The Effects of Biological and Psychosocial Explanations for Depression on Prognostic Pessimism and Preferred Treatment

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Master's Thesis in Psychology
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Åbo Akademi University, 2021

ÅBO AKADEMI UNIVERSITY – FACULTY OF ARTS, PSYCHOLOGY AND THEOLOGY

Subject: Psychology	
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Abstract:
Prognostic pessimism is the belief that mental health problems are untreatable and relatively permanent. Earlier studies suggest that biological explanations for depression, compared to psychosocial explanations, increase prognostic pessimism. Studies also suggest biological explanations for depression could lead to a preference of psychotropic drugs as a treatment method. In the current study, we examined if different explanations (biological, psychosocial and biopsychosocial) for depression influenced prognostic pessimism and preferred treatment in a Finnish sample. We investigated this conducting an online vignette-experiment with 122 Finnish, non-depressed adults aged 18 to 40 years. The vignettes were based on the Finnish national guidelines for treating depression. Contradictory to previous studies, we found no statistically significant differences in prognostic pessimism and preferred treatment depending on the explanation for depression. However, we found a statistically significant difference in how credible the participants found the explanations for depression, with the biopsychosocial explanation for depression being rated most credible. Further, it is important to take into consideration that the vignettes were relatively mildly manipulated which also may explain the lack of effect.

Keywords: Prognostic Pessimism, Preferred Treatment, Explanation for Depression

Date: 02.11.2021	**Page count:** 37
Level: Master's thesis	

ÅBO AKADEMI – FAKULTETEN FÖR HUMANIORA, PSYKOLOGI OCH TEOLOGI

Ämne: Psykologi	
Författare: Amanda Salenius	
Arbetets titel: Effekten av biologiska och psykosociala orsaksförklaringar till depression på prognostisk pessimism och föredragen behandling	
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Abstrakt:
Prognostisk pessimism är uppfattningen att psykiska störningar är relativt permanenta och inte går att bota. Tidigare forskning har visat att biologiska orsaksförklaringar till depression framkallar mera prognostisk pessimism än psykosociala orsaksförklaringar. Vidare har forskning visat att biologiska orsaksförklaringar till depression kunde leda till en preferens för psykofarmaka som behandlingsmetod. I den här studien undersöktes om olika orsaksförklaringar (biologiska, psykosociala och biopsykosociala) till depression påverkar prognostisk pessimism och föredragen behandling i ett finskt sampel. Studien utfördes som ett vinjettexperiment online och nätenkäten besvarades av sammanlagt 122 finska, icke-deprimerade vuxna i åldern 18 till 40 år. Vinjetterna var baserade på de finska nationella riktlinjerna för vård av depression. I kontrast till tidigare forskningsfynd fanns det inga statistiskt signifikanta skillnader i prognostisk pessimism och föredragen behandling beroende på orsaksförklaring till depression. Däremot fanns det en statistiskt signifikant skillnad i hur trovärdiga de olika orsaksförklaringar till depression uppfattades av deltagarna. Den biopsykosociala orsaksförklaringen till depression upplevdes mest trovärdig. Utöver skillnader i vinjetternas trovärdighet bör det även beaktas att vinjetterna var relativt svagt manipulerade vilket eventuellt kunde förklara avsaknaden av effekt.

Nyckelord: Prognostisk pessimism, Föredragen behandling, Orsaksförklaring till depression	
Datum: 02.11.2021	**Sidoantal:** 37
Nivå: Pro-gradu avhandling	

Acknowledgements

Turku, November 2021

As a former team player, there is nothing I value more than having a supportive team around me. Luckily, that is exactly what I have had throughout this writing process, and it is finally time to express my gratitude to the people who have supported me during this time. First and foremost, a huge thank you to my supervisors Mira Karrasch and Jan Antfolk. I am profoundly grateful for the guidance and encouragement that I have received throughout this project. I also want to thank my thesis seminar group, led by Mira Karrasch and Matti Laine, for providing me with valuable feedback and suggestions for improvement. An additional sincere thank you to Martin Lagerström and Julia Simonsen who have offered their generous help with statistics related questions. Finally, I want to thank all friends, family and fellow students who in any way have helped me finish this project. Thank you, team.

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Introduction

In recent decades, the biomedical paradigm of mental illness has been prominent across the globe (Lebowitz & Appelbaum, 2019; White, 2013). This prominence has been broadly embraced by the public (Pescosolido et al., 2010; Schomerus et al., 2012). According to the biomedical model, which highlights neurobiology and genetics in the development of psychopathology, mental disorders are seen as brain diseases that require pharmacological treatment (Deacon, 2013). Furthermore, studies have shown that these increasingly biological explanations for mental illness have led people to hold more positive views on psychotropic drugs (Deacon, 2013; Iselin & Addis, 2003; Phelan et al., 2006; Schomerus et al., 2012). It was initially thought that this shift towards a biomedical perspective could reduce stigmatization of people with mental illnesses by decreasing attributed responsibility from the affected person and, thus, also decreasing blame and self-blame (Deacon, 2013; Deacon & Baird, 2009; Haslam, 2011; Kvaale et al., 2013a, 2013b). Research has, however, suggested that the biomedical perspective simultaneously increases prognostic pessimism, that is, the belief that mental health problems cannot be treated and that they are relatively permanent (Deacon & Baird, 2009; Kemp et al., 2014; Kvaale et al., 2013b; Lebowitz et al., 2013; Phelan, 2005; Phelan et al., 2006). This means that an entirely biological explanation for mental illnesses like major depressive disorder (hereafter 'depression'), could be harmful if it, in fact, increases prognostic pessimism. The reason is that increased prognostic pessimism could lead to a factually worse recovery prognosis, by decreasing motivation to seek and receive help and, as a result, becoming more stigmatized due to persistent disease. In light of this, the aim of the current study was to explore if different explanations for depression influence prognostic pessimism and preferred form of treatment.

Explanations for Depression and Prognostic Pessimism

Dar-Nimrod and Heine (2011) and Haslam (2011), among others, argue that genetic essentialism is a potential explanation for why a biological explanation for depression elicits pessimistic beliefs regarding the prognosis. They describe genetic essentialism as the tendency to think that an individual's characteristics and behaviors are a consequence of their genes, which can increase the belief that these characteristics and behaviors are unchangeable. Thus, biological explanations for depression, including information about the genetic components, might insinuate that depression is untreatable and incurable. Furthermore, a more recent study conducted by Berent and Platt (2021) found that people spontaneously essentialize psychiatric conditions that are linked to the brain, even when the biological

explanation presented provided absolutely no information about the genetic components of the illness, showing the strength of the essentialist bias.

There are many reasons as to why this biological explanation for mental illness, and more specifically depression, has become so prominent (e.g., Deacon, 2013; Kvaale et al. 2013b; Lebowitz & Appelbaum, 2019; Leo & Lacasse, 2008; Phelan et al., 2006). One likely reason is the direct-to-consumer marketing of prescription drugs in the United States and New Zealand, through which companies have been able to market antidepressants as a quick fix for a chemical imbalance in the brain. An et al. (2009) found that direct-to-consumer marketing of prescription drugs affects peoples' views of depression and preferred treatment. In addition to this type of marketing legislation, media reporting has also been a factor that has made it possible for the biological perspective to gain ground in the public debate and thereby increase the preference for psychotropic drugs as treatment method, according to Leo and Lacasse (2008). However, a study conducted in Finland (Sundqvist, 2017), where direct-to-consumer marketing of prescription drugs is not allowed, did not find equally strong evidence for subjectively held biological explanations for depression. In fact, psychosocial explanations were endorsed more than biological explanations. The most supported explanation for depression was current and previous life stress, especially problems related to relationships. This indicates a cultural variation in how common different causal beliefs of depression are.

Studies have shown that biological explanations for depression are associated with more prognostic pessimism than psychosocial explanations (Deacon & Baird, 2009; Kvaale et al., 2013b; Lam et al., 2005; Loughman & Haslam, 2018; Phelan, 2005; Phelan et al., 2006). The most recent studies suggest that a more balanced and malleable biopsychosocial explanation (i.e., including both biological and psychosocial aspects) for depression could be a more effective way to decrease prognostic pessimism (Deacon, 2013; Deacon & Baird, 2009; Kemp et al., 2014; Lebowitz et al., 2013; Lebowitz & Appelbaum, 2019; Loughman & Haslam, 2018; Walker & Read, 2002). This research indicates that people’s view of their prognosis of mental illness is influenced by their causal beliefs about etiology. Further, this allows for the possibility that the explanation for depression is more important than, or at least as important as, the depression itself when it comes to prognosis. This applies both to people’s own beliefs about their prognosis and the actual likelihood of recovering. A more malleable biopsychosocial explanation for depression that induces less prognostic pessimism than a biological explanation is also clinically supported by Rutherford et al. (2010), who found evidence of higher expectation for therapeutic improvement in patients leading to greater improvement.

