

Exploring Alternative Pain Management Methods: Challenges and Concerns in Athletes and
Women during Labor
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Abstract for Masters

Exploring Alternative Pain Management Methods: Challenges and Concerns in Athletes and Women during Labor

Stefania Nudo

This thesis reports the results of a Systematic Review with Meta-Analysis, and a research study. The Systematic Review compared the efficacy of topical versus oral medications to a placebo in athletes. A meta-analysis was conducted calculating Hedges' g and forest plots with 95% CI were created. There was a significant pooled effect size indicating a reduction in pain for the topical medication compared to the placebo, but not for the oral medication compared to the placebo. The conclusion was that topical medication was more effective for pain reduction than oral medication amongst athletes.

The research study aimed to analyze the correlation between the Pain Catastrophizing Scale (PCS), the Labor Pain Relief Attitude Questionnaire for pregnant women (LPRAQ-p), the Edinburgh Postnatal Depression Scale (EPDS), and pain measured on the Visual Analogue Scale (VAS). Women were asked to fill out the PCS, the LPRAQ-p, and the EPDS in the prenatal period, and the EPDS once again in the postpartum period, along with the VAS regarding labor pain and current pain. The hypotheses were that the PCS would be correlated to pain, the LPRAQ-p, and the EPDS, and that the LPRAQ-p and the EPDS would be correlated. The only significant correlations amongst the questionnaires were the prenatal EPDS being correlated to the postpartum VAS, and the prenatal and postpartum EPDS being correlated. A secondary exploratory analysis showed that EPDS scores were higher amongst nulliparous women compared to multiparous women, and that the LPRAQ-p was higher in women who received analgesia.

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CONTRIBUTION OF AUTHORS

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Stefania Nudo wrote chapter 1 and Geoffrey Dover provided the edits.

Chapter 2:

Stefania Nudo, John-Alexander Jimenez-Garcia, and Geoffrey Dover wrote chapter 2. John-Alexander Jimenez-Garcia and Geoffrey Dover also provided edits, and John-Alexander Jimenez-Garcia provided the statistical analysis.

Chapter 3:

Stefania Nudo wrote chapter 3. Geoffrey Dover provided the edits. Both Stefania Nudo and Geoffrey Dover provided the statistical analysis.

Chapter 4:

Stefania Nudo wrote chapter 4.

All authors reviewed the final manuscript and approved of the contents.

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Chapter 1: Introduction

Pain is a major health issue that affects a large portion of the population. It can be influenced by a multitude of factors, including but not limited to psychosocial influences, age, sex, and genetics (Fillingim, 2017). Pain varies in perception and intensity from person to person, resulting in it being a unique experience with no objective measurement (Bushnell et al., 2013; Fillingim, 2017). In Western cultures, pain is traditionally managed with pharmacology (Bushnell et al., 2013). In Canada and the United States, there has been a significant rise in the prescription of opioids for pain management in the last two decades (Degenhardt et al., 2019), with this class of medications having become the most commonly prescribed (Jannetto, 2021). With the continued increase in pharmacological pain management, there has also been a sharp rise in opioid related deaths and opioid addiction (Kolodny et al., 2015). There are adverse effects associated with less addictive pain medication such as Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) as well. Oral NSAIDs act on prostaglandins and can negatively impact the gastric mucosal barrier, renal blood flow, endothelial tone, circulatory system, kidneys, and liver (Derry et al., 2017; Maniar et al., 2018). This makes an important case for an investigation into alternative methods to conventional pain management.

While pain is prevalent in the general population, athletes specifically have their own challenge with pain. Athletes commonly suffer from acute or chronic sport injuries which are associated with pain (Moseid et al., 2018). Athletes often take NSAIDs or paracetamol for pain relief (Harle et al., 2018; Pedersen et al., 2022). Oral medications are more frequently used by athletes, in comparison with topical medications (Harle et al., 2018). Despite the wide use of pain medications in athletes, there are still concerns related to the safety of the medication. For example, paracetamol can cause liver toxicity (Watkins et al., 2006) and NSAIDs are associated with gastrointestinal difficulties, as previously stated. Moreover, there is some evidence that

NSAIDs may be more dangerous in some sports than others. For example, there may be more severe bleeding following trauma which is a concern for athletes in contact sports (Lundberg & Howatson, 2018). Marathon runners who take NSAIDs have a five time greater likelihood of the typical adverse effects with these analgesics, but also cardiovascular events (Küster et al., 2013). Many opioids are banned substances in competition (Vernece et al., 2017). Regardless of that fact, opioid use remains common in professional sports, and even between 25-50% of high school student athletes have reported having taken an opioid for pain (Ekhtiari et al., 2020).

In addition to the concern with medication in athletes, there is another population where pain medication is a concern which are women who are expecting. The most common management of labor pain is medication (Koyyalamudi et al., 2016). Labor pain medications can include regional anesthesia through epidural or spinal injections, or combined spinal-epidural (CSE) analgesia (Schrock & Harraway-Smith, 2012), and intramuscular or intravenous opioids (Smith et al., 2018). Epidural use, including standard epidural and CSE, has steadily been on the rise over the last decades in Canada (Moola, 2018). Bupivacaine, with or without an opioid such as fentanyl or sufentanil, is the commonly used anesthetic with epidural (Nanji & Carvalho, 2020). Combined Spinal Epidural has been shown to provide a more rapid onset of pain relief (Simmons et al., 2012). There are, however, more adverse effects associated with CSE versus traditional epidural injection. Bradycardia and fetal heart-rate changes have been linked to CSE (Hattler et al., 2016). Either type of epidural use has been associated with more instrumental vaginal births and cesarean section births due to fetal distress, as well as urinary retention, low blood pressure, fever, and motor block (Moola, 2018). Opioids provide another type of analgesia during labor, which can be used independently of epidural analgesia or in conjunction with it (Schrock & Harraway-Smith, 2012). There are, nonetheless, side effects involved in the use of opioids during labor. Opioids used during labor can affect both the mother and baby. The mother may experience nausea, vomiting, sedation, pruritus, and respiratory depression (Moola,

2018). The fetus or newborn may experience bradycardia, loss of heart rate variability, sedation, respiratory depression, and difficulties with breastfeeding (Moola, 2018). There is also the possibility that a newborn being born to a woman having received opioid analgesia will require resuscitation including the need for naloxone (Moola, 2018). Neither method is perfect, with problems being reported with both techniques. Epidural users experience more hypotension, fever, urinary retention, assisted vaginal birth, and longer first and second stages of labor compared to opioid users (Anim-Somuah et al., 2018). Conversely, opioid users required more oxygen assistance, nausea and vomiting than epidural users (Anim-Somuah et al., 2018).

The trend towards medication use for pain management above alternative therapies is concerning when considering the number of reported adverse effects and potential for addiction. Pain management is a complex issue that requires a comprehensive approach and should include safer and non-pharmacological interventions. This thesis investigates two groups that require improved alternatives and more information regarding pain management beyond pharmacological options. Chapter 2 is a systematic review comparing the efficacy of oral versus topical analgesic medication compared to a placebo in injured athletes. Despite the widespread use of medication in athletes as described above, few studies have been done that examine the efficacy of pain medication in athletes who are recovering from an injury. Chapter 3 is a research study that compares multiple questionnaires in an effort to make associations between catastrophizing, pain experience, epidural use, and postpartum depression. The objective of this thesis is to evaluate if medication is effective in treating injured athletes compared to a placebo, and to identify pregnant women who may experience greater pain during childbirth, providing better options for these groups.

Chapter 2: Efficacy of Topical versus Oral Analgesic Medication Compared to a Placebo in Injured Athletes: A Systematic Review with Meta-Analysis

2.1 Abstract

Background: Athletes are injured frequently and often take analgesic medication. Moreover, athletes commonly use non-prescription topical and oral medications with little guidance.

Despite wide use, limited studies exist on the efficacy of pain medication in injured athletes compared to a placebo.

Objective: To determine efficacy of topical or oral medications in pain reduction compared to a placebo in injured athletes.

Study Design: A systematic review and meta-analysis

Methods: We conducted an electronic search using Medline/Pubmed, Web of Science, Ovid, and SportDiscus for all literature relating to topical or oral medications in athletes for pain management post-injury. Two reviewers screened the studies and measured their quality. To determine efficacy, we calculated the Hedges' g value.

We created forest plots with 95% CI to graphically summarize the meta-analyses.

Results: There was a significant pooled effect size reflecting a reduction in pain outcomes for the topical treatment versus placebo ($g=-0.64$; 95% CI $[-0.89, -0.39]$; $p < 0.001$). There was not a significant reduction in pain outcomes for the oral treatment versus placebo ($g=-0.26$; 95% CI $[-0.60, 0.17]$; $p = 0.272$).

Conclusion: Topical medications were significantly better at reducing pain compared to oral medications versus a placebo in injured athletes. These results are different when comparing to other studies that used experimentally induced pain versus musculoskeletal injuries. The results from our study suggest that athletes should use topical medications for pain reduction, as it is more effective, and there are less reported adverse effects compared to oral medication.

Key Words (3-8): NSAID, Sport, Drug, Pain, Injury, Muscle, Inflammation, Game

2.2 Introduction

Elite athletes are defined as competing in a sport at a high level for their age category (von Rosen et al., 2018). There is a high prevalence of sports injuries among elite athletes (Moseid et al., 2018). Sport injuries can be acute or chronic and are often associated with pain (Moseid et al., 2018). Athletes need to quickly manage their pain to be able to return to play quickly. Regardless of pain tolerance, most athletes will typically seek pain management, often including but not limited to analgesic medications. Athletes frequently use analgesic medications early on in the management of an injury as they are available over-the-counter (OTC), and do not require a prescription (Feucht & Patel, 2010). One study indicated that 46% of NCAA female athletes and 38% of NCAA male athletes who were experiencing pain were taking non-steroidal anti-inflammatory drugs (NSAIDs) (Christopher et al., 2020). Among them, 70% of female athletes had purchased the NSAIDs themselves, versus 61% of male athletes, suggesting that they might not be part of their medical record or known by the medical staff. Another study reported that 62% of collegiate athletes use non-prescription drugs for pain management (Stache et al., 2014). Many of these athletes do not consult healthcare practitioners prior to self-medicating and are unaware of the potential adverse effects associated with medication use (Feucht & Patel, 2010). They are also not properly informed on which medication can be the best for their injury management. While many athletes will use analgesic medications specifically for sustained injuries, it is also reported that several athletes use them prophylactically before competition (Gorski et al., 2011). There are limited studies done on the use of analgesics drugs in injured athletes for pain management.

Athletes experience pain differently than non-athletes, often showing a higher pain tolerance than non-athletes (Geisler et al., 2020; Tesarz et al., 2012). It is unclear whether athletes would respond to NSAIDs the same way a non-athlete would, considering the

differences in their pain threshold. Previous studies on the efficacy of pain medications in athletes have been completed using various experimentally induced pain models including delayed onset muscle soreness (DOMS). Experimentally induced pain may not have the same inflammation cascade or psychological impact as an actual musculoskeletal injury (Hotfiel et al., 2018; Petersen-Felix & Arendt-Nielsen, 2002). In fact, experimental models inducing pain or DOMS are done to control for psychological factors that are present post-injury (Petersen-Felix & Arendt-Nielsen, 2002). While some suggest the inflammation response is similar in DOMS, the mechanism of DOMS is still unknown (Hotfiel et al., 2018). In addition, the recovery after DOMS is more defined compared to an actual injury (Armstrong, 1984; Rae & Orchard, 2007). During a musculoskeletal injury, the symptomology begins at the time of the mechanism or during continued exercise (Hotfiel et al., 2018). In DOMS, the symptomology begins 6-12 hours post-exercise and increases to reach a maximum pain level at 48-72 hours (Hotfiel et al., 2018). It is thus difficult to say if a medication response to DOMS would be the same as a medication response to a musculoskeletal injury (Hotfiel et al., 2018).

