged to touch their perpetrators body in a sexual way, an attempt to have any type of sexual intercourse, and actually having sexual intercourse (Dube et al., 2005). Perpetrators can be relatives, family friends, or strangers, male or female. Prevalence rates estimate that 20% to 30% of female children have experienced some type of sexual abuse (Dube et al., 2005), while these estimates range from 1% to 16% in males (Briere & Elliott, 2003). Again, these rates can be assumed to be underestimates as they only include reported cases of sexual abuse (Briere & Elliott, 2003).

Indicators for childhood sexual abuse can vary among males and females. During adolescence, male children who were sexually abused are more likely to demonstrate externalizing behaviors such as delinquency and heavy drinking, while internalizing behaviors such as suicide ideation and disorder eating are more common among females. An increased risk for such trauma exists in families who also experience divorce, domestic violence, substance abuse, and engage in emotional unavailability. Not surprisingly, many of these indicators contribute to a variety of negative long term consequences associated with childhood sexual abuse. For example, the literature provides strong evidence supporting childhood sexual abuse as a risk factor for alcohol problems, illicit drug use, suicide attempts, marrying an alcoholic, and adult marital and family problems equally evident in males and females (Dube et al., 2005). Additional evidence indicates childhood sexual abuse to be a risk factor for a range of psychiatric problems and disorders including depression, phobias, obsessive-compulsive disorder, panic disorder, posttraumatic stress disorder, and sexual disorders (Briere & Ellott, 2003). Though age of occurrence, severity, and relatedness to abuser can all impact symptomology, it is clear that long term consequences of childhood sexual abuse impact adult function in a variety of domains.

Emotional neglect

While emotional abuse is a form of psychological maltreatment, emotional neglect is a distinct trauma characterized by emotional deprivation or the absence of a nurturing environment (Spertus et al., 2003). A child is emotionally neglected when he or she is not made to feel important, special, or loved by family members. Neglect may be occurring in families that do not feel close, look out for each other, or provide strength and support (Dong et al., 2004). One study reported that approximately 15% of children encounter emotional neglect. Furthermore, findings from studies support that emotional neglect is rarely the only form of maltreatment occurring, and is often worsened by concomitant adverse childhood events (Dong et al., 2004).

The importance of a nurturing and supportive environment to ensure proper and healthy development is a common principle shared by various psychological theorists. Certainly, emotional neglect is in direct opposition to these models, and therefore poses detrimental long-term effects. Indicators of such neglect include the child's lack of self-worth and negative beliefs regarding themselves and their efficiency, as well as minimal adaptive behaviors and poor self care. Long-term consequences are similar to those of emotional abuse, and include an array of physical and emotional distress, and lifetime exposure to trauma. Though not considered post-traumatic stress disorder, long-term symptomology is very similar but lacks life-threatening nature (Spertus et al., 2003). Studies have also supported an association between childhood emotional neglect and the development of personality disorders in adolescence and adulthood. For instance, both paranoid personality disorder and avoidant personality disorder symptoms are associated with emotional neglect experienced in childhood (Johnson, Smailes, Cohen, Brown, & Bernstein, 2000). With this, it is clear that a lack of a loving childhood can be an equally devastating form of abuse.

Physical neglect

Contrary to physical abuse characterized by aggressive and forward acts from adults, physical neglect is defined by the lack of parental presence (Yates, Dodds, Sroufe, & Egeland, 2003). This includes incompetent and irresponsible management of a child's daily care, inadequate nutrition and health care, and dangerous home environment due to insufficient supervision by a primary caregiver (Yates et al., 2003). A child is physically neglected if he or she does not have enough to eat, is not taken care of or protected, whose parent's drug use interferes with their care, has to wear dirty clothes, or is not taken to the doctor (Dong et al., 2004). With 30 out of every 1000 children in the United States suffering from physical neglect, this form of abuse

is considered the most common form of child maltreatment (Hilyard & Wolfe, 2002).

Several risk factors are associated with physical neglect and can be considered potential indicators. These factors include unemployed, single-parent families, larger family size, parental substance abuse problems, maternal depression or chronic mental illness, parental criminal history (Hartley, 2002), chronic poverty, homelessness, family break up and poor pre- and post- natal care (Hilyard & Wolfe, 2002). Unlike trauma types such as abuse, which occur as incidences, neglect is typically a chronic situation less easily identifiable. Long-term consequences of childhood physical neglect include lower IQ scores, higher levels of delinquency, adult criminal behavior and violent criminal behavior, and increased likelihood for a diagnosis of a personality disorder (Hilyard & Wolfe, 2002). Studies have found an especially strong association between physical neglect and Cluster A Personality disorders, including schizotypal personal disorder developing in late adolescence or early adulthood (Johnson et al., 2000). Still though, due to the comorbid nature of neglect and childhood maltreatment, identifying specific long-term consequences of childhood trauma types is a difficult task.

Executive Function

Executive functions (EF) are a set of cognitive processes key to self-regulation (Williams, Suchy, & Rau, 2009). Unlike many of the automatic brain processes, this skill set is classified by effortful and controlled cognitions, actions, and responses. Processes such as working memory, present and future planning, organization, inhibition, and initiating and monitoring one's own behavior all constitute executive functions. Collectively, these skills support every-day efficiencies and adaptive responses that aide in goal-oriented behavior (Williams et al., 2009).

While these skills are less complex individually, when functioning together they allow for the solving of novel problems, the use of new information to modify behavior, the development of strategies for complex actions, overriding emotions, and follow-through with plans. EF skills are vital to critical thinking and stress regulation: functions that are especially important for students and individuals in the workforce. However, like any skill, various levels of functioning exist and individual differences are evident (Williams et al., 2009).

Measuring Executive Functioning

Gathering both objective and subjective measures provides the most robust assessment of executive functioning. While objective psychometric measures are most frequently used in research studies, subjective behavioral reports provide additional information and can serve as useful comparisons (Biederman et al., 2008). Notably, previous studies that utilized both methods of measurement have yielded findings that suggest a low to moderate correlation. In most cases, it appears that behavioral measures are more sensitive, identifying a larger with-deficit group as compared to what is identified by objective, performance-based measures (Biederman et al., 2008). This inconsistency between measures creates a clinical challenge, as these measures are often used to formulate diagnoses and treatment decisions. Overall, the extant literature provides evidence that objective and subjective measures cannot be used interchangeably, as they have been shown to identify different groups within a singular population (Biederman et al., 2008). With this, test reliability and validity are threatened, test mistrust is warranted (Jahedi & Mendez, 2013), and the need for investigation of objective versus subjective measure is evident.

Objective. The advantage of objective measures is the standardized, normed, and evidence-based nature of these measures (Delis, Kaplan, & Kramer, 2001). In terms of disadvantages, the controlled environment in which testing is completed makes the situation less realistic or true to normal functioning. For example, perhaps a child is able to demonstrate organization in a controlled lab setting, but fails in the potentially chaotic classroom setting. A second example of a potentially disadvantageous element is the game-like structure of many objective measures (Delis et al., 2001). While experimenters want their subjects to be engaged, perhaps the interest obtained in a game-like task does not generalize to real-life stress situations.

Some neuropsychological tests that are commonly used to assess executive functioning include the Wisconsin Card Sorting Test (WCST), the Trail Making Test-Part B (TMT-B), and verbal and figural fluency measures. Experimental tasks are also advantageous measures, utilizing cognitive control tests such as switching tasks, Go/No-Go and reversal learning, and Stroop tasks. Additional tests assess attention, working memory, and emotional decision-making (Suchy, 2009). The current study utilizes the Delis-Kaplan Executive Function System (D-KEFS)

to objectively measure executive function, which is effective in isolating fundamental skills by using subtests that are specifically designed to examine select cognitive components. The various measures within the D-KEFS account for initiation of problem-solving behavior, verbal and non-verbal concept-formation skills, the transfer of concepts into action, abstract expression of conceptual relationships, and flexible thinking and behavioral response (Delis et al., 2001).

Subjective. Subjective measures provide a way to collect data in situations where objective measurement is difficult or too costly to obtain, and when subject matter is vaguely defined (Jahedi & Mendez, 2013). In regard to EF, it is the isolation of each function that poses a challenge to researchers. Advantages of subjective measures include simplicity and cost-effectiveness (Biederman et al., 2008), as well as the ability to capture some of the implicit events that cannot be obtained by objective measures (Jahedi & Mendez, 2013). However, there are also several disadvantages that have been cited in literature. First, subjective behavioral reports have been shown to be subject to responder bias related to order, scale, recall, and even psychological factors. These measures depend on the responses provided by participants, which are susceptible to personality, memory, opinion, and an overall uncontrolled norm. Secondly, there is minimal and even sometimes negative correlation with independent, objective measures. And third, the data collected can often be difficult to interpret, particularly when ordinal scales are used (Jahedi & Mendez, 2013). The current study utilizes the Behavior Rating Inventory of Executive Function- Adult Version (BRIEF-A) to subjectively measure executive function, which considers the individual's everyday environment at home, in school, or at work as important venues for observing executive function, rather than relying on a singular performance (Gioia, Isquith, & Kenworthy, 2000)

Childhood Trauma as a Potential Threat to Executive Functioning

A compelling body of research has supported the relationship between childhood trauma and various cognitive and psychosocial outcomes in both the short and long term. With a focus on the behavioral, mental, and social consequences, an increased risk for substance abuse, personality disorders, and suicide are among a variety of problems that have been identified subsequent to childhood trauma. Furthermore, many psychiatric disorders that have been linked to childhood disturbance are in fact characterized by cognitive impairment, such as depression and PTSD. Given these findings, more recent studies have extended investigation to potential cognitive deficits, including working memory, executive function, psychomotor speed and sustained attention, as well as academic achievement (Majer et al., 2010). One research team found both childhood emotional abuse and physical neglect to be significantly associated with impaired working memory, and sexual abuse and physical neglect to be negatively associated with academic achievement. Notably, though, the results of their study revealed minimal deficits in executive function assessed by tasks that required shifting tasks and rule acquisition (Majer et al., 2010). Since many definitions of EF include working memory (Suchy et al., 2009), there is a need for continued investigation of childhood trauma in association with executive function. Overall, these results provide evidence for the relationship between childhood trauma and cognitive performance, so further study is warranted.

Other studies have focused on more general childhood stress, but include all of the trauma types mentioned. Early life stressors (ELS) are defined as the exposure to a single or multiple events during childhood that exceeds coping resources (Pechtel & Pizzagalli, 2011). Several investigations of ELS have found decrements in a range of executive functions, particularly inhibitory control and planning, as well as overall global cognitive functioning. For instance, one study focusing on these tasks and their respective brain regions found longer reaction times in participants who experienced early life stressors, indicated by performance as well as activations in the inferior frontal cortex, which is associated with cognitive inhibitory control, and the striatum, which is associated with response control. Interestingly, the study also revealed that such impairments are found in related psychopathologies, such as depression (Pechtel & Pizzagalli, 2011).

Because of the challenge posed for researchers to purposefully measure executive function prior to and after a traumatic event, findings usually rely on comparisons between normal and traumatized populations. Due to these methodological constraints, exact decrement in individual executive function cannot be measured. However, one team of researchers studied the effect of therapeutic treatment on post-traumatic-stress-disordered women, finding particular improvements in neuropsychological tests of executive

function. Following therapy, group effects were found for enhancements in cognitive flexibility, set shifting, and organization and planning (Walter, Palmieri, & Gunstad, 2010). Though this exploratory study did not utilize randomization or a controlled group, the findings suggest that executive function improvements can occur in traumatized populations if they participate in therapy.

The present study aims to contribute to the understanding of types of trauma and their individual effect on executive functioning. This study will add to the literature by separating five trauma types, and treating each as an individual trauma to more specifically investigate the resulting sequelae. This is an important topic because findings may allow for more personalized identification and support for these individuals, and therefore more positive outcomes. Results may contribute to the development of predictive measures based on type of trauma experiences, allowing for the most immediate and evidence-based treatment.

Secondly, the present study is novel in that it utilizes a college-enrolled sample. Participants are within a small age range from a variety of backgrounds and experiences, though at a level of education that demands and relies on executive function skills. Furthermore, previous studies present conflicted findings regarding college samples. Where one team found impairment in executive function in college-aged victims of repeated childhood sexual assault females (Navalta, Polcari, Webster, Boghossian, & Teicher, 2006), another study found no difference in executive functioning in groups diagnosed with post-traumatic-stress disorder following a traumatic childhood experience when compared to their normal counterparts (Twamley, Hami, & Stein, 2004). Understanding the individual effect of trauma type in college-aged students could allow for improved university resources and better equip university officials and counselors to optimize student functional outcomes.

Finally, the present study utilizes both objective and subjective measures of executive function. As previously mentioned, both self-report and performance based assessment add value to investigations. Because this study employs both methods, the individual effect of trauma types can be examined per behavioral data, psychometric data, and collectively.

We hypothesize that participants who report childhood trauma will also report greater executive function decrements and achieve lower scores on measures of executive function performance. Secondly, we hypothesize that groups of individual trauma types will not be impacted equally, and specific trauma types will emerge as more sensitive to deficits. Thirdly, we hypothesize that subjective and objective measures of executive function will not significantly correlate.

Method

Participants

Subjects in this study were 125 undergraduate college students (34 men [27.2%] and 91 women [72.8%]) from a private, urban university. The sample had an average age of 20.44 years (SD = 1.4), with ages ranging from 18 to 23 years (3 participants turned 23 years old between recruitment and testing). Demographic information was collected using a brief questionnaire designed for this study, in which participants were asked to report their age, gender, race/ethnicity, medical information such as medical and/or psychiatric diagnoses and treatments, and history of drug and alcohol use. Recruitment for the study consisted of university-wide emails, classroom solicitations, and poster advertisements. Respondents completed a 5-part eligibility questionnaire to determine their undergraduate status, age, English language proficiency, head injury status, and status of psychotropic medication use. Exclusion criteria included non-undergraduates, age outside the range of 18 to 22 years, non-proficient in English, head injury that resulted in loss of consciousness for more than 15 minutes or over night hospitalization, and the current use of psychotropic medication.

Within our sample, 72 participants (58%) self-reported some type of childhood trauma ranging anywhere from low to severe, while 53 (42%) did not endorse experiencing trauma of any kind. Based on responses to the retrospective measure of trauma exposure, the frequencies of types of trauma identified were as follows: 36.8% indicated being emotionally abused (n=46), 22.4% reported physical abuse (n=28), 11.2% revealed being sexually abused (n=14), 40% reported emotional neglect (n=50), and 16.8% reported physical neglect (n=21). Participants reported these traumas as low, moderate, or high in level of severity. In order to utilize the most robust sample size for analyses, we combined these categories to represent a single category of positive past history of childhood abuse. These frequencies are displayed in Figure 1.

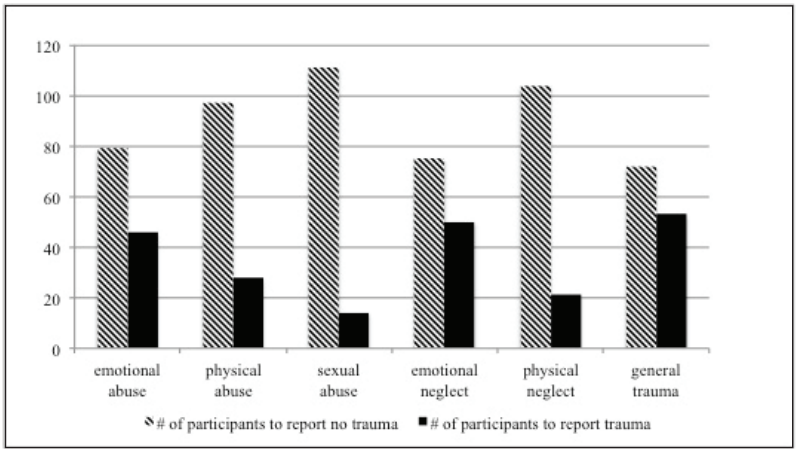

Figure 1. Frequencies of reported trauma by type as measured by the CTQ.

Procedure

Qualifying participants scheduled a two-hour time slot with study staff, in which the study purpose and procedures were explained and informed consent was obtained. Following this, each measure was individually administered in a specified order, starting with the study-designed demographic questionnaire. Next, we administered an objective measure of intelligence, the Wechsler Abbreviated Scale of Intelligence (WASI). Then two questionnaire-style measures were administered: the Childhood Trauma Questionnaire (CTQ) to classify type and severity of childhood trauma, and the Beck Anxiety Inventory (BAI). The objective measure of executive function was administered next, using four subtests of the Delis-Kaplan Executive Function Scales (D-KEFS): the Trail Making Test, Verbal Fluency, Color-Word Interference, and the Tower Test. Finally, the following two subjective self-report questionnaires were administered: the Beck Depression Inventory (BDI) and the Behavior Rating Inventory of Executive Function – Adult (BRIEF-A). Study procedures took approximately 90 minutes to two hours, and participants were compensated with a 40 dollar gift card for their time.

Analyses will include a t-test to compare scores amongst participants who have experienced general childhood trauma verses those who have not experienced general childhood trauma. Linear regressions will then be utilized to investigate the individual impact of each trauma type when controlling for the other types of trauma. Square root and inverse transformations wills be utilized to adjust skewed variables.

Measures

CTQ. The Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998) was used to measure childhood trauma. This measure is a self-report questionnaire identifying five dimensions of childhood trauma: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. The 25 items utilize a Likert scale ranging from "never true" to "very often true." Items within each subscale are a mix of references to experiences growing up and "I believe" statements. For example, sample items within the emotional abuse dimension include: "People in my family called me things like "stupid," "lazy," or "ugly,"' as well as "I believe that I was emotionally abused." The measure also considers the effects of minimization responses by including three items that detect minimization when "very often true" is selected. An example of this item type is "I had the best family in the word."

The CTQ demonstrates satisfactory to excellent reliability coefficients, ranging from .66 to .92 across the different abuse type scales. The measure is validated through confirmatory factor analyses, with all robust comparative fit indices greater than .90, ensuring viable and coherent constructs (Bernstein & Fink, 1998). Besides being a sound measure, the CTQ is especially important to this investigation because it classifies the traumas experienced by each participant, which base our differential analyses.

D-KEFS. Executive function was measured objectively using four subtests of the Delis-Kaplan Executive Function Scales (D-KEFS; Delis, Kaplan, & Kramer, 2001). The D-KEFS assesses various components of executive function using a game-like task design. The Trail Making Test subtest measures set-shifting, the Verbal Fluency subtest measures verbal domain, the Color-Word Interference Test measures inhibition, and the Tower Test subscale measures planning, rule-learning, and inhibition. The objective nature of the D-KEFS allowed for executive function scores to be obtained based on one-time performance. Reliability coefficients (Cronbach's alpha) for the five conditions of the Trail-Making subtest ranged from .20 to .82; the three conditions of Verbal Fluency ranged from .24 to .81; the four conditions of Color-Word Interference from .52 to .90; and the Tower Test Achievement Score reliability coefficient ranged from .41-.51 depending on age. The various subscales within the D-KEFS have been empirically validated against each other, as well as to other executive function measures such as the California Verbal Learning Test-II (CVLT-II) and the Wisconsin Card Sorting Test (Delis et al., 2001). The

D-KEFs is especially crucial to this study because it objectively measures various abilities that constitute executive function as a whole, and represents our psychometric measure of executive performance.

BRIEF-A. Executive function was measured subjectively using the Behavior Rating Inventory of Executive Function – Adult (BRIEF-A; Gioia, Isquith, Guy, & Kenworthy, 2000). This measure is a self-report rating scale consisting of 75-items pertaining to nine theoretically and empirically derived subscales: Inhibit, Shift, Emotional Control, Self-Monitor, Initiate, Working Memory, Plan/Organize, Task Monitor, and Organization of Materials. The BRIEF-A yields an overall score called Global Executive Composite (GEC). T-scores have been normed to a sample composed of 1050 self- and 1200 informant reports (Gioia et al., 2000).

Participants provide self-reported information regarding a variety of everyday behaviors associated with executive function skills. Each statement is rated as "never," "sometimes," or "always." Sample questions include "I am disorganized," "People say that I am easily distracted," and "I have good ideas but cannot get them on paper." The BRIEF-A was selected for this research because it allows participants to assess their own executive functioning based on every day occurrences, and it serves as our behavioral measure of executive function.

BAI. The Beck Anxiety Inventory (BAI; Beck & Steer, 1993) was used to measure emotional functioning. This self-report measure consists of 21 items in which symptoms of anxiety are rated from "not at all" to "severely." The BAI is psychometrically cogent, as internal consistency measured using Cronbach's alpha ranges from .92 to .94 in adult populations. Additionally, the BAI has been validated against several other anxiety scales (Fydrich, Dowdall, & Chambless, 1992).

BDI. The Beck Depression Inventory (BDI; Beck, Steer, & Brown, 1996) was also used to measure emotional functioning. This self-report measure consists of 21 items in which symptoms of depression are rated on a 0 to 3 scale. The inventory has achieved good internal consistency, measured by Cronbach's alpha as .90 for the total scale, and has been validated against the State-Trait Anxiety Inventory of Depression (STAI-D) and the State-Trait Anxiety Inventory of Anxiety (STAI-A) (Storch, Roberti, & Roth, 2004).

Though the BAI and BDI are not essential to the main research questions, these measures are important to our investigation as extant literature supports the increased rates of anxiety and depression in childhood trauma victims. Collecting these extra measures will be useful for further investigation in the future.

WASI. The Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) was used to measure intelligence. This is a widely used and accepted measure sampling performance abilities on verbal tasks: vocabulary and similarities; and performance tasks: block design and matrix reasoning. The measure is highly reliable, with reliability coefficients for the targeted age range varying from .88 to .94 across the four subtests. It is also validated through moderate to strong correlations with the Wechsler's Adult Intelligence Scale-III (WAIS-III), the Wechsler Intelligence Scale for Children-III (WISC-III), and the Wechsler Individual Achievement Test (WIAT) (Wechsler, 1999). The WASI is important to this study to collect baseline intelligence data and serves as a measure to further investigate executive function.