Whereas several studies have investigated the effect of explanation for depression on prognostic pessimism in clinical populations, Deacon and Baird (2009) conducted their study in a non-clinical population consisting of 90 undergraduate students. The study consisted of a thought experiment where the participants were to imagine themselves as feeling depressed and receiving a depression diagnosis from a doctor. They then received, in a counterbalanced order, both a chemical imbalance explanation and a biopsychosocial explanation for their symptoms and were asked to rate their expectancies on the credibility of the different explanations, perceptions of self-stigma, prognosis and treatment. The results showed that the chemical imbalance explanation, compared to the biopsychosocial explanation, led to more prognostic pessimism and to a belief that nonbiological, psychosocial treatment options would be ineffective.

The Current Study

Our aim with the current study was to investigate if different explanations for depression influence prognostic pessimism and preferred form of treatment (hereafter 'preferred treatment'). To our knowledge, this is the first study that investigates this in a Finnish sample. To maintain high ecological validity, we used adapted versions of the actual Finnish national guidelines for treating depression (The Finnish Medical Society Duodecim [Depression: Current Care Guidelines], 2021) in the online experiment. To probe for laypeople's opinion, we did not include depressed individuals who could have known more about the etiology and treatment of depression.

Using a vignette-design, we investigated if different explanations for depression influence prognostic pessimism and we compared three different explanations for depression: 1) biological, 2) psychosocial, and 3) biopsychosocial. We also investigated if these three different explanations for depression influence the preferred treatment. The treatment options were psychotropic drugs (medication), psychotherapy, a combination of both or changes in attitude and lifestyle. The participants reading the biopsychosocial explanation for depression served as a control group. The following hypotheses were tested:

i. Participants reading the biological explanation for depression will report higher prognostic pessimism than participants reading the psychosocial and biopsychosocial explanations for depression.
ii. Participants reading the psychosocial explanation for depression will report lower prognostic pessimism than participants reading the biological and the biopsychosocial explanations for depression.

iii. Participants reading the biological explanation see psychotropic drugs as a more effective treatment method than participants reading the psychosocial explanation.

iv. Participants reading the psychosocial explanation see psychotherapy as a more effective treatment method than participants reading the biological explanation.

Method

Participants

To provide enough statistical power to detect a small effect (f = .03 with 1-β = .08 and α = .05, assuming equal group sizes in all three groups) the total number of participants had to be at least 111. A total of 199 participants took part in our online experiment. We excluded participants (n = 32) who were feeling depressed before the online experiment by directly guiding them to the debriefing section. We also excluded participants not meeting the age criterion (18–40 years; n = 4), participants answering two or more of the four attention-check questions incorrectly (n = 36), and participants meeting both previous exclusion criteria (n = 5). We excluded a total of 77 participants from the online experiment (Figure 1). The completion rate for the study was 61.2%. The final sample consisted of 122 non-depressed Finnish speaking adults aged 18 to 40 years (M = 24.98, SD = 4.48, range = 18–40), of whom 47 were placed in the biological condition (38.5%; age: M = 24.87, SD = 4.73, range = 18–39), 38 in the psychosocial condition (31.2%; age: M = 25.45, SD = 4.42, range = 18–40) and 37 in the biopsychosocial condition (30.3%; age: M = 24.65, SD = 4.31, range = 18–40). There was no significant difference in age between the conditions, p = .729. Table 1 presents the additional demographic variables of the sample (n = 122).

Figure 1

Flowchart of the Sample Selection

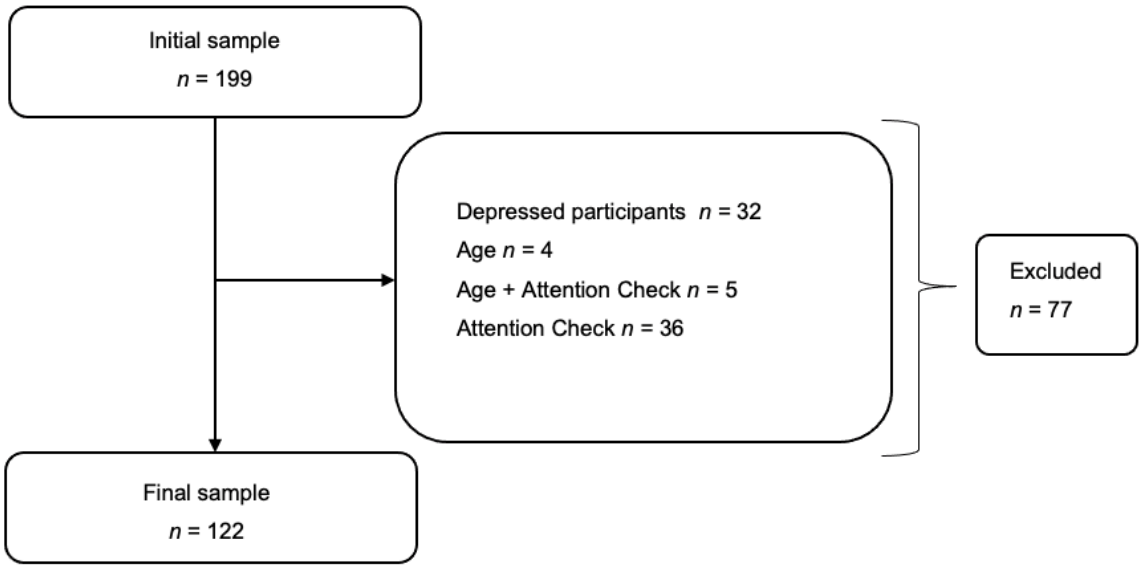


Table 1

Sample Characteristics

Demographic variables	BIO (*n* = 47)		PS.SOC (*n* = 38)		BIO.PS.SOC (*n* = 37)		*p*
	n	*%*	*n*	*%*	*n*	*%*	
Gender							.725
Women	34	72.3	28	73.7	29	78.4	
Men	12	25.5	10	26.3	8	21.6	
Other	1	2.1	0	0.0	0	0.0	
Occupation							.804
Student	33	70.2	21	55.3	24	64.9	
Employed	12	25.5	16	42.1	12	32.4	
Unemployed	0	0.0	1	2.6	0	0.0	
Other	2	4.3	0	0.0	1	2.7	
Socioeconomic status							.605
10 (Top)	0	0.0	1	2.6	1	2.7	
9	8	17.0	7	18.4	8	21.6	

	n	*%*	*n*	*%*	*n*	*%*	*p*
8	16	34.0	15	39.5	4	10.8	
7	13	27.7	5	13.2	6	16.2	
6	3	6.4	4	10.5	15	40.5	
5	3	6.4	4	10.5	1	2.7	
4	4	8.5	1	2.6	2	5.4	
3	0	0.0	1	2.6	0	0.0	
2	0	0.0	0	0.0	0	0.0	
1 (Bottom)	0	0.0	0	0.0	0	0.0	
Psychotropic drugs							**.042**
Yes	0	0.0	0	0.0	0	0.0	
No	40	85.1	37	97.4	36	97.3	
No, but have before	7	14.9	1	2.6	1	2.7	

Note. N = 122. The demographics of the sample presented by condition. Significant values bolded. BIO = The biological condition; PS.SOC = The psychosocial condition; BIO.PS.SOC = The biopsychosocial condition.

Ethical Statement

Prior to data collection, the current study was granted ethical permission by the Institutional Review Board at the Department of Psychology and Logopedics at Åbo Akademi University in March 2021. Participants were told that they were taking part in a study on their beliefs about the prognosis of depression. All participants were 18 years or older and gave informed consent prior to participating in the study.

Procedure

We created our online experiment on a secure online survey platform and pre-registered the study on aspredicted.org. The data collection began on the 6th of April 2021 and ended on the 13th of May 2021. We recruited the participants via social media platforms and online forums. All participants were asked to give their informed consent, and they were informed that participation is voluntary and that they can terminate their participation at any point during the experiment. Additionally, they were told that the results would be reported only at group level.