The placebo effect makes evaluating pain treatment challenging (Benedetti, 2008). Studies aiming to show if a drug is effective at reducing pain will often include a placebo group (Benedetti, 2008), and are in some instances required to do so (Skierka & Michels, 2018). Many of these studies are done on non-athletes. Knowing that athletes experience pain differently than non-athletes, one may question whether athletes also feel the placebo effect differently than non-athletes. One study concluded that athletes experience the placebo effect less compared to non-athletes when provoked with painful stimulation (Geisler et al., 2020). It is unclear whether this finding translates to sustained injuries as well. The purpose of this review is to analyze the placebo effect in athletes treating a musculoskeletal injury with topical or oral over the counter medication. This will help athletes as well as the healthcare practitioners working with them to make better-informed decisions about the management of their injury. Furthermore, there are less reported adverse effects with the use of topical medications

compared to oral counterparts, and as such we would want to know if these medications are of equal effectiveness in athletes (Leppert et al., 2018). If they are, then topical medications would be the safer choice.

2.3 Methods

2.3.1 Data Sources and Search Strategy

We used the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) guidelines to conduct and report this study (Moher et al., 2009). We searched four electronic databases: Web of Science, Ovid/Medline, PubMed/NCBI, and SPORTDiscus from their inception to February 2022. Our search strategy was based on a combination of key terms, synonyms, Boolean conjunction, and truncation (Appendix 1). Two reviewers independently screened titles and abstracts of potential articles in the initial search. In case of disagreement, a third reviewer, who is an expert in pain research, helped to decide if a study could be included. Following this, the two reviewers screened full texts for eligibility. We also performed hand-searching of references and times-cited lists of included articles and authors' bibliographies to find relevant articles not identified using the predefined search strategy (Figure 1).

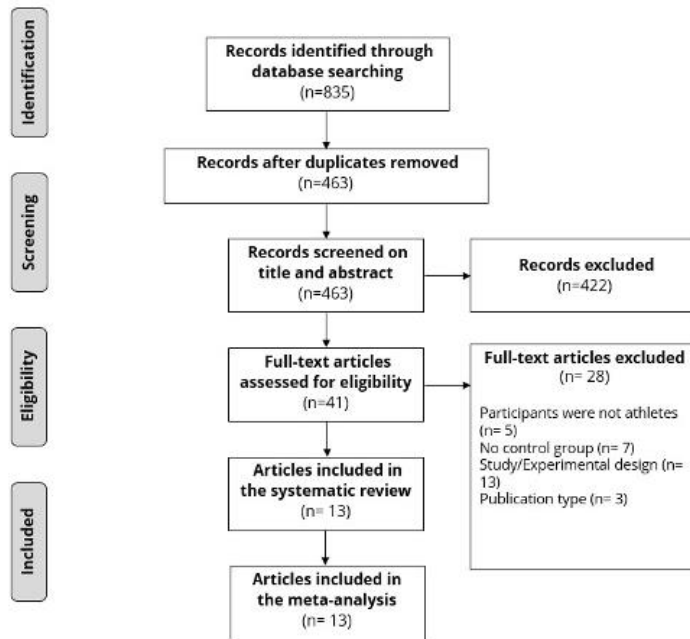


Figure 2-1. PRISMA flow diagram indicating process for identification, screening, eligibility, and inclusion of articles for the meta-analysis.

2.3.2 Inclusion and Exclusion Criteria

We used the PICOS approach (population, intervention, comparison, outcome, study design) to analyze titles and abstracts (Huang et al., 2006). We included studies that met all the following criteria: 1) population: the study included athletes (elite or recreational) who experienced a musculoskeletal injury without limits for age, sex, or level; 2) intervention: the study included an intervention group receiving either a topical or oral OTC medication; 3) control: the study included a control group receiving a placebo; 4) outcomes: the study measured pain and functional improvements; 5) study design: the study was a randomized controlled trial.

Exclusion criteria were: 1) the study included a non-active population; 2) the study was done on animals; 3) the study used natural or alternative analgesics; 4) the study used

analgesic medications for delayed onset muscle soreness (DOMS); 5) the study was done on induced injury or soreness; 6) the study was not available in English.

2.3.3 Data Extraction

Two reviewers extracted the following data from included articles: authors, year, sample size, setting, intervention characteristics, type of medication, placebo, dosage, pain measurement, point estimates, standard deviations (SD), and confidence intervals (CI). If authors only reported outcomes using figures, we used plot digitizer (<http://plotdigitizer.sourceforge.net/>) to extract data. This method was used for Åström & Westlin, 1992, Galer et al., 2000, Giani et al., 1989, May et al., 2007, Predel et al., 2004, Predel et al., 2016, and Wetzel et al., 2002. When authors reported data in multiple time points, we used the values for the “best day” characterized by the biggest difference in the outcomes between groups. We focused on the outcomes reported at rest; however, if authors only reported outcomes measured during activity, then those outcomes were used.

When authors reported standard errors (SE) instead of SD, we calculated the SD using the following formula: $SD=SE*N$. Similarly, when authors reported CI instead of SD or SE, we calculated the SD using the following formula: $SD=N*(upper\ limit-lower\ limit)/3.92$. We computed the intraclass correlation coefficient (ICC) to evaluate the agreement between reviewers in the data extraction process. We used Mendeley to manage the references, Rayyan QCRI to conduct the data screening process, and a spreadsheet to extract the data.

2.3.4 Outcomes

We studied the effects of topical or oral analgesic medications when compared to a placebo in athletes. The independent variable was the type of medication, and the dependent variable was pain improvement measured using different scales including the visual analogue scale (VAS) for pain or 5-point (Likert) function scales.

2.3.5 Risk of Bias Assessment of Individual Studies

We assessed study quality using the Downs and Black checklist (DBC) (Downs & Black, 1998). The DBC measures quality of reporting, external validity, internal validity (bias and confounding), and power. The maximal quality index (QI) is 28. The DBC is a 27-item checklist, in which each item can have a score of 1 or 0, except for question 5, which may score 2. We scored question 27 (power) as 0 or 1 the authors reported a power calculation. QI scores of >20 were considered good, 11–20 moderate, and <11 poor. We did not exclude any article based on the DBC scores. We evaluated the level of agreement between reviewers in the quality assessment using the ICC.

2.3.6 Data Synthesis - Meta Analysis.

We used R 4.1.3 (<https://www.r-project.org/>) and the package meta 5.2-0 (<https://cran.r-project.org/web/packages/meta/index.html>) to conduct the meta-analyses (Balduzzi et al., 2019). We computed bias-corrected standardized mean differences (Hedges' g) of the change scores with 95% CI. We assumed that included studies were methodologically different, so we used an inverse-variance with random-effects model and the DerSimonian and Laird estimator to pool effect sizes and estimate between-study-variance (τ^2) (Borenstein et al., 2010). We created forest plots with 95% CI to graphically summarize the meta-analyses. We estimated statistical heterogeneity using Cochran Q and the I^2 statistic; we interpreted I^2 as follows: 25%, 50%, and 75% reflecting low, moderate, and high heterogeneity, respectively (Higgins, 2003). We used funnel plots and Egger's regression tests to assess the risk of publication bias. We set an alpha level of 0.05 for all statistical tests.

2.4 Results

2.4.1 Search Results

We found 835 articles after using our search strategy (Appendix 2). After screening potential articles and removing duplicates, we used 13 articles (Figure 1) on this study. The

agreement between reviewers in the data extraction process was ICC = 0.805 95%CI[0.643, 0.910].

2.4.2 Risk of Bias of Individual Studies

We reported DBC results for each study in Table 1., 1230 DBC scores ranged from 13 to 25. Seven articles obtained QI scores above 20 in the DBC, and six articles obtained QI scores between 11 and 20 in the DBC. No study scored below 11 in the DBC. The agreement between reviewers when using the DBC was ICC = 0.840 95%CI[0.645, 0.933].

| Study (year) | Participants (n); | Age (mean) | Information for athletic population | Intervention Type | Intervention | Length of study | Pain Scale | Dows and Black Score |
|-----------------------|--|--|---|-------------------|--|---|-------------|----------------------|
| Åstrom et al. (1992) | 67; INT: 34 piroxicam (11 female); CON: 33 placebo (8 female) | Mean 35; INT:18-58 piroxicam; CON: 20-57 placebo | Engaged in various sports | Oral | INT: Piroxicam 40mg 2 days and 20mg thereafter; CON: Placebo | 28 days (2 weeks mandatory, 2 weeks optional) | VAS 100mm | 12 |
| Dupont et al., (1987) | 61; INT: 30 ibuprofen (14 female); CON: 31 placebo (8 female) | INT: 23.9 +/- 6.4 Ibuprofen; CON: 23.7 +/- 5.7 placebo | Physically active individuals, Sports Medicine clinic | Oral | INT: 600 mg ibuprofen, 4 times per day; CON: Placebo (lactosa) | 28 days | Scale 0-3 | 21 |
| Galer et al., (2000) | 213; INT: 106 diclofenac (28 female); CON: 107 placebo (37 female) | INT: 31.15 diclofenac; CON: 29.9 placebo | Sports related sprain, strain, contusion | Topical | INT: 1.3% diclofenac; CON: Placebo | 14 days | VAS 100mm | 19 |
| Giani et al., (1989) | 45 (6 female) | Mean 20.87 | Athletes | Oral | INT1: Diclofenac; INT2: Suprofen; | 7 days | VAS 0-100mm | 18 |

| CON: Placebo | | | | | | | | |
|----------------------------------|--|---|--|---------|---|--------|--------------|----|
| Malmgaard-Clausen et al., (2021) | 68; INT: 33 naproxen (11 female); CON: 35 placebo (9 female) | INT: 41+/-2.1 Naproxen; CON: 40.7 +/- 1.7 Placebo | Active sports participants | Oral | INT: Naproxen, 500mg 2 times per day; CON: Placebo | 1 year | NRS 0-10 | 17 |
| May et al., (2007) | 36; INT: 20 diclofenac; CON: 16 placebo (only men analyzed) | INT: 36 +/-12 Diclofenac; CON: 39 +/- 10 Placebo | Long distance kayakers | Topical | INT: 2.5g 1% diclofenac gel; CON: Placebo gel (sorbolene and 10% propyl alcohol) | 5 days | Scale 1-5 | 21 |
| Predel et al., (2017) | 132; INT: 64 ibuprofen (31 female); CON: 68 placebo (39 female) | INT 33.2 +/- 12.1 ibuprofen; CON: 30.8 +/- 11.2 placebo | Acute injury in sport, immediate recruitment | Topical | INT: Ibuprofen 200mg plaster; CON: Placebo | 6 days | VAS 0-100 | 25 |
| Predel et al., (2004) | 120; INT: 60 diclofenac (22 female); CON: 60 placebo (25 female) | INT: 31.6 diclofenac; CON: 31.7 placebo | Soccer, handball, and basketball competitions/training camps | Topical | INT: 140mg diclofenac sodium; CON: Placebo | 7 days | VAS 0-100 | 25 |