Results

To examine the association between general trauma and executive function, scores on the four subtests within the D-KEFS and the composite score of the BRIEF-A were compared to scores on the trauma questionnaire. Independent samples t-tests revealed that participants with history of trauma had significantly lower scores on the Color-Word Interference subtest of the D-KEFS [$t(123) = 2.50$, $p = 0.01$], which assesses inhibition and switching ability. Independent samples t-test were run for the other three subtests administered in the D-KEFS, but did not yield a significant difference in the means between participants with and without history of trauma, as shown in Figure 2. Findings from independent samples t-tests also revealed significantly greater self-reported executive function problems for participants with self-reported trauma as measured by the BRIEF-A GEC score [$t(116) = -3.92$, $p < 0.01$], as shown in Figure 3. This limited baseline associated may have impacted following analyses.

Our next analysis sought to further explore the independent relationships between executive function decrements and specific types of childhood trauma. Linear regression analyses were used to investigate if certain trauma types predicted any of the D-KEFS subtests or the BRIEF-A GEC score, and the results are shown Tables 1 though 5. Physical neglect was found to significantly predict poorer switching ability as measured by the D-KEFS Color Word Interference test [$b = 0.27$, $t(123) = 2.58$,

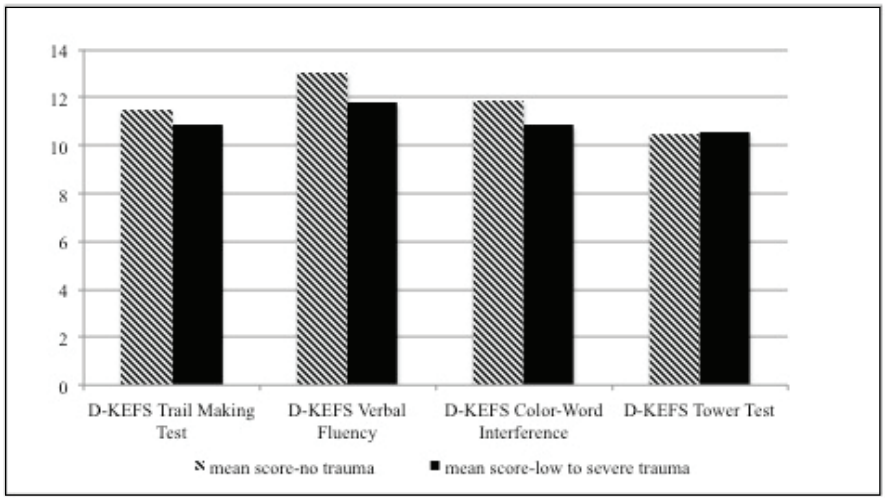

Figure 2. Between groups mean comparisons of unadjusted D-KEFS subtest scores. The Color-Word Interference subtest was the only subtest with significantly different means.

p = 0.01], as well as the D-KEFS Trail Making Test [b = 0.21, t(123) = 1.99, p = 0.05] when controlling for all other trauma types. The linear regression did not indicate physical, sexual, or emotional abuse or emotional neglect to be independent predictors of executive function as measured by the D-KEFS. However, emotional abuse was found to significantly predict self reported executive function deficit as measure by the BRIEF-A Global Executive Composite [b = -.44, t(123) = -3.192, p = 0.002], when controlling for all other trauma types. Physical and sexual abuse and physical and emotional neglect did not emerge as predictors for self-reported executive dysfunction. The overall lack of significance could be due to limited sample sizes of specific trauma types under powering analyses.

Discussion

Though a negative relationship between childhood trauma and executive function is supported in the literature (Majer et al., 2010), we felt it necessary to establish this effect within our specific sample before further investigating trauma type predictors. As hypothesized, these baseline analyses revealed both subjective and objective measures to support this relationship, indicating that participants who did endorse general trauma performed significantly worse on executive function measures than those who did not endorse trauma of any kind. From this, each trauma type was analyzed separately to determine among which group or groups the effect was strongest. Interestingly, physical neglect and emotional abuse were the only trauma types to emerge as significant predictors of executive function decrement. Our findings suggest that victims of physical neglect and emotional abuse are more likely to experience these deficits in young adulthood, and provide support for early assessment and treatment of these individuals. However, our study does not provide strong evidence to support such intervention among victims of physical or sexual abuse or emotional neglect. These results support the hypothesis that executive function performance is not affected equally among different types of trauma; however, our hypothesis that several if not all trauma types would significantly predict some level of executive function deficit is not supported.

The second aim of the present study was to investigate the association between subjective and objective measures of executive function. Previous research suggests self-report measures may be better able to detect executive function deficits as compared to objective measures of EF, and that these measures correlate at a lesser degree than may be expected (Biederman et al., 2008). Subjective and objective measures had widely varying outcomes in this investigation, as objective measures indicate only physical neglect as a predictor, and subjective measure indicate only emotional abuse, and neither measure lending support for the findings of the other. These results contribute to the literature questioning the differences between subjective and objective measures, and support the need to utilize both measures to obtain a more inclusive and complete representation of level of executive function. Furthermore, differences in

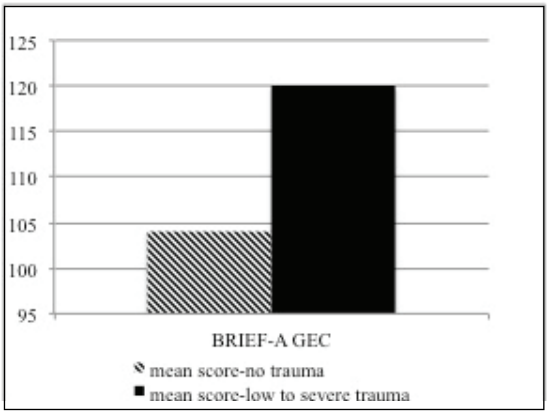

Figure 3. Between groups mean comparison of unadjusted BRIEF-A Global Executive Composite.

Table 1

Regression Results for D-KEFS Trail Making Test

Variable	*B*	SE(*B*)	t	Sig (*p*)
Emotional Abuse	0.62	1.03	0.60	0.55
Physical Abuse	-0.55	0.91	-0.60	0.55
Sexual Abuse	0.31	0.92	0.34	0.74
Emotional Neglect	-1.00	0.88	-1.14	0.26
Physical Neglect	1.97	0.99	1.99	0.049*

Notes. $R^2 = 0.04$

Table 2

Regression Results for D-KEFS Verbal Fluency

Variable	*B*	SE(*B*)	t	Sig (*p*)
Emotional Abuse	-0.41	8.75	-0.05	0.96
Physical Abuse	6.94	7.73	0.90	0.37
Sexual Abuse	13.80	7.76	1.78	0.08
Emotional Neglect	0.78	7.50	0.10	0.92
Physical Neglect	4.42	8.40	0.53	0.60

Notes. $R^2 = 0.06$

Table 3

Regression Results for D-KEFS Color Word Interference

Variable	*B*	SE(*B*)	t	Sig (*p*)
Emotional Abuse	0.29	1.15	0.25	0.80
Physical Abuse	-0.91	1.01	-0.90	0.37
Sexual Abuse	1.76	1.02	1.73	0.09
Emotional Neglect	-0.32	0.98	-0.33	0.75
Physical Neglect	2.83	1.10	2.58	0.01*

Notes. $R^2 = 0.08$

Table 4

Regression Results for D-KEFS Tower Test

Variable	*B*	SE(*B*)	t	Sig (*p*)
Emotional Abuse	-0.56	1.08	-0.52	0.60
Physical Abuse	0.51	0.95	0.54	0.59
Sexual Abuse	0.97	0.96	1.02	0.31
Emotional Neglect	-0.41	0.92	-0.44	0.66
Physical Neglect	-0.11	1.03	-0.10	0.92

Notes. $R^2 = 0.08$

Table 5

Regression Results for BRIEF-A GEC

Variable	*B*	SE(*B*)	t	Sig (*p*)
Emotional Abuse	-215.87	67.63	-3.19	0.002*
Physical Abuse	50.48	60.51	0.83	0.41
Sexual Abuse	-42.72	60.28	-0.71	0.48
Emotional Neglect	-8.86	57.30	-0.16	0.88
Physical Neglect	-2.98	65.03	-0.05	0.96

Notes. $R^2 = 0.08$

individual scores help to identify perceived versus pathological deficits.

In terms of limitations, several factors offer potential explanations for non-significant findings. Perhaps most importantly is sample size. Though our main study did in fact have a large sample, separating groups into individual trauma type minimized the sample first by eliminating participants who did not experience trauma, and furthermore by the varying frequencies of trauma types reported. For example, 49 participants reported emotional neglect, while only 14 reported sexual abuse. Our analyses were likely underpowered within groups that were less frequently endorsed, as 14 participants is not enough to reliably determine an effect. Furthermore, emotional neglect was the most frequently endorsed and coincidently the only significant predictor of EF. Perhaps other trauma types could be found to predict executive function deficits with an increased number of participants in each respective group. Future studies should control for this through targeted recruitment methods or increasing general sample size.

Secondly, the CTQ classifies self-reported trauma severity as none, mild, moderate, or severe. As previously stated, we dichotomized trauma as 'yes' or 'no,' essentially combining mild, moderate or severe trauma into one group in order to analyze the largest possible sample. However, mild trauma verses severe trauma may not impact executive function and all resulting sequelae equally. The effect of a moderate or severe experience of a particular trauma type on executive function performance may have been dulled by those who experienced only a mild incidence. Further analyses might seek to group these ratings where none and mild is dichotomized as 'no,' and moderate and severe as 'yes,' or to utilize the Likert scale with different statistical methods. Though our dichotomization was effective in optimizing sample size, it eliminated the severity factor entirely, which may have impacted the results.

Another limitation to our findings stems from the baseline data analysis determining a relationship between general trauma and executive function deficits within our sample. The Trail Making and the Color-Word Interference were the only subtests of the four subtests within the D-KEFS that significantly supported this

relationship. Though these measures target the prominent executive function skills of inhibition, switching, and rule-learning, they do not encompass executive function as a whole.

Finally, comorbidity of the five trauma types may have prevented clear distinction among groups. Previous research supports that most children who experience childhood trauma have likely been exposed to more than one type of trauma (Spertus et al., 2003), and this finding is true within our sample. With this, executive function scores of participants who reported more than one type of trauma were included in analyses for each type, resulting in trauma type groups that were not independent of each other. This could weaken an argument claiming one type of trauma is a predictor of executive function deficit, or make another type appear as a predictor that independently is not supported by the data.

To conclude, results of this study offer implications for both future research as well as clinical practice. Findings support that childhood trauma does in fact negatively affect executive function in young adulthood, and a differential impact based on type of trauma may exist. These findings could contribute to clinical intervention in cases of physical neglect and/or emotional abuse. Furthermore, university administration and professors should utilize these findings to effectively identify and support affected individuals. Second, these findings encourage continued investigations into the effect of childhood trauma in later adulthood. Our study's use of a college sample targeted a population that heavily relies on executive function skills for academic success, but future research is needed amongst adult populations utilizing executive function in careers and within families.

Finally, the minimal correlations found between objective and subjective measures indicate the need for utilization of both measures in future research. Furthermore, these discordant findings warrant the investigation of the sensitivity, reliability, and validity of these measures. Researchers should chose these measures based on specific research questions, and determine if self-report, psychometric, or both measures are appropriate.

Taken together, our study supports that childhood trauma could negatively impact certain executive function skills within college students, and this is potentially detrimental to academic success. Understanding the cognitive sequelae associated with childhood trauma in general and by type allows for the proper intervention and care for these individuals. Emotional abuse has emerged as one of the more common types of childhood trauma, and offers an explanation for executive function difficulties. This is especially useful as it is the form of trauma that will likely never leave a physical sign and is somewhat subjective amongst families (Spertus et al., 2003). On the other hand, physical neglect almost always leaves evidence or obvious signs of maltreatment (Yates et al., 2003), and so understanding the importance of and implementing immediate intervention could help to minimize cognitive deficits. In addition to helping current college students facing these deficits, these findings offer support for immediate executive function intervention when emotional abuse or physical neglect is discovered in childhood.

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Anissa Ghafarian & Jasmine Ho

University of California, Los Angeles

Anissa Ghafarian was born and raised in Quebec, Canada, where she attained fluency in the English and French languages. She recently graduated Magna Cum Laude from the University of California, Los Angeles with a Bachelor of Arts in Psychology. She is currently working as a Project Coordinator examining the effects of social connections in threatening situations in the Social Affect and Neuroscience Lab under the supervision of Dr. Naomi Eisenberger and graduate student Erica Hornstein. She is also currently working as a Research Assistant at the Semel Institute for Neuroscience and Human Behavior at the University of California, Los Angeles in the Wood lab, assisting with the administration of cognitive-behavioral treatment for children and adolescents with high functioning Autism Spectrum Disorder and comorbid anxiety under the supervision of Dr. Jeffrey Wood. She aspires to attain a Ph.D in clinical psychology focusing on generalized anxiety disorder. In the future, she would like to continue collaborations with Jasmine T. Ho and merge the worlds of social and clinical psychology. In her spare time, Anissa likes to go hiking and travel. She enjoys watching basketball and rooting for her Los Angeles Lakers.

Jasmine Ho graduated Magna Cum Laude from the University of California, Los Angeles in 2013 with a Bachelor of Arts in Psychology and minor in Cognitive Science. She concurrently works at UCLA in two labs, one as a project coordinator in Dr. Naomi Eisenberger's Social Affective Neuroscience Lab, examining the effects of social connection on threatening experiences. Also, she works in Drs. Rena Repetti and Ted Robles' Relationships and Health Lab, which investigates how family environments affect physiological indicators of stress and immune functions. Intrigued by the interdisciplinary field of social-cognitive neuroscience, Jasmine plans to focus her future research interests on the neural substrates of reasoning and decision-making. After completion of a Masters, Jasmine will pursue a Ph.D. in Neuroeconomics or Social Psychology. Ideally, she will continue researching the neural basis of cognitive processes and collaborating with her lab partner, Anissa Ghafarian, on future projects. When she is not running experiments or analyzing data, Jasmine enjoys riding horses competitively, attending musical festivals, and traveling, especially to Switzerland, where she was born and raised.

Anissa Ghafarian

What interests you the most about psychology?

I find that psychology provides a holistic approach and that its all-encompassing nature allows me to see the "whole picture" when analyzing different situations, disorders, and relationships.

What is your dream job?

My dream job is to be a Clinical Psychologist. I would like to continue research with children primarily focusing on anxiety and other comorbid disorders.

Besides psychology, what other fields interests you?

I am also interested in the field of international law, primarily focusing on human rights.

Besides research, what do you do for fun?

I enjoy hiking with my friends and traveling to places I've never been to before and learning about different cultures.

Was there any particular experience that sparked your research interests?

In a health psychology class, my professor spoke of individuals that reported more social support in their daily lives had lower cortisol responses to a stressful situation. I thought it would be interesting to see if the effects of social connection on a similar stressful situation would produce similar results.

Jasmine Ho

Was there any particular experience that sparked your research interests?

A cognitive psychology course I took at UCLA examined psychological questions from an artificial intelligence and neuroscience perspective, an approach I found particularly intriguing. Examining the change in personality and cognition of brain-damaged patients sparked my interest in the biological basis of psychology.

How do you think you've grown as an author/researcher by going through the publication process?

The publication process involves several revisions by different editors, each of which contributes to the paper with different comments and critiques. This process pushed me to truly scrutinize the paper in more detail, while learning how to improve on future work.

Besides research, what do you do for fun?

I have been very active in the equestrian community since the age of 7, riding horses both for pleasure and competitively. I also enjoy exploring new places and traveling overseas, when time and finances allow for such activities!

Besides psychology, what other fields interests you?

I am very intrigued by the interdisciplinary field of cognitive science, particularly the connection between neuroscience and psychology. I hope to integrate these fields in my future research.

What inspired your project?

Previous studies examined the effects of social connection on threatening experiences before and after the threatening events, but no one had yet looked at how people feel during the threat itself. Our study is the first to collect physiological data while people undergo a negative experience.

Social Connection and Threat: Stress Reduction for Men and Women During A Shared Experience of Threat

Jasmine T. Ho, Anissa Ghafarian, Erica Hornstein, and Naomi I. Eisenberger
University of California, Los Angeles

In this study, we examined whether sharing a negative experience changes an individual's perception of the experience and reduces stress. We hypothesized that going through a threatening experience at the same time as another would lead to increased feelings of social connectedness and thus alleviate self-reported and physiological stress across individuals. Participants were assigned to one of three conditions: sharing a threatening experience (electric shock) with another, undergoing a different threatening experience than another, or undergoing the threatening experience alone. We collected self-report ratings of perceived discomfort and negativity and measured Galvanic Skin Response (GSR) to evaluate changes in perceptions of the experience and physiological stress. Current results, while not significant, display trends in the data. We found a trending main effect of condition on subjective ratings of the overall experience, and participants in the shared condition reported a lower overall negative experience when anticipating and receiving electric shock. We also found a trending main effect of condition on GSR, such that participants in the shared condition displayed lower physiological stress arousal when anticipating a shock. Further examination is required, but current data suggests that sharing a negative experience attenuates both physiological and subjective stress responses during a threatening experience.

Human beings are motivated to share their reality with others; both to foster relationships and to better understand and predict the world around them (Echterhoff, Higgins, & Levine, 2009). Previous research has demonstrated greater feelings of in-group membership and more pro-social behavior towards an individual sharing the same experience, if people expect to share a threatening experience with another person (Dovidio & Morris, 1975). This indicates that shared negative experiences may increase feelings of connection. Similarly, individuals display greater preference for activities or interactions that allow them to affiliate with others after a threatening experience (Taylor et al., 2000). However, these studies have focused on people's responses before or after a shared negative experience, whereas no research has yet examined social connection and stress responses during the event itself. In order to better understand feelings of social connection and its implications on human stress responses, we sought to examine whether undergoing the same experience as another leads to reduced stress and subjective negative experience during the event.

The effects of social support on stress attenuation have been studied by Coan et al. (2006), who used functional magnetic resonance imaging to examine whether spousal hand-holding, stranger hand-holding, or no hand-holding would reduce neural threat systems activation during a threatening experience. Results indicated neural attenuation in the right anterior insula, superior frontal gyrus, and hypothalamus during spousal hand-holding, with a more limited attenuation during stranger hand-holding. Furthermore, the extent of neural attenuation varied as a function of marital satisfaction, suggesting that, although interpersonal relationships reduce threat-related neural activity, the quality of social relationships moderates the extent of stress response attenuation during threat.

The presence of attachment figures during a negative experience may also serve as a safety stimuli to reduce stress responses, as examined in a study investigating the relationship between partner's pictures and activity in neural regions associated with safety-signaling (Eisenberger et al., 2011). Results indicated that viewing a partner's picture during a painful experience led to reductions in self-reported pain and activity in pain-related neural regions. Furthermore, the length of the relationship and the perceived quality of partner support mediated stress reduction and was associated with greater ventromedial prefrontal cortex activity. This study builds upon previous research by highlighting the importance of quality

social relationships in the attenuation of self-reported and neural threat responses to negative experiences.

Previous studies identified that social relationships affect physiological responses. Kamarck, Manuck & Jennings (1990), discovered that subjects accompanied by a friend during a psychological challenge exhibited reduced heart rate compared to their counterparts in the alone condition,suggesting that interpersonal support also affects cardiovascular activity. Furthermore, a meta-analytic review of 81 studies concluded that social support attenuates distress-related activity across several domains, such as cardiovascular, endocrine, and immune systems (Uchino, Cacioppo, Kiecolt-Glaser , 1996). Thus, social support during a negative experience has been thoroughly investigated, and although this multidimensional construct necessitates further research to identify the context-specific conditions under which it may prove most beneficial, a general consensus recognizes the favorable effects of interpersonal relationships on stress attenuation.

Beyond such positive effects of social support in familiar dyads (e.g., friends, spouse, family) and unfamiliar dyads (e.g., stranger), even mere feelings of belongingness to unfamiliar others have been identified to increase helping behavior. Gump & Kulik (1997), exposed female participants to a threat with another person they believed were facing either the same or a different situation. Results indicated that threat increased affiliation particularly in those subjects who believed to be facing the same situation as another. Similarly, a previous study by Dovidio & Morris (1975) studied the effects of stress and commonality of fate (i.e., believing that the other participant is facing the same negative experience) on helping behavior by exposing participants to the threat of electric shock together with a confederate believed to be either waiting for electric shock as well or for an inconspicuous word association task. The results indicated that participants who believed they were about to share the experience (i.e., the electric shock) with another, were more likely to display helping behavior than those who thought they were going through a different experience, if the anticipated threat was not as stressful (i.e., innocuous word-association task), or if the confederate was perceived as dissimilar from the participant.

The results of these studies raise interesting implications of how sharing the same negative experience with another person – particularly if that person is perceived as similar to oneself – might elicit pro-social behavior. However, past research on social connection and threat has focused on participants' behavior before undergoing a threatening experience, whereas experiences during such a shared negative experience remain to be examined. Social support has demonstrated stress alleviation in self-reported pain and pain-related neural activity (Coan, Schaefer & Davidson 2006; Eisenberger et al., 2011) during threat and furthermore, interferes with fear responses during the experience of threatening or painful stimuli (Hornstein & Eisenberger, in prep). Coupled with previous findings demonstrating an increase in pro-social behavior following the anticipation of a shared negative experience, such findings suggest that sharing a negative experience increases feelings of mere belongingness and social connection, which in turn may reduce stress as well.

To expand upon previous studies and shed light on the effects of social connection and commonality of fate on stress, we examined the effects of sharing a negative experience on participants' physiological stress responses during threat (electric shock) using Galvanic Skin Response to evaluate changes in stress, and further attained self-reports during the experience to evaluate subjective experiences. We hypothesized a main effect of shared experience, such that sharing a negative experience with another would reduce both emotional and physiological distress during that experience.