After the informed consent, the participants answered the question: “Are you feeling depressed at the moment?” with a “yes” or a “no”. The participants who answered “yes” were

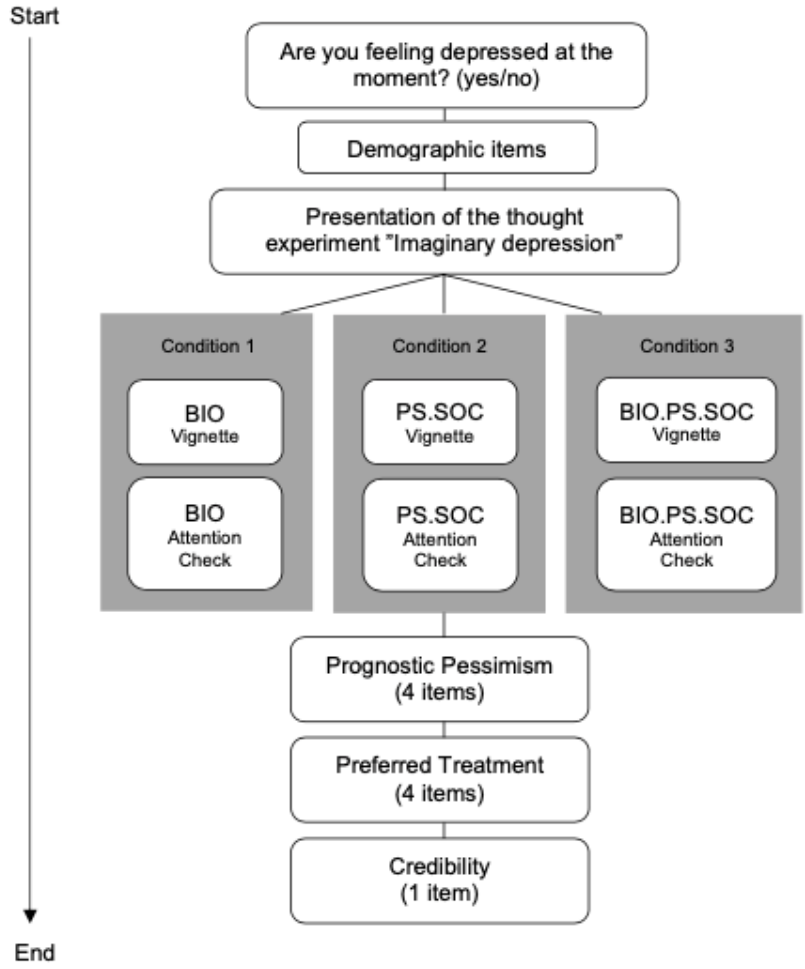
excluded from the online experiment and guided straight to the debriefing section. Participants answering “no” were asked to provide demographic information regarding age, gender, occupation, socioeconomic status, and whether they currently were or had been using any psychotropic prescription drugs, for example antidepressant medication. After this, a short description of the actual thought-experiment of an imaginary depression was presented, which was based on the Deacon and Baird study (2009). The aim of the thought experiment was to induce a sense of what it would feel like to be depressed and receive a diagnosis of depression, which was important for the remaining data collection. After this, the participants were allocated into three conditions based on their reported birth month and were instructed to read different versions of the etiological part of the Finnish national guidelines for depression (Depression: Current Care Guidelines, 2021) and to answer questions about this vignette afterwards. Condition 1 read a biological vignette; Condition 2 read a psychosocial vignette; and Condition 3 (control condition) read the whole biopsychosocial part of the guidelines. In other words, all conditions read “the etiology of depression” part of the Finnish national guidelines for depression, but a part of the description had been left out on purpose in the vignettes for Condition 1 and 2 to highlight either the biological (Condition 1) or the psychosocial (Condition 2) parts of the description.

After the vignette, we conducted an attention check to ensure that the participants had read the vignette carefully. The attention check consisted of four sentences that either corresponded to a sentence in the vignette or not. The participants had to answer two or more of the four questions correctly to be included in the analyses.

We then asked the participants both about how they viewed their own prognosis for recovery from this imaginary depression diagnosis, and about their preferred treatment. This was done using questions about prognosis and preferred treatment based on Deacon and Baird (2009; see Measures). Lastly, the participants were asked to answer an additional question on how similar the vignette they had read was to their own belief of the etiology of depression. After the experiment, we presented a debriefing section. See Figure 2 for the experimental procedure.

Figure 2

Schematic Overview of the Experimental Procedure



Note. BIO = The biological condition, PS.SOC = The psychosocial condition, BIO.PS.SOC = The biopsychosocial condition.

Measures

Prognostic Pessimism

We measured prognostic pessimism using four questions based on questions Deacon and Baird (2009) used in their study. See Table 2. The items were rated on a 5-point Likert scale from zero (*not at all*) to four (*extremely*). Additionally, we created a prognostic pessimism sum score (ProgPesSum) consisting of all four questions ($\alpha = .58$). Two of the four questions (LongtermTreatment and ChronicDepression) were reverse scored when calculating the sum scores.

Preferred Treatment

To measure preferred treatment, we used four questions based on those used by Deacon and Baird (2009). See Table 2. The items were rated on a 5-point Likert scale from

zero (*not at all*) to four (*extremely*). Additionally, we created a preferred treatment sum score (TreatmentSum) consisting of all four questions (α = .50).

Credibility

To measure how similar the vignette the participants read was to their own belief of the etiology of depression, we used a credibility question. See Table 2. The item was rated on an 8-point Likert scale from zero (*not at all*) to seven (*extremely*).

Table 2

Items for Prognostic Pessimism and Preferred Treatment

Question	Name
Prognostic Pessimism	
1. To what extent would you believe you could eventually recover from your depression?	BelieveRecovery
2. To what extent would you feel able to effectively control the depression on your own?	ControlDepression
3. *To what extent would you believe long-term treatment is necessary to overcome your depression?	LongtermTreatment
4. *To what extent would you expect your depression to be a chronic problem that persists for years?	ChronicDepression
Preferred Treatment	
5. How effective would you expect medication to be in treating your depression?	Medication
6. How effective would you expect psychotherapy to be in treating your depression?	Psychotherapy
7. How effective would you expect a combination of psychotherapy and medication to be in treating your depression?	Combination
8. To what extent would you believe that making changes in your attitudes and lifestyle would improve your depression?	AttitudeLifestyle
Credibility	
9. How well did the vignette match your own beliefs of depression?	Credibility

Note. *Items 3 and 4 were reverse scored when calculating the sum scores.

Statistical Analyses

Our statistical analyses were completed using IBM SPSS Statistics 26.0. As a preliminary step, we examined possible relations between the demographic variables and both the prognostic pessimism sum score and the preferred treatment sum score. We examined

gender and occupation using two different independent samples one-way analyses of variance (ANOVAs) and we examined age and socioeconomic status using two different linear regressions. Lastly, we examined the use of psychotropic drugs and since no participant answered "yes" on that variable we did an independent samples *t*-test based on the answers "no" and "no, but have before".

We examined each of the four hypotheses using independent samples one-way ANOVAs. The grouping variable was condition (biological, psychosocial and biopsychosocial). For hypotheses 1 and 2, the outcome variable was prognostic pessimism and for hypotheses 3 and 4 it was preferred treatment. Following Deacon and Baird (2009), we conducted independent samples one-way ANOVAs both for each of these eight items independently and with sum scores of the four items belonging to each measurement (prognostic pessimism and preferred treatment). As we put forth specific hypotheses, the independent samples one-way ANOVAs were followed by pairwise comparisons between all conditions.

Since we found a significant difference between the three conditions regarding the use of psychotropic drugs potentially acting as a confound, we conducted sensitivity tests, excluding participants who had used psychotropic drugs before (n = 9).

We also conducted an independent samples one-way ANOVA for the credibility item, which was statistically significant, and therefore followed by pairwise comparisons between all conditions.

Results

Preliminary Analysis

There were no statistically significant effects between the prognostic pessimism sum score and the demographic variables age, B = -.03, SE = .06, p = .568, socioeconomical status, B = .12, SE = .18, p = .501, gender, $F(14,107) = 1.44$, p = .147, occupation, $F(14,107) = .91$, p = .547 and the use of psychotropic drugs, $t(120) = 1.32$, p = .188. Moreover, there were no statistically significant effects between the preferred treatment sum score and the demographic variables age, B = -.02, SE = .05, p = .646, socioeconomical status, B = .09, SE = .16, p = .554, gender, $F(14,107) = .96$, p = .499, occupation, $F(14,107) = .70$, p = .769 and the use of psychotropic drugs, $t(120) = -.19$, p = .847.

Descriptive Statistics

Table 3 presents mean scores, standard deviations and ranges of the different items and sum scores.