| | | | | | | | | |
|----------------------------|--|--|--|---------|---|-------------|-----------------------------------|----|
| Predel et al., (2016) | 168; INT: 84 diclofenac (33 female); CON: 84 placebo (33 female) | INT: 33.39 +/- 11.12 diclofenac; CON: 33.31 +/- 11.45 placebo | Patients recruited on sports grounds | Topical | INT: 140 mg diclofenac sodium patch; CON: Placebo | 7 days | VAS 0-100 | 24 |
| Predel et al., (2018) | 130; 66 ibuprofen (22 female); 64 placebo (20 female) | INT: 34.09 ibuprofen; CON: 30.08 placebo | Acute injury in sport, immediate recruitment | Topical | INT: 200 mg ibuprofen plaster; CON: Placebo | 5 days | VAS 0-100 | 24 |
| Reynolds et al., (1995) | 44; INT1: 13 meclofenamate (0 female); INT2: 16 diclofenac (1 female); CON: 14 placebo (0 female) | INT1: 33.8 +/- 10.6 meclofenamate; INT2: 31.8 +/- 9.9 Diclofenac; CON: 30.7 +/- 7.9 Placebo | Acute sports related injury | Oral | INT1: 50 mg meclofenamate; INT2: 25 mg diclofenac; CON: Placebo | 7 days | VAS 0-10 | 16 |
| Steunebrink et al., (2013) | 33; INT: 16 GTN (5 female); CON: 17 placebo (3 female) | INT: 31.9 +/- 9.6 GTN; CON: 33.8 +/- 10.5 Placebo | Recreational or competitive athletes, playing various sports | Topical | INT: Topical glyceryl trinitrate 5mg; CON: Placebo | 12 weeks | VAS 0-10 (reverse order) | 19 |

| | | | | | | | | |
|-----------------------|---|--|---|---------|--|----------|-------------|----|
| Wetzel et al., (2002) | 156; INT1: 54 escin 1% (17 female); INT2: 51 escin 2% (17 female); CON: 51 placebo (10 female) | INT1: 29.2 escin 1%; INT2: 31.3 escin 2%; CON: 30.7 placebo | Soccer, karate, handball competitions | Topical | INT1: Escin 1%, 5% diethylammoni um salicylate, 5000 IU heparin; INT2: Escin 2%, 5% diethylammoni um salicylate, 5000 IU heparin; CON: Placebo | 24 hours | VAS 0-10 | 24 |
|-----------------------|---|--|---|---------|--|----------|-------------|----|

Table 1

2.4.3 Characteristics of the Interventions and Participants

There were 13 studies included in this article with 16 interventions. The studies done by Giani et al., 1989, Reynolds et al., 1995, and Wetzal et al., 2002, all had two interventions. We reported information for each article in Table 1. The articles involved 1304 participants from which 1273 received an intervention (410 females). A weighted average was used to calculate the average age of participants, which was 31.07 years, with the youngest participants being 17.5 years-old and the oldest being 58 years-old. 13 articles reported data from both males and females, but May et al., 2007, only analyzed data from males.

All articles reported an intervention compared to a placebo group Åström & Westlin, 1992; Dupont et al., 1987; Galer et al., 2000; Giani et al., 1989; Malmgaard-Clausen et al., 2021; May et al., 2007; H. G. Predel et al., 2004, 2016; H.-G. Predel et al., 2017, 2018; Reynolds et al., 1995; Steunebrink et al., 2013; Wetzal et al., 2002. Eight interventions used a topical medication, and five interventions used an oral medication. There was a wide range of intervention duration, from 24 hours to one year. The post intervention pain level used to calculate the effect size was the day of the peak difference from the beginning of treatment. For most, this occurred within two weeks from the onset of pain.

2.4.4. Meta-Analysis

We observed a significant pooled effect size reflecting a reduction in pain outcomes for the topical treatment ($g=-0.64$; 95% CI $[-0.89, -0.39]$; $p < 0.001$). The meta-analysis for the topical treatment (Figure 2) presented high heterogeneity ($I^2=71\%$) indicating high variability between the effect sizes of included studies.

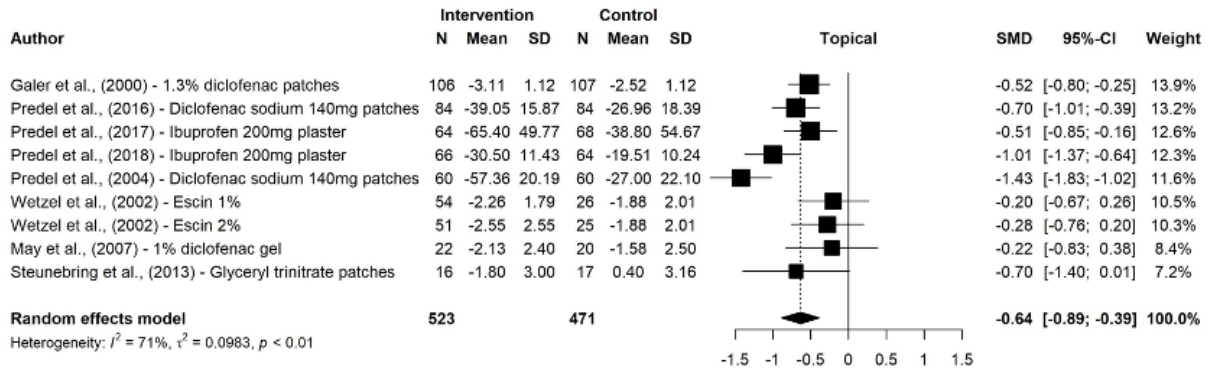


Figure 2-2. Forest plot indicating the efficacy of topical analgesic medication for injured athletes compared to a placebo. There was a significant improvement in pain in the athletes receiving the topical medication compared to the placebo ($p < 0.001$).

We did not observe a significant reduction in pain outcomes for the oral treatment ($g = -0.26$; 95% CI [-0.60, 0.17]; $p = 0.272$). The meta-analysis for the oral treatment (Figure 3) presented moderate heterogeneity ($I^2 = 55\%$) indicating moderate variability between the effect sizes of included studies.

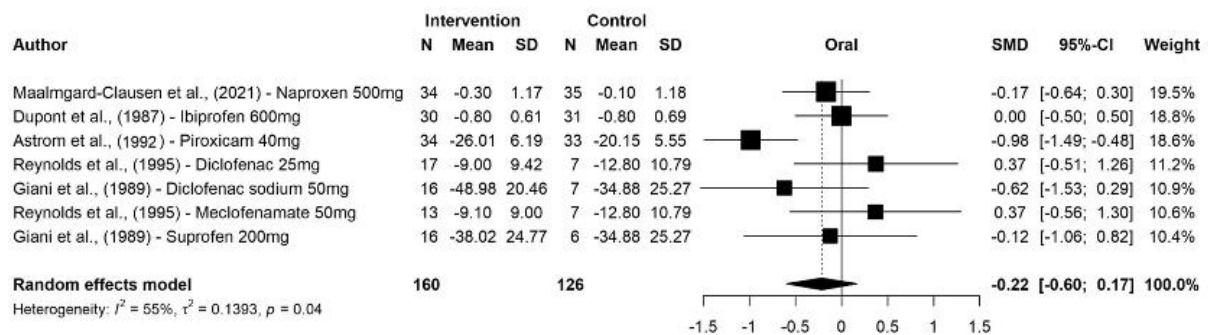


Figure 2-3. Forest plot indicating the efficacy of oral analgesic medication for injured athletes compared to a placebo. There was not a significant improvement in pain in the athletes receiving the oral medication compared to the placebo ($p = 0.272$).

When conducting a meta-analysis for the topical and oral treatments (Figure 4), we observed a significant pooled effect size ($g = -0.49$; 95% CI [-0.71, -0.27]; $p < 0.001$) with a

moderate heterogeneity ($I^2=69\%$). Although the pooled effect size for the topical treatment was significant ($p < 0.001$) and the pooled effect size for the oral treatment was not significant ($p = 0.272$), we did not observe statistical differences between the topical and oral treatments ($p=0.072$).

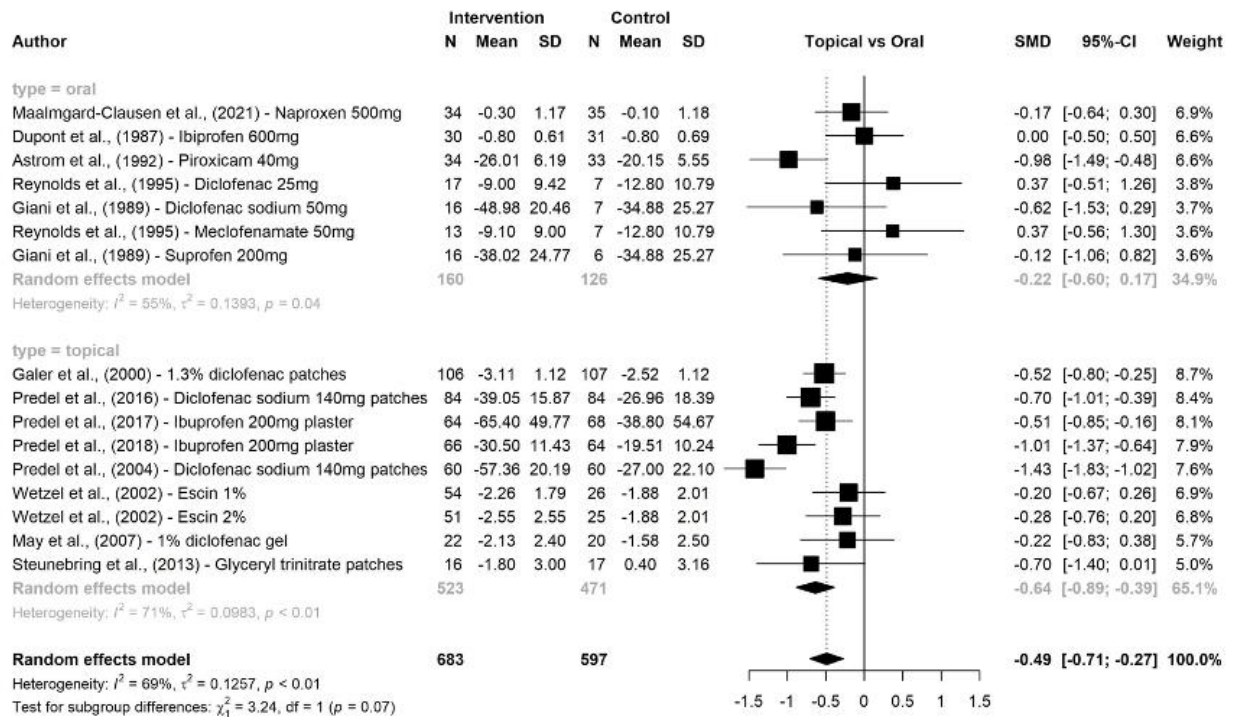


Figure 2-4. Forest plot indicating the efficacy of topical and oral analgesic medications for injured athletes compared to a placebo. There was a significant pooled effect size ($p < 0.001$), but we did not observe statistical difference between the topical and oral treatments ($p=0.072$).