Method

Participants

15 undergraduate students (13 females, two males, mean age = 20.2 years, age range = 18-28 years, median age = 19 years) at the University of California, Los Angeles participated in this study to fulfill extra credit or psychology course requirements. All undergraduate students at UCLA were eligible to sign up for the study. The majority of the participants were psychology majors, however, other majors such as art history, biology, linguistics, and undeclared were also included.

Design

The experiment consisted of a 3 (commonality condition: shared, unshared, alone) x 3 (negative experience: electric shock, pictures of faces, pictures of objects) mixed model design. Participants in the shared condition were convinced that they were simultaneously undergoing the same negative experiences (i.e., pictures of faces, pictures of objects, and electric shock) as the other participants Those in the unshared

condition, in contrast, were told that they while they were experiencing pictures of faces, objects, and an electric shock the other participants were seeing pictures of faces and objects, but not receiving an electric shock. In the alone condition, a single participant experienced pictures of faces, objects, and electric shock. Subjective evaluations of the negative experience were collected using self-report ratings, and Galvanic Skin Response (GSR: a physiological index of stress) was used to measured sympathetic nervous system arousal. Lastly, participants filled out individual difference questionnaires. Additionally, in the shared and unshared conditions participants completed an affiliation questionnaire asking about the other participant.

Materials and Apparatus

In the shared and unshared conditions, a research assistant served as a confederate, posing as a second subject participating in the experiment. The experiment room contained the stimulator apparatus for electric shock, a computer for the experimental procedures, and a BioPac System used for GSR data collection. The negative experience sessions were constructed using E-Prime and consisted of pictures of angry or fearful faces, pictures of threatening objects (guns pointed at the viewer), or mild electric shock. Electric shock was calibrated to each participant's individual set point (range 30-60 volts) but never exceeded the maximum of 60 volts, even if the participant conveyed that he or she could endure greater voltage. The three negative experience sessions consisted of six trials each, with a ten second countdown that served as an anticipation period preceding each trial. After the countdown, the participant saw a fixation cross and subsequently would either experience a negative event (picture of faces or objects, or receive an electric shock) or not–3 trials culminated in a negative event and 3 trials culminated in simply viewing a fixation cross. After each trial, subjective ratings regarding the previous experience were collected which assessed how aversive the anticipation period felt, how aversive the shock felt, how positive/negative perception of the overall experience during the previous trial was, and how strong the shock was. After all three sessions were completed, the participants filled out questionnaires, including the Beck Depression Inventory (item 9 removed), a demographic questionnaire, Revised Eysenck Personality Questionnaire, Positive and Negative Affect Schedule, Risky Families, State Anxiety Scale, Trait Anxiety Scale, and the UCLA Loneliness Scale Version 3. Lastly, an affiliation questionnaire in the shared and unshared conditions served as an indicator of how much the participant liked, desired to interact, and perceived himself to be similar to the other participant (confederate).

Measures

The following questionnaires were administered to participants after completing the threatening experiences to find possible correlations between the these measures and social connection. However, this data pertaining to the possible correlations remains to be analyzed.

Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery (1979)

The BDI is a 21-item self-report measure that assesses the intensity of depression on a 4-point scale ranging from 0 to 3, with higher scores indicating more severe depressive symptoms. Sample items include "I do not feel sad (0), I feel sad (1), I am sad all the time and I can't snap out of it (2), I am so sad or unhappy that I can't stand it (3)" and participants are asked to report on their symptoms over the past week. The BDI is an empirically supported measure of depression with a Cronbach's alpha of .90 (Steer, Beck, Riskind, & Brown, 1987).

Revised Eysenck Personality Questionnaire (EPQ-R; Eysenck, 1985)

In order to find possible correlations in the current research between personality traits and social connection, the researchers administered the revised Eysenck Personality Questionnaire, which is an abbreviated 21-item self-report instrument that assesses,three biologically based independent dimensions of temperament: extraversion, neuroticism, and psychoticism. The EPQ-R has been well-validated in a large represented sample for the extraversion scale (males: a = .84, females: a = .80), neuroticism scale (males: a =0.88, females: a = .84), and psychoticism scale (males: a = .62, females: a = .61) (Anton, Garcia, & Garcia, 2002).

Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988)

The PANAS is a 20-item scale that assesses short periods of positive affect (PA) or negative affect (NA). PA is characterized by the extent an individual experiences pleasant interactions with the environment, while NA is depicted by subjective distress

and unpleasant interactions with the environment. Emotions such as enthusiasm and alertness are inherent of high PA and lethargy and sadness are intrinsic of low PA. The participants indicated the extent to which they felt each of the listed emotion during the past week on a five-point Likert scale ranging from, with 1 = Not at All to 5 = Extremely (Crawford & Henry, 2004). The PANAS is a reliable measure with Cronbach's a = .89 for the PA scale and .85 for the NA scale (Crawford &Henry, 2004)

Risky Families Questionnaire (RFQ; Taylor, Eisenberger, Saxbe, Lehman, & Lieberman 2006)

The RFQ is an 11-item questionnaire that measures participants' childhood experiences of coldness, neglect, and conflict in the family environment. Sample questions include the following: "would you say the household you grew up in was chaotic and disorganized?" Items are scored on a 5-point Likert-type scale, with (higher/lower) scores indicating greater/less conflict. This instrument possesses satisfactory psychometric properties (Cronbach's a =.77) (Loucks et al., 2011).

State-Trait Anxiety Inventory (STAI, Spielberger, 1983)

The STAI is composed of 20 self-report items to evaluate state and 20 self-report items to evaluate trait anxiety. State Anxiety is conceived as a transient condition of unpleasant, consciously perceived feelings of tension, nervousness, apprehension that alter in intensity and vary in time as a reaction to threatening circumstances (Spielberger, 1983). The respondent rates the intensity at that given moment of a feeling, such as "I feel calm," on a scale ranging from 1 "not at all" to 4 "very much so." Trait anxiety is conceptualized as the general level of stress that is characteristic to the individual. Trait anxiety describes the personality of the individual rather than a transitory feeling. The respondent rates the intensity at that given moment of a feeling on a scale ranging from 1 "almost never" to 4 "almost always." The STAI has excellent psychometrics (eg. Cronbach's a = .90) and is considered to be an empirically grounded measure for anxiety (Spielberger, 1983).

UCLA Loneliness Scale Version 3 (Russell, 1996)

The UCLA Loneliness Scale Version 3 is a 20-item questionnaire that assesses participants' subjective feelings of loneliness and feelings of isolation. The measure consists of 11 negatively worded (lonely) items and nine positively worded (non-lonely) items, and participants are asked to rate each item as "I often feel this way," " I sometimes feel this way," "I rarely feel this way," or "I never feel this way." The UCLA Loneliness Scale Version 3 is a reliable measure with a Cronbach's a =.89 (Russell, 1996).

Procedure

Participants were led to believe the main goal of the study was to examine people's perceptions of emotional experiences and that the confederate was another participant who had signed up for the study. After explanation of the study procedures (including a comment that participants may be receiving electric shock), the experimenter left the room to allow for a brief interaction between the participants, during which the confederate asked the participant whether he/she had ever been shocked before. When the experimenter returned, participants were either assigned to the same experimental procedure (shared condition: both see pictures of faces and objects and receive electric shock) or the unshared condition (participant sees pictures of faces and objects and receives electric shock, whereas confederate only sees pictures of faces and objects). In the alone condition the experimenter simply assigned the participant to conditions. The experimenter explained that each participant would be placed in one of two separate but adjacent rooms and that the experimenter would monitor the control equipment from the participant's room. Once in the room, the experimenter explained the stimulator apparatus to the participant and attached the electrode to the underside of the radius-area on the right hand. Participants were informed that the goal would be to feel a very uncomfortable tingling or buzzing sensation without being painful, and that the experimenter would approach this personal set point in small increments. Shock levels began at 30 volts and were increased by 2.5 – 5 volt increments until the participant conveyed that he or she had reached a point at which the level of shock was uncomfortable enough without being painful. This was used throughout the rest of the experiment as the participant's set point. After this level was identified, the voltage was returned back to zero to avoid any extraneous nervousness of being shocked by accident. The experimenter subsequently explained the non-invasive BioPac equipment, which consisted of two electrodes placed on the middle and ring finger of the left hand to measure electrical skin conductance activity. In the shared and unshared conditions, the experimenter pretended

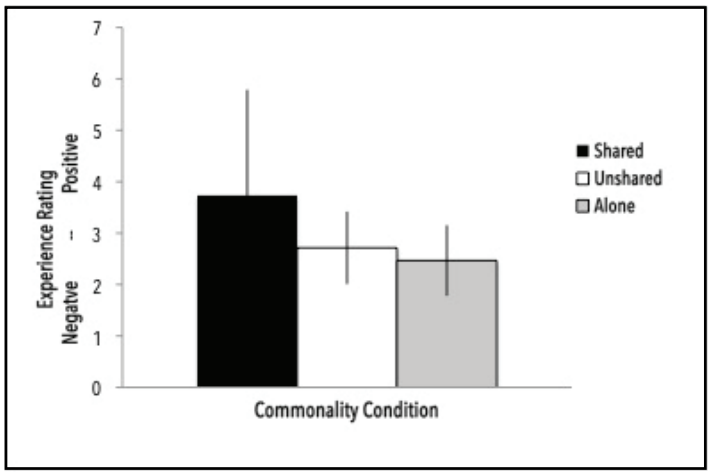

Figure 1. Self-report rating of overall shock experience.

to set up the equipment on the confederate as well. Once the participant was hooked up properly to both stimulator and GSR equipment, the experimenter ran the three sessions (pictures of faces, pictures of objects, or electric shock), the order of which was randomized across subjects. Each session consisted of six pseudo-randomized trials (pseudo-randomized in order to avoid more than 2 shocks being applied in succession), during which the participant viewed a ten second countdown from 10-1 (anticipation period), and subsequently either received the negative stimulus (i.e., seeing a picture of a face, a picture of an object, or receiving electric shock) or simply saw a fixation cross, without accompanied negative stimulus. After each trial, they were asked to rate the previous experience, including aversion of the anticipation period and negative event (picture of face, object or shock) on a 0-19 scale, with following explanations of the numerical values included: 0 = neutral, 5 = slightly unpleasant, 6 = slightly annoying, 7 = unpleasant, 8 = annoying, 9 = slightly distressing, 10 = very unpleasant, 15 = intolerable, 17 = very intolerable. Furthermore, subjects were asked to rate the positivity or negativity of the overall experience and the perceived strength of the shock, each on a 7-point scale (1 = extremely negative, 7 = extremely positive). In the shared and unshared conditions, the experimenter pretended to check on the confederate between sessions. After completing all three sessions, participants were unhooked from the stimulator and BioPac equipment and asked to complete the questionnaires. In the shared and unshared conditions, participants were also asked to complete an affiliation questionnaire that included items on the 'other participant' (confederate) to measure levels of liking, perceived similarity, and desire to interact with the other participant. After completion of the questionnaires, participants were debriefed and informed of the study's true research interest. Participants in the alone condition underwent the same study procedure but with no other participant present.

Results

Figure 1 depicts the self-report rating of the overall shock experience and current results indicate that participants in the shared condition reported less negative overall experience after the countdown (anticipation period) and shock (experience period) were both completed. Similarly, physiological data indicated less stress arousal (as measured by lower GSR) during the anticipation period for participants in the shared condition (Figure 2).

To test these effects, the data were analyzed using two one-way ANOVA's for the shared, unshared, and alone conditions. The first 3x1 ANOVA examined the subjective rating of the overall shock experience and revealed a trending main effect of condition on the overall experience of shock, such that participants in the shared condition rated the overall experience of receiving shock as less negative/more positive than participants in the unshared ($M = 2.71$, $SD = 0.71$) and alone ($M = 2.47$, $SD = 0.69$) conditions ($M = 3.73$, $SD = 2.06$), $F(2, 14) = 2.08$, $p = 0.17$. The second 3x1 ANOVA analyzed the physiological data collected from the GSR during the anticipation period and similarly, results display a trending main effects of condition on stress during the anticipation period, such that participants in the shared condition ($M = 0.17$, $SD = 0.09$) displayed lower GSR and thus less physiological stress-arousal than those in the unshared ($M = 0.23$, $SD =$

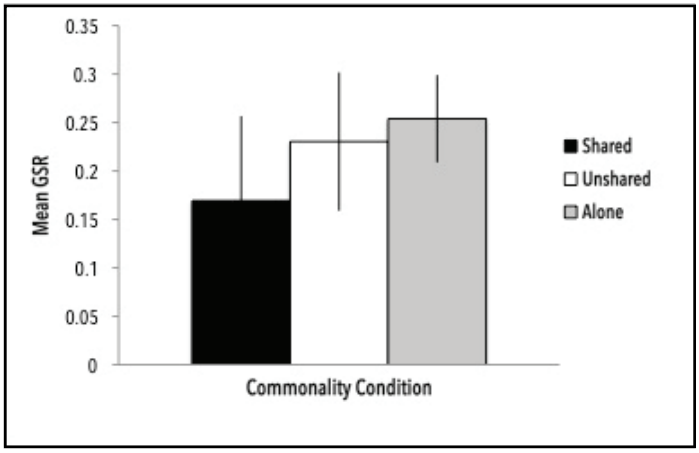

Figure 2. Mean GSR during anticipation period for all trials.

0.07) and alone (M = 0.25, SD = 0.05) conditions during the anticipation period for shock F (2, 14) = 1.87, p = 0.20.

Discussion

The results of this study do not suggest that sharing a threatening experience ameliorates the negative effects of that event. However, preliminary data suggest that participants who believed they were sharing an experience with another displayed lower levels of physiological distress when anticipating the event and rated the overall experience as less negative after the event was completed. Although further data collection is required to conclude statistical significance and to examine whether feelings of affiliation or liking play a role in this effect, current findings display trends for the beneficial effects of social connection on emotional and physiological stress alleviation. Pair-wise comparisons between shared, unshared, and alone conditions could suggest potential differences between unshared and alone conditions, further shedding light on the ways in which social connection or contact play a role in stress reduction.

The participants rated the overall experience as less negative after the event was completed. A possible limitation of the experiment could be the small sample size of 15 participants, which limited the power of the present study. By increasing the sample size, researchers could possibly find a significant effect of social connection on a threatening experience that could be applicable to the entire population.

Stress alleviation during shared experience of threat could expand upon existing social support research, such as the stress-reducing effects of social support identified alleviation of neural (Inagaki & Eisenberger, 2012) and cardiovascular (Lepore & Evans (1993) responses to threat. Furthermore, the mere availability of social support (but absence of enacted support) moderates cardiovascular response to an acute stressor (Uchino & Garvey, 1996), suggesting that the physical presence of another person is not necessary to promote adaptive responses to stress. Such results pose interesting implications for the effectiveness of mere social connection on stress reduction. A possible limitation in the current study could have been the social connection administrated. The brief interaction the participant and confederate had might not have been a strong enough social connection to ameliorate the stresses of a threatening situation. Future efforts to facilitate stronger social connections (e.g., have the participants play a game before separating them into separate rooms), are needed. Another possible limitation of the present study could be that the participant did not visually see the confederate during the experiment, which may hinder the establishment of a social connection. To create a stronger connection, the researchers could administer breaks during the experiment, such that the participant and confederate could interact.

It seems that the form in which social support is offered also plays a key role in its positive effect, as a study by Strazdins & Broom (2007) showed that certain types of social support may exacerbate stress in certain contexts. Helping behavior (i.e., assistance with anger, stress, or conflict) led to more depressive symptoms, whereas companionship (i.e., increased feelings of belonging) resulted in fewer depressive symptoms. These results could potentially offer a possible link between social support and social connection, suggesting that a feeling of belongingness and connection to another person may be a determining factor in stress alleviation.

If the belief that another person understands one's own experience of an event fosters feelings of group membership and belongingness, it may possibly act to buffer individuals against the stress experience during the experiment. Furthermore, a study by Bolger & Amarel (2007) identified that social support outside a person's awareness (invisible support) or very subtle social support may prove more effective than visible support (which entails an emotional cost on behalf of the recipient). Social connection, on the other hand, does not bear such emotional cost to either person and instead targets feelings of belongingness outside a person's awareness, similar to invisible support. Therefore, future research on the context-specific variations of social support and stress will help better understand people's conscious and unconscious coping mechanisms to stressors and connect these findings to research on social connection.

In addition to shedding light on how social support and connection, and the forms these interactions take, might reduce stress in the face of negative events, this research may also help to illuminate differences in the ways in which males and females respond to social connection. This research may also help to illuminate differences in the ways in which males and females respond to social connection. Past research has suggested that there may be sex differences in how individuals respond to stress, such that females exhibit a tend-and-befriend behavioral pattern (Taylor et al., 2000; Turton & Campbell, 2007),

whereas males engage in fight-or-flight responses (Smeet, Dziobek & Wolf, 2009). In addition to these behavioral studies, Smeet et al. (2009) found differing effects of glucocorticoids on social cognition in men and women, further implicating that the sex differences in behavioral patterns observed by previous researchers may match bio-behavioral underpinnings. Therefore, once more data is collected, comparison between men's and women's responses in this study could suggest differences in how social connection impacts men and women during threat by allowing us to examine subjective experience and physiological reactions to stress across shared, unshared, and alone conditions.

However, many factors may influence an individual's feelings of social connection. Examination of the anxiety, attachment style, and loneliness questionnaires will afford greater insight as to how individual characteristics can influence stress responses. Additionally, affiliation questionnaire analysis of participants' feelings toward their experience partner could potentially elucidate whether subjects in the shared condition felt more connected and similar to their partner. We expect that the mere expectation of sharing a negative event will cause participants in the shared condition to feel more similar and connected to their experience partner. These findings could coincide with prior research findings on increased helping behavior if participants anticipate a shared experience of threat (Dovidio et al., 1975) and decreased aggression if subjects believed their partner's feelings were similar to their own (Berkowitz, Schrager & Dunand 2006).

Future research on shared experiences will include examination of neural responses using functional magnetic resonance imaging (fMRI) to pinpoint brain regions correlated with sharing negative events. Past research by Eisenberger et al. (2011) identified greater ventromedial prefrontal cortex (VMPFC) activity while viewing partner's pictures while receiving painful stimulation. However, amount of VMPFC activity varied according to relationship length and perceived partner support, suggesting that the brain's reward system will differentiate according to the quality of social support. After examination of neural regions responsive to social connection, we might expect to find similar differential activation of those regions in shared and unshared negative experiences.

The potentially beneficial effects of social connection on stress reduction have far-reaching implications for both individuals and groups as a whole. Sharing a negative experience with another can prove a valuable resource in cognitive-behavioral therapy during treatment for phobias and other anxiety disorders. Group therapy displays a considerable success rate, and such effects may be due in part to patients feeling more similar, connected, and integrated in a group that shares the same suffering (Emrick, Tonigan, Montgomery & Little, 1993). If an individual forms a connection to a group that is undergoing a similar threatening situation, potential beneficial strategies to overcoming the stressful situation can be implemented. Being a part of group may give an individual a sense of universality, such that he or she does not feel along in facing the experience. Another potential benefit of social connectedness in group therapy is catharsis, such that if an individual feels connected to another person or a group's experience, feelings of pain and stress can be released. Continued research on social connection may also elucidate individual characteristics that affect stress alleviation during a shared negative experience, and, furthermore, help explicate group phenomena (such as social cohesion from natural disasters) to better understand group behavioral mechanisms in societies as a whole.

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Molly Hodul

University of California, Los Angeles

Understanding the brain has captured my interest for most of my life; I find it fascinating that a relatively small amount of biological material in the brain accounts for the inestimable possibilities for human thoughts, emotions, memories, and actions. My experience as a psychology major and neuroscience minor at UCLA has provided me an understanding of behavioral neuroscience in terms of both philosophical and scientific approaches to the workings of the brain. I have also had the opportunity to work in multiple research labs and volunteer in the medical center. I have spent the past two years studying fear learning and memory with Professor Michael Fanselow. After my graduation from UCLA, I will be attending Tufts Sackler School of Biomedical Sciences to obtain a Ph.D in neuroscience. I plan on continuing my studies on the detailed biochemical pathways behind memory and behavior in graduate school to contribute to my long-term goal of translating this understanding into clinical practice by reprogramming chemical pathways. My goal is to continue research throughout my career as a professor of neuroscience.

Who has influenced you the most?

The biggest influence I have had would be my mother. She is a professor of chemistry at UC Berkeley, and has constantly encouraged me to look at the world from a scientific perspective. Many of my aspirations come from wanting to follow in her footsteps.

What interests you the most about psychology?

I am very interested in the neural mechanisms behind behavior, or, put more simply, what makes people tick. It is fascinating to me that different people behave uniquely in the same situation, and that the same person may not behave the same way each time. I am interested in what causes those differences within the brain.

What are your favorite journals?

I enjoy Science, Nature, and Neuroscience.

Explain the process of putting your manuscript together?

My paper is addressing the effects acetylcholine has on memory. This topic has been addressed by many pronounced neuroscientists in the past, but generally memory is attributed to other neurochemicals. I wanted to analyze the data that other researchers have put forward to look more critically at just the neurobiology of acetylcholine to support the cholinergic hypothesis.

What inspired your project?

I became interested in the effects of different neurotransmitters in the brain when I took a Behavioral Neuropharmacology class I took with Professor David Jentsch earlier this year. I decided to study memory because I already have a strong background and interest from my work in Professor Michael Fanselow's lab.

Where do you envision yourself in 10 years?

In 10 years, I hope to be working at a university studying behavioral neuroscience and hopefully teaching undergraduates the importance of this field and the importance of research. Neuroscience is such a quickly developing field, and with each new breakthrough we find so many more things to learn and study. I am hoping in 10 years I will be able to be a part of the frontier on neuroscientific research.

Besides psychology, what other fields interests you?

I am really interested in physics. I am fascinated by the different rules of the way things work and move and how so much of the world can be broken down into much smaller puzzles. I have been able to apply my interest in physics somewhat to neuroscience with electrophysiology.