Table 3

Mean, Standard Deviation, and Range for Items and Sum Scores per Condition

Items	Range	BIO		PS.SOC		BIO.PS.SOC	
		M	*SD*	*M*	*SD*	*M*	*SD*
Prognostic Pessimism							
BelieveRecovery	0–4	2.17	1.15	1.92	1.08	2.35	1.39
ControlDepression	0–4	1.53	1.00	1.66	1.17	1.57	1.07
LongtermTreatment	0–4	0.94	0.92	1.00	0.93	1.16	1.21
ChronicDepression	0–4	1.74	1.07	1.82	1.06	2.27	1.12
ProgPesSum	0–16	6.38	2.78	6.39	2.99	7.35	2.98
Preferred Treatment							
Medication	0–4	1.96	0.93	1.74	1.06	1.89	0.99
Psychotherapy	0–4	2.72	0.99	3.00	0.90	3.03	0.83
Combination	0–4	3.06	1.03	2.87	0.91	2.84	1.07
AttitudeLifestyle	0–4	2.83	1.09	2.61	1.20	2.59	1.30
TreatmentSum	0–16	10.57	2.50	10.21	2.67	10.35	2.70
Credibility							
Credibility	0–7	4.57	1.41	4.61	1.39	5.35	1.16

Note. BIO = The biological condition; PS.SOC = The psychosocial condition; BIO.PS.SOC = The biopsychosocial condition.

Hypothesis-testing Analyses

Prognostic Pessimism

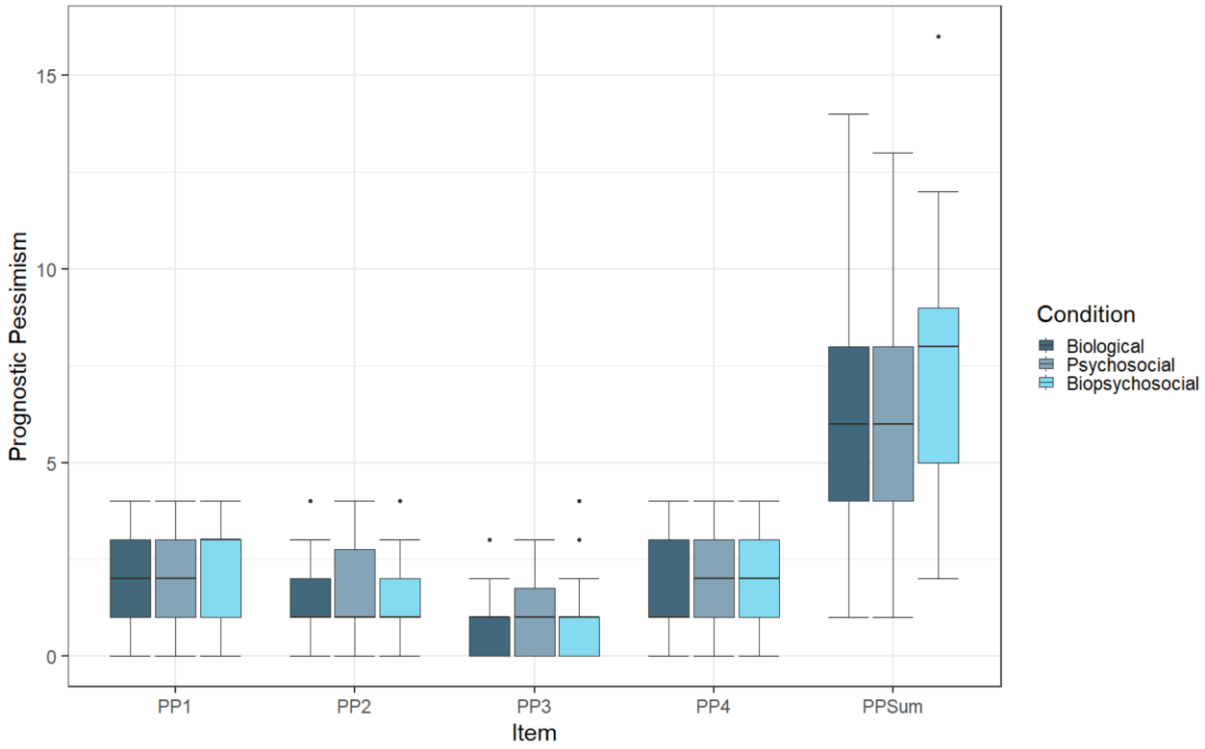
We found no main effect of condition in the independent samples one-way ANOVA with condition as the grouping variable and the sum score of the prognostic pessimism items (ProgPesSum) as outcome variable, $F(2,119) = 1.41$, $p = .247$. Corresponding results for the other four prognostic pessimism items were: BelieveRecovery, $F(2,119) = 1.24$, $p = .292$,

ControlDepression, $F(2,119) = .15$, $p = .862$, LongtermTreatment, $F(2,119) = .52$, $p = .593$ and ChronicDepression, $F(2,119) = 2.72$, $p = .070$. Thus, neither the sum score nor the four items reached significance. As would then be expected, the items did not reach significance in the Bonferroni post-hoc tests.

The sensitivity test controlling for use of psychotropic drugs revealed no significant effects for the five prognostic pessimism items: ProgPesSum, $F(2,110) = .99$, $p = .375$, BelieveRecovery, $F(2,110) = 1.53$, $p = .222$, ControlDepression, $F(2,110) = .28$, $p = .755$, LongtermTreatment, $F(2,110) = .12$, $p = .892$ and ChronicDepression, $F(2,110) = 1.77$, $p = .176$.

Figure 3

The Prognostic Pessimism Items and the Sum Score for the Three Different Conditions



Note. The figure visualizes the individual prognostic pessimism items that makes the sum score and the prognostic pessimism sum score by condition. The range of the prognostic pessimism items are 0–4 and for the prognostic pessimism sum score it is 0–16. The error bars represent values 1.5 interquartile range from the nearest quartile. The dots/outliers represent values 1.5 interquartile range from the upper or the lower quartile. PP1 = BelieveRecovery; PP2 = ControlDepression; PP3 = LongtermTreatment; PP4 = ChronicDepression; PPSum = ProgPesSum.

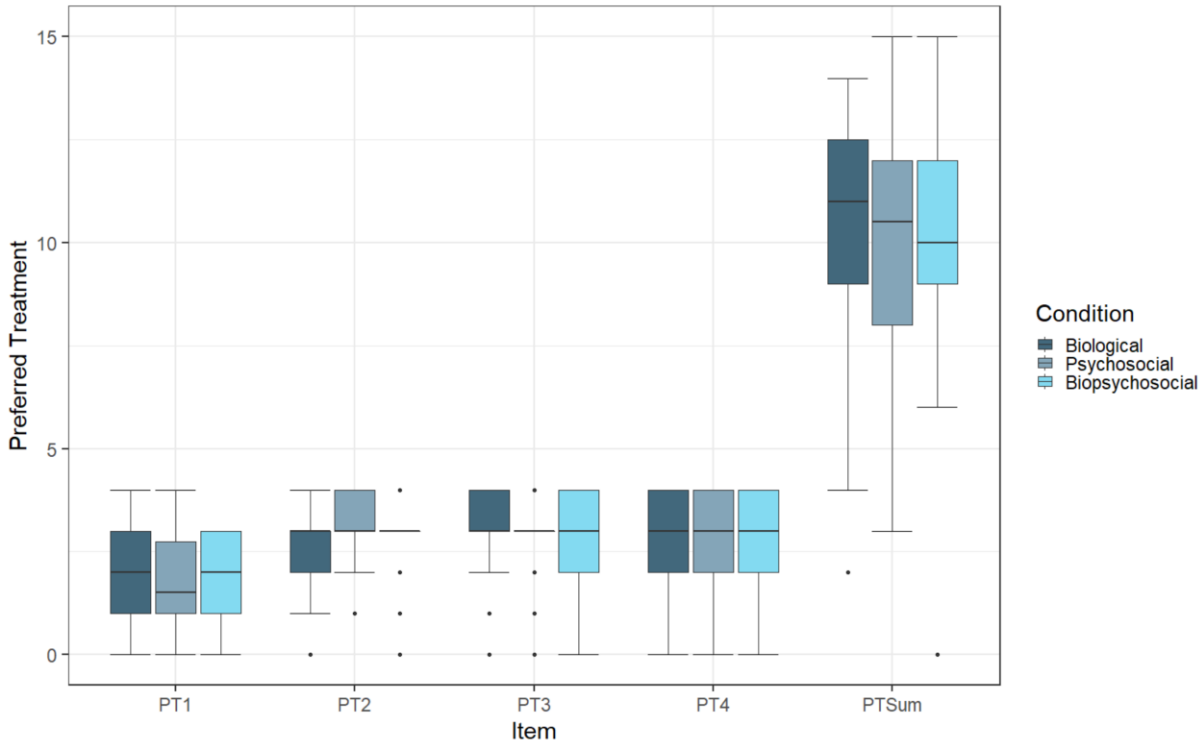
Preferred Treatment

The second independent samples one-way ANOVA, with condition as the grouping variable and the sum score of the preferred treatment items (TreatmentSum) as outcome variable failed to reach significance, $F(2,119) = .21$, $p = .811$. The results for the other four preferred treatment items were: Medication, $F(2,119) = .54$, $p = .587$, Psychotherapy, $F(2,119) = 1.45$, $p = .239$, Combination, $F(2,119) = .54$, $p = .585$ and AttitudeLifestyle, $F(2,119) = .54$, $p = .585$. Again, neither the sum score nor the four items reached significance. As suspected, the items did not reach significance in the Bonferroni post-hoc corrected test.