Funnel plots did not suggest risk of publication bias and the Egger's regression tests did not indicate funnel plot asymmetry for the topical treatment (Figure 5, $p=0.699$) and for the oral treatment (Figure 6, $p=0.461$).

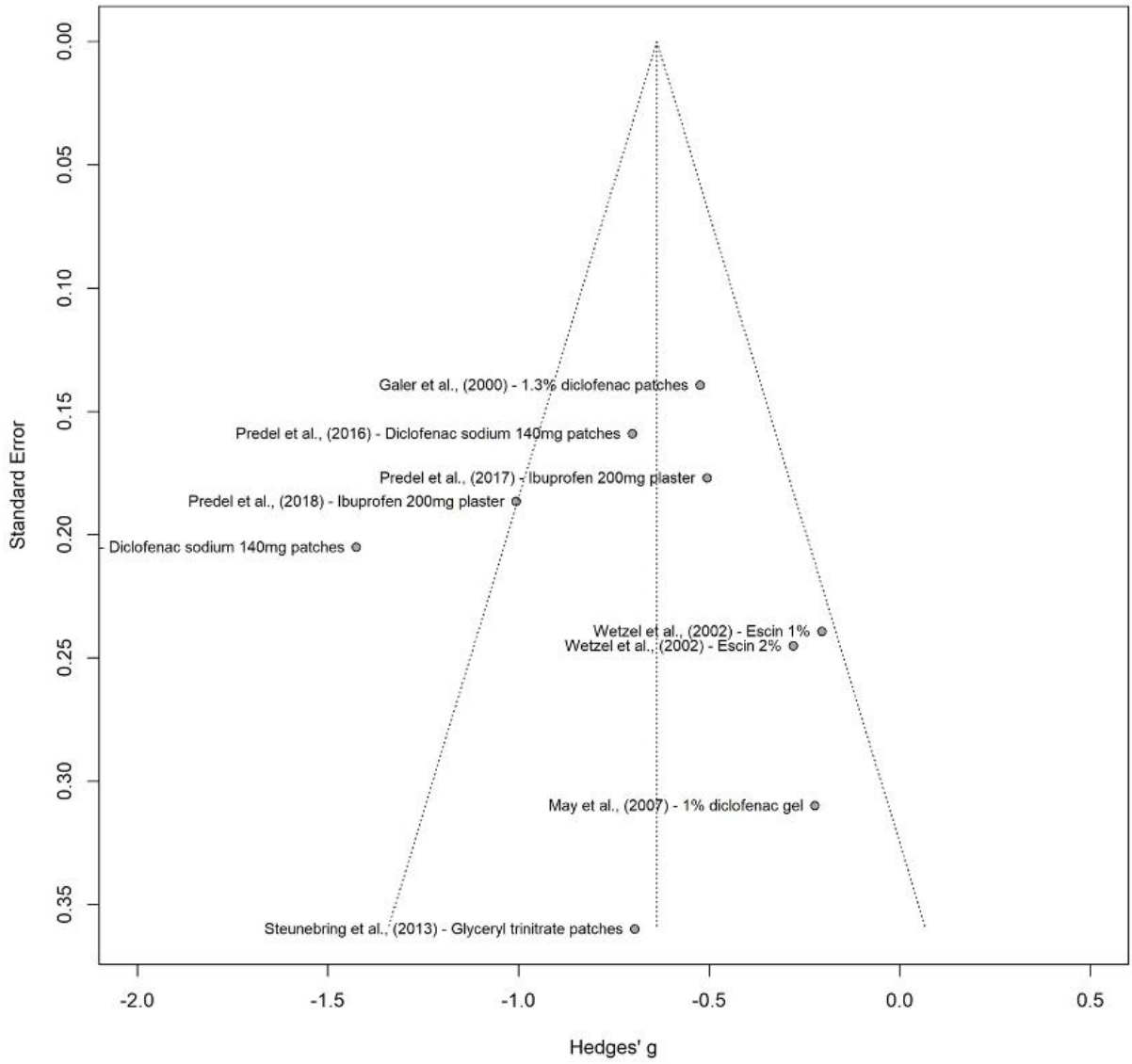


Figure 2-5. Funnel plot based on standardized effect sizes of the topical treatments.

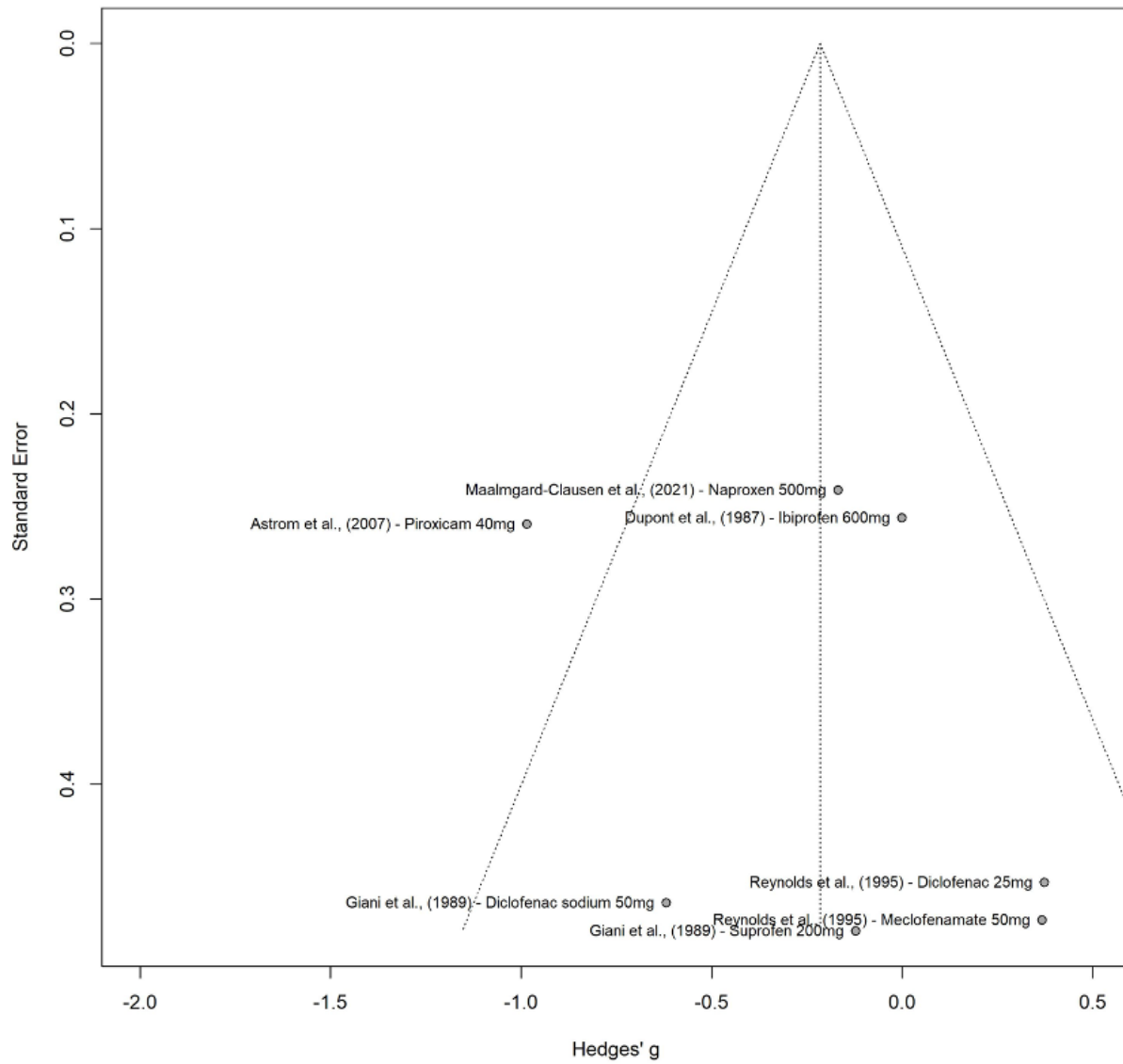


Figure 2-6. Funnel plot based on standardized effect sizes of the oral treatments.

We transformed the mean and standard deviations of the pain outcomes to a 100mm VAS to favor comparability between outcomes.

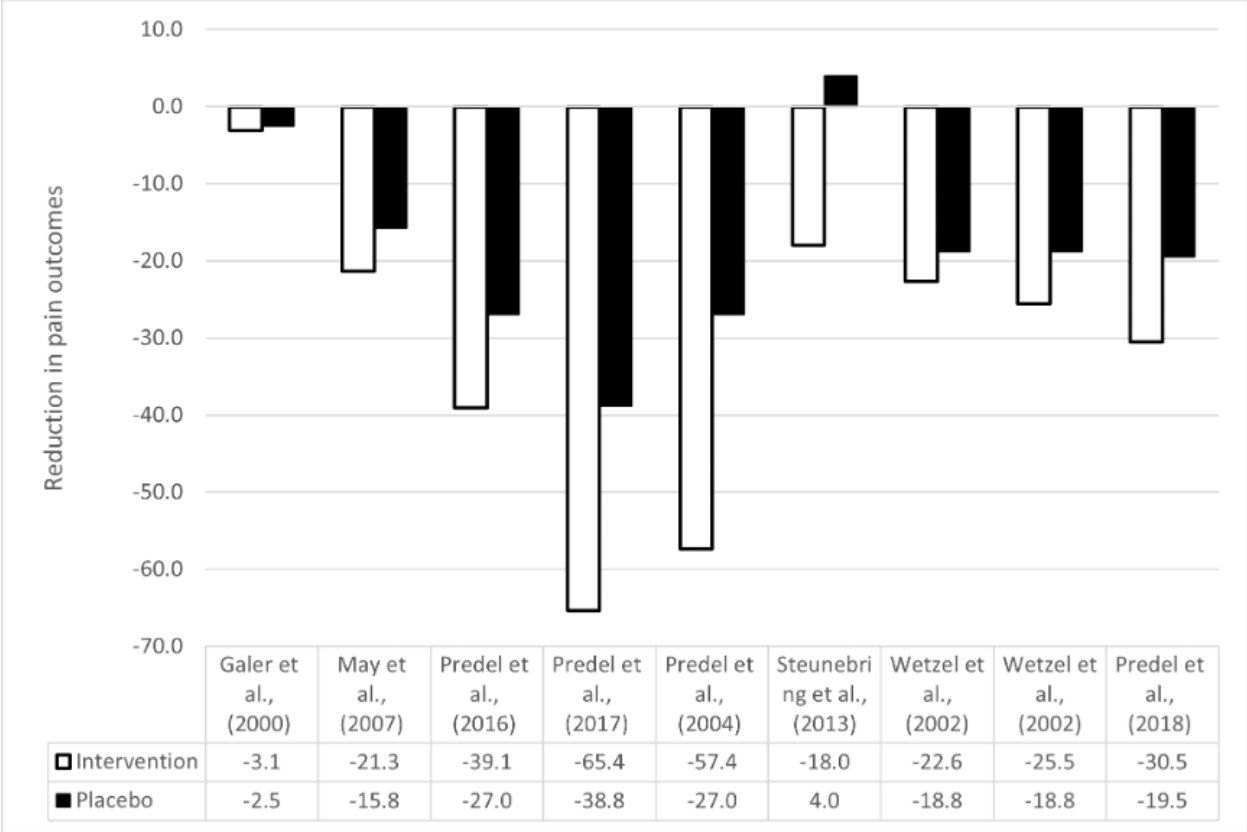


Figure 2-7. Reduction in pain outcomes for topical treatment versus placebo based on change scores in a 100mm VAS for the topical treatment.