The Relationship of Acetylcholine and Memory

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There is mounting evidence that low levels of acetylcholine in the brain are associated with memory deficits. This has led to the cholinergic hypothesis for memory, which is the idea that acetylcholine is not only correlated to, but necessary for memory functions. There is strong interest in pharmacology on the implications for increasing acetylcholine level to reverse memory problems in memory-impaired individuals. This article reviews extant evidence supporting the cholinergic hypothesis to show that: (1) Lowering levels of acetylcholine causes memory deficits; (2) Increasing levels of acetylcholine is correlated with an increase in memory; (3) Lesions of cholinergic nuclei in the basal forebrain produce memory deficits; and (4) Physiological symptoms of organic dementias include destruction of cholinergic cells and decreased levels of acetylcholine in affected brains. Future research may be of great benefit, especially for geriatric populations who are experiencing the onset of Alzheimer's disease and a variety of other dementias.

Acetylcholine is an important neurotransmitter that functions both in the peripheral and central nervous system. Peripherally, it causes contractions at the neuromuscular junction and is important in the parasympathetic division of the autonomic nervous system. In the central nervous system, acetylcholine is a key factor in sensory perceptions and in sustaining attention. There is growing evidence from clinical studies that acetylcholine is also important in an array of higher levels of cognitive function such as arousal, reward, sleep, and mood (Wallace & Bertrand, 2013; Jackson, Sanjakader, Muldoon, McIntosh, & Damaj, 2013; Alfaro-Rodríguez, González-Piña, Bueno-Nava, Ávila-Lunca, & Arch-Tirado, 2013). Furthermore, researchers have hypothesized for many decades that acetylcholine is important for learning and memory functions.

Support for the cholinergic hypothesis originated from research on organic memory loss. Some of the first studies of the effects of acetylcholine on short-term memory showed that there was a significant reduction of the neurotransmitter in humans with dementia, and that some memory impairment in rats could be reversed using cholinergic grafts (Dunnett, 1990). LeBlanc et al. (1999) used similar grafts to increase cholinergic innervation in the hippocampus to show that there is a significant correlation between the amount of cholinergic connections in the hippocampus and higher cognitive function, specifically memory.

Recent studies have continued to indicate that cholinergic deficits seem to be the centerpiece in the development of a variety of dementias including Alzheimer's disease, dementia with Lewy bodies, vascular dementia, and dementia associated with Parkinson's disease (Klein et al., 2010). A recent review cited that Alzheimer's disease, for example, is characterized by decreases in cholinergic storage in the cortex, receptor binding, and neuronal connections (Nikolaus, Antke, & Müller, 2009). Studies of dementia have suggested that the neurological origins of memory have strong roots in cholinergic levels in both the cortex and the hippocampus (Mukhin, 2013). While many proponents of learning and memory are looking into glutamatergic explanations, there has continued to be strong evidence that cholinergic inputs interact with both the hippocampal and cortical functions related to memory. There are numerous pieces of biological evidence to support the cholinergic hypothesis for memory, as detailed below.

The relationship between acetylcholine and memory has been studied by administering drugs that change acetylcholine levels, by examining lesions of cholinergic nuclei in the basal forebrain, and by studying the causes of organic dementia (Blokland, 1995; Broks et al., 1988; Collerton, 1986; Perry, Irving, & Perry, 1991; Capurro et al., 2013; Chambon, Jatzke, Wegener, Gravius, & Danysz, 2012; Ma et al., 2009; Buccafusco, Letchworth, Bencherif, & Lippiello, 2005; Smith, 1998; Francis, Palmer, Snape, and Wilcock, 1999; Birks, McGuinness, & Craig, 2013; Sakr et al., 2014; Hirano, Shinotoh, & Eidelberg, 2012; Dugger & Dickson, 2010; Perry et al., 1978). These studies

point to four types of evidence that support the cholinergic hypothesis: (1) Drugs that lower levels of acetylcholine, namely receptor antagonists, choline acetyltransferase inhibitors, and choline transporter protein inhibitors, cause deficits in memory and other cognitive functions; (2) An increase in cholinergic transmission (e.g., via acetylcholinesterase inhibitors, positive allosteric modulators, or receptor agonists) is correlated with an increase in memory; (3) Lesions of cholinergic nuclei in the basal forebrain produce memory deficits; and (4) Clinical studies of the structure of the forebrain of patients with Alzheimer's disease, which has the hallmark symptom of memory loss, and other dementias show destruction of cholinergic cells and decreased levels of acetylcholine. Each of these four types of evidence is described further below.

Neurobiology of Acetylcholine

Acetylcholine is a small organic molecule that is an ester of acetic acid and choline, with receptors in almost all areas of the brain. It is known to be very important in the brain due to its numerous nuclei and projections. The two main systems of cholinergic nuclei are in the brain stem, whose nuclei are referred to as the pendunculopontine tegmentum and the laterodorsal tegmentum, and in the basal forebrain, whose nuclei include the medial septum, diagonal band of Broca, substantia innominata, and the nucleus basalis. The medial septal nucleus has a direct projection to the hippocampus, indicating that it is most likely important in memory.

These nuclei project along multiple pathways throughout the brain to targets that have cholinergic receptors (muscarinic and/or nicotinic). There are many muscarinic receptors in the midbrain, medulla, and pons. Nicotinic receptors can be found in the substantia nigra, locus coeruleus, and septum. A mix of both can be found in a wide variety of brain areas, such as the corpus striatum, cerebral cortex, thalamus, hypothalamus, cerebellum, and most notably, the hippocampus.

The septohippocampal projections from the medial septal area to the hippocampus are an area of interest for researchers for the effects of acetylcholine on memory, as it is already known that the hippocampus is one of the main memory and learning centers in the brain (Ruvio & Mellor, 2013). Mitsushima, Sano, and Takahashi (2013) have found that activation of muscarinic receptors proliferates long-term potentiation (the strengthening of hippocampal excitatory pyramidal synapses); they also found that nicotinic receptors play an important role in the strengthening of inhibitory synapses related to learning in the hippocampus. Furthermore, it has been shown that the inhibition of cholinergic receptors produces a decrease in hippocampal acetylcholine release (Gorman, Pang, Frick, Givens, & Olton, 1994). This is consistent with the hypothesis that there are important cholinergic inputs into the hippocampus that affect memory. The information about acetylcholine and its pathways on the molecular level allows us to further understand how we can alter its levels; researchers have applied this in the following studies to understand the larger story of acetylcholine and memory.

Decreases in Cholinergic Transmission

If acetylcholine is correlated with memory functions, then a decrease in synaptic levels of the neurotransmitter should, in turn, cause a deficit in short-term memory. This would show that acetylcholine is correlated with memory and is present in hippocampal pathways related to memory. Three techniques have been used to reduce acetylcholine concentration at the synapse, and all three have been shown to create memory deficits. Many of the studies that examine the inhibition of cholinergic pathways use muscarinic antagonists, which are molecules that bind to cholinergic receptors and physically block acetylcholine from binding to the receptor (Blokland, 1995; Broks et al., 1988; Perry et al., 1991). Other studies have included inhibition of choline acetyltransferase, an enzyme that produces acetylcholine from choline and acetyl-CoA (Collerton, 1986). Lastly, depriving the neuron of precursors to the neurotransmitter has been used to study the effects of decreased neurotransmitter at the synapse.

The targets for muscarinic receptor antagonists used for research are found in numerous places in the brain, as detailed above. Scopolamine, one of these antagonists, is used commonly in experimental studies to produce amnesia in subjects. Cognitive deficits can be observed directly after introduction of scopolamine into the nervous system (Blokland, 1995). There is significant memory impairment displayed by subjects on an array of tasks that have been shown to accurately assess learning, including list learning and paired-association tasks. Studies also indicate that major functions associated with memory are severely impaired by scopolamine (Broks et al., 1988). Although it seems to influence multiple behaviors, the negative affects of scopolamine on memory have been well established for many decades (Pazzagli & Pepeu, 1965;

Vogel, Hughes, & Carlton, 1967; Glick & Zimmerberg, 1972; Lewis & Bregman, 1972; Wiener & Messer, 1973). Other anti-muscarinic agents such as atropine also have been shown to disrupt both the acquisition and performance of learned behavior (Perry et al., 1991).

The inhibition of choline acetyl transferase (ChAT), another disruption to cholinergic transmission, also produces detrimental behavioral effects associated with memory. Observing subjects who have memory impairments has revealed that they are more likely to have a severe reduction in cortical levels of ChAT. In fact, reduction in ChAT levels is correlated with the intensity of intellectual decline in terms of memory and other cognitive factors (Broks et al., 1988). Decreased activity of ChAT is one of the most consistent symptoms of Alzheimer's disease, giving strong evidence that acetylcholine is near the heart of the disease (Blokland, 1995). Moreover, when researchers induce a reduction of ChAT with inhibitors such as trans-4-(I-napthylvinyl) pyridinium methiodide and trans-4-(I-naphylvinyl) pyridinium hydroxyethyl bromide, the effects parallel the organic loss of ChAT and show a decrease in memory function when administered (Collerton, 1986).

A third way of inhibiting the cholinergic pathway is by decreasing the levels of precursor molecules. Acetylcholine is generated in the axon terminal from choline and acetyl-CoA. Neurons obtain choline from the extracellular matrix. The inhibition of the membrane-bound protein that drives choline from the extracellular matrix into the cell causes a decrease in the main precursor for acetylcholine. Decreases in the precursor molecules, choline and acetyl-CoA, have a direct effect on the levels of acetylcholine, and subjects show decreased performance on short-term memory tasks (Collerton, 1986).

The behavioral effects caused by the mechanisms described above (muscarinic receptor antagonists, decrease in ChAT levels, cholinergic synthesis inhibition) are the expected results if acetylcholine is involved in memory. The inhibition of three different parts of the pathway of cholinergic transmission has been shown to cause significant decreases in memory, supporting a hypothesis that cholinergic transmission is correlated with memory function.

Increases in Cholinergic Transmission

Further evidence for the cholinergic hypothesis for memory has been provided by studies that examine the effects of increased cholinergic transmission, especially those aimed at reversing antagonist-induced deficits (Blokland, 1995; Perry et al., 1991; Capurro et al., 2013; Chambon et al., 2012; Ma et al., 2009; Buccafusco et al., 2005; Levin et al., 2006). If acetylcholine is related to memory, increased transmission should improve performance on memory tasks. This would show that acetylcholine is sufficient to produce memory changes, suggesting that it is a centerpiece for the memory pathway. A target for research is acetlycholinesterase (AChE), an enzyme that breaks down acetylcholine in the synapse (Blokland, 1995; Perry et al., 1991; Capurro et al., 2013). Another intervention is to use positive allosteric modulators, which bind to receptors and amplify the signal from acetylcholine (Chambon et al., 2012; Ma et al., 2009). Lastly, agonists at nicotinic and muscarinic receptors can directly increase cholinergic signals to boost memory function.

The enzyme AChE is found in the synapse and can be inhibited by many molecules. Studies have shown that AChE inhibitors can reverse deficits in acetylcholine by slowing the breakdown of acetylcholine (Blokland, 1995). Moreover, the reversal of these deficits is shown to improve cognition and memory, implying that the deficit is due to removal of acetylcholine (Blokland, 1995). Perry et al. (1991) have furthered these results, showing that physostigmine can actually augment performance in learning and memory tasks for those who do not have a deficit. However, these results are still being investigated. In addition to physostigmine, memoquin has been tested for its properties as an AChE inhibitor, and has shown to improve cognition, most notably in those with memory dysfunctions associated with Alzheimer's disease (Capurro et al., 2013).

Positive allosteric modulators are molecules that increase the strength of synaptic transmission, and should therefore increase memory capabilities. Benzyl quinolone carboxlic acid (BQCA), one of these modulators, reduces the overall concentration of acetylcholine required to create an action potential in the post-synaptic cell. BQCA acts at the muscarinic M1 receptor, and studies indicate that it has potential as a cognitive enhancer where a cholinergic deficit is already expressed (Chambon et al., 2012). For example, conclusions drawn from behavioral studies in a rodent model suggest that BQCA successfully reversed scopolamine-induced memory deficits in a spontaneous alternation task (Chambon et al., 2012). Spontaneous alternation tasks are designed to test spatial learning and memory by teaching a rodent to use a

T-shaped maze and testing their later performance, which gives a measure of their memory for the maze. Other studies have shown that BQCA recruits beta-arrestin to muscarinic M1 receptors, providing further evidence for its role in the formation and preservation of memory by increasing acetylcholine levels (Ma et al., 2009).

Receptors also can be stimulated directly by the action of nicotinic agonists. Nicotinic agonists have shown potential in attenuating memory deficits, providing further evidence for the relationship of the cholinergic pathways and memory. Agonists of nicotinic receptors have been shown to induce long-term potentiation (LTP), a phenomenon that refers to an increase in synaptic connections linked with learning and memory formation (Buccafusco et al., 2005). These agonists cause many changes within the cell that have been associated with LTP, such as increases in intracellular calcium, activation of second messenger systems, increased transcription and translation, and improved release of neurotransmitter (Buccafusco et al., 2005). This is extremely interesting because while current research in the field has its main focus on the glutamatergic processes behind LTP, nicotinic agonists provide evidence that acetylcholine may be an important modulator in LTP and thus memory. There is also evidence that nicotinic agonists improve working memory and learning by interacting with both alpha-4/beta-2 and alpha-7 type receptors, which seem to have crucial roles in memory (Levin et al., 2006). Nicotine and other agonists have shown potential as cognitive enhancers, implying that the systems where they are present must be associated with learning and memory.

Lesion Studies

Small-scale changes at the synapse associated with increasing or decreasing acetylcholine can only give so much information; further studies have used lesions to investigate the effects of the loss of major cholinergic nuclei in the brain. If these nuclei have been abolished or in another way taken offline, researchers should expect to see that acetylcholine levels are severely depleted, and that this depletion causes memory loss. As it would be unethical to lesion cholinergic nuclei in humans, many researchers use a rodent model to study the cholinergic nuclei in the brain. Rodents allow for very strict variable control, and they are easier to study and perform surgeries on. As mammals, rodents have very similar DNA to humans, meaning that the hippocampus and cholinergic nuclei and pathway are relatively preserved between species.

As listed above, the two main systems of cholinergic nuclei are in the brain stem and basal forebrain. Cholinergic dysfunction can be induced by introducing a lesion in one of these major nuclei or any of the major pathways associated with them. Researchers have shown that an array of memory and learning functions are affected by lesions such as delayed visual discrimination and short-term spatial memory (Andrews, Grützer, & Stephens, 1992; Andrews, Jansen, Linders, & Princen, 1994). Furthermore, lesions to the nucleus basalis, a primary cholinergic afferent, have been shown to decrease ChAT activity and cause aphasia, adiposia, and both long-term and short-term memory as tested by a radial arm maze, a similar paradigm to the spontaneous alteration tasks (Smith, 1998). Smith (1998) describes another study indicating involvement of cholinergic nuclei in memory where lesions were created in the nucleus basalis and drugs were used to modulate the levels of learning ability. In this study, scopolamine aggravates the deficit in memory and AChE inhibitors improve memory, as expected on the basis of the evidence provided in the sections on decreases and increases in cholinergic transmission (Smith, 1998).

These studies in which brain lesions were created to destroy major cholinergic nuclei have provided clear evidence of the relationship between these nuclei and memory function. Moreover, it is possible to control memory function by either increasing or decreasing cholinergic transmission at the site of the lesion.

Organic Dementias

One of the most important mechanisms to study the impact of aceytylcholine on memory is to examine the cause of natural memory loss. Alzheimer's disease (AD) is characterized by the loss of cholinergic neurons in the brain, caused by neurofibrillary tangles and amyloid plaques in the forebrain. Many other dementias, including vascular dementia, Parkinson's disease with dementia, and dementia with Lewy bodies, are also associated with the loss of cholinergic function (Sakr et al., 2014; Hirano et al., 2012; Dugger & Dickson, 2010).

There is substantial evidence for the relationship between the symptoms associated with dementia and cholinergic decline (Bartus, Dean, Beer, & Lippa, 1982). First, a reliable decline in the major cholinergic markers, ChAT and acetylcholine, is observed (Blokland, 1995; Birks & Craig,

2006; Birks et al., 2013). In patients with dementias such as AD, reductions in activity of ChAT and levels of acetylcholine are correlated with the degree of cognitive impairment (Francis et al., 1999). The levels of these markers are also correlated with other physiological symptoms of dementia, such as mean plaque count (Perry et al., 1978).

Reductions of other processes related to acetylcholine are also correlated with the progression of AD. Alongside deficits in ChAT and acetylcholine, there are also reductions in choline uptake, acetylcholine release, and overuse of phosphotidylcholine. The direct depletion of acetylcholine at the synapse caused by a reduction in the release and uptake are shown to increase with increased memory deficit (Francis et al.,1999). In AD patients, there is an increase of phospholipid metabolites (Blokland, 1995). This increase leads to a disruption of the metabolism of choline in the cell, which in turn causes overuse of phosphotidylcholine to keep up with the neuron's demand for choline (Blokland, 1995). This process severely harms cholinergic pathways, and, again, is one of the main physiological symptoms shown in degenerative diseases in which memory loss is a key symptom.

Finally, those with dementia show a decline in the number of both nicotinic and muscarinic M2 receptors. Muscarinic M1, M3, and M5 receptors are found post-synaptically and are GQ-coupled, while M2 and M4 are GI-coupled, and M2 is expressed pre-synaptically as an autoreceptor. There is also evidence that the G-protein coupled messenger system supported by the activation of M1 receptors is negatively affected by AD (Francis et al., 1999). These researchers have shown that transfection of additional M1 receptors in combination with M1 agonists reduced the phosphorylation of tau, a protein that is instrumental in causing the plaques and tangles associated with AD (Francis et al., 1999). This suggests that M1 receptors are instrumental in causing key physiological problems in AD.

While Alzheimer's disease shows a strong relationship to acetylcholine, numerous other dementias also show significant correlations. Vascular dementia has been shown to be alleviated with increased acetylcholine levels, and to be correlated with the same reductions in cholinergic molecules listed above (Sakr et al., 2014). The development of dementia for those with Parkinson's disease has also been shown to be dependent on the decline in levels of acetylcholine (Hirano et al., 2012). In fact, many of the same brain areas that are responsible for the motor symptoms in Parkinson's are also areas that contain cholinergic nuclei. Lastly, work has been done showing that dementia with Lewy bodies is also closely tied to cholinergic decline in interesting ways. Lewy bodies, which are abnormal masses of proteins within cells, seem to disrupt neurotransmitter production by appropriating the necessary enzymes (Dugger & Dickson, 2010). These clinical studies of dementia show reduced cholinergic activity, which indicate that these pathways are probably a major component in the development of the quintessential symptoms in dementia that are thought to cause the memory deficits.

Critiques of the Cholinergic Hypothesis

While there is mounting evidence linking the decrease in memory to decline in cholinergic function in AD, there is also evidence that the deficits associated with AD are dissimilar to those caused reversibly by antagonists such as scopolamine. Scopolamine acts in the short-term and does not cause memory deficits that are nearly as strong as those produced by organic dementias. While this might cause confusion about the cholinergic hypothesis for the reduction of memory in AD, the difference can be explained by the fact that the disease involves the chronic destruction of many important facets of the cholinergic system, as described above (Perry et al., 1991). While muscarinic agonists cannot replicate organic dementia exactly, both the short-term and long-term memory losses are clearly related to cholinergic function.

Another critique of studies involving directly antagonizing or agonizing the synapse is that the behavioral effects may not be due to the specific pathway as claimed by the researchers. Cholinergic pathways, while found in the two main areas in the central nervous system, are also found in abundance in the peripheral nervous system. Acetylcholine is the neurotransmitter used in the parasympathetic nervous system, and is therefore important for growth, relaxation, digestion, and other healing or relaxing actions done at rest. The somatic system is also cholinergic, using acetylcholine to peripherally contract muscles. A good argument is that the deficits seen using drugs that increase or decrease acetylcholine may have behavioral changes caused by connections outside of the central nervous system. To determine the system involved in the behavior, Collerton studied the use of peripheral cholinergic agonists, such as prostigmine and neostigmine, in comparison with the central cholinergic agonist physotigmine (1986). He found that peripheral cholinergic agonists had at most

very small and insignificant effects, especially as compared to central cholinergic agonist (Collerton, 1986). This study indicates that deficits seen in memory are due to the cholinergic system within the brain and are not caused by changes in the peripheral nervous system or at the neuromuscular junction.

Further Studies and Conclusions

The cholinergic hypothesis of learning and memory has been tested by many different methodologies and has proven to be quite robust. Depleting acetylcholine at the synapse using mechanisms like muscarinic antagonists, inhibiting ChAT, and decreasing precursor molecules has been shown to cause declines in learning and memory. Increasing the neurotransmitter's signal using AChE inhibitors, positive allosteric modulators, and agonists has been shown to increase memory, especially in circumstances where there was already a decline. Memory decline is shown when main cholinergic nuclei are severed, providing further evidence of the key role that acetylcholine plays in memory. Lastly, organic memory losses associated with diseases such as AD have been shown to be related to declines in the cholinergic system in numerous ways, including decrease of markers and death of cholinergic cells. These four methods of studying the behavioral effects of varying levels of acetylcholine provide strong evidence of its relationship to memory in mammals.

The neurobiological data clearly indicates that the cholinergic system is essential for memory. Armed with this knowledge, researchers need to look deeper into the specific causes of cholinergic decline in memory disorders in hopes of treating them. Our current medications have a variety of targets and some level of efficacy in terms of slowing deterioration. Many are AChE inhibitors, such as galantamine and rivastigmine (Birks & Craig, 2006; Frölich, 2010). The success of these drugs, however, is debatable due to the low internal validity from high drop out rates of clinical trials. Inhibiting AChE also has no effect on the decrease in precursor molecules and enzymes to make acetylcholine, and thus is only partially bandaging the problem. Memantine, another medication, is a glutamate NMDA and nicotinic receptor antagonist. While it has been shown to reduce aggressiveness and excitement, researchers have not found that treatment helps prevent memory impairment (Hattori, 2013). It has, however, been shown to increase hippocampal neurogenesis (Ishikawa et al., 2014). The effects of both AChE inhibitors and memantine do not have convincing evidence in clinical trials, despite the fact that the proposed neurobiology of the drugs is intuitively correct (Frölich, 2010). Lastly, researchers have been studying Ladostigil, an anti-apoptotic drug that has been shown to regulate amyloid protein processing and seems to have a clear positive effect for patients with AD and dementia with Lewy bodies (Weinreb, Amit, Bar-Am, & Youdim, 2012).