The sensitivity test controlling for use of psychotropic drugs revealed no significant effects for the five preferred treatment items as following: TreatmentSum, $F(2,110) = .06$, $p = .945$, Medication, $F(2,110) = .21$, $p = .813$, Psychotherapy, $F(2,110) = 1.75$, $p = .179$, Combination, $F(2,110) = .15$, $p = .857$ and AttitudeLifestyle, $F(2,110) = .75$, $p = .474$.

Figure 4

The Preferred Treatment Items and the Sum Score for the Three Different Conditions



Note. The figure visualizes the individual preferred treatment items that makes the sum score and the preferred treatment sum score by condition. The range of the preferred treatment items are 0–4 and for the preferred treatment sum score is 0–16. The error bars represent values 1.5 interquartile range from the nearest quartile. The dots/outliers represent values 1.5

interquartile range from the upper or the lower quartile. PT1 = Medication; PT2 = Psychotherapy; PT3 = Combination; PT4 = AttitudeLifestyle; PTSum = TreatmentSum.

Exploratory Analyses

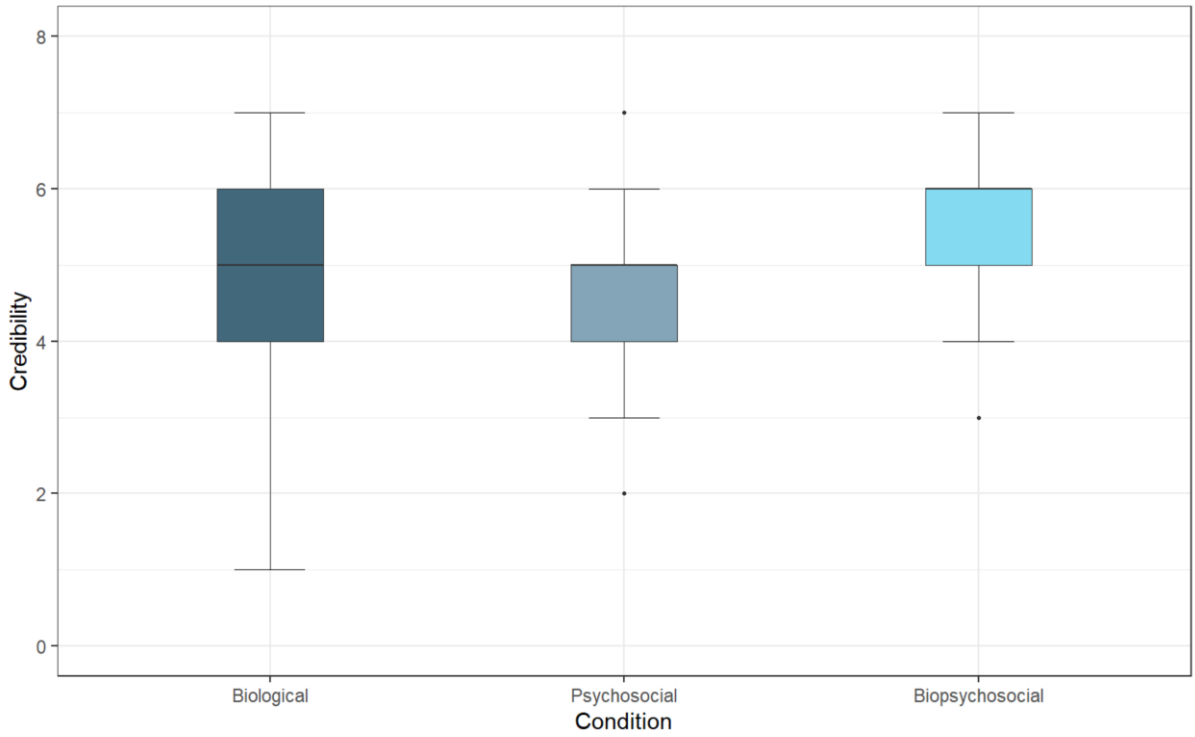
Credibility

In the independent samples one-way ANOVA with condition as the grouping variable and the credibility item as outcome variable, we found a main effect of condition, indicating that there was a significant difference in the responses to the credibility question, $F(2,119)= 4.24$, $p = .017$. The Bonferroni corrected post-hoc test showed that the biopsychosocial condition had significantly higher scores on the credibility question than the biological condition ($p = .027$), indicating that the vignette the participants in the biopsychosocial condition read matched better with their own belief of depression than the vignette the biological condition read.

Consistent with the previously mentioned independent samples one-way ANOVA, the sensitivity test controlling for the use of psychotropic drugs also revealed a significant effect for the credibility item, $F(2,110) = 3.69$, $p = .028$.

Figure 5

The Credibility Item Presented by Condition



Note. The credibility item score presented by condition. Higher scores indicate a more credible view of the vignette the participants read. The range of the credibility item is 0–7. The error bars represent values 1.5 interquartile range from the nearest quartile. The dots/outliers represent values 1.5 interquartile range from the upper or the lower quartile.

Discussion

The aim of the current study was to examine if different explanations for depression influence prognostic pessimism. We further explored if these different explanations for depression influence the preferred form of treatment. Based on earlier studies, mostly conducted in the United States and New Zealand, a biological explanation for depression induces more prognostic pessimism than a psychosocial explanation for depression. Furthermore, studies have shown that biological explanations for depression have led people to hold more positive views on psychotropic drugs. This indicate that people's view of their prognosis for depression is influenced by their causal beliefs about etiology. Further, this allows for the possibility that the explanation for depression is more important than, or at least as important as, the depression itself when it comes to prognosis. To test these previous research findings in a new cultural setting, we investigated this in a Finnish sample using adapted versions of the actual Finnish national guidelines for depression (Depression: Current Care Guidelines, 2021) as explanations for depression. Additionally, we examined how credible the different explanations for depression were experienced by the participants.

Main Findings and Interpretations

We found no statistically significant differences between the different explanations for depression on prognostic pessimism and preferred treatment. However, we found a significant difference between the conditions regarding how credible they found the vignette they read.

Initially, we found no support for our first hypothesis, although the participants in the biological condition did report higher prognostic pessimism compared to the participants in the psychosocial and biopsychosocial conditions. The direction of the non-significant results was thus in line with previous research, according to which a biological explanation for depression increases prognostic pessimism more than a psychosocial explanation (Deacon & Baird, 2009; Kvaale et al., 2013b; Loughman & Haslam, 2018; Phelan, 2005; Phelan et al., 2006).

Contrary to our second hypothesis, the participants in the psychosocial condition did not report lower prognostic pessimism than the participants in the biopsychosocial condition,

but lower than the participants in the biological condition. None of these differences were statistically significant. As mentioned above, the direction of our result of the psychosocial condition reporting lower prognostic pessimism than the biological condition is in line with previous research (Deacon & Baird, 2009; Kvaale et al., 2013b; Loughman & Haslam, 2018; Phelan, 2005; Phelan et al., 2006). Interestingly, the participants in the biopsychosocial condition, that worked as a control condition, reported lower prognostic pessimism than the participants in the psychosocial condition. This non-significant result is, however, not surprising, since there is growing evidence that speaks for the effectiveness of a more malleable biopsychosocial explanation for depression to best decrease prognostic pessimism (Deacon, 2013; Deacon & Baird, 2009; Kemp et al., 2014; Lebowitz et al., 2013; Lebowitz & Appelbaum, 2019; Loughman & Haslam, 2018; Walker & Read, 2002).