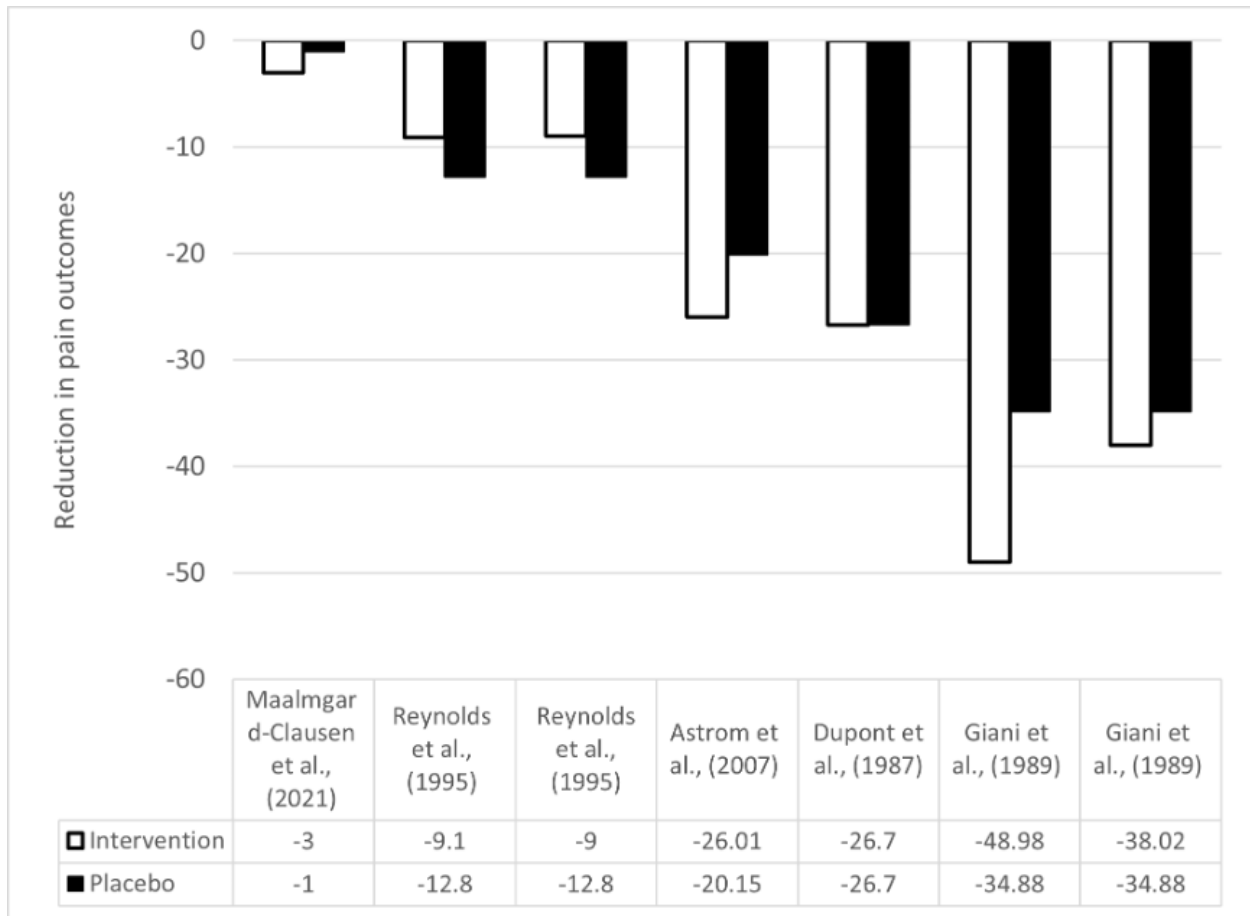


Figure 2-8. Reduction in pain outcomes for oral treatment versus placebo based on change scores in a 100mm VAS.

2.5 Discussion

The objective of this study was to determine if topical and oral medications were effective compared to a placebo in athletes. As noted in the forest plots, the results of this meta-analysis suggest that topical analgesics are more effective compared to a placebo in reducing pain in an athletic population suffering from musculoskeletal injuries. It is important to note that the sustained injuries in this review were not associated with DOMS or induced pain but actual musculoskeletal injuries. When athletes suffer an injury, it is essential to know what medication would be effective in reducing pain. In addition, oral analgesic medications were not effective in reducing pain in injured athletes compared to a placebo. The forest plots also illustrate that both

the topical and oral effect sizes are slightly more skewed to the left than the placebo medications, but only the topical is skewed enough to be statistically significant.

2.5.1 Mechanisms of action of the medications

The medications used in the studies included in this review were NSAIDs (ibuprofen, piroxicam, naproxen, and diclofenac) as well as diethylammonium salicylate with Escin, and triglyceryl nitrate, which all reduce pain in different ways. NSAIDs reduce pain by inhibiting the cyclooxygenase (COX) enzyme activity (Ghlichloo & Gerriets, 2022; Lundberg & Howatson, 2018). COX enzymes are responsible for the production of prostaglandins following tissue injury (Lundberg & Howatson, 2018). COX is responsible for the conversion of arachidonic acid into thromboxanes, prostaglandins, and prostacyclins (Vane, 1971). Thromboxanes are required for platelet aggregation, while prostaglandins are vasodilators, increase the hypothalamus temperature, and have a role in pain relief (Ghlichloo & Gerriets, 2022). By blocking these actions, NSAIDs decrease pain (Ghlichloo & Gerriets, 2022).

The isoenzymes COX-1 and COX-2 are the ones that are typically targeted by NSAIDS (Ghlichloo & Gerriets, 2022). COX-1 enzymes are essential in the body, while COX-2 enzymes are present during anti-inflammatory response (Chaiamnuay et al., 2006). Some NSAIDs are selective and target only COX-2 (Chaiamnuay et al., 2006), but all of the NSAID medications in this present study are non-selective and target both COX-1 and COX-2 enzymes. The mechanism of action for NSAIDs are the same for both oral and topical medications.

Diethylammonium salicylate is a type of rubefacient which is thought to decrease pain by causing counter-irritation to the skin (Tallo, 2016). This counter-irritation causes a vasodilation, resulting in a warming sensation (Derry et al., 2014). This drug is related to NSAIDs but works by a different mechanism when applied topically (Derry et al., 2014). The cutaneous irritation produces sensory nerve irritation, which is believed to decrease pain in the musculoskeletal

structures innervated by the same nerves (Derry et al., 2014). Escin has been shown to decrease inflammation, but the mechanism in humans is still unclear (Gallelli, 2019).

Glyceryl trinitrate (GTN) liberates nitric oxide (NO) in the tissue (Agvald et al., 2002). NO is thought to influence tendon healing by being involved in processes such as blood flow, host defense, and collagen synthesis (Bokhari & Murrell, 2012).

2.5.2 Why would topical medications be more effective than oral?

While oral NSAIDs act systemically to inhibit COX activity, topical analgesics act locally to reduce pain. An acute musculoskeletal injury is accompanied by a local inflammatory reaction (H. G. Predel et al., 2016). Oral NSAIDs only target the affected area after large quantities of the drug enter systemic circulation, whereas topical NSAIDs can deliver direct relief (H. G. Predel et al., 2016). Topical medications also interact with nociceptors in the outer layers of the skin at the site of the injury (Choi et al., 2020). They penetrate the stratum corneum in the epidermis to reach unmyelinated A δ and C-fibers, which transmit the sensation of pain (Choi et al., 2020). This direct interaction with the pain site may offer an explanation as to why the topical medication was more effective than the oral medications in this study.

There are some side effects of oral NSAIDs. By acting on prostaglandins, NSAIDs can adversely affect the gastric mucosal barrier, renal blood flow, endothelial tone, circulatory system, kidneys, and liver (Derry et al., 2017; Maniar et al., 2018). The rationale behind topical NSAIDs is that they can act locally to inhibit COX activity with minimal systemic effect (Derry et al., 2017). Topical NSAID application does reach high enough levels to inhibit COX-2 activity, all while being found at low levels of plasma concentration (Derry et al., 2017; Maniar et al., 2018). Because of this, there should be less adverse effects with the use of topical NSAIDs.

The other types of medication in this review, diethylammonium salicylate and glyceryl trinitrate, are used exclusively topically for pain management.

2.5.3. Why was the oral not effective compared to the placebo?

In this review, the group that received the oral placebo experienced a similar amount of pain reduction compared to the medication group. As mentioned earlier, many previous studies have shown effectiveness of oral medications over placebo. There the analysis of the oral studies supports the idea that athletes have a higher placebo experience than nonathletes. However, the topical medications were overall more effective than the placebo, which contradicts this hypothesis. Though it has been previously demonstrated that athletes did not have a greater placebo effect than non-athletes in a study involving pain induction (Geisler et al., 2020), we expected the results of our study to be different as the subjects are injured athletes. The pain you experience from an injury will be different than pain induction because it will impact the return to play of the athlete. Furthermore, the mechanisms of injury are not the same when comparing sustained injury to induced injury or DOMS (Mackey et al., 2012). It is thus not recommended to generalize the results from studies on the effect of medications on DOMS or induced injury to sustained injuries (Mackey et al., 2012). As such, this current review only analyzes sustained athletic injuries.

An individual can experience placebo analgesia because of verbal cues alluding to pain relief (Colloca et al., 2013). This can be due in part to the individual remembering previous experiences of pain relief (Colloca et al., 2013). Those who frequently use medication may be more conditioned to experience an analgesic effect similar to that of an active drug when a placebo is used (Colloca et al., 2013). As previously stated, athletes are subject to frequent injuries (Moseid et al., 2018), and analgesic medications that are available over-the-counter are frequently used by athletes (Feucht & Patel, 2010). The most common form of analgesic medication that athletes take are oral NSAIDs (Harle et al., 2018). An athlete may thus be more used to taking oral analgesic drugs than topical for their injuries to help with their pain management and as such are conditioned to respond the same way to an oral placebo medication.

Since the 1960s, studies have suggested that the placebo effect is a result of the release of endogenous neuromodulators, such as opioids, cholecystokinin, cannabinoids, dopamine, as well as the activation of the vasopressin and oxytocin systems (Colloca, 2019; Colloca et al., 2013). Placebo drugs have been shown to activate the rostral anterior cingulate cortex (rACC) and the orbitofrontal cortex (OrbC) on positron emission tomography (PET) (Benedetti, 2006). Studies have shown that there is a descending pain-modulating pathway involving the rACC, the periaqueductal grey (PAG), and the rostral ventromedial medulla (RVM) (Benedetti, 2006). Functional magnetic resonance imaging of the brain during placebo analgesia showed decreased activity in areas involved in pain transmission such as the thalamus, the anterior insula (aINS), and the caudal rACC (Wager et al., 2004).