Further studies and clinical trials could allow us to see the way cholinergic nuclei and pathways behave throughout the progression of dementia. It is not enough to know the neurobiology of acetylcholine; researchers have already shown that it is directly related to memory and is clearly correlated with organic dementia, but the medicines predicted on the basis of this understanding have not been significantly successful. Focuses of further studies should be on the analysis of why clinical trials are failing, and if there is any way we can improve the current medications. Another line of focus should be in the analysis of possible drugs that could proliferate cholinergic transmission or inhibit proteins that are causing decays in transmission. These studies could improve the quality of life for countless patients with a wide variety of dementias.

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Min Su Kang is a junior at Duke University majoring in Psychology. She is also a research assistant at Duke Center for Eating Disorders (Durham, NC) and a clinical trials assistant at Duke Center for Developmental Epidemiology (Durham, NC). She wants to pursue Ph.D. in clinical psychology after graduation. Her research interests include obesity, binge eating, food addiction, and food policy in the U.S. Her latest project is on identifying genetic and neural substrates for food addiction.

How do you juggle all of your responsibilities (your research, school, etc.)?

I am fortunate to be at Duke, because of the focus on research and all the opportunities. In order to find balance between research and academics, I enrolled in independent study courses, in which students conduct research and prepare manuscript with a faculty for a course credit. Also, undergraduate students have access to graduate-level seminars with refined topics. In fact, the manuscript I submitted is a term paper I wrote for one of these courses, which was really killing two birds with one stone.

Who has influenced you the most?

Kelly D. Brownell, Ph.D. -- I had the opportunity to meet him as a guest speaker in one of my classes last year. He was the first person to introduce to me about the concept of food addiction, which is one of my main research interests, as well as the policy-level intervention for treating obesity. He is now the Dean of Sanford School of Public Policy at Duke and no longer does his own research, but his previous work has inspired me a lot and I plan to use his book *Food Addiction* as a guidance for my future research.

What are your favorite journals?

My personal favorite is Appetite. Appetite is an international research journal specializing in nutrition, food, food choices, and our consummatory behavior. Topics covered in this journal includes everything from genetics to preference in diet, something that both professional researchers and lay people can easily find interest in. In fact, one of my mentors, Dr. Nancy Zucker, is an editor of the journal so I had the opportunity to be involved in the publication process as a guest reviewer, which was an incredibly rewarding and valuable experience.

What interests you the most about psychology?

What I love about psychology is that everyone is the expert of their own mind. Unlike many of the hard sciences, I can explain psychology to my grandma and she will know what I mean -- to an extent. What I truly enjoy about learning and doing research in psychology is the fact that I can share my thoughts and ideas with others, who may not necessarily have expertise in the topic. With psychology, especially the ones involving food, everyone has first-hand experience with it and will be less intimidated, and will have a less difficult time conceptualizing relative to other areas of science.

Besides psychology, what other fields interests you?

Besides psychology, I am very intrigued by the neuroscience and genetics behind our behavior. For my senior honors thesis, I will be identifying the genetic and neural substrates of food addiction and comparing them to those of substance abuse at Duke NeuroGenetics Lab.

What is your dream job?

My dream job would be to work in regulating food policy in the U.S. Unlike in many other countries, we have a highly industrialized food environment in which high-calorie foods are available at very low costs while fruits and vegetables are not. Our economy is so closely tied with the food industry that despite all the recent movements to raise awareness to these issues, the system is resistant to change. One day I hope to convince people on the psychological and medical implications of the toxic food environment and work on implementing practical changes to our environment so that everyone in the country can have access to healthy foods.

Psychotherapies for Binge Eating Disorder: How to Address the Heterogeneity

Min Su Kang
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Formerly diagnosed as Eating Disorder Not Otherwise Specified (ED-NOS), Binge Eating Disorder (BED) is now recognized as its own category in DSM-5. A number of clinical trials have shown treatment efficacy for BED. However, pharmacological treatments are often associated with high rates of side effects and the follow up data for psychological treatments are inconclusive. The aim of this paper is to provide a review of the current literature on the etiology and treatment of binge eating disorder. Several clinical trials using Cognitive Behavioral Therapy (CBT), Interpersonal Psychotherapy (IPT), Dialectical Behavior Therapy (DBT), Acceptance and Commitment Therapy (ACT), and Motivational Interviewing (MI) are evaluated in the context of treatment for BED to assess both short-term and long-term efficacy, as well as to determine the potential benefit as a treatment that addresses the heterogeneity within the disorder.

Binge eating disorder (BED) has recently become a separate category for eating disorders in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (APA, 2013). BED is characterized by consuming unusually large amounts of food and feeling a sense of loss of control, in the absence of significant compensatory behaviors such as self-induced purging and excessive exercise. BED is the most prevalent eating disorder in adults, with a prevalence rate of 3.5% of women and 2.0% of men in the United States (Iacovino, Gredysa, Altman, & Wilfley, 2012). Although BED is more common than bulimia nervosa (1%) or anorexia nervosa (0.3%), it is the most under-treated condition among all eating disorders (Hoek & Van Hoeken, 2003; Hudson, Hiripi, Pope, & Kessler, 2007; Carrard et al., 2011). For instance, less than half of individuals who meet the diagnostic criteria for BED seek treatment, and the disorder is rarely screened by physicians despite serious health implications associated with BED such as obesity, type 2 diabetes, and cardiovascular diseases (Crow, Peterson, Levine, Thuras, & Mitchell, 2004). The adverse effect of BED is not limited to physical health; as with other eating disorders, it is characterized by lifetime psychiatric comorbidity, reduced quality of life, and impaired social functioning (Wilfley, Wilson, & Agras, 2003; Iacovino et al., 2012). Despite the increasing prevalence rates, research results on the treatment of BED are inconclusive (Iancavo et al., 2012). Because many researchers consider the general overevaluation of body shape and weight as mainly cognitive in nature, Cognitive Behavioral Therapy (CBT) has been widely accepted as the first-line treatment for Eating Disorders Not Otherwise Specified (ED-NOS), a former DSM category for BED (Murphy, Straebler, Cooper, & Fairburn, 2010; APA, 2013). However, a recent study proposed a four-class model of BED, suggesting that BED is a highly heterogeneous disorder (Sysko, Hildebrandt, Wilson, & Wilfley, 2010). According to the authors, the four classes are characterized by (1) lower body mass index (BMI) and increased physical activity; (2) high levels of binge eating, body dissatisfaction, and compensatory behaviors; (3) high levels of binge eating but lower levels of compensation; and (4) highest BMI, most frequent overeating episodes in the absence of compensatory behaviors. In fact, each class reported different remission rates to Interpersonal Psychotherapy (IPT), guided self-help based Cognitive Behavioral Therapy (CBTgsh), and Behavioral Weight Loss (BWL). This finding suggests that different treatment approaches that cater to individual needs may be necessary in order to effectively address the variability within the disorder. The aim of this paper is to provide a review of current literature on the etiology and different modalities of psychotherapy for BED to evaluate whether CBT is a comprehensive treatment that addresses problems in all four classes of BED. In fact, this review concludes that individuals with BED can benefit from Cognitive Behavioral Therapy (CBT), Interpersonal Psychotherapy (IPT), Dialectical Behavior Therapy

(DBT), Acceptance and Commitment Therapy (ACT), and Motivational Interviewing (MI) depending on the patient's etiology and profile. For future investigation, it is necessary to further consolidate the theoretical classes of BED as well as to empirically test which psychotherapy is most effective in each class.

Etiology

As the nomenclature of the disorder entails, core symptoms of BED are recurrent episodes of binge eating, marked psychological distress associated with the binge, and progressive weight gain. Because the physical and psychological manifestations of the disorder are highly similar to those of bulimia nervosa and obesity, BED was regarded as a noncompensatory subtype of bulimia and behavioral subtype of obesity in the past (Devlin, Goldfein, & Dobrow, 2003). For the clinical assessment of BED, the most commonly used instrument is Eating Disorder Examination (EDE; Cooper & Fairburn, 1987). EDE is a semi-structured interview with four subscales on Dietary Restraint, Eating Concern, Weight Concern, and Shape Concern. However, because of the secretive and subjective nature of a binge episode, clinicians face many challenges in the assessment of BED (Grilo, Masheb, & Wilson, 2001). Hence, some argue that the EDE Questionnaire (EDE-Q), a self-report version of the EDE, may be a better diagnostic tool that yields more accurate responses and costs relatively less to administer (Wilfley, Schwartz, Spurrell, & Fairburn, 1997; Grilo et al., 2001).

The etiology of BED is multidimensional; biological, psychological, and environmental factors all contribute to the development of the disorder (Polivy & Hermann, 2002). According to a recent review of family studies, twin studies, and molecular genetics studies indicate that there is a significant genetic contribution in the onset of BED (Trace, Baker, Peñas-Lledó, & Bulik, 2013). Imaging studies indicate that the neural signatures underlying BED are hypersensitivity to food reward and increased activation in the orbitofrontal cortex, suggesting that the reinforcing value of palatable foods is learned more readily in BED (Schienle, Schäfer, Hermann, & Vaitl, 2009). Disruption in several neurotransmitters including serotonin and dopamine, as well as appetite-regulating hormones–such as leptin, ghrelin, and insulin–has been identified to predict the onset and severity of the disorder (Schienle et al., 2009; Geliebter, Yahav, Gluck, & Hashism, 2004). In addition, a composite of psychological factors including personality traits predisposes individuals to engage in binge eating. Examples of these factors are body image disturbance, perfectionism, overvaluation of physical appearance, negative affect, depressive symptoms, low self-esteem, and impulsivity among many others (Stice, Presnell, & Spangler, 2002; De Zwaan et al., 1994). Lastly, social and environmental influences play a role as a trigger to individuals who are already biologically and psychologically predisposed to developing BED. Strongest risk factors are dieting, pressure to be thin, modeling of eating disturbances, lack of social support, food environment, and exposure to media that promotes thinness as the ideal body image (Stice et al., 2002; Allison & Timmerman, 2007; Polivy & Hermann, 2002). As with other eating disorders and many psychiatric disorders, it is important to understand that rather than a single cause, biological, psychological, and environmental factors all interact with one another and lead to the development of BED (Bulik, 2005).

For an individual who is already at high biological, psychological, and environmental risks for BED, the core principle of "vicious cycle" initiates, maintains, and even exacerbates the disorder. One of the vicious cycles is emotional eating and negative affect; experiencing negative affect–a psychological risk factor for BED–leads one to engage in emotional binge eating episode, which subsequently produces heightened negative feelings, such as guilt and shame. In order to eliminate these feelings, a patient of BED again turns to emotional eating, which has become a negative reinforcement (Stice, 2002). Similar patterns apply to dietary restraint, another significant predictor of the disorder. After a binge episode, many individuals with BED attempt to compensate for the excess caloric intake by subsequent dieting. However, intensive dieting in these individuals tends to result in a relapse binge rather than actual weight loss (Howard & Porzelius, 1999).

In a study of BED patients, psychiatric comorbidity was found to be the single most significant predictor of symptom severity, duration of illness, and treatment resistance (Fichter, Quadflieg, & Hedlund, 2008). Other predictors were body image disturbance, impulsivity, history of sexual abuse, and childhood obesity (Cachelin et al., 1999). Body image dissatisfaction and compensatory self-starvation also predicted severity of the symptoms, as measured by larger amounts of food consumption and increased frequency of binge episodes (Wolf & Crowther, 1983). Fortunately, overall

prognosis for BED upon receiving treatment is relatively better than anorexia and bulimia (Fairburn et al., 2000). However, because untreated BED gradually and progressively leads to weight gain and subsequent weight-related diseases, it is imperative for patients to receive treatment in the early stage of the disorder for better prognosis within BED cases (Wilfley, Wilson, & Agras, 2003).

Treatment

Several pharmaceutical treatments using antidepressants, appetite suppressants, and anticonvulsants have shown efficacy in reducing the frequency of binge episodes in patients (Carter et al., 2003). Widely used as a treatment for obesity, topiramate and sibutramine have been found to successfully reduce frequency of binge eating and body mass index (McElroy et al., 2003; Appolinario et al., 2003). Researchers found similar outcomes in several selective serotonin reuptake inhibitors that have been associated with weight loss, such as sertraline, fluvoxamine, and fluoxetine (McElroy et al., 2000; Hudson et al., 1998; Arnold et al., 2002). A review of pharmaceutical trials for BED concluded that although medications successfully reduce the binge frequency, they do not usually lead to complete abstinence (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007). Despite the relative symptom improvements, studies also report negative side effects associated with the antidepressants–such as dry mouth, headache, nausea, insomnia, and dry mouth–and inconclusive data on long-term remission (Gartlehner et al., 2008). There have been mixed findings regarding whether adjunctive psychotherapy to medication is more effective than either psychotherapy or medication alone, suggesting a need for finding the optimal combination of the two approaches (Brownley et al., 2007). This section of the paper aims to review different strategies in behavioral treatment for BED to evaluate their potentials as a candidate for either the adjunct or sole treatment.

Cognitive Behavioral Therapy (CBT)

Originally developed as a treatment for depression, CBT is a structured, short-term, present-oriented psychotherapy that directs patients toward identifying and modifying dysfunctional thoughts and behaviors. Multiple adaptations of CBT have been proposed and proven to be equally effective in treating other psychological disorders (Beck, 1964; Alford & Beck, 1997). The core principles of CBT for BED are based on the restraint model, which postulates that disturbed eating patterns and extreme concerns about weight and body shape result in dietary restriction, subsequently followed by binge eating (Telch, Agras, Rossiter, Wilfley, & Kenardy, 1990; Wilson & Shafran, 2005). The goal of CBT is to promote structured eating patterns, to improve weight and shape concerns, and to encourage healthy behaviors for weight control, such as self-monitoring and physical exercise to ultimately stop such diet-binge cycles (Iacovino et al., 2012).

Prior randomized controlled trials have shown CBT to be equally, if not more, effective relative to any other eating disorder treatments to which it has been compared (Wilfley & Cohen, 1997). In addition, evidence from a number of studies supports that CBT has both short-term and long-term positive outcomes. For example, after receiving weekly CBT for as short as seven months, individuals with BED demonstrated significant improvement on binge frequency and general psychological well-being, mood, attitude toward one's body, and sense of control over the eating behavior. Follow-up data from this particular study indicated that these results were maintained for an average of 3.5 years following the treatment (Vanderlinden et al., 2012). Nonresponders to CBT can be identified as early as during the first four therapy sessions and may need tailored interventions during the initial treatment phase in order to respond well to later CBT sessions (Munsch, Meyer, & Biedert, 2012).

Compared to pharmacological and other psychological treatments, CBT is believed to be most effective in reducing binge frequency, as well as pathological concerns about eating, weight, and body shape despite no significant evidence of weight reduction (Vocks et al., 2010). However, unlike pharmacotherapies, evidence suggests that CBT may achieve complete abstinence; as few as 15 sessions of CBT compared to waitlist-control demonstrated 63% and 18% abstinence rates respectively. The same study reported that the majority (80%) of the individuals who received CBT were abstinent at one-year follow-up (Dingemans, Spinhoven, & Van Furth, 2007). A study on the efficacy of CBT combined with fluoxetine found that individuals who received CBT only demonstrated higher abstinence rates relative to both fluoxetine-only and combined condition, suggesting its superiority over different treatment techniques (Grilo & Masheb, 2005).

In comparison to Behavioral Weight Loss (BWL), a regimen of decreasing energy intake and increasing energy

expenditure, CBT again showed higher rates of abstinence (80% for CBT vs. 36% for BWL). Similar results were observed at a one-year follow-up, supporting the long-term superiority of CBT to BWL (Munsch et al., 2007). These results suggest that emphasis on addressing the cognitive distortions that prevail in BED is important in improving overall treatment outcome, rather than focusing on weight loss alone.

For patients who have limited access to mental health care, guided self-help CBT (CBTgsh) has been identified as a low-cost, effective alternative treatment for traditional CBT (Iacovino et al., 2012). CBTgsh is usually delivered by a self-help manual, Overcoming Binge Eating by Christopher Fairburn, and regular meetings with a therapist to produce similar outcomes as traditional CBT (Carter & Fairburn, 1998). However, CBTgsh was shown to have reduced effect on individuals with severe eating disorder pathology, such as low self-esteem, negative affect, body image disturbance, and shape concerns (Wilson, Wilfley, Agras & Bryson, 2010). Recently, CBTgsh coupled with the use of technology has been proposed for increased treatment accessibility and equally significant outcomes. Internet-based CBTgsh intervention for a six-month period showed a decrease in dysfunctional behaviors, such as emotional eating, binge eating, and thinking about bingeing. This intervention also showed a reduction in binge frequency, shape concern, and body dissatisfaction (Carrard et al., 2011). Despite the high abstinence rates and long-term effectiveness, some patients fail to respond to CBTgsh (Munsch et al., 2012). This may be in part due to the individual variability within the disorder, suggesting the need to explore other treatment platforms for the nonresponders (Sysko et al., 2010).

Dialectical Behavior Therapy (DBT)

Originally developed for women with borderline personality disorder, DBT is a comprehensive treatment that specifically teaches adaptive skills necessary to target emotion regulation (Telch, Agras, & Linehan, 2000). The rationale of DBT adapted for BED is based on the affect regulation model of binge eating. This model is built upon a theory that binge eating occurs in response to intolerable emotional experiences in the absence of more adaptive coping mechanisms (Polivy & Herman, 1993). By focusing on four areas–mindfulness, distress tolerance, emotion regulation, and interpersonal effectiveness–DBT empowers the patient to learn adaptive emotional regulation skills and to subsequently prevent emotional eating and binge episodes (Wiser & Telch, 1999; Telch & Agras, 2001).

Despite the small sample size and lack of comparison group, early studies on using DBT to treat BED is promising. Researchers found that 82% of the women who received DBT were no longer binge eating but were reporting improvement in emotion regulation. This pattern of improvement was maintained at three-month and six-month follow-up (Telch et al., 2000). In a subsequent randomized controlled trial (RCT) by the same group of researchers, DBT demonstrated an abstinence rate of 89%, compared to 12.5% for control. However, there was a significance drop in the abstinence rate in DBT group from 89% to 56% at six-month follow-up, suggesting that long-term efficacy of DBT may not be comparable to other psychological treatments (Telch et al., 2001). Similar results were found in RCT that examined effectiveness of DBT in BED comorbid with Borderline Personality Disorder. Although participants reported reduced binge eating behavior, associated eating disorder concerns, self-injury, suicidal behavior, and improved social functioning at the end of the treatment, the results were not maintained at six-month follow-up. One possible interpretation of such long-term inadequacy may be due to relatively short duration of the treatment, and stronger dosage of DBT is suspected to show more promising results (Chen, Matthews, Allen, Kuo, & Linehan, 2008). Predictors of relapse following successful DBT were early onset and higher EDE Restraint scores at baseline (Safer, Lively, Telch, & Agras, 2002).

Because DBT heavily incorporates group-based skill learning, comparing DBT to individual therapy may be confounding. In order to address this issue, researchers conducted a comparative study between DBT and Active Comparison Group Therapy (ACGT). 20 group sessions of DBT and ACGT were delivered to individuals with clinical diagnoses of BED (Safer, Robinson, & Jo, 2010). Post-treatment abstinence rates were 64% and 36%, respectively. Although similar rates were maintained at 12-month follow-up, the DBT group was less likely to drop out of the study (4%), compared to ACGT (33.3%). Two moderators of post-treatment outcome were avoidant personality disorder and early onset of overweight or dieting (Robinson & Safer, 2012). Evidence suggests that a modified version of DBT that caters to the core struggles associated with BED may be more effective than the traditional DBT. In fact, one study showed that DBT with focus on appetite (DBT-AF) was more effective than the standard DBT for binge eating

disorders. After as little as six weeks of treatment, participants who received DBT-AF showed significant improvements on BED symptoms relative to the group that received standard DBT. This finding suggests that further modification of DBT that focuses on other core aspects of BED, such as cognitive distortions, emotional eating, and negative affect may be promising (Hill, Craighead, & Safer, 2011).

Interpersonal Psychotherapy (IPT)

The theoretical foundation of IPT is that when an individual's needs for attachment are not being met, psychological problems arise (Stuart & Robertson, 2003). Initially developed for the treatment of unipolar depression, IPT has been modified for the treatment of various mental disorders including eating disorders, but mostly for bulimia nervosa and anorexia nervosa (Hilbert & Brahler, 2012). The principle of IPT for BED is based on the interpersonal model of binge eating, which posits that individuals use binge eating as a coping mechanism to poor interpersonal relationships, such as unfulfilling social interactions and isolation (Rieger et al., 2010). The goal of IPT for BED is to replace such maladaptive response with healthier mechanisms by supporting the development of adaptive interpersonal skills and promoting a positive self-image (Wolfe, Baker, Smith & Kelly-Weeder, 2009).

A randomized comparison study found that the effectiveness of IPT is equivalent to that of CBT (Wilfley et al., 2002). Both IPT and CBT group received 20 sessions of treatment and reported abstinence rates of 79% and 73%, respectively. Although IPT and CBT yielded similar results post-treatment, long-term effect of IPT indicated slightly superior results at 1-year follow-up (62% vs. 59%). Areas of improvements were reduced binge episodes, eating disorders and psychiatric symptoms, decreased dietary restraints, and maintenance of goals. Similar effects were observed in another study for up to four years in individuals who received IPT, suggesting the long-term efficacy of the treatment relative to CBT (Hilbert, Bishop, Stein, Tanofsky-Kraff, & Swenson, 2012). Although IPT and CBT yielded similar long-term results, researchers found some differences in when such treatment outcomes begin to emerge. In a randomized controlled trial for simplified behavioral modification, CBT, and IPT, researchers found that IPT took longer to achieve the equivalent, substantial, and lasting outcomes that are comparable to CBT. This result can be interpreted as that CBT and IPT yield equivalent outcomes through different mediating mechanisms (Fairburn, Jones, Peveler, Hope, & O'Connor, 1993).