From a cultural perspective, potential reasons for the non-significant effects in our study are of interest. One reason for our biological condition not significantly reporting higher prognostic pessimism than the two other conditions could be the relatively weak experimental manipulation of the explanations for depression used in this study. Another reason could be the fact that direct-to-consumer marketing of prescription drugs is not allowed in Finland, while it is allowed for example in the United States and New Zealand (Leo & Lacasse, 2008). The lack of direct-to-consumer marketing in Finland could have limited the extent of the debate about the biomedical perspective on mental illness in Finnish media. Furthermore, this may indicate that there are cultural differences in how prominent and influential the biomedical perspective on mental illness is. Future studies could test these two possible explanations for the lack of an effect in our experiment by investigating if the experimental manipulation was too weak to produce an effect or if the effect is moderated by whether direct-to-consumer marketing of prescription drugs is allowed or not.

In line with our third hypothesis, the participants in the biological condition saw psychotropic drugs as a more effective treatment method than the participants in the psychosocial condition, but the difference was not significant. Again, the direction of our result is in line with previous research that has found an association between biological explanations for depression and the preference for psychotropic drugs as treatment method (Deacon, 2013; Iselin & Addis, 2003; Phelan et al., 2006; Schomerus et al., 2012).

Complementary to our third hypothesis, our fourth hypothesis showed that the participants in the psychosocial condition saw psychotherapy as a more effective choice of treatment than the participants in the biological condition. As with the other hypotheses, the difference between the conditions was not significant, but the direction of the result is in line

with previous research. Iselin and Addis (2003) found that treatment was experienced more helpful when it was congruent with one's causal belief of depression. That indicate, that an individual with a psychosocial causal belief of depression would experience psychotherapy as a more helpful treatment method than, for instance, a biological treatment method like the use of psychotropic drugs.

The prohibition of direct-to-consumer marketing of prescription drugs in Finland could be a potential explanation to why we did not find any statistically significant preference for the use of psychotropic drugs as treatment method in our Finnish sample. As discussed before, this could influence the public discussion about treatment overall but also about the specific use of psychotropic drugs, by not giving that as much space in the public debate as in other cultural settings like in the United States or New Zealand (Leo & Lacasse, 2008).

Exploratory Findings Regarding Credibility

We found a significant difference between the conditions regarding how well the vignette matched the participants' own beliefs of depression. The vignette that the participants in the biopsychosocial condition read matched better with the participants' own beliefs of depression than the vignettes both the participants in the biological and the psychosocial condition read. In the light of our otherwise non-significant results, this is an important finding that warrants discussion. A potential reason for this difference in credibility could lie in our choice to use the Finnish national guidelines for depression (Depression: Current Care Guidelines, 2021) as the explanation for depression in our experiment. Our Finnish sample may have been familiar with these actual guidelines, and therefore also with this biopsychosocial explanation for depression that we used in the biopsychosocial condition, as it was taken straight from the guidelines. Logically then, participants in the biological and the psychosocial conditions found their vignettes less credible, if they were formerly familiar with the biopsychosocial explanation in the Finnish national guidelines for depression (Depression: Current Care Guidelines, 2021).

Clinical Implications

Based on our findings there is no reason to avoid the biological or the psychosocial explanation for depression considering clients' prognostic pessimism and preferred form of treatment. However, it is noteworthy that the biopsychosocial explanation for depression was experienced most credible.

From a clinical point of view, it is also important to acknowledge that the sample of the present study consisted of non-depressed people. Future research conducted in Finland could explore whether the results are generalizable in a clinical sample.

Limitations

There are some limitations in our study that need to be acknowledged. To begin with, the setup of an online experiment is in some ways uncontrollable, despite several practical advantages. It is, for example, difficult to control the level of focus and attention of the participant during the experiment, which could lead to a reduced effect of the experimental manipulation. We took this into account by adding an additional attention check after the vignette, allowing us to exclude participants unlikely to have focused enough when reading the vignette. Furthermore, it was challenging to control how much the participants knew about depression beforehand. Our way of controlling this was to choose a non-depressed sample, by asking the participants whether they felt depressed currently or not, and thereafter exclude the individuals who did feel depressed. An alternative way to take this issue into account would have been to screen participants for depression with for example BDI-II (Beck et al., 1961) or QIDS SR-16 (Rush et al., 2003). These screening questionnaires could, however, have given the participants new information about depression before the actual experiment.

One additionally important aspect to take into consideration is the strength of the manipulation of the explanation for depression. As we wanted as realistic results as possible, we chose to keep the manipulation somewhat weak, by taking the different explanations straight from the actual Finnish national guidelines for depression (Depression: Current Care Guidelines, 2021). In any case we can exclude that an effect exists in the context we studied this in, that is with a relatively weak manipulation of text-based information. Further, it is important to consider the reliability and validity of the measurements used. Our sum scores showed low internal consistency and one potential reason could be the few items per each sum score. Worth mentioning here is also that Deacon and Baird (2009) did not compose a sum score at all for the preferred treatment items, but analyzed them separately, since they differed that much in what they measured.

Lastly, the sample sizes were relatively small in the three conditions, which may lead to possible differences between the conditions not being found. However, we did have statistical power to find an effect if it had been large enough to have played any practical role.

Conclusions

The current study examined if different explanations for depression influence prognostic pessimism and preferred treatment in a Finnish sample. Contrary to earlier research findings, we found no statistically significant differences between biological, psychosocial and biopsychosocial explanations for depression on prognostic pessimism and preferred treatment. However, we found a statistically significant difference in how credible the participants found the explanations for depression, with the biopsychosocial explanation for depression being rated most credible. Future research could examine if a stronger experimental manipulation could explain this lack of effect or if the effect is moderated by whether direct-to-consumer marketing of prescription drugs is allowed or not. Furthermore, it could be of clinical importance to study this in a clinical setting.

Summary in Swedish – Svensk sammanfattning

Effekterna av biologiska och psykosociala orsaksförklaringar till depression på prognostisk pessimism och föredragen behandling

Introduktion

Det biomedicinska paradigmet kring mental ohälsa har under de senaste decennierna blivit allt mera framträdande globalt (Lebowitz & Appelbaum, 2019; White, 2013). Den biomedicinska modellen betonar neurobiologi och genetik inom psykopatologi och ser psykiska störningar som hjärnsjukdomar (eng. *brain disorders*) som kräver psykofarmakologisk behandling (Deacon, 2013). Grundidén med främjandet av det biomedicinska perspektivet var ursprungligen att minska stigmatisering av individer med mentala störningar bland annat genom att minska den drabbades ansvar och därigenom minska både skuld och självklander (eng. *self-blame*; Deacon, 2013; Deacon & Baird, 2009; Haslam, 2011; Kvaale m.fl., 2013a, 2013b).

Parallellt med att främjandet av det biomedicinska perspektivet på mental ohälsa hittills haft för avsikt att minska stigmatiseringen av de drabbade verkar det emellertid även öka mängden prognostisk pessimism. Prognostisk pessimism är uppfattningen att psykiska störningar är relativt permanenta och inte går att bota (Deacon & Baird, 2009; Kemp m.fl., 2014; Kvaale m.fl., 2013b; Lebowitz m.fl., 2013; Phelan, 2005; Phelan m.fl., 2006). Tidigare forskning visar exempelvis att psykosociala orsaksförklaringar till depression framkallar mindre prognostisk pessimism än biomedicinska orsaksförklaringar (hädanefter ”biologiska förklaringar”; Deacon & Baird, 2009; Kvaale m.fl., 2013b; Lam m.fl., 2005; Loughman & Haslam, 2018; Phelan, 2005; Phelan m.fl., 2006) och att det finns en effekt mellan biologiska orsaksförklaringar till depression och en mera positiv syn på psykofarmaka som behandlingsalternativ (Deacon, 2013; Iselin & Addis, 2003; Phelan m.fl., 2006). Många tidigare studier är dock gjorda i USA och i Nya Zeeland där fri marknadsföring av receptbelagda läkemedel till allmänheten är lagligt, det vill säga även marknadsföringen av psykofarmaka**.** An med kolleger (2009) fann i sin studie att denna typ av marknadsföring av receptbelagda läkemedel påverkar individers syn på depression och föredragen behandling. Vidare har medierapporteringen i tillägg till lagstiftningen varit en faktor som möjliggjort att det biomedicinska perspektivet vunnit mark i den allmänna samhällsdebatten samt ökat

preferensen för psykofarmaka som behandlingsmetod i just länder som USA och Nya Zeeland, enligt Leo och Lacasse (2008).