2.5.4 Previous studies comparing medication to placebo

To date, there have been various systematic reviews and meta-analyses analyzing the effectiveness of topical or oral analgesic medications on pain in adults compared to a placebo, but none exclusively done on athletes, and not all include a comparison of both topical and oral medications. There are varying results amongst these non-athlete studies. A systematic review and meta-analysis analyzing the effectiveness of oral and topical analgesic medications for ankle sprains stated that overall, both oral and topical medications were effective at reducing short term pain in adults (van den Bekerom et al., 2015). A systematic review and meta-analysis studying the effectiveness of topical NSAIDs in acutely injured adults noted a significant overall effect size for diclofenac, ibuprofen, ketoprofen, piroxicam, and indomethacin compared to placebo (Derry et al., 2015). Another review reported that among six randomized-controlled trials, topical and oral NSAIDs were statistically significant over placebo medications at treating chronic lower back pain in adults (Enthoven et al., 2016). They do however state that the quality of this evidence is low (Enthoven et al., 2016). A review on lateral epicondylitis in adults showed low-quality evidence that there may be some benefit of topical and oral NSAIDs over placebo

medication (Pattanittum et al., 2013). These reviews all include subjects suffering from acute or chronic pain, not experimentally induced pain or DOMS.

There have also been various studies on the effectiveness of topical and oral medications other than the reviews listed above. These studies compare topical to oral medications in various injured populations. A study on topical and oral ibuprofen in older adults with chronic knee pain showed that both formulations were equally effective at pain reduction (Underwood et al., 2008). A study measuring the effectiveness between topical and oral ibuprofen in acute soft-tissue injuries also concluded equal success (Whitefield et al., 2002). Two systematic reviews showed no difference in effectiveness between oral and topical NSAIDs for acute and chronic pain (Derry et al., 2015).

As our review exclusively looks at the effectiveness of analgesic medications in an athletic population, the results were hypothesized to be different than the regular population. The following are the reasons why this may be.

Humans often use analgesic medications to reduce pain but are also able to inhibit pain through their own endogenous pain-inhibition system (Basbaum & Fields, 1978). There are numerous ways in which this occurs, including use of placebo medications or the activation of this system through conditioned pain modulation (CPM). Chronic pain may develop as a result of reduced endogenous pain inhibition (Edwards, 2005). One systematic review demonstrated that chronic pain patients had a reduced CPM (Lewis et al., 2012). This was demonstrated in the non-athletic population. Conversely, it has been shown that endurance athletes have a higher CPM effect than non-athletes (Flood et al., 2017; Geva et al., 2017; Geva & Defrin, 2013). There is a high prevalence of sports injuries among athletes (Moseid et al., 2018). These injuries can be acute or chronic, and can be accompanied by pain (Moseid et al., 2018). These athletes will oftentimes play through their pain (Barrette & Harman, 2020). Athletes have been shown to have a higher pain tolerance than non-athletes (Geisler et al., 2020; Tesarz et al., 2012). These factors may contribute to the higher CPM seen in athletes. This further suggests

that athletes have a stronger endogenous pain-inhibition system than non-athletes. It has been suggested that CPM and the placebo effect occur via the same mechanism (Damien et al., 2018; Sprenger et al., 2011). As such, it is plausible to believe that athletes would also have a higher placebo effect than non-athletes.

2.5.5 Variability in studies in this review

The forest plot shows that there is a high heterogeneity amongst the topical and oral studies. The variance in these studies could thus be due to something other than chance. The topical studies had generally higher sample sizes than the oral studies, and there were more topical studies included in the analysis which met the search criteria.

None of the oral medication studies except Åström & Westlin, 1992, were statistically significant. The confidence intervals shown on the forest plot were high for each oral study, indicating less precision.

There are several reasons for the variability amongst the studies. The studies measured pain levels on different days. Some studies were conducted over the course of several months, and others just over a few days. This could contribute to the variability in the results, as the natural course of pain is such that pain may improve on its own the more time has elapsed since the injury date. Some studies allowed the athletes to receive concurrent therapy such as the use of ice, physical therapy, or rescue medication (typically acetaminophen). As such, the decrease in pain can be affected by other factors than solely the medication used. The use of concurrent therapies is possibly due to ethical concerns regarding withholding treatment from participants. Other factors that could affect the variability in the results are that there are different injuries being studied, as well as different pain levels at the beginning of the study amongst groups.

Additional factors that may affect the variability of the studies are the type of medication used and the location of application for topical analgesics. The studies within this review used

different types of medications, thus there could be differences in the efficacies. Furthermore, it has been suggested that the bioavailability of the topical drug used differs depending on the location of the application (Shah et al., 1996). In this study, ketoprofen applied to the back and arm produced statistically significantly higher plasma levels than in the knee of male subjects (Shah et al., 1996). Some studies in this review observed the effect of a certain drug on a common type of injury, but many others included various musculoskeletal conditions. Thus, it is possible that the topical drug applications may have been more effective with some injuries and others less so.

2.5.6 Future directions

To show that a medication is superior to a placebo, it is best practice to include an experimental drug group, a placebo group, and a natural history (NH) group, taking no medication at all (Klinger et al., 2018). The purpose of the NH group is to show that the reduction in pain is not due to other factors such as the natural course of the injury or spontaneous healing (Klinger et al., 2018). None of the studies in this review included an NH group, presumably for the ethical reason of not withholding a treatment that could potentially help the individual's pain. As such, there are two speculations that we can make. The first is that if athletes do not in fact experience the placebo effect as highly as non-athletes do even in sustained injuries, then the insignificant results between the experimental and placebo groups for oral medications could be because neither are truly effective, and that the injury took its natural healing course. If the results of the Geisler study on pain provocation cannot be extrapolated to sustained injuries in athletes, then the results of this study would show that the experimental analgesic oral drugs do not reduce pain statistically significantly more than placebo medications.

2.5.7 Limitations

As stated previously, there were more topical than oral studies used in this meta-analysis. Many oral medication studies in athletes were excluded for reasons such as not including a placebo group, or not including enough data to be able to carry out the meta-analysis. Most subjects in all the studies used are male.

2.6 Conclusion

Topical analgesic medications are more effective than oral medications at reducing pain in athletes. There are less reported adverse effects with the use of topical medications. If given the choice, athletes should elect to take topical medications instead of oral medications to help reduce pain after injury.

2.7 Conflict of Interests

The authors have no conflicts of interest to declare.

Appendix 1. Search strategy

| Database | Search Strategy |
|----------------|--|
| Web of Science | TOPIC: (comparison OR efficacy) AND TOPIC: (medication OR drug OR analgesic) AND TOPIC: (ketorolac OR Toradol OR anti-inflammatory OR NSAID OR ibuprofen OR acetaminophen OR paracetamol OR aspirin OR acetylsalicylic acid OR corticosteroid OR diclofenac OR piroxicam OR indomethacin OR naproxen OR ketoprofen) AND TOPIC: (placebo) AND TOPIC: (athlete OR sport OR game OR athletic injuries OR sports medicine OR athlet* OR injur*) AND TOPIC: (oral OR topical) |

| | |
|--------------|---|
| Ovid/Medline | (comparison OR efficacy) AND (medication OR drug OR analgesic) AND (ketorolac OR Toradol OR anti-inflammatory OR NSAID OR ibuprofen OR acetaminophen OR paracetamol OR aspirin OR acetylsalicylic acid OR corticosteroid OR diclofenac OR piroxicam OR indomethacin OR naproxen OR ketoprofen) AND (placebo) AND (non-steroidal) AND (athlete OR sport OR game OR athletic injuries OR sports medicine OR athlet* OR injur*) AND (oral OR topical) |
| SPORTDiscus | (comparison OR efficacy) AND (medication OR drug OR analgesic) AND (ketorolac OR Toradol OR anti-inflammatory OR NSAID OR ibuprofen OR acetaminophen OR paracetamol OR aspirin OR acetylsalicylic acid OR corticosteroid OR diclofenac OR piroxicam OR indomethacin OR naproxen OR ketoprofen) AND (placebo) AND (non-steroidal) AND (athlete OR sport OR game OR athletic injuries OR sports medicine OR athlet* OR injur*) AND (oral OR topical) |
| PubMed | (comparison[Title/Abstract] OR efficacy[Title/Abstract]) AND (medication[MeSH Major Topic] OR drug[Title/Abstract] OR analgesic[Title/Abstract]) AND (ketorolac[Title/Abstract] OR Toradol[Title/Abstract] OR anti-inflammatory[Title/Abstract] OR NSAID[Title/Abstract] OR ibuprofen[Title/Abstract] OR acetaminophen[Title/Abstract] OR paracetamol[Title/Abstract] OR aspirin[Title/Abstract] OR acetylsalicylic acid[Title/Abstract] OR corticosteroid[Title/Abstract] OR diclofenac[Title/Abstract] OR piroxicam[Title/Abstract] OR indomethacin[Title/Abstract] OR naproxen[Title/Abstract] OR ketoprofen[Title/Abstract]) AND (placebo[MeSH Major Topic] AND (non-steroidal[Title/Abstract]) AND (athlete[MeSH Major Topic] OR sport[MeSH Major Topic] OR game[Title/Abstract] OR athletic injuries[MeSH Major Topic] OR sports medicine[MeSH Major Topic] OR athlet* OR injur*) AND (oral[Title/Abstract] OR topical[Title/Abstract]) |

Appendix 2. Records by database

Database

**Records identified using the
search strategy**

| | |
|----------------|-----|
| PubMed | 237 |
| Scopus | 185 |
| Web of Science | 235 |
| SPORTDiscus | 178 |
| Total | 835 |

Appendix 3. Downs and Black Checklist

| Article | Q 1 | Q 2 | Q 3 | Q 4 | Q 5 | Q 6 | Q 7 | Q 8 | Q 9 | Q 10 | Q 11 | Q 12 | Q 13 | Q 14 | Q 15 | Q 16 | Q 17 | Q 18 | Q 19 | Q 20 | Q 21 | Q 22 | Q 23 | Q 24 | Q 25 | T ot al | | | | |
|---------------------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|---------|---|---|----|----|
| Galer et al. (2000) | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 19 |
| Predel et al. (2004) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 25 |
| Astrom & Westlin (1992) | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 12 | |
| Dupont et al. (1987) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 21 |
| Malmgaard-Clausen et al. (2021) | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 0 | 17 | |
| Reynolds et al. (1995) | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 1 | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 16 | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|------------------------------|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|----|---|---|---|---|----|
| Giani et al. (1989) | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 18 |
| May et al. (2007) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 0 | 1 | 0 | 21 | | | | | |
| Predel et al. (2017) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 25 |
| Predel et al. (2016) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 24 |
| Predel et al. (2018) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 24 |
| Steunebrink et al. (2013) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 19 | | | | | |
| Wetzel et al. (2002) | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 24 |

Chapter 3: The Association between the Labor Pain Relief Questionnaire for Pregnant Women, the Pain Catastrophizing scale, and the Edinburgh postnatal Depression Scale

3.1 Abstract

Background: The fear of childbirth is a common concern among many women, often due to the fear of labor pain. Women who have high levels of pain catastrophizing before birth may experience more pain during labor. The use of epidural analgesia during labor can result in negative effects, such as prolonged labor, non-spontaneous deliveries, and newborns with low APGAR scores and higher NICU admissions. To better understand why there has been a rise in epidural use, the Labor Pain Relief Attitude Questionnaire for pregnant women (LPRAQ-p) was created and validated in 2020. This questionnaire measures women's attitudes towards labor pain relief. A pilot study suggested that a high score on the questionnaire was associated with a higher likelihood of using pain relief medication during delivery. This questionnaire has not been used for the same purpose since this pilot study. There is a possibility that epidural analgesia and pain catastrophizing are risk factors in the development of postpartum depression.