Because of the heterogeneity within the disorder (Sysko et al., 2010) and evidence that CBT and IPT work through different pathways, some researchers have believed that patients who do not respond to CBT may benefit from IPT. In a quasi-experimental study (Agras et al., 1995), non-responders following 12 weeks of CBT were randomly assigned to an additional 12 weeks of IPT or wait-list. The results indicated that these patients showed no significant improvement regardless of the IPT or wait-list condition. Predictors of failure to respond were early onset and severe eating disorder pathology. Such findings indicate that the two most effective treatments for BED (CBT and IPT) still fail to address a comprehensive population affected by the disorder, suggesting the necessity for a novel treatment for individuals with early onset of and severe BED. To date, the clinical consensus is to treat these individuals who failed to respond to both CBT and IPT with higher dosages, such as more expanded or intensive CBT, which usually takes place in an inpatient setting, rather than exploring a new psychotherapy (Wilson, 1996).

Acceptance and Commitment Therapy (ACT)

The foundation of ACT is based on the theory that negative emotions do not necessarily have to be eliminated, reduced, or suppressed, and attempts to do so may induce or exacerbate maladaptive thoughts and behaviors. Therefore, ACT skill-based treatment to develop adaptive skills and to replace such avoidance-directed strategies with acceptance and goal-directed strategies (Wilson & Roberts, 2002).

Compared to CBT, DBT, and IPT, there is limited research investigating the efficacy of ACT on BED. To date, there are no randomized controlled trials that investigated the efficacy of ACT on individuals with clinical diagnoses of BED (Masuda & Hill, 2013). However, several principles of ACT, such as self-acceptance and mindfulness training, may be applicable to the treatment of binge eating. According to the principles of ACT, experiential avoidance is the process of avoiding, escaping, or otherwise altering unwanted private events, such as weight gain and feelings associated with it. Greater experiential avoidance was shown to contribute to the covariation of problem behaviors, suggesting that such aspect of ACT may be a key process to target in the management of maladaptive eating behaviors (Kingston, Clarke, & Remington, 2010). In

fact, one randomized controlled trial found that after a 1-day acceptance-based workshop, participants reported reductions in binge eating as well as changes in experiential avoidance. Specific improvements include reduction in disordered eating attitudes, body anxiety, and preoccupations with eating, weight, and shape. In addition, unlike CBT and IPT, which had little effect on weight loss, participants in this experiment reported subsequent changes in weight, which is most likely to have been mediated by changes in experiential avoidance (Lillis, Hayes & Levin, 2011). In a case study exploring the role of mindfulness practice in treatment, a client with subthreshold BED reported significant improvements at both post-treatment and six-month follow-up (Baer, Fischer, & Huss, 2005). Although these results are preliminary and require further investigations using larger samples RCT's, core principles of ACT have shown to improve BED pathology significantly.

A correlational study on food thought suppression found that women are more likely to suppress food-related thoughts than men, as well as dieters compared to non-dieters (Barnes & Tantleff-Dunn, 2010; Barnes, Masheb, & Grilo, 2011). In addition, suppression of food thoughts predicted binge eating, food cravings, and other eating disorder symptoms in women. In fact, there was an interesting gender difference in the relationship between food-related thought suppression; higher levels of food thought suppression in women with BED predicted higher frequency of binge eating, whereas in men with BED it predicted lower frequency. Such findings suggest that an acceptance-based treatment to address avoidance and suppression may be promising for female patients with BED. Researchers also found that effects of ACT for patients who underwent bariatric surgery include significant improvements on emotional eating behaviors. Compared to the traditional behavior treatment following bariatric surgery, patients who received ACT following the surgery showed greater improvements on eating disordered behaviors, body dissatisfaction, quality of life, and acceptance for weight-related thoughts and feelings compared to controls. Such results are also associated with more effective weight maintenance post-treatment (Weineland, Aarvidsson, Kakoulidis, & Dahl, 2012). Furthermore, ACT may be more effective than CBT in regards to reducing cravings for food, particularly sweets. A study of overweight women indicated that acceptance-based intervention was superior to a standard cognitive reappraisal intervention in lowering cravings and consumption of sweets. This acceptance-based strategy was particularly effective in individuals with greater emotional eating and mindless eating (Forman, Hoffman, Juarascio, Butryn, & Herbert, 2013).

Unfortunately, empirical evidence on the effect of ACT for BED is very limited. However, data from recent obesity-related studies suggest that ACT has potential as an effective treatment for not only reducing the symptoms of BED, but also for promoting weight loss and maintenance (Weineland, et al., 2012; Forman et al., 2013). Whereas CBT–the most widely used treatment for BED–has no effect on weight loss, ACT has promise in addressing one of the important limitations of CBT.

Motivational Interviewing (MI)

According to the Spiral Model of motivation, individuals move up and down the five stages of change: pre-contemplation, contemplation, preparation, action, and maintenance (Prochaska & DiClemente, 1992; Prochaska, DiClemente, & Norcross, 1992). Derived from the model, researchers found that poor outcomes of treatments for eating disorders are largely associated with lack of motivation to change, and denial of a problem, as well as ambivalence to treatment engagement, elimination of disordered behavior, and weight restoration (Treasure & Schmidt, 2008; Vitousek, Watson, & Wilson, 1998). In order to overcome such lack of motivation for treatment, it has been proposed that increasing patient's motivation to change may result in enhanced treatment adherence and decreased relapse rates (Wade, Frayne, Edwards, Robertson, & Gilchrist, 2009).

A randomized controlled trial demonstrated that Motivational Enhancement Therapy (MET) was associated with increased readiness to change for BED compared to self-help control (Dunn, Neighbors, & Larimer, 2006). Additional results from this study included changes in eating attitudes and frequency of binge eating, although these results were not as significant as change in motivation. This study may suggest that MI as an adjunctive or preceding treatment to another form of psychotherapy can be effective in individuals with BED who show lower levels of motivation in the early stage. Similar interpretations can be made from another randomized controlled trial, in which 108 women received either 16 weeks of Adapted Motivational Interviewing (AMI) or control. Results indicated that participants who were in the AMI group reported more confidence in their ability to change their behaviors, as well as improvement in binge eating, mood, self-esteem, and

quality of life. Furthermore, 27.8% of the AMI group showed abstinence compared to control (11.1%), and 87% no longer met the binge frequency on DSM-IV (Cassin, von Ranson, Heng, Brar, & Wojtowicz, 2008). Despite the prevalence of lack of motivation to change in patients with eating disorders, very few studies have investigated how to address this issue in BED (Dunn et al., 2006; Cassin, von Ranson et al., 2008). However, the results from these studies clearly indicate that increasing readiness to change can benefit many patients.

Conclusion

Most likely due to the latest addition in the DSM-5, more recent studies are beginning to recognize BED as its own disorder and to investigate the efficacy of different treatment strategies. Fortunately, the results are quite promising. The superiority of one treatment over another cannot be determined without a randomized controlled trial inclusive of all psychotherapies. Numerous studies have shown evidence that CBT and IPT are the two most effective psychological treatments for BED. Although their short-term and long-term outcomes are consistently positive, there is evidence that a portion of this clinical population fails to respond to neither treatment (Munsch et al., 2012). This directs researchers to further investigate the variability in outcome of other forms of treatments for BED, such as DBT, ACT, and MI. For instance, although evidence indicates that DBT has little long-term effect on improving BED symptoms, it was more effective within certain demographic groups (Chui et al., 2007). Similarly, although there are no randomized controlled trials examining the effectiveness of ACT in treating BED, existing studies on anorexia and bulimia suggest acceptance-based approach is most effective in reducing body image dissatisfaction and disturbance, suggesting the possible implications in BED (Heffner, Sperry, Eifert, & Detweiler, 2002). Lastly, although MI as a sole treatment may not yield significant improvements, it can be effective as an adjunctive treatment for individuals with lack of motivation for change–a recurrent problem that interferes with treatment outcome in many individuals suffering from eating disorders (Vitousek et al., 1998). For future research, it is necessary to systematically investigate the heterogeneity to determine which psychotherapy may be best suited for each subcategory of BED.

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Briana O'Leary

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Briana O'Leary is a recent UCLA class of 2014 graduate, with her bachelor's degree in Psychobiology. Her interest in Psycholobiology stems from her dual interests in molecular cell biology and human cognition. While at UCLA, she was heavily involved with Peer Health Exchange, through which she taught health education in inner-city Los Angeles high schools. Her passion for both the biological sciences and community health has led her to pursue a career in medicine. She will be attending Tulane University as a part of the medical school class of 2018, where she hopes to continue her work in psychology and community health. In her spare time, Briana enjoys being on the go. Hiking, yoga, and racquetball are just a few of the activities she enjoys most.

Was there any particular experience that sparked your research interests?

Overall, my research paper was initially inspired by an assignment for my behavioral neuropharmacology course, which required us to write a paper concerning a psychoactive drug. However, my specific focus on carbamazepine resulted from more personal interests, as I had been prescribed carbamazepine in the past, for atypical pain of the trigeminal nerve.

What interests you most about psychology?

Originally a biology major, my classes focused on understanding the mechanisms underlying life. However, I found that this wasn't completely fulfilling; I also wanted to understand the larger picture of human behavior, perception and cognition, which led me to the field of psychology.

Besides research of course, what do you do for fun?

Whether hiking or playing racquetball or practicing yoga, I enjoy being on the move. I also love to read novels, work on crossword puzzles and spend time with my family.

Explain the process of putting your manuscript together?

Before I started writing my paper, I researched my topic and collected relevant articles. As part of this initial phase, I ensured that there was enough information to flesh out my article and confirmed that the topic was complex enough to warrant a review paper. Once this was determined, I outlined my paper, organized the information by disorder and proposed mechanism, and began the process of writing and editing.

How do you juggle all your responsibilities?

I juggle my responsibilities by staying organized and by getting a head start on my work whenever possible. I also make sure to stay active and take time out my week to decompress, both of which minimize stress and help me stay focused. But above it all, my drive comes from my enthusiasm for my work, as I make sure to get involved in activities that are personally meaningful.

Analysis of Carbamazepine's Mechanisms of Action

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Carbamazepine, an anticonvulsant and mood-stabilizing drug, is primarily used in the treatment of epilepsy, trigeminal neuralgia and manic-depressive disorder. Although the drug's mechanisms of action in the treatment of the first two disorders are relatively well understood, the pathway underlying the drugs mood-stabilizing effects remains unclear. This paper focuses on explaining the drug's mechanisms of action in the treatment of epilepsy and trigeminal neuralgia, while exploring multiple hypotheses concerning its efficacy in bipolar treatment.

Anticonvulsants, traditionally used in the mitigation of epileptic action, are now widely used in the treatment of psychiatric indications and pain disorders. However, the mechanisms of action underlying the mood-stabilizing and anti-nociceptive properties of specific anticonvulsants remain unclear despite their documented success in the treatment of bipolar disorder and trigeminal neuralgia. Understanding the mechanisms of action behind such therapeutic properties may enable the creation of new and more efficacious treatment options. Therefore, this paper will review existing literature concerning the mechanisms of action of the anticonvulsant carba-mazepine in the treatment of epilepsy, trigeminal neuralgia, and bipolar disorder in order to provide a comprehensive summary of existing evidence and hypotheses.

Epilepsy

Seizure Pathology

Epileptic seizures typically result from hyper-activity of neurons in the brain, which can be caused by an imbalance of excitatory and inhibitory neural mechanisms (Lasón, Chlebicka & Rejdak 2013). Animal models of seizure have confirmed that both excessive excitatory neuronal activity and diminished inhibitory neuronal activity have the capacity to produce seizures resembling those of a human epilepsy patient (Lasón et al., 2013). The hyperactivity of neurons during seizures typically results from one of two mechanisms: excessive glutamate transmission or disturbances in either sodium or calcium ion channels (Lasón et al., 2013). On the other hand, the diminished inhibitory mechanism underlying seizures typically results from diminished GABAergic transmission (Lasón et al., 2013).

GABA and Sodium Channels

GABAergic neurons, which comprise the main inhibitory system in the brain, act as interneurons between pyramidal glutamate cells and prevent large-scale excitatory firing in the brain, a characteristic of seizures (Lasón et al., 2013). $GABA_A$ receptors are ionotropic receptors that, when active, flux chloride ions into neurons. This cellular influx of negatively charged ions causes neuronal hyperpolarization and thus cessation of action potential generation (Lasón et al., 2013). Carbamazepine utilizes this pathway as a mechanism of anti-epileptic action by acting as a positive modulator of $GABA_A$ receptors (Granger et al., 1995). The resulting increase in the $GABA_A$ receptor chloride current upon carbamazepine binding, either through an increase in the receptor's affinity for GABA or a prolonged "open" receptor conformation upon GABA binding, serves in carbamazepine's anticonvulsant effects by impeding the hyperactivity of neurons (Granger et al., 1995).

The sustained and high frequency firing of neurons during epileptic seizures is also made possible due to persistent neuronal depolarization which lies below the threshold level for voltage-gated sodium channel inactivation (Lasón et al., 2013). This causes the sodium channels, which generate action potentials through Na^+ influx and membrane depolarization, to remain active for prolonged periods of time. This sustained sodium channel activation consequently maintains neuronal depolarization and results in high frequency neuronal firing during seizures (Lasón et al., 2013). Such channel malfunctions during seizures provide an avenue through which carbamazepine predominantly exerts its anticonvulsant effects (Rogawski & Löscher, 2004). Acting

as a voltage-gated sodium channel blocker, carbamazepine binds with specificity to the inactive sodium channel state in a frequency dependent manner (Rogawski & Löscher, 2004). The increase in neuronal firing associated with epilepsy increases the frequency of the channel's inactive state, a conformation that brings specific amino acid residues into contact and facilitates carbamazepine's binding (Lasón et al., 2013). Therefore, during seizures, carbamazepine binds more frequently to sodium channels due to an increase in neuronal firing and a consequent increase of receptors facilitating carbamazepine binding (Rogawski & Löscher, 2004). Once bound to the receptor, carbamazepine stabilizes the inactive state, decreases the availability of voltage-gated sodium channels during seizures, and reduces neuronal hyperactivity (Rogawski & Löscher, 2004). The resulting decrease in epileptic hyperactivity, through both carbamazepine's inactivation of sodium channels and potentiation of hyperpolarizing chloride currents, also serves to indirectly decrease glutamate release (Granger et al., 1995). Carbamazepine also directly decreases glutamate release via NMDA receptor modulation, but this mechanism plays an insignificant role in epilepsy treatment in comparison to voltage-gated sodium channel inactivation (Rogawski & Löscher, 2004).

Chronic Pain and Sodium Channels

In addition to their role in seizure generation, voltage-gated sodium channels have also been implicated in nociception. Recent studies on pain insensitivity revealed that individuals with this disorder lack the voltage gated sodium channel 1.7 (Nav1.7), implicating this channel as a critical aspect of the pain pathway (Cummins, Sheets & Waxman, 2007). In addition, researchers have shown that in transgenic mice models, knocking out the gene *SCN9A*, which encodes NAv1.7, reduces sensitivity to acute pain. (Cummins et al., 2007). Point mutations in the gene *SCN9A* have also been implicated in the chronic pain disorder erythromelalgia (Cummins et al., 2007). The point mutations associated with this condition have been shown to increase neuronal firing rates by decreasing the action potential threshold and increasing the length of time required for channel deactivation (Cummins et al., 2007). From these results, it is hypothesized that repetitive firing of nociceptive neurons due to voltage-gated sodium channels (such as Nav1.7) translates into pain sensation (Cummins et al., 2007).

Trigeminal Neuralgia and Sodium Channel Hyper-excitability

This implication of voltage-gated sodium channels and repetitive neuronal firing in nociception helps explain the use of carbamazepine in the treatment of trigeminal neuralgia, a chronic pain condition affecting the trigeminal nerve. The efficacy of carbamazepine in the treatment of both epilepsy and neuralgia supports the theory that shared neuronal hyper-excitability underlies both disorders, as blocking repetitive firing of sodium channels mitigates the symptoms of both disorders (Bialer, 2012). It thus stands to reason that if this sodium channel pathophysiology does indeed underlie both conditions, then a large percentage of anti-epilepsy drugs should also be effective treatments of neuralgia. However, this is not the case. Only a small number of such anticonvulsants effectively treat trigeminal neuralgia, suggesting that the causal overlap between epilepsy and neuralgia may not be as large as initially assumed (Bialer, 2012). Such discrepancies between anticonvulsant efficacy in trigeminal neuralgia and epilepsy raises questions about the mechanism through which anticonvulsants help relieve the pain associated with this disorder. Although it is possible that anticonvulsants with analgesic properties bind to sodium channels specific to sensory neurons involved in pain perception, more research should be conducted in order to explain the differences in anticonvulsant efficacy in trigeminal neuralgia treatment.

Manic-Depressive Disorder

GABA Receptors

The last FDA approved use of carbamazepine is in the treatment of manic-depressive disorder, a condition characterized by swings between depressive and manic states. The initial hypotheses behind the success of carbamazepine and other anticonvulsants in the treatment of both bipolar disorder and epilepsy rely upon on the hyper-excitability shared by these seemingly otherwise disparate conditions (Bialer, 2012). Thus, researchers propose that carbamazepine's ability to reduce hyper-excitability in epileptic conditions explains the drug's ability to prevent the excessive excitability of the manic state associated with bipolar disorder (Bialer, 2012). One such hypothesis centers upon carbamazepine's agonistic effect on GABA transmission, as polymorphisms in the GABA gene correlate strongly with bipolar disorder (Grunze, 2010). Researchers found that both carbamazepine and valproate, efficacious treatments for bipolar disorder, did exhibit GABA

agonistic behaviors, as described above (Granger et al., 1995). However, the lack of efficacy of other GABAergic anticonvulsants (e.g. gabapentin and tiagabine) in the treatment of bipolar disorder undermine this mechanistic hypothesis, which suggests that another mechanism underlies carbamazepine's mood-stabilizing effects (Grunze, 2010).

NMDA Receptors

Researchers continue to study carbamazepine and other mood-stabilizing anticonvulsants in order to pinpoint additional shared mechanisms that may explain carbamazepine's efficacy in treating bipolar disorder. Following the potential link between hyper-excitability and manic episodes, researchers analyzed the effects of carbamazepine, valproate and lamotrigrine on NMDA receptors, receptors that flux cations (Na^+ or Ca^{2+}) upon glutamate binding and subsequently cause neuronal depolarization (Grunze, 2010). Studies found that chronic use of both carbamazepine and valproate decreased NMDA receptor activity through the arachidonic acid cascade (Grunze, 2010). In this cascade, glutamate binds to NMDA receptors and activates Ca^{2+} dependent enzymes such as phospholipase A, which in turn increases levels of arachidonic acid and its downstream effectors (COX-2 and PGE) (Basselin, Chang, Chen, Bell & Rapoport, 2008). Through this attenuation of COX activity, NMDA receptor activity and overall glutamate release diminishes, a mechanism that will be discussed in more detail during the following analysis of the arachidonic acid cascade (Basselin et al., 2008). Consequently, this link between the excitation hypothesis and arachidonic acid cascade hypothesis supports the inhibition of excitatory amino acid signaling as a potential contributor to the use of anticonvulsants in the treatment of bipolar disorder.

Neurotransmitter Pathways

With the basic inhibitory and excitatory pathways explored and limited explanations accrued for carbamazepine's mood-stabilization mechanism, researchers turned to other neurotransmitter pathways associated with bipolar disorder. For instance, a genetic variation that decreases synthesis of tyrosine hydroxylase, the rate-limiting enzyme in serotonin synthesis, has been implicated as a risk factor for bipolar disorder (Grunze, 2010). With this connection in mind, researchers hypothesized that efficacious treatment for bipolar disorder may act to increase serotonergic transmission. Results confirmed this hypothesis, demonstrating that lithium, valproate, and carbamazepine all amplify serotonin levels (Grunze, 2010). Such evidence serves to validate increased serotonergic signaling as a potential mechanism of action. However, if this pathway does act as the primary driver of carbamazepine's mood-stabilization effects, then one might expect that providing 5-HTP to patients with bipolar disorder would alleviate manic symptoms. On the other hand, one would expect that decreasing serotonergic transmission in the brain (via a receptor agonist or depletion of tryptophan) would result in manic-like symptoms. Evidence for such results has not been found, raising questions about the importance of serotonergic transmission in treatment of bipolar disorder.

Inositol

With a lack of support for glutamatergic, GABAergic and serotoninergic mechanisms, the answer to carbamazepine's mood-stabilizing properties may be found through inositol. Lithium, the original mood-stabilizer used in treatment of bipolar disorder, inhibits inositol monophosphatase, an enzyme required for inositol recycling (Kim & Thayer, 2009). The resulting decrease in myo-inositol also has downstream consequences, as the molecule plays a critical role in inositol phosphate based cellular signaling (Kim & Thayer, 2009). Recent studies of carbamazepine and valproate have confirmed similar effects of drug use on inositol levels, as the two drugs both decrease inositol turnover, while valproate additionally inhibits inositol monophosphate synthesis (Granger et al., 1995). The shared effects of all three drugs in this pathway provide promising support for an inositol based mechanism of action, as decreased myo-inositol levels exhibit downstream effects, such as reduced Ca^{2+} calmodulin activation and reduced CAM Kinase II activity, an enzyme implicated in synaptic changes and cyoskeleton restructuring (Grunze, 2010). These downstream effects were even evidenced through findings that all three drugs increase neuronal growth cone spreading, a change abolished with inositol reintroduction (Kim & Thayer, 2009). Furthermore, MRI studies present support for this mood-stabilization hypothesis of attenuated inositol phosphate signaling and synapse formation, as bipolar patients demonstrate significant increases in gray matter with lithium treatment (Kim & Thayer, 2009). Thus, the evidence linking efficacious bipolar treatments to myo-inositol depletion and increased growth cone spreading presents a strong argument for a synaptic restructuring mechanism of action.