Baserat på premissen att individers syn på sin egen depressionsprognos påverkas av vad de tror att orsaken till insjuknandet är kan man argumentera att orsaksförklaringen är viktigare, eller åtminstone lika viktig, som själva depressionen. Detta är av betydelse både för individers egen syn på sin prognos och för den statistiska sannolikheten att återhämta sig från en depression. Med andra ord kan en ensidigt biologisk orsaksförklaring till depression vara skadlig om den ökar mängden prognostisk pessimism hos individer. Konsekvenser av detta kunde följaktligen vara att individers faktiska återhämtningsprognos blir sämre, att de drabbade blir mindre motiverade att söka och få hjälp samt att de fortsättningsvis blir stigmatiserade av andra i samhället på grund av ihållande sjukdom. I nyare forskning föreslås att det bästa sättet att minska prognostisk pessimism skulle vara mera balanserade och formbara biopsykosociala orsaksförklaringar (Deacon & Baird, 2009; Lebowitz m.fl., 2013; Walker & Read, 2002).

Syftet med den här studien var att undersöka hur olika orsaksförklaringar till depression påverkar prognostisk pessimism och föredragen behandling (eng. *preferred form of treatment*) i ett finskt sampel och med de finska nationella riktlinjerna för vård av depression (The Finnish Medical Society Duodecim [Depression: Current Care Guidelines], 2021) som bas för experimentet. Baserat på tidigare forskningsresultat undersöktes följande hypoteser:

i. Deltagare som läser den biologiska orsaksförklaringen till depression kommer att rapportera mera prognostisk pessimism än deltagare som läser den psykosociala eller den biopsykosociala orsaksförklaringen till depression.
ii. Deltagare som läser den psykosociala orsaksförklaringen till depression kommer att rapportera mindre prognostisk pessimism än deltagare som läser den biologiska eller den biopsykosociala orsaksförklaringen till depression.
iii. Deltagare som läser den biologiska orsaksförklaringen till depression ser psykofarmaka som en mera effektiv behandlingsmetod än deltagare som läser den psykosociala orsaksförklaringen till depression.
iv. Deltagare som läser den psykosociala förklaringen till depression ser psykoterapi som en mera effektiv behandlingsmetod än deltagare som läser den biologiska förklaringen till depression.

Metod

Studiens data samlades in via en nätenkät som riktade sig till finska individer i åldern 18 till 40 år. Sammanlagt 199 individer svarade på nätenkäten. De deltagare som upplevde att de för tillfället var deprimerade exkluderades ur studien. Övriga exklusionskriterier var en ålder utanför åldersspannet samt flera än två fel av fyra frågor på en uppmärksamhetskontroll i mitten av enkäten. Det slutgiltiga antalet deltagare var 122.

I första delen av nätenkäten ombads deltagarna svara på ifall de för tillfället känner sig deprimerade och de som svarade nej på frågan fick sedan fortsätta med att fylla i demografisk information som ålder, kön, huvudsyssla, socioekonomisk status samt huruvida de äter eller tidigare har ätit antidepressiv medicin. De deltagare som svarade att de upplever sig deprimerade blev hänvisade direkt till slutet av enkäten. Därefter presenterades själva tankeexperimentet, baserat på Deacon och Bairds (2009) tankeexperiment, där deltagarna ombads att föreställa sig att de är deprimerade och får en depressionsdiagnos. Idén med själva tankeexperimentet var att framkalla en känsla av hur det är att vara deprimerad och få en depressionsdiagnos hos deltagarna, vilket var av betydelse för den resterande delen av experimentet. Deltagarna delades därefter in i tre grupper baserat på deras födelsemånad och fick läsa olika uppskruvade versioner (hädanefter ”vinjetter”) av den etiologiska delen av de finska nationella riktlinjerna för vård av depression (Depression: Current Care Guidelines, 2021). Med andra ord läste alla grupper samma riktlinjer men i två av de tre grupperna hade delar av riktlinjerna utelämnats för att skruva upp den experimentella manipulationen. En grupp läste en biologisk vinjett, en grupp en psykosocial vinjett och sista gruppen, kontrollgruppen, läste en biopsykosocial vinjett. I denna sammanfattning refereras grupperna hädanefter till som den biologiska gruppen, den psykosociala gruppen och den biopsykosociala gruppen. Efter tankeexperimentet följde en uppmärksamhetskontroll med fyra frågor om vinjetten för att kontrollera att deltagarna läst den tillräckligt noggrant. För att inkluderas i studien måste deltagarna svara korrekt på två eller flera av kontrollfrågorna. Efter dessa kontrollfrågor fick deltagarna svara på frågor om sin egen prognos och på frågor om vilken sorts behandling de föredrar. Slutligen, före en kort återgivningssektion, fick deltagarna svara på hur väl vinjetten de läst stämde överens med deras egen tanke om orsaksförklaringar till depression.

För att samla in data om hur olika orsaksförklaringar till depression påverkar prognostisk pessimism och föredragen behandling användes åtta frågor baserade på ett

självskattningsformulär som Deacon och Baird (2009) använt sig av i sin studie. Både prognostisk pessimism och föredragen behandling mättes med fyra frågor var. Därtill utformades varsin summavariabel för prognostisk pessimism och föredragen behandling inkluderande sina respektive fyra frågor. Slutligen användes en egenformulerad fråga för att samla in data om hur trovärdig deltagarna upplevde att vinjetten de läste var jämfört med deras egen tanke om orsaksförklaringar till depression.

Studiens hypoteser undersöktes med oberoende envägs-variansanalyser. Grupptillhörighet fungerade som oberoende variabel. För hypotes ett och två var den beroende variabeln prognostisk pessimism och för hypotes tre och fyra var den föredragen behandling. I trovärdighetsfrågan fungerade grupptillhörighet som oberoende variabel och trovärdighet som beroende variabel. Studiens analyser utfördes i IBM SPSS Statistics 26.0.

Resultat

Resultaten visar att den biologiska gruppen upplevde mera prognostisk pessimism än både den psykosociala och den biopsykosociala gruppen, men skillnaden var icke-signifikant, *ProgPesSum*, $F(2,119) = 1.41$, $p = .247$. Den biopsykosociala gruppen upplevde minst prognostisk pessimism. Vidare visar resultaten om föredragen behandling att den biologiska gruppen upplevde användningen av antidepressiva läkemedel som en mera effektiv behandlingsmetod än både den psykosociala och den biopsykosociala gruppen, men skillnaden var icke-signifikant, *Medication*, $F(2,119) = .54$, $p = .587$. Den psykosociala gruppen upplevde psykoterapi som behandlingsmetod som mera effektiv än den biologiska gruppen men inte mera effektiv än den biopsykosociala gruppen. Även här var resultatet icke-signifikant, *Psychotherapy*, $F(2,119) = 1.45$, $p = .239$. En signifikant skillnad hittades dock mellan grupperna om hur trovärdig deltagarna ansåg vinjetterna vara jämfört med deras egen syn på orsaksförklaringar till depression*, Credibility,* $F(2,119) = 4.24$, $p = .017$. Bonferroni post hoc-testet visade att den biopsykosociala gruppen upplevde sin vinjett signifikant mera trovärdig än hur den biologiska gruppen upplevde sin vinjett ($p = .027$). Resultaten av analyserna sammanfattas i tabell 1 i form av medeltal, standardavvikelser samt intervall för de olika enkätfrågorna och summavariablerna.

Tabell 1

Medelvärde, standardavvikelse och intervall för enkätfrågorna och deras summavariabler baserat på grupptillhörighet

Variabler	Intervall	BIO		PS.SOC		BIO.PS.SOC	
		M	*SD*	*M*	*SD*	*M*	*SD*
Prognostisk pessimism							
BelieveRecovery	0–4	2.17	1.15	1.92	1.08	2.35	1.39
ControlDepression	0–4	1.53	1.00	1.66	1.17	1.57	1.07
LongtermTreatment	0–4	0.94	0.92	1.00	0.93	1.16	1.21
ChronicDepression	0–4	1.74	1.07	1.82	1.06	2.27	1.12
ProgPesSum	0–16	6.38	2.78	6.39	2.99	7.35	2.98
Föredragen behandling							
Medication	0–4	1.96	0.93	1.74	1.06	1.89	0.99
Psychotherapy	0–4	2.72	0.99	3.00	0.90	3.03	0.83
Combination	0–4	3.06	1.03	2.87	0.91	2.84	1.07
AttitudeLifestyle	0–4	2.83	1.09	2.61	1.20	2.59	1.30
TreatmentSum	0–16	10.57	2.50	10.21	2.67	10.35	2.70
Trovärdighet							
Credibility	0–7	4.57	1.41	4.61	1.39	5.35	1.16

Note. BIO = den biologiska gruppen; PS.SOC = den psykosociala gruppen; BIO.PS.SOC = den biopsykosociala gruppen; M = medelvärde; SD = standardavvikelse; BelieveRecovery, ControlDepression, LongtermTreatment, ChronicDepression = de fyra frågorna som mätte prognostisk pessimism; ProgPesSum = summavariabeln för prognostisk pessimism; Medication, Psychotherapy, Combination, AttitudeLifestyle = de fyra frågorna som mätte föredragen behandling; TreatmentSum = summavariabeln för föredragen behandling.