The research project aims to measure the association between pain and the LPRAQ-p scale, between catastrophizing, labor pain relief, and pain before and after delivery, and between the Edinburgh Postnatal Depression Scale (EPDS) and the LPRAQ-p scale. The hypotheses are that catastrophizing will lead to more pain, the PCS scale and the LPRAQ-p scale will be correlated, and the LPRAQ-p questionnaire and the EPDS will be correlated.

Methods: The participants in this study (n=31) were pregnant women in their third trimester (27 to 40 weeks of gestation). Follow up measurements were conducted on the participants 4-6 weeks postpartum. The participants completed the LPRAQ-p questionnaire, PCS scale, and EPDS at the first meeting. At 4-6 postpartum, participants were asked to report current pain and

a delayed recall of pain at the time of delivery on the Visual Analogue Scale (VAS) as well as the EPDS again.

Results: The PCS was not correlated with the VAS during labor (VAS2) ($r=-0.187$, $p=0.313$), the VAS postpartum (VAS1) ($r=0.181$, $p=0.331$), or the LPRAQ-p ($r=-0.160$, $p=0.390$). The LPRAQ-p was additionally not correlated with the VAS during labor ($r=0.119$, $p=0.525$), the VAS postpartum ($r=-0.135$, $p=0.470$), or with the EPDS postpartum ($r=-0.070$, $p=0.708$).

Discussion: Catastrophizing was not associated with pain, the LPRAQ-p, or potential postpartum depression. The PCS may not be the best tool for pregnancy as previously thought, potentially because women do not view pregnancy as an injury and know the end date. The EPDS was also not associated with the LPRAQ-p. The EPDS may not be the optimal tool to use in the prenatal period to determine potential use of epidural analgesia, or pain experience during labor, though it has been validated for use in the prenatal period to flag potential development of postpartum depression. A secondary analysis showed that higher scores on the LPRAQ-p were associated with use of epidural analgesia, and that EPDS were higher amongst nulliparous than multiparous women.

3.2 Introduction

The fear of childbirth, or tokophobia, is experienced by many women from childhood into old age (Hofberg, 2003). There are multiple factors that contribute to this fear, one of them being the fear of pain during labor (Hofberg, 2003). This fear can be so great for some women that they choose to have an elective cesarean section, despite the potential known risks (Dehghani et al., 2014; Jodzis et al., 2022). Some suggest that the expectation of pain during delivery may be associated with pain catastrophizing (Flink et al., 2009). A previous study used the pain catastrophizing scale (PCS) in eighty-two women to assess pain before and after labor (Flink et al., 2009). They noted that women who had a higher pain catastrophizing in the period before birth experienced more pain during labor (Flink et al., 2009).

Anything that can increase pain or the use of pain medication during childbirth is a concern. An increase in pain during labor may lead to more epidural analgesia use. The anticipation of labor pain is also a factor in the choice for receiving epidural analgesia during labor (Smorti et al., 2020). Anything that can contribute to less medication during labor is the preferred outcome since epidurals can lead to adverse effects such as the prolongation of labor and an increase in nonspontaneous deliveries (Zimmer et al., 2000), as well as newborns with lower APGAR scores and higher NICU admissions (Høtoft & Maimburg, 2021). Epidural analgesia has also been associated with a delayed onset of lactation (Segura-Pérez et al., 2022). A 2016 meta-analysis showed that combined-spinal epidural analgesia significantly affected the risk of non-reassuring Fetal Heart Rate (FHR) (Hattler et al., 2016). There are different possibilities of analgesic medication that can be administered by epidural during labor, but to date, there is no standard medication accepted which can optimize analgesia while minimizing adverse effects (Halliday et al., 2022).

While there is preliminary evidence indicating the relationship between catastrophizing and labor pain, a more specific scale was developed to measure the construct that the two are

linked. A new scale was developed and validated in 2020 called the Labor Pain Relief Attitude Questionnaire for pregnant women (LPRAQ-p) (Hulsbosch et al., 2020). This questionnaire was created to measure pregnant women's attitude toward labor pain relief in an attempt to have a greater understanding of why there has been an increase in the use of epidural analgesia despite the potential negative outcomes associated with its use (Hulsbosch et al., 2020). The results of this pilot study showed that a high score on the questionnaire during the prenatal period was related to a higher likelihood of using pain relief medication during delivery (Hulsbosch et al., 2020).

To date, no further studies have been done using the LPRAQ-p questionnaire to measure a woman's likelihood of selecting epidural analgesia. Furthermore, the pilot study reported that a higher score predicted a woman's likely choice to opt for analgesic medication but did not measure if a higher score would correlate to a higher pain intensity experienced during labor.

The Edinburgh Postnatal Depression Scale (EPDS) is a tool that has been validated and is used in both the pre- and post-natal period to flag women who are more likely to experience or be experiencing postpartum depression (Cox et al., 1987). There is currently conflicting research regarding whether or not receiving epidural analgesia during labor affects postpartum depression, with two recent systematic reviews concluding opposite results (Almeida et al., 2020; Liu et al., 2022). Both the Almeida et al. and the Liu et al. reviews only included observational studies, and it is possible that there were other confounding variables not taken into account amongst these included studies (Almeida et al., 2020; Liu et al., 2022). There is however evidence supporting that increased pain during labor is associated with a higher incidence of postpartum depression (Lim et al., 2020). Another study found that higher pain catastrophizing was a significant predictor of postpartum depression, while pain during delivery was not (Ferber et al., 2005). Postpartum depression can negatively impact the mother's

physical health, psychological well-being, and decrease her quality of life (Slomian et al., 2019). Further research is needed on factors affecting the incidence of postpartum depression.

For our research project, we included multiple maternal outcomes to make associations amongst them. Therefore, the objectives of our research project were:

- 1) Measure the association between pain and the LPRAQ-p scale.
- 2) Measure the association between catastrophizing, labor pain relief, and pain before and after delivery.
- 3) Measure the association between the EPDS and the LPRAQ-p scale.

Our hypotheses were:

- 1) Catastrophizing will be associated with more pain: higher score on the LPRAQ-p scale during pregnancy would predict a high amount of pain felt during delivery
- 2) The PCS scale and the LPRAQ-p scale will be positively correlated
- 3) The LPRAQ-p questionnaire and the EPDS will be positively correlated.

If the LPRAQ-p scale accurately predicts pain during labor, it could be an excellent, quick screening tool to use during pregnancy to flag women who may be at a higher risk of experiencing more pain and who are higher pain catastrophizers, and who are at a higher risk for postpartum depression. This would be practical as it is a shorter questionnaire than the PCS scale and the EDPS. If a labor practitioner (obstetrician, midwife, doula) could quickly identify women at risk of experiencing both higher pain and higher catastrophizing, they could be referred to an appropriate support system to prevent future labor complications and the potential development of postpartum depression.

3.3 Measures

3.3.1 Psychosocial self-report measures: Pain catastrophizing, Postpartum depression and labor pain

Pain catastrophizing was measured using the Pain Catastrophizing Scale which was developed and validated in 1995 (Sullivan et al., 1995). It has been used in a multitude of studies since its development, including with pregnant women (Flink et al., 2009). The PCS has 13 questions which can be answered from 0-4. Therefore, the lowest possible value would be 0 and the highest is 52.

The EPDS was first developed and validated in 1987 as a tool to recognize women at risk of suffering from postpartum depression (Cox et al., 1987). It can be administered in the prenatal period as well to raise a flag for women who may potentially suffer from postpartum depression after the birth of their child. The scale has further been validated in recent years and is still widely used today (Hewitt et al., 2010; Smith-Nielsen et al., 2018). The EPDS consists of 10 questions which can be answered from 0-4, thus score possibilities range from 0-40.

The LPRAQ-p is a questionnaire developed and validated in 2020 to measure pregnant women's attitude toward labor pain relief in an attempt to have a greater understanding of why there has been an increase in the use of epidural analgesia despite the potential negative outcomes associated with its use (Hulsbosch et al., 2020). The LPRAQ-p consists of six questions with answers ranging from 1-5. The lowest score for the LPRAQ-p is 6 and the highest is 30.

3.3.2 Pain Measurement

The VAS is a tool for pain measurement that has been shown to be reliable and has been validated multiple times (Bijur et al., 2001; Thong et al., 2018). The VAS ranges from 0 to 10. This scale has also been used previously in the context of assessing pain amongst pregnant and postpartum women (East et al., 2012; Marín-Jiménez et al., 2019).

3.3.3 Birth and activity information

Type of delivery was defined as regular (1), induced (2), or cesarean section (3). We were interested in prior activity level but were wary of increasing the demand of adding another scale to for the participants to complete. Therefore, we just measured prior level of activity before birth but during pregnancy using a 4-point Likert scale, with choices being not active (0), somewhat active (1), moderately active (2), and very active (3). Birth preparation and pain during labor were assigned a 0 for no and 1 for yes. Birth preparation included examples like attending prenatal classes, seeing a pelvic floor physiotherapist, or preparation with a doula. For prior births, if nulliparous, then the variable was 0. Multiparous women were assigned a variable of 1.

3.3.4 Procedures

Participant recruitment was done through social media posts, advertising in various Athletic Therapy, Osteopathy, Physiotherapy, and Acupuncture clinics as well as in Birthing Centers, and amongst prenatal groups. Fifty-one participants completed a screening questionnaire during their pregnancy. Participants were excluded if they had a previous mental health condition diagnosis (regardless of whether there was a prescription medication for said condition) or classification as a high-risk birth. Participants were also excluded if they were scheduled for an induction or a cesarean-section. Participants needed to be fluent in English as not all the questionnaires had been validated in other languages. Eleven participants were

initially excluded (see Figure 1). The remaining 40 agreed and were contacted for a Zoom meeting either entering or while in their third trimester. They were instructed to let the interviewer know if any of their conditions changed before the meeting could take place (i.e. if they were not initially classified as a high-risk pregnancy but then became high-risk). Seven participants were lost to follow-up for the first meeting. Thirty-three participants completed the first Zoom meeting in which they were asked to fill out an informed consent document, as well as the PCS, the EPDS, and the LPRAQ-p. They were instructed to fill out the PCS in relation to any pain related to pregnancy that they were currently in. The instructions for the EPDS were that the participant was to answer based on how she had been feeling in the last seven days. The meeting lasted approximately 15 minutes. The participant had indicated in their screening questionnaire their expected date of delivery and agreed to be contacted for a second Zoom meeting after their delivery. At 4-6 weeks postpartum, the participants were contacted for a follow-up. One more participant was lost at this point. The remaining 32 participants met on Zoom to fill out the EPDS again, as well as the VAS based on their current perception of pain and their pain at the time of delivery. This meeting lasted approximately 5-10 minutes. The following demographic information was also collected from participants at the second meeting: Type of delivery, prior level of activity before birth, number of prior births, whether they prepared for labor in any way, and whether they were administered pain medication. Data from all questionnaires were transferred into a numerical response in an excel document.