One problem with the inositol depletion mechanism described above concerns the research conducted by Kim & Thayer (2009). In this study, which primarily corroborates the aforementioned evidence underlying the inositol phosphate signaling hypothesis of mood-stabilization, there was found to be a discrepancy between the onset of synapse formation and therapeutic relief from manic-depressive symptoms (Kim & Thayer, 2009). It is possible that such a discrepancy results from differences in rate of inositol depletion between mice and humans or differences in rate of synaptic change between in vitro and in vivo models (Kim & Thayer, 2009). However, at this time, the therapeutic lag associated with mood-stabilizing drugs (such as carbamazepine) in relation to the rapid onset of inositol depletion and synaptic change shown in this experiment destabilizes this mechanistic hypothesis (Kim & Thayer, 2009). Furthermore, additional research explaining the potential relationship between increased synaptic connections and mood are necessary to strengthen this hypothesis, as decreased synaptic connections may not be the causal pathophysiology of bipolar disorder.

Arachidonic Acid Cascade

Finally, researchers have studied the connection between mood-stabilizing drugs and the arachidonic acid cascade in the brain, which was previously discussed in concert with carbamazepine's inhibition of NMDA receptors. This cascade begins with stimulation of G-protein coupled receptors, which activates phospholipase A (PLA) and causes the production of arachidonic acid, a secondary messenger involved in gene regulation and neuronal excitation (Duncan & Bazinet, 2010). Both chronic lithium and carbamazepine use affect genes involved in this cascade, causing a decrease in PLA transcription, which in turn decreases arachidonic acid production and signaling (Duncan & Bazinet, 2010). In addition, both drugs also decrease availability of arachidonic acid's downstream effectors, COX-2 and prostaglandin E (PGE), through an inhibition of transcription, an effect also seen with chronic valproate administration (Duncan & Bazi-net, 2010). This reduction in COX-2 enzymes by carbamazepine and valproate contributes to their ability to decrease NMDA signaling as previously discussed, as the activity of COX enzymes is critical to NMDA's receptor response to drug binding (Basselin et al., 2008). As a result, carbamazepine and valproate's ability to attenuate signaling by the arachidonic acid cascade acts as a mechanism through which the drugs simultaneously regulate neuronal excitation and gene expression, both potential mechanisms of mood-stabilization. (Basselin et al., 2008)

In order to support this attenuation of arachidonic acid signaling as a potential mood-stabilization mechanism, researchers also analyzed the effects of anti-depressants such as fluoxetine, which increase the risk of mania in bipolar patients, on the arachidonic acid cascade (Duncan & Bazinet, 2010). As predicted by this hypothesis, fluoxetine increases turnover of arachidonic acid and PLA activity, as did imipramine, an anti-depressant that shares the risk of mania inducement in bipolar patients (Duncan & Bazinet, 2010). On the other hand, when researches studied anti-depressant bupropion, which does not carry the same risk of mania inducement, no change in PLA activity or arachidonic acid turnover was seen (Duncan & Bazinet, 2010). This tight correlation between a drug's effects on PLA and arachidonic acid and its risk of inducing manic states supports the hypothesis that arachidonic acid signaling might underlie the efficacy of carbamazepine in the treatment of bipolar disorder (Duncan & Bazinet, 2010).

Further research on the arachidonic acid cascade

Despite the evidence indicating that lithium, carbamazepine and valproate all decrease signaling via the arachidonic cascade, further research needs to be conducted before this hypothesis can be confirmed. For instance, although there have been studies implicating the arachidonic signaling cascade in neuronal hyper-excitability and regulation of gene expression, further information about the link between this signaling pathway and mood-stabilization will need to be acquired. Specifically, more information regarding the arachidonic acid cascade's genetic targets and their connection to mood would fill gaps between this mechanism and carbamazepine's mood-stabilizing effects. In addition, another problematic aspect of this hypothesis concerns the stimulation of arachidonic acid production by serotonin (Duncan & Bazinet, 2010). If carbamazepine administration increases serotonin levels and serotonin stimulates the production of arachidonic acid, then this serotonergic effect of carbamazepine appears contradictory to carbamazepine's reduction of arachidonic acid levels. Although it may be possible to reconcile this discrepancy, as carbamazepine blocks the downstream effectors of arachidonic acid, it would be beneficial to have

evidence that serotonin-induced arachidonic acid production is insufficient to overcome carbamazepine's block on COX-2 and PGE. Finally, studies concerning the time frame of arachidonic acid reduction and onset of carbamazepine's therapeutic effects would be informative. For instance, if arachidonic acid levels and NMDA excitation drop rapidly upon carbamazepine administration, but mood-stabilization effects develop after a lengthy time period, the existence of therapeutic lag could provide support for arachidonic acid regulation of genetic expression (a slow process). However, the existence of therapeutic lag could also undermine the arachidonic acid hypothesis in favor of a different mechanistic pathway. Such unanswered ques-tions reveal the importance of further studies and the lack of understanding surrounding carbamazepine's efficacy in manic-depressive treatment.

Conclusion

Although carbamazepine's mechanisms of action in the treatment of epilepsy and trigeminal neuralgia are relatively well understood, there exist multiple proposals to explain the drug's mood-stabilizing properties. The most supported hypotheses center upon the inositol phosphate signaling pathway and the arachidonic acid cascade, as these mechanisms are common and specific to the three efficacious treatments of bipolar disorder: lithium, carbamazepine, and valproate. However, despite the evidence supporting these pathways as mechanisms of action, the links between inositol-phosphate signaling, the arachidonic acid cascade, and carbamazepine's efficacy in manic-depressive disorder treatment remain unclear. Thus, further research must be conducted in order to elucidate the pathways contributing to carbamazepine's mood-stabilizing effects in the hopes that the knowledge will enable the development of new therapeutic options for the treatment of manic-depressive disorder.

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Kira Radstrom is a fourth-year undergraduate psychology student at the University of California, Los Angeles. She is also pursuing a minor in cognitive science with a focus on the biological basis of human cognition. Kira works as a research assistant in the UCLA Addictions Research Laboratory under Dr. Lara Ray. Currently, she is assisting with clinical pharmacogenetic research examining the effects of genetic factors and the drug naltrexone on craving for alcohol in Asian Americans. In her spare time, Kira enjoys visiting museums, hiking, practicing yoga, and volunteering with a women's community service organization. Kira plans to attend graduate school within the next few years to pursue an additional degree in a psychology- or health-related field. Until then, she plans to travel and work as she narrows down her academic interests.

What are your favorite journals?

My interests within psychology include brain development, abnormal psychology, and addiction in particular. A few publications that I am consistently drawn to are Behavior and Brain Science, Journal of Abnormal Psychology, and Addiction.

Was there any particular experience that sparked your research interests?

A close family member of mine has struggled with alcohol use problems since I was a child. We have a great relationship, but I have always desired to learn more about the mechanisms behind addiction in order to better understand his disorder. Attending UCLA and joining the Addictions Research Laboratory has allowed me to further develop my research interests, and I hope to continue investigating these issues post-graduation.

Besides research, what do you do for fun?

Visiting museums, hiking with my dog, practicing yoga, and exploring downtown Los Angeles are a few ways that I like to spend my free time. When I have time off from work, research, and school, I love taking long trips on the open road with friends and camping along the way.

Besides psychology, what other fields interests you?

A few other fields that interest me are cognitive science, genetics, and art history. Cognitive science is particularly fascinating to me due to its interdisciplinary nature. Studying cognitive science at UCLA has allowed me to explore diverse topics such as artificial intelligence, philosophy, and neuroscience, all under the umbrella of the study of intelligent systems.

What part of research do you find most fun/challenging?

One of my responsibilities as a research assistant in the UCLA Addictions Research Laboratory is to conduct in-lab assessment visits with participants who have drug or alcohol use problems. Working directly with a clinical population provides both a challenge and an opportunity to gain valuable insight into the subjective experience of addiction.

Comorbid Alcohol Use Disorder and Anxiety Disorders: Etiology and Treatment Implications

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Anxiety disorders and alcohol use disorder (AUD) frequently co-occur. Individuals with anxiety disorders are more susceptible to the initial development of alcohol dependence, and often experience an accelerated transition from non-pathological alcohol use to dependence. Additionally, a pre-existing AUD increases an individual's susceptibility to anxiety. Several explanations for the observed rates of comorbidity have been proposed, including self-medication to alleviate anxiety symptoms, development of substance-induced anxiety, and a common etiology of anxiety and AUD. The implications of comorbidity on treatment for alcoholism remain contentious; however, psychiatric anxiety interventions and pharmacotherapies combined with traditional AUD programs have been suggested as possible treatment strategies.

Anxiety disorders are frequently observed in alcohol-dependent populations, yet the mechanisms linking these disorders remain elusive. Individuals with comorbid alcohol use disorder (AUD) and anxiety disorders often experience accelerated development of alcohol dependence, face greater challenges in treatment, and are more susceptible to alcohol relapse than their non-comorbid counterparts (Hobbs, Kushner, Lee, Reardon, and Maurer, 2011; Kushner, Abrams, Thuras, Hanson, Brekke, and Sletten, 2005; Kushner, Maurer, Menary, and Thuras, 2011). Thus, the comorbidity of anxiety disorders and AUD has clinical implications and provides a challenge for patients, clinicians, and researchers alike. Several theories have been proposed to explain the co-occurrence of AUD and anxiety disorders, but no definitive consensus has been reached. Similarly, although several lines of treatment for comorbid AUD and anxiety have been empirically examined, no standard intervention has been established.

Much of the existing literature in this field has relied on the distinct classifications of substance abuse and substance dependence, as outlined in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The DSM-V has combined and strengthened the criteria for these two categories into a single "substance use disorder" (American Psychiatric Association, 2013). This review refers to alcohol dependence and alcohol use disorder, although the latter is the current clinical classification.

Prevalence of Comorbid Anxiety and AUD

Individuals with an anxiety disorder are two to four times more likely to develop alcohol dependence than those without an anxiety disorder (Kushner et al., 2011). Similarly, the rate of comorbid anxiety disorders in patients being treated for alcohol or substance use disorders has been estimated to lie between 42.3% and 50% (Kushner et al., 2005; Kushner et al., 2011; Schadé, Marquenie, Van Balkom, De Beurs, Van Dyck, and Van Den Brink, 2003). However, data regarding rates of comorbidity between specific anxiety disorder subtypes (or multiple concurrent anxiety disorders) and AUD have been mixed (Crum, La Flair, Storr, Green, Stuart, Alvanzo, Lazareck, Bolton, Robinson, Sareen, and Mojtabai, 2013; Kushner et al., 2005; Schadé et al., 2003). In a review of epidemiological perspectives on comorbidity, Kushner, Krueger, Frye, and Peterson (2008) suggest that generalized anxiety disorder (GAD) has the highest rate of comorbidity with AUD, followed by agoraphobia and social phobia. However, in a community survey of 3021 (2548 at follow-up) adolescents in Munich, panic disorder and social phobia were associated with subsequent development of alcohol problems, but other anxiety disorders did not significantly predict AUD incidence or outcomes (Zimmerman, Wittchen, Höfler, Pfister, Kessler, and Lieb, 2003). The inconsistency in these findings may be due to unique characteristics of clinical samples that limit the generalizability of results to other populations. However, the current body of research suggests that the presence of an anxiety

disorder, regardless of subtype, may influence the course of development of AUD. Generally, individuals with anxiety disorders appear to be more vulnerable to the development of alcohol dependence, and are more likely to experience an accelerated transition from regular, non-pathological alcohol use to alcohol dependence (Kushner et al., 2011; Zimmerman et al., 2003). The presence of AUD may also increase one's vulnerability to the development of an anxiety disorder and contribute to the maintenance of anxiety symptoms (Allan, 1994; Kushner et al., 2011; Smith et al., 2012).

Etiology of Comorbidity

Self-Medication: AUD as a Consequence of Anxiety

The directionality of the relationship between pathological anxiety and AUD is an important etiological factor to consider, and several pathways of comorbidity have been proposed. The self-medication model is a prominent explanation for the high rate of comorbidity between AUD and anxiety disorders. This model purports that individuals suffering from an independent anxiety disorder attempt to alleviate anxiety symptoms by drinking alcohol. Repeated attempts to self-medicate anxiety create a cycle of alcohol consumption, thus fostering the development of AUD (Crum et al., 2013; Robinson, Sareen, Cox, and Bolton, 2009; Smith et al., 2012). Anxiety disorders may be classified as independent from substance use if their onset predates the onset of AUD, or if symptoms do not resolve even after periods of abstinence lasting one month or more (Kushner et al., 2011; Smith et al., 2012). The relationship between anxiety and AUD may vary across anxiety subtypes. For example, phobias and dual diagnoses (the presence of multiple anxiety disorders) tend to predate AUD, while panic disorder and GAD tend to develop after the initial onset of AUD (Crum et al., 2013; Schadé et al., 2003).

Evidence for this model is provided by data directly linking self-medication with AUD. Crum and colleagues (2013) conducted analyses of self-report data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a national household comorbidity survey, which revealed that drinking to self-medicate anxiety was significantly associated with subsequent development and persistence of alcohol dependence ($p < .001$). Robinson and colleagues (2009) also utilized the NESARC to analyze prevalence of self-medication with alcohol across specific anxiety disorder subtypes. Their analyses indicated that 79.1% of individuals with comorbid AUD and anxiety disorder reported using alcohol to self-medicate. Together, these findings demonstrate an association between self-medication and problems with alcohol in a nationally representative sample. Additionally, in a study of anxiety in individuals receiving treatment for alcohol dependence, Kushner and colleagues (2011) found that anxiety disorders were not correlated with an overall increase in alcohol consumption but instead were related to increased problems with alcohol due to drinking to alleviate negative affect. This suggests that anxiety disorders may be more directly linked to drinking motives rather than the amount of alcohol consumption.

Substance-Induced Anxiety Disorders

Contrary to the self-medication model, which postulates that individuals with primary anxiety disorders develop alcohol dependence through a cycle of repeated attempts to alleviate anxiety symptoms with alcohol, the substance-induced anxiety model proposes that a primary AUD triggers and maintains anxiety symptoms (Allan, 1994). Chronic cycles of consumption and withdrawal may alter the nervous system in ways that induce or exacerbate anxiety. Although acute alcohol consumption produces an anxiolytic effect by boosting activity of the neurotransmitter a-aminobutyric acid (GABA), persistent and excessive alcohol use leads to a general GABA deficiency. This deficiency counteracts the anxiolytic effect of acute alcohol exposure and may generate anxiety symptoms. Frequent episodes of withdrawal may also result in neurological changes that increase individuals' vulnerability to the development of anxiety over time. (Smith et al., 2012).

Although the majority of anxiety disorders occurring in the general United States population, as well as in alcohol-dependent populations, are independent of substance use disorders, AUD may elicit anxiety symptoms in some individuals (Kushner et al., 2005). In such cases, AUD predates the anxiety disorder, and anxiety symptoms diminish after a period of abstinence lasting one month or more (Kushner et al., 2011).

Common Etiology of Anxiety and AUD

Another possibility is that AUD and anxiety stem from a common etiology. This model posits that AUD and anxiety do not cause one another but instead co-occur as a result of an unknown third variable (Zimmerman et al., 2003). Genetic vulnerability, personality traits, anxiety sensitivity,

and environmental factors have been proposed as possible common factors of AUD and anxiety (Crum et al., 2013; Kushner et al., 2000; Smith et al., 2013). Support for this model is limited, as most of the existing literature only indirectly substantiates its claims. Family and twin studies provide some correlational evidence for the common influence of genetic and environmental factors, but consistent overlap in specific risk factors relevant to both disorders has not been established (Kushner et al., 2000; Smith et al., 2013).

Alternatively, the presence of one disorder may make individuals more susceptible to the other disorder indirectly through an unknown third variable (Robinson et al., 2009). Due to the elusive nature of examining such an unknown variable, there is a lack of data regarding this theory. Currently, the collective literature does not exclusively support nor exclude any of the proposed models, suggesting that the nature of comorbidity may be heterogeneous or subject to individual differences. It is possible that anxiety and AUD may develop independently as a result of an unknown common factor. However, the timeline of onset of AUD versus anxiety may provide insight into the possible mechanisms of comorbidity on a case-by-case basis. For example, a pre-existing anxiety disorder may increase an individual's vulnerability to the development of AUD through a variety of mechanisms. Conversely, an individual suffering from primary AUD may develop symptoms of anxiety as a consequence. The relationship between anxiety and AUD may also be bidirectional, such that anxiety symptoms are exacerbated by pathological alcohol use, while alcohol consumption is maintained as an attempt to alleviate anxiety and negative affect.

Treatment Implications

Treatment of comorbid AUD and anxiety disorders may require specialized strategies. Alcoholism patients with a comorbid anxiety disorder experience increased vulnerability to relapse after alcoholism treatment and may require unique intervention strategies. Kushner and colleagues (2005) found that patients in an alcoholism treatment program with a baseline anxiety disorder experienced significantly greater relapse rates following treatment than did patients without a baseline anxiety disorder. Baseline anxiety patients were also faster to relapse, as indicated by fewer days to first drink and fewer days to first drinking binge. Moreover, anxiety disorder persisted without a relapse to alcohol dependence ten times more often than relapse to alcohol dependence occurred without a persistent anxiety disorder. The persistence of anxiety symptoms despite prolonged abstinence from alcohol indicates that anxiety was likely to be independent of AUD in this sample. The presence of specific anxiety subtypes such as panic disorder and social phobia predicted relapse to alcohol use and dependence, but the psychiatric severity of the anxiety disorder(s) predicted persistence of anxiety symptoms post-treatment (Kushner et al., 2005).

Comorbidity appears to complicate treatment for both disorders. Reducing anxiety symptoms may enhance AUD treatment, but studies directly examining the effects of anxiety interventions on AUD outcomes are scarce (Kushner, Donahue, Sletten, Thuras, Abrams, Peterson, and Frye, 2006; Schadé et al., 2003). Furthermore, it is possible that anxiety disorders with co-occurring AUD require unique treatment. Anxiety-focused interventions may be particularly important in cases where anxiety is substance-independent, while substance-induced anxiety may resolve with AUD treatment alone (Kushner et al., 2005). The use of pharmacotherapies for anxiety in comorbid AUD patients also requires special consideration, as this population is particularly vulnerable to substance abuse (Schadé et al., 2003). A standard course of treatment for comorbid anxiety and AUD has yet to be established, but both psychiatric and pharmacological interventions have been examined.

Supplemental Anxiety Interventions

Clinical outcomes for both anxiety and AUD in comorbid populations may be improved by supplementing traditional AUD treatment with psychiatric anxiety interventions. A meta-analysis of supplemental interventions for depressive and anxiety disorders in alcoholism treatment programs revealed that supplemental treatments for internalizing disorders resulted in small but significant improvements in alcoholism treatment outcomes, particularly in terms of reduced relapse sensitivity. Traditional anxiety interventions were moderately effective at reducing anxiety in alcohol-dependent populations, but the effects were smaller than in non-comorbid populations (Hobbs et al., 2011). The reduced effectiveness of traditional anxiety interventions in comorbid versus non-comorbid populations suggests that anxiety disorders that co-occur with AUD may also require specialized treatment.

Integrated cognitive-behavioral therapy (CBT) has been

examined as a possible psychiatric treatment for comorbid anxiety in AUD patients. Kushner et al. (2006) designed an AUD treatment program with a supplemental CBT intervention containing psychoeducation, cognitive restructuring, and cue exposure components. Participants that underwent the supplemental CBT intervention experienced significant improvements in anxiety symptoms. The CBT intervention was also reduced the severity of relapse to alcohol use, although the overall rate of relapse was not affected. These findings suggest that supplemental anxiety treatments may be more effective for reducing the severity of relapse than for preventing future alcohol use completely.

The collective evidence is stronger for the benefits of anxiety interventions on anxiety symptoms than for the benefits of such interventions on AUD outcomes. In one randomized controlled trial (RCT) examining the efficacy of anxiety treatment in comorbid AUD and phobic disorder patients, anxiety treatment reduced anxiety symptoms but did not have an effect alcohol relapse rates (Schadé et al., 2003). Methodological inconsistencies such as differences in specific psychiatric anxiety treatments may contribute to the disagreement of results across studies.

It is currently feasible for psychiatric anxiety interventions to be integrated into conventional AUD treatment programs in a way that is both economically and clinically beneficial (Hobbs et al., 2011; Kushner et al., 2006). Early diagnosis of comorbid anxiety in patients receiving treatment for AUD would provide information essential for the assessment of relapse risk and the implementation of an appropriate treatment regimen. This could be accomplished with a clinical screening at the initiation of treatment (Kushner et al., 2005). Questions remain as to how narrowly or broadly an anxiety treatment component should be tailored to specific anxiety disorder subtypes, whether group or individual treatment is superior, and whether outpatient or inpatient programs are more effective. Kushner et al. (2006) suggest that a broadband, group-format, outpatient treatment program would be the most effective and economical. Additional randomized controlled trials are needed to assess the efficacy of such a program.