Diskussion

Syftet med studien var att undersöka hur olika orsaksförklaringar till depression påverkar prognostisk pessimism och föredragen behandling i ett finskt sampel. Utöver detta

undersöktes hur trovärdiga de olika orsaksförklaringarna till depression uppfattades av deltagarna. I motsats till studiens för övrigt statistiskt icke-signifikanta resultat fanns det en statistiskt signifikant skillnad i hur trovärdiga orsaksförklaringarna uppfattades vara.

Resultaten visade ingen signifikant skillnad i mängden rapporterad prognostisk pessimism beroende på orsaksförklaring till depression. Men den biologiska gruppen rapporterade mest prognostisk pessimism följt av den psykosociala gruppen och sist den biopsykosociala gruppen. Därtill hittades varken statistiskt signifikant stöd för att den biologiska gruppen skulle se psykofarmaka som en mera effektiv behandlingsmetod än den psykosociala gruppen eller för att den psykosociala gruppen i sin tur skulle se psykoterapi som en mera effektiv behandlingsmetod än den biologiska gruppen. Resultaten i denna studie skiljer sig därmed från tidigare studier då inga av studiens hypoteser kunde bekräftas statistiskt signifikanta. Trots det har de flesta av resultaten i denna studie samma riktning som tidigare studier har påvisat, det vill säga att biologiska orsaksförklaringar till depression ger upphov till mera prognostisk pessimism än psykosociala orsaksförklaringar till depression (Deacon & Baird, 2009; Kvaale m.fl., 2013b; Loughman & Haslam, 2018; Phelan, 2005; Phelan m.fl., 2006) och att en biologisk orsaksförklaring till depression är associerad med en preferens för psykofarmaka som behandlingsmetod (Deacon, 2013; Iselin & Addis, 2003; Phelan m.fl., 2006; Schomerus m.fl., 2012). Den nyssnämnda skillnaden i statistisk signifikans bör tas i beaktande då det gäller kulturella perspektiv och ekologisk validitet, eftersom de flesta av de tidigare studierna kring detta tema har gjorts i USA och Nya Zeeland.

Att det inte fanns statistiskt signifikanta skillnader i prognostisk pessimism eller föredragen behandling beroende på orsaksförklaring till depression i den här studien kan möjligen bero på att det i Finland inte är lagligt att fritt marknadsföra receptbelagda läkemedel (såsom antidepressiva läkemedel), vilket det är exempelvis i USA och Nya Zeeland. Den biomedicinska debatten om psykiska störningar har eventuellt inte kunnat etablera sig lika brett, högljutt och ensidigt biologiskt i det finska samhället då medierna i Finland inte lika fritt får marknadsföra receptbelagda läkemedel. I och med detta har det biomedicinska perspektivet på psykiska störningar eventuellt inte i samma utsträckning kunnat påverka individernas preferenser och åsikter gentemot depression och dess etiologi. Med detta i åtanke kunde framtida forskning undersöka ifall effekten av orsaksförklaringar till depression på prognostisk pessimism och föredragen behandling är modererad av huruvida friare marknadsföring av receptbelagda läkemedel är tillåtet eller inte.

En annan potentiell orsak till de statistiskt icke-signifikanta resultaten i studien kan eventuellt kopplas ihop med att den biopsykosociala gruppen upplevde sin vinjett statistiskt signifikant mera trovärdig än hur de övriga två grupperna upplevde sina vinjetter. Detta resultat kan härröra från valet att använda de finska nationella riktlinjerna för vård av depression (Depression: Current Care Guidelines, 2021) som bas för studiens experiment då den biopsykosociala vinjetten var en exakt kopia av dessa riktlinjers del om etiologin bakom depression. En del av studiens finska sampel var möjligen bekanta med de nationella riktlinjerna i sin helhet redan innan experimentet, vilket kan ha påverkat att den biologiska gruppen och den psykosociala gruppen ansåg deras vinjetter som bristfälliga medan den biopsykosociala gruppen som fick hela den riktiga versionen av riktlinjerna upplevde vinjetten mera trovärdig.

Denna studie hade även vissa begränsningar. Bland annat utfördes experimentet på nätet, vilket är relativt okontrollerbart, trots att det även finns praktiska fördelar med det tillvägagångssättet. Det kan vara svårt att kontrollera att deltagarna inte störts av någon yttre faktor, vilket eventuellt kunde minska effekten av den experimentella manipulationen. Detta försökte minimeras med en extra uppmärksamhetskontroll i mitten av experimentet, för att möjliggöra exkluderandet av deltagare som förmodligen inte fokuserat tillräckligt noga på uppgiften. Därtill var det utmanande att kontrollera hur mycket deltagarna visste om depression från förut. För att minimera risken att deltagarna skulle ha varit djupt insatta i ämnet från förut, och därmed även antagligen bekanta med orsaksförklaringar till depression, valdes ett icke-deprimerat sampel till studien. En ytterligare aspekt som måste beaktas är styrkan i manipulationen av orsaksförklaringarna till depression. För att få så verklighetstrogna resultat som möjligt hölls manipulationen något svag. På detta sätt kan man enbart utesluta att det finns en effekt i denna studies explicita kontext, det vill säga med en relativt svag manipulation av textbaserad information. Framtida forskning kunde undersöka om den här studiens experimentella manipulation var för svag för att ge någon effekt.

Syftet med studien var att undersöka hur olika orsaksförklaringar till depression påverkar prognostisk pessimism och föredragen behandling i ett finskt sampel. Resultaten visar att olika (biologiska, psykosociala och biopsykosociala) orsaksförklaringar till depression inte har en statistiskt signifikant effekt på prognostisk pessimism och föredragen behandling. Nämnvärt är dock att den biopsykosociala orsaksförklaringen till depression upplevdes mest trovärdig. Framtida forskning kunde undersöka ifall studiens något svaga

experimentella manipulation kan förklara denna avsaknad av effekt eller om effekten modereras av huruvida friare marknadsföring av receptbelagda läkemedel är tillåtet eller inte. Därtill kunde det vara av klinisk relevans att genomföra studien med ett kliniskt sampel.

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PRESSMEDDELANDE

Effekterna av biologiska och psykosociala orsaksförklaringar till depression på prognostisk pessimism och föredragen behandling
Pro-gradu avhandling i psykologi
Fakulteten för humaniora, psykologi och teologi, Åbo Akademi

Resultaten från en pro-gradu avhandling i psykologi vid Åbo Akademi tyder på att olika orsaksförklaringar till depression inte signifikant påverkar prognostisk pessimism eller föredragen behandling i ett finskt sampel. Prognostisk pessimism är uppfattningen att psykiska störningar är relativt permanenta och inte går att bota. Dessa resultat är motstridande till tidigare forskningsfynd, främst gjorda i USA och Nya Zeeland, där man funnit att biologiska orsaksförklaringar till depression framkallar mera prognostisk pessimism än psykosociala orsaksförklaringar och att biologiska orsaksförklaringar leder till en preferens för psykofarmaka som behandlingsmetod framom andra alternativ. Syftet med avhandlingen var att undersöka om biologiska, psykosociala och biopsykosociala orsaksförklaringar till depression påverkar prognostisk pessimism och föredragen behandling i ett finskt sampel. Studien utfördes som ett vinjettexperiment online och nätenkäten besvarades av sammanlagt 122 icke-deprimerade vuxna i åldern 18 till 40 år. Vinjetterna var baserade på de finska nationella riktlinjerna för vård av depression för att vara så verklighetsbaserade som möjligt. Resultaten visar att den biopsykosociala orsaksförklaringen till depression upplevdes signifikant mera trovärdig av deltagarna än de andra orsaksförklaringarna. En ytterligare faktor som bör beaktas vid tolkningen av resultaten är att experimentets vinjetter var relativt svagt manipulerade vilket eventuellt kunde förklara avsaknaden av effekt. Avhandlingen är den första som undersöker om olika orsaksförklaringar till depression påverkar prognostisk pessimism och föredragen behandling i ett finskt sampel.

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