Pharmacotherapy

Supplementing AUD treatment with pharmacotherapies for anxiety may also improve treatment outcomes. Medications commonly prescribed for anxiety include benzodiazepines, monoamine oxidase inhibitors (MAO-Is), tricyclic antidepressants (TCAs), and serotonergic-based drugs such as selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and the serotonin (5-HT1a) partial agonist busiprone (Smith et al., 2013). Although these drugs are well-established anxiety treatments, there is limited data on their efficacy in comorbid AUD populations because clinical trials tend to exclude this demographic (Book, Thomas, Randall, and Randall, 2007). In light of this lack of information, clinicians must carefully consider the risks of anxiety medications for this particular population. Benzodiazepines pose the greatest risk of abuse for comorbid anxiety and AUD individuals because alcoholics are more sensitive to their rewarding properties. Therefore, benzodiazepines should be prescribed with great care and close monitoring. MAO-Is and TCAs must also be used with caution due to the risk of adverse interactions with alcohol. However, limited research has suggested that SSRIs, SNRIs, and busiprone may be safe alternatives (Book et al., 2007; Shadé et al., 2003; Smith et al., 2013). Book and colleagues (2007) found that the SSRI paroxetine was effective at reducing anxiety symptoms in a comorbid AUD and social anxiety sample but did not affect AUD outcomes. A meta-analysis of the efficacy of anxiety treatments in comorbid samples conducted by Shadé and colleagues (2003) also concluded that paroxetine improved anxiety symptoms but did not improve AUD outcomes. Busiprone reduced anxiety symptoms in three out of four studies but did not significantly influence alcohol dependence (Schadé et al., 2003). Although there is some evidence for the benefits of anxiety medications in this population, additional research is required to determine whether combined pharmacotherapy and AUD treatment would further improve AUD and anxiety outcomes.

Conclusions

The prevalence of comorbid anxiety and AUD demonstrates a clear need for examination by the scientific community. Currently, no definitive consensus regarding the causal pathways or clinical implications of this comorbidity has been established. Possible pathways of development that have received attention in research include self-medication for anxiety, substance-induced anxiety, and common etiology. Several treatment strategies for anxiety and AUD have been empirically assessed, and an integrated format of treatment

appears to be most effective for anxiety- focused psychiatric interventions (Kushner et al., 2006; Smith et al., 2013). Although several medications have been shown to reduce anxiety symptoms in comorbid AUD populations, integrated pharmacological interventions may provide additional benefits and require further research (Book et al., 2007; Shadé et al., 2003; Smith et al., 2013). Methodological limitations emerge from the existence of multiple pathways to comorbidity that may influence responses to treatment across individuals, in addition to the possibility of differences in treatment requirements for specific anxiety subtypes. Randomized controlled trials assessing the various treatment strategies in comorbid populations would provide the greatest benefit to the body of knowledge regarding this issue. The prevalence of comorbid anxiety and AUD, lack of conclusive evidence for one specific explanation of comorbidity, and uncertainty surrounding treatment implications make the conundrum of comorbidity a worthy subject for empirical examination.

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Jean Stafford

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I am currently completing my final year of an undergraduate psychology degree at the University of Edinburgh. Some modules which I have found particularly interesting during my degree include 'psychological therapies', 'abnormal psychology' and 'memory, aging and the brain'. During my degree I also gained a place on an international exchange programme. I spent my exchange at McGill University in Montreal, which broadened by horizons both academically and personally as I adjusted to life within a different culture. At McGill I took classes in 'human motivation' and 'child and adolescent psychopathology' which I found fascinating. During the exchange programme I gained experience working as a research assistant in a laboratory and also gained practical experience working in a clinical environment. After completing my degree I am going on to do an MSc in child and adolescent mental health which has a practical component. Following this I plan to complete a doctorate in clinical psychology and to work towards becoming a clinical psychologist. Aside from psychology I am also interested in music- I play piano, guitar and enjoy singing. In my spare time I enjoy spending time with friends and family, travelling, reading novels, cooking, keeping fit and learning French.

Where do you envision yourself in 10 years?

In ten years I see myself practicing as a clinical psychologist after having completed a postgraduate degree in clinical psychology. I also hope to be involved in conducting research within the field of clinical psychology.

Was there any particular experience that sparked your research interests?

My interest in research was sparked while volunteering as a research assistant during summer. The laboratory I worked in focussed on child and adolescent social abilities and mental health, which I found very interesting. I gained a variety of experience while working at the laboratory, including running participants and data entry.

What interests you the most about psychology?

An aspect of psychology that I find particularly interesting is the fact that it is made up of varied sub-disciplines, which provide different perspectives on understanding human behaviour.

Besides research, what do you do for fun?

Aside from keeping up-to-date with current research in psychology, in my spare time I enjoy playing piano, guitar and singing. I also enjoy taking French language classes, reading novels and keeping fit.

Besides psychology, what other fields interests you?

I am very interested in the field of sociology. I feel that it addresses some of the same issues as psychology but from a different perspective. I am also interested in philosophy and the philosophical origins of psychology. Having studied English literature for two years I also find this field very interesting.

Why do older adults exhibit patterns of 'over-recruitment' in the prefrontal cortex in functional neuroimaging studies?

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The present review examined encoding and retrieval studies of episodic memory to synthesize findings on prefrontal over-recruitment with aging. The review focused particularly on the ventrolateral prefrontal cortex and dorsolateral prefrontal cortex as in some studies these regions are the sites of increased activity in older adults. This over-recruitment pattern has been linked to both successful and poorer performance. Three explanations for over-recruitment were considered: (1) the compensatory hypothesis which suggests over-recruitment occurs as an adaptive process to counter neural deficits, (2) the de-differentiation hypothesis which views over-recruitment as a symptom of reduced neural specificity, and (3) the partial-compensation hypothesis which posits that adaptive compensation occurs, but not sufficiently to fully compensate for deficits. Findings gathered from a review of the literature assessing age-related changes in prefrontal over-recruitment were mixed and often contradictory. Findings supported over-recruitment in the presence of poor task performance, and therefore the compensatory hypothesis does not fully account for over-recruitment. Instead, it is suggested that partial compensation may account for the link between over-recruitment and poor performance. Nonetheless, de-differentiation cannot be ruled out based on present findings. Finally the importance of using transcranial magnetic stimulation in future studies is emphasised as a means of teasing apart potential explanations of over-recruitment.

Episodic memory, broadly defined as memory of past events and their contexts, shows marked decline with age (Nilsson, 2003). Simple deficit theories posit that this decline in episodic memory is caused by a decrease in neural activity in associated regions, such as the frontal lobes (Wheeler, Stuss, & Tulving, 1997). However, this theory is inconsistent with an emerging pattern of increased neural activity observed in regions of the prefrontal cortex (PFC) in older adults (Spreng, Wojtowicz, & Grady, 2010). This pattern has been labeled 'over-recruitment' and can be observed in both encoding and retrieval episodic memory studies (Eyler, Sherzai, Kaup, & Jeste, 2011). Mixed findings have linked over-recruitment to both successful performance (Dennis, Kim, & Cabeza, 2007) and poorer performance (Duverne, Motamedinia, & Rugg, 2009). Various hypotheses have been proposed to explain over-recruitment, some of which appear to contradict each other. One common interpretation of over-recruitment is the compensatory hypothesis, which posits that older adults exhibit more activity in areas of the PFC, such as the dorsolateral prefrontal cortex (DLPFC) (Gutchess et al., 2005), than younger adults in order to compensate for deficits present in other regions including the medial temporal lobes (MTL) (Reuter-Lorenz, & Lustig, 2005) and the left ventrolateral prefrontal cortex (VLPFC) (Dennis et al., 2007).

Conversely, alternate accounts, such as the de-differentiation hypothesis, propose that over-recruitment reflects neural deterioration with aging (Li, Brehmer, Shing, Werkle-Bergner, & Lindenberger, 2006). According to this hypothesis, functional brain activity becomes less specialized and, hence, more generalized with age (Goh, 2011). This hypothesis posits that older adults tend to show more similar patterns of neural activity across different tasks; whereas younger adults show a more diverse activation pattern specific to the task they are completing.

Another potential explanation for over-recruitment is the partial compensation hypothesis (De Chastelaine, Wang, Minton, Muftuler, & Rugg, 2011). According to this hypothesis, the fact that over-recruitment is sometimes observed in poorer performing older adults does not imply deterioration and does not provide evidence against the occurrence of compensation. Instead, this hypothesis posits that compensation still occurs in these poorer performers but not sufficiently to bring their

performance up to the standard of higher performers (Duverne et al., 2009). Disparity exists between theories pertaining to prefrontal over-recruitment exhibited by older adults in episodic memory tasks. Clarification is needed to determine whether over-recruitment is beneficial to older adults, symptomatic of deterioration, or reflects partial compensation.

This review seeks to synthesize findings regarding different theories of prefrontal over-recruitment. Briefly, findings that would support these theories include the following. The compensatory hypothesis would be supported by studies showing a correlation between over-recruitment and successful task performance in older adults, which should not be present in younger adults. Evidence for de-differentiation could be taken from studies in which older adults show bilateral prefrontal activity, which is similar across different types of task and is associated with poorer performance while younger adults should show more differential activation across tasks and higher levels of performance. Evidence for partial compensation would be observed in studies where success effects are still positive in the over-recruited regions but are seen more in poorer performers (Duverne et al., 2009). Studies pertaining to prefrontal over-recruitment in the encoding and retrieval of episodic memory have been selected for this review with the aim of gaining a deeper insight into this activity pattern. This review will group previous theories published in the neuroscience of memory and aging by the following themes: over-recruitment as compensatory, over-recruitment as de-differentiation, over-recruitment as partial compensation and the link between de-differentiation and compensatory theories.

The compensatory hypothesis

There is evidence that over-recruitment functions as compensation for declines in processing and structural integrity (Park & Reuter-Lorenz, 2009). It is argued that over-recruitment represents an adaptive response to cognitive decline and numerous encoding and retrieval studies concur with this.

Encoding

An encoding study using functional magnetic resonance imaging (fMRI) to examine the neural correlates of true and false memory encoding provides evidence for the compensatory hypothesis was conducted (Dennis, Kim, & Cabeza, 2007). Older adults showed decreased activity in the MTL and left VLPFC, but increased activity in the right VLPFC with subsequent true memories. Given that increased activity correlated with more successful performance in older adults suggests that older adults may have compensated for activity decreases in the MTL and left VLPFC by exhibiting extra activity in the right VLPFC.

Moreover, increased activity in the right VLPFC in older adults was found during a deep vs shallow encoding task (Rosen et al., 2002). Higher performing older adults showed this increased activity pattern while younger adults and poorer performing older adults did not. This concurs with the Dennis et al. (2007) study in which researchers argue that this pattern may be compensatory.

Another study yielded concurrent results, which involved a subsequent memory paradigm along with a scene encoding task, in which both the MTL and frontal regions were engaged (Gutchess et al., 2005). The DLPFC was engaged more by older adults overall, and older adults that exhibited less activity in the MTL were more likely to engage the DLPFC. This suggests prefrontal regions could serve a compensatory function for declines in MTL activity with age. Yet, this study is limited in that age differences in correlations were not directly examined; therefore the way these processes vary between age groups is not entirely clear.

Retrieval

Findings pertaining to the compensatory hypothesis have also been observed in retrieval studies. For example, positron emission tomography (PET) was used to measure PFC activity in relation to age and performance during recall and source memory tasks (Cabeza, Anderson, Locantore, & McIntosh, 2002). Researchers found that younger adults and lower performing older adults exhibited right PFC activity for source memory. However, higher performing adults showed more bilateral frontal activation. It was argued that poorer performing older adults engaged the same areas as young adults but with less efficiency, whereas higher performing older adults compensated with bilateral activity to make up for deficits in the region which would typically be activated, allowing them to preserve performance.

Evidence for the compensatory hypothesis was also found using transcranial magnetic stimulation (TMS). TMS is a useful technique that allows researchers to measure causal relationships between performance and regional activity. This

is not often possible based on findings from neuroimaging studies, which are often correlational (Rossi et al., 2004). Rossi et al. (2004) applied TMS to the right and left sides of the DLPFC to compare effects on recognition memory in older and younger subjects. They found that in younger subjects, TMS of the right DLPFC interfered more with retrieval than stimulation of the left DLPFC did. In older adults, the interference was more bilateral for recognition memory. This bilateral pattern may reflect compensation for deficits in function.

Concurrent findings emerged from a study in which fMRI was used to examine episodic retrieval. Researchers found evidence of reduced occipital activity and increased frontal activity in older adults (Davis et al., 2008). Furthermore, age-related increases in frontal activity were positively correlated with task performance and negatively correlated with age-related decreases in occipital activity. This finding, along with the others presented alongside it, provides evidence in support of the compensatory hypothesis as an explanation for prefrontal over-recruitment in older adults.

Alternative accounts

One issue with many studies that appear to provide evidence for the compensatory hypothesis is that they tend to be correlational. Simply because there is a correlation between brain activity and task performance does not mean that a causal relationship can be inferred. It could be the case that these 'over-recruited' regions, rather than providing compensation, are merely engaged alongside other regions that are aiding performance.

Persson et al. (2006) found evidence from cross-sectional studies suggesting frontal over-recruitment was present in older adults. However, they also found evidence from longitudinal studies for under-recruitment with advancing age. It is unclear whether over-recruitment or under-recruitment is more influential on episodic memory with age. Given that most studies within this area are cross-sectional, using longitudinal studies to observe regional brain change within individuals throughout the life span would be an important step in adding more validity to current findings or refuting them.

De-differentiation of function

Another argument in opposition to the compensatory hypothesis is that additionally recruited areas might not aid performance, but instead, this activation pattern could imply de-differentiation of function. This theory posits that extra activity observed in areas of the PFC represents less specialized neural activity that occurs with age-related decline (Li et al., 2006). Perhaps, as argued by Rajah and D'esposito (2005), older adults tend to activate the PFC across tasks in a less specific manner than younger individuals.

This is supported by a study that used fMRI to examine encoding in self-initiated (intentional) encoding and semantic elaboration conditions with age (Logan, Sanders, Snyder, Morris, & Buckner, 2002). Researchers found that older adults showed bilateral over-recruitment with the use of semantic elaboration in the deep encoding task. Over-recruitment was non-selective and was associated with poorer performance, which was argued to reflect de-differentiation and cognitive decline with age.

Further support for the de-differentiation hypothesis was found in a study where researchers analyzed episodic encoding data by determining which activity patterns in the PFC best predicted subsequent performance success (Morcom & Friston, 2012). Results indicated that young adults exhibited more left-lateralization while older adults showed more bi-lateral activity (e.g., Cabeza, 2002). Morcom and Friston (2012) also found support for less regional specificity with aging as they found a wider distribution of activity in older adults, which was linked to poorer memory performance. These results, along with the others discussed in this section, could be interpreted as supporting the de-differentiation hypothesis as an account of over-recruitment.

Partial compensation

Conversely, other researchers have argued that over-recruitment in the presence of poorer performance in older adults does not necessarily indicate de-differentiation of function and neural decline. Instead, such results could be interpreted as indicating partial compensation, where compensation occurs but not sufficiently to bring performance up to the level of higher performers. A study conducted by Duverne et al. (2009) examined animacy decisions on words that were visually presented. A pattern of bilateral frontal activity was observed in older adults, which concurs with previous findings. However, this activation pattern was only present in poorer performing adults, which contrasts with previous findings where over-recruitment has been associated with improved performance (e.g., Dennis et al., 2007). Initially, these findings appear to

provide evidence against the compensatory hypothesis given that older adults that performed well did not exhibit significant activity in the right PFC. However, this does not necessarily imply that right prefrontal over-recruitment is not adaptive (Duverne et al., 2009). Compensation for age-related decline could still occur in these poorer performing older adults but not enough to enable those exhibiting it to perform at the same level as higher performers.

Further support was found in a study conducted by de Chastelaine et al. (2011), in which fMRI was used to examine the neural correlates of encoding. Researchers expected that right callosal integrity would be correlated negatively with right prefrontal over-recruitment and with performance. However, older adults exhibiting subsequent memory effects in the right PFC also had preserved anterior callosal integrity but showed poorer performance. This over-recruitment may reflect the use of processes that are only able to partially compensate for neural decline of the left PFC. The attempted compensation does little to make up for deficits in the left PFC because this area is crucial for episodic encoding of verbal information (de Chastelaine et al., 2011). A limitation in this study is that the brain–behavior effects were not compared directly with younger adults hence it is unclear if this is an age specific finding. As suggested by de Chastelaine et al. (2011), further study involving TMS would be particularly useful to determine the consequences of interfering with right prefrontal activation in encoding tasks.

Co-occurrence of de-differentiation and compensation

A final consideration is the idea that although the de-differentiation hypothesis and the compensatory hypothesis are distinct ideas, perhaps they co-occur within the PFC. Support for examining these hypotheses comes from a PET study of encoding and retrieval in which researchers compared cerebral blood flow in older and younger adults as they completed encoding and retrieval tasks (Cabeza et al., 1997). Older adults showed greater bilaterality during the retrieval task and a reduced difference in cerebral blood flow between recall and recognition tasks compared with younger subjects. Decreased specificity of neural activity with age may signify less efficient processing. These findings also imply that compensation occurs with age, which results in increased bi-laterality to counteract the observed decline in processing efficiency.

Results from a study of individuals with Alzheimer's disease (AD), which is marked by impairments in episodic memory among other impairments, further illustrate this possibility (Grady et al., 2003). Researchers found that those with AD showed increased activity in the PFC relative to controls, which, on the surface, appears to suggest over-recruitment is linked to decline of cognitive function. Yet when comparing individuals within the group of AD suffers itself, over-recruitment in the network observed in individuals with AD was associated with better performance on episodic memory tasks. This is an example of the co-occurrence of degeneration and potential compensatory processes.

Examining the co-occurrence of these hypotheses may account for the very mixed findings within the literature. This concurs with findings from the review article by Rajah and D'Esposito (2005), which argues that de-differentiation of function and functional compensation co-occur in different regions of the PFC. Further studies must be conducted in this area to determine whether or not these processes occur in conjunction.

Discussion

Many studies discussed in the present review provide evidence for prefrontal over-recruitment in older adults, yet there is a lack of convergence on how to interpret these findings. Results from numerous studies reviewed support the compensatory hypothesis with findings showing a correlation between over-recruitment and task success in older adults (e.g., Dennis et al., 2007; Gutchess, et al., 2005). However, based on other findings examined in this review (e.g., Morcom & Friston, 2012), results are too mixed to provide full support for the compensatory hypothesis. As argued by Rajah and D'Esposito (2005), the compensatory hypothesis alone cannot account for the complex age-related changes in neural activity. Other studies emphasized de-differentiation of function as an explanation for over-recruitment, with some finding associations between over-recruitment and poorer task performance (Logan et al., 2002; Morcom & Friston, 2012).

An alternative interpretation of the association between over-recruitment and poor task performance is the partial compensation hypothesis (De Chastelaine et al., 2012). Perhaps older adults are compensating for their deficits in an adaptive way but the compensation is simply not sufficient to bring their performance up to the level of their higher performing older counterparts or younger adults. Yet, neither of these hypotheses

explains the numerous positive correlations found between over-recruitment and task performance in other studies.

A key difficulty in determining the cause of over-recruitment is that this pattern can be differentially interpreted by researchers as either compensatory or reflecting de-differentiation of function. This becomes particularly apparent in findings where over-recruitment is associated with poorer subsequent performance. This pattern would be expected as a result of either partial-compensation or de-differentiation of function, and could thus be interpreted as supporting either of these hypotheses. Perhaps, as observed by Duverne et al. (2009), it might not be possible to distinguish between these theories with correlational methods alone. In light of this, the use of TMS will be crucial in future studies in order to directly measure which brain regions contribute to task performance. If compensation is occurring in areas of the PFC, then when TMS is applied to such regions, subjects that show a pattern of over-recruitment in the targeted area should show more impairment than those that do not. Hence, the use of TMS could help researchers to tease apart these conflicting hypotheses regarding over-recruitment. Moreover, further longitudinal studies regarding regional changes in brain activity within the individuals throughout their lifespans would provide an alternative to cross-sectional designs, as findings differed in Persson et al. (2006). Another possibility explored was that mixed findings on over-recruitment could lead to the conclusion that de-differentiation and compensation co-occur in different regions of the PFC (Rajah & D'Esposito, 2005). Given that there is evidence to support both these theories, perhaps it would be helpful to consider the possibility that they play a joint role in over-recruitment.

To summarize, the compensatory hypothesis may not entirely account for the pattern of over-recruitment observed in the PFC in older adults, although further research must be conducted in this domain before drawing conclusions. Results explored in this review lent more support to the partial compensation hypothesis, although neither the partial compensation nor de-differentiation of function could be ruled out as potential explanations of over-recruitment. The present study emphasises the importance of further study, particularly longitudinal studies and studies involving TMS which would allow a deeper understanding of the way de-differentiation and the partial compensation hypothesis relate to over-recruitment. In particular, TMS could be used to explore the cause of over-recruitment in a way that cannot be determined using the correlational methods, which have been employed in many previous studies. It is important to determine whether processes such as compensation and de-differentiation of function co-occur within the PFC or whether they can be teased apart. Gaining insight into the cause of prefrontal over-recruitment could improve our understanding of both healthy mental aging and decline in neural function. If over-recruitment was found to represent a form of neural compensation that benefited performance, this could alter perceptions of neural aging as a linear process marked largely by decline and deterioration. Additionally, if over-recruitment was found to represent compensation, it would be useful to examine why some people are able to compensate for neural deficits while others are not. This information could prove particularly beneficial for older adults who exhibit episodic memory impairments. For example, further research into compensation could benefit those suffering from AD, where episodic memory decline is one of the earliest and most prominent symptoms (Gold, 2009).

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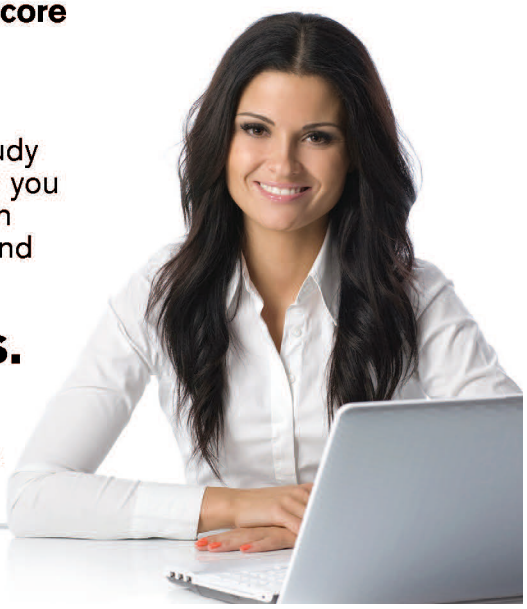

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