Originally, we planned on excluding participants if they were not originally scheduled for induction or cesarean-section but were ultimately induced into labor, if their contractions were induced, or if they had to have a cesarean section, since that could affect the results in how much pain they experience during birth. However, we decided to change this because after starting the study, a large portion of our participants were either induced or received a cesarean section. We still excluded participants if they had a scheduled induction or cesarean, but included those who had a birth with this type of intervention at the second meeting. After data

collection, since there was such a discrepancy in the type of delivery and other demographic information that was collected, it was decided that a secondary exploratory analysis should be completed examining the difference in pain and psychosocial variables amongst the induced, non-induced, and Cesarean section groups. We decided to also do a secondary exploratory analysis for the activity level groups, the prior births groups, the labor preparation groups, and the analgesic medication groups. This was only a secondary analysis because some of these subcategories had very few participants, and not all the statistical assumptions were met with a small number of subjects.

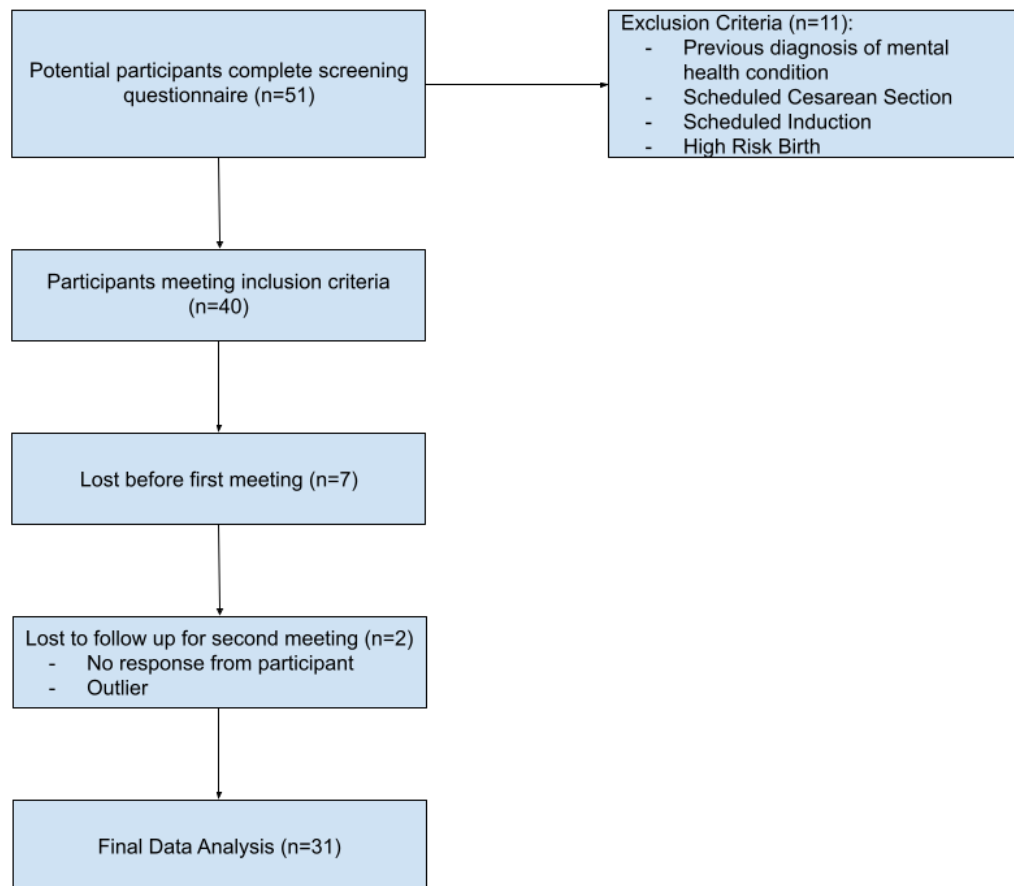


Figure 3-1. Flow chart indicating process for screening, eligibility, and inclusion of participants

3.4 Statistical Analysis

Thirty-two participants completed the study and their data were initially analyzed, however it was visually noted after examining the scatter plots that there may have been an outlier. The Z score was calculated for this data point for the PCS and the EPDS2. The mean PCS score with the potential outlier for the 32 participants was 5.81 ± 6.463 , and the data point for the outlier PCS was 33, giving a Z score of 4.2. Since this is higher than 3, this was considered to be an outlier point. The original mean EPDS2 score was 7.03 ± 4.068 and the outlier data point was 21, giving a Z score of 3.43. Once again, this was higher than 3, and confirmed that this participant was an outlier. The final data set was analyzed with 31 participants. IBM SPSS Statistics version 29.0.2.0 was used for the data analysis. The primary objective was to determine if there was a correlation between the PCS, LPRAQ-p, EPDS, and VAS. For the secondary exploratory analysis, multiple one-way ANOVAs were carried out to identify differences in the questionnaire responses amongst the delivery type, prior births, prior level of activity, labor preparation, and pain medication groups. A p-value <0.05 was considered statistically significant.

3.5 Results

| | |
|-----------------------------|--|
| Age | Mean = 32.9 ± 3.4 yrs |
| Type of Delivery | Not Induced=7 Induced=17 C-Section=7 |
| Prior Activity Level | NA = 3 SA = 9 MA = 11 VA = 8 |
| Prior Births | 0 = 19 |

| | |
|----------------------------------|----------------------|
| | 1+ = 12 |
| Labor Preparation | No = 7 Yes = 24 |
| Pain Medication | No = 5 Yes = 26 |
| Mean Questionnaire Scores | PCS = 4.9 ± 4.2 |
| | LPRAQ-p = 12.8 ± 3.7 |
| | EPDS1 = 5.6 ± 3.0 |
| | VAS1 = 0.8 ± 1.4 |
| | VAS2 = 7.1 ± 2.9 |
| | EPDS2 = 6.6 ± 3.2 |

Table 1. Participant demographic information and descriptive statistics for questionnaires for 31 participants. Age, type of delivery (regular, induced, cesarean section), prior activity level (not active, somewhat active, moderately active, very active), prior number of births, labor preparation, and use of analgesic medication during labor was recorded. PCS and LPRAQ-p were measured in prenatal women. EPDS was measured before (EPDS1) and after delivery (EPDS2). VAS for pain was measured during (VAS2) and after delivery (VAS1).

3.5.1 Results of hypotheses testing

The results indicated that the PCS was not correlated with the VAS during labor (VAS2) ($r=-0.187$, $p=0.313$), the VAS postpartum (VAS1) ($r=0.181$, $p=0.331$), or the LPRAQ-p ($r=-0.160$, $p=0.390$). The LPRAQ-p was additionally not correlated with the VAS during labor ($r=0.119$, $p=0.525$), the VAS postpartum ($r=-0.135$, $p=0.470$), or with the EPDS postpartum ($r=-0.070$, $p=0.708$). See Table 2 below for all correlation values.

| | PCS | LPRAQ-p | EPDS1 | VAS1 | VAS2 | EPDS2 |
|---------|-----|----------------------|----------------------|----------------------|-----------------------|----------------------|
| PCS | 1 | r=-0.160; p=0.390 | r=0.159; p= 0.394 | r=0.181; p=0.331 | r=-0.187; p= 0.313 | r=-0.027; p=0.887 |
| LPRAQ-p | | 1 | r=0.043; p=0.818 | r=-0.135; p=0.470 | r=0.119; p=0.525 | r=-0.070; p=0.708 |
| EPDS1 | | | 1 | r=0.474; p=0.007* | r=0.112; p=0.548 | r=0.520; p=0.003* |
| VAS1 | | | | 1 | r=-0.174; p=0.348 | r=0.165; p=0.375 |
| VAS2 | | | | | 1 | r=0.190; p=0.305 |

Table 2. Correlations amongst Questionnaires. *r*=Pearson correlation, *p*-value was considered statistically significant below 0.05. *indicates a significant correlation between EPDS1 and VAS1, as well as between EPDS1 and EPDS2.

There were some significant correlations that were not a part of the a priori hypotheses.

For example, the mean prenatal EPDS scores (5.6 ± 3.0 , N=31) were correlated with postpartum pain measured on the VAS ($0.8 + 1.4$, N=31, $p=0.007$). The other significant correlation was between the prenatal EPDS and the postpartum EPDS ($6.6 + 3.2$, N=31, $p=0.003$).

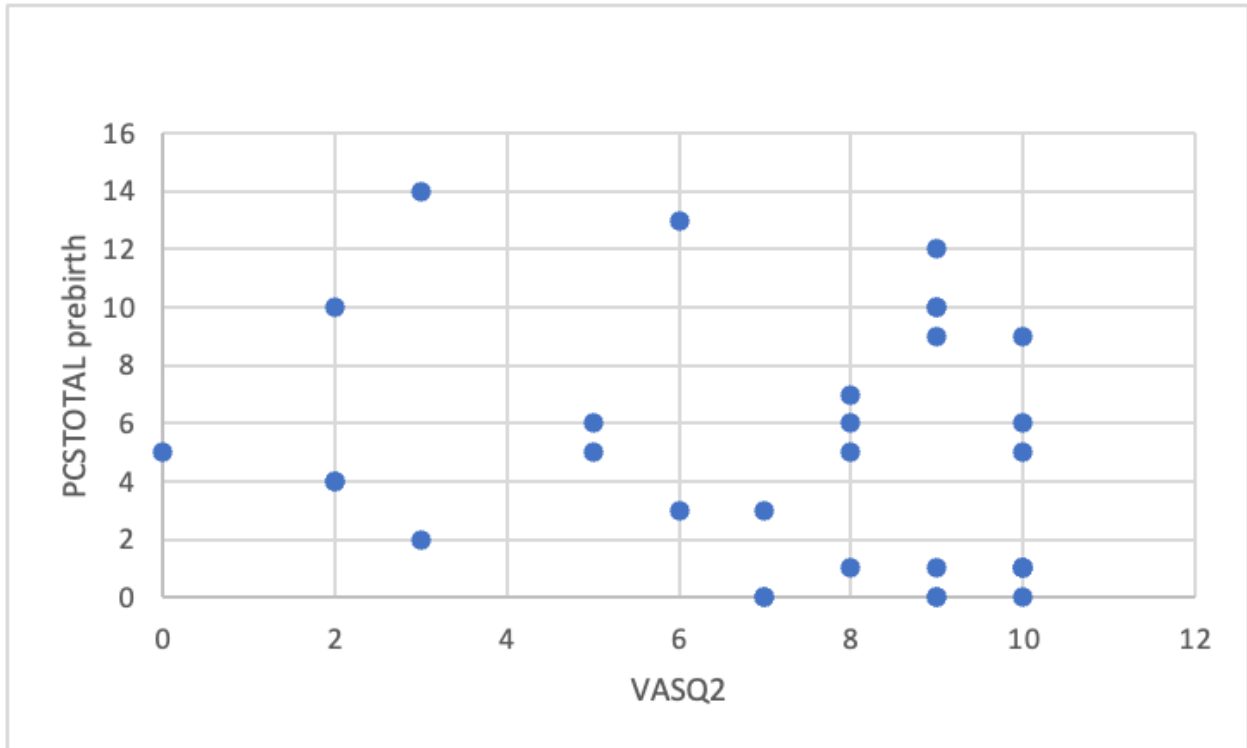


Figure 3-2. Scatter plot for 31 participants showing correlation between PCS mean scores and pain during labor on the VAS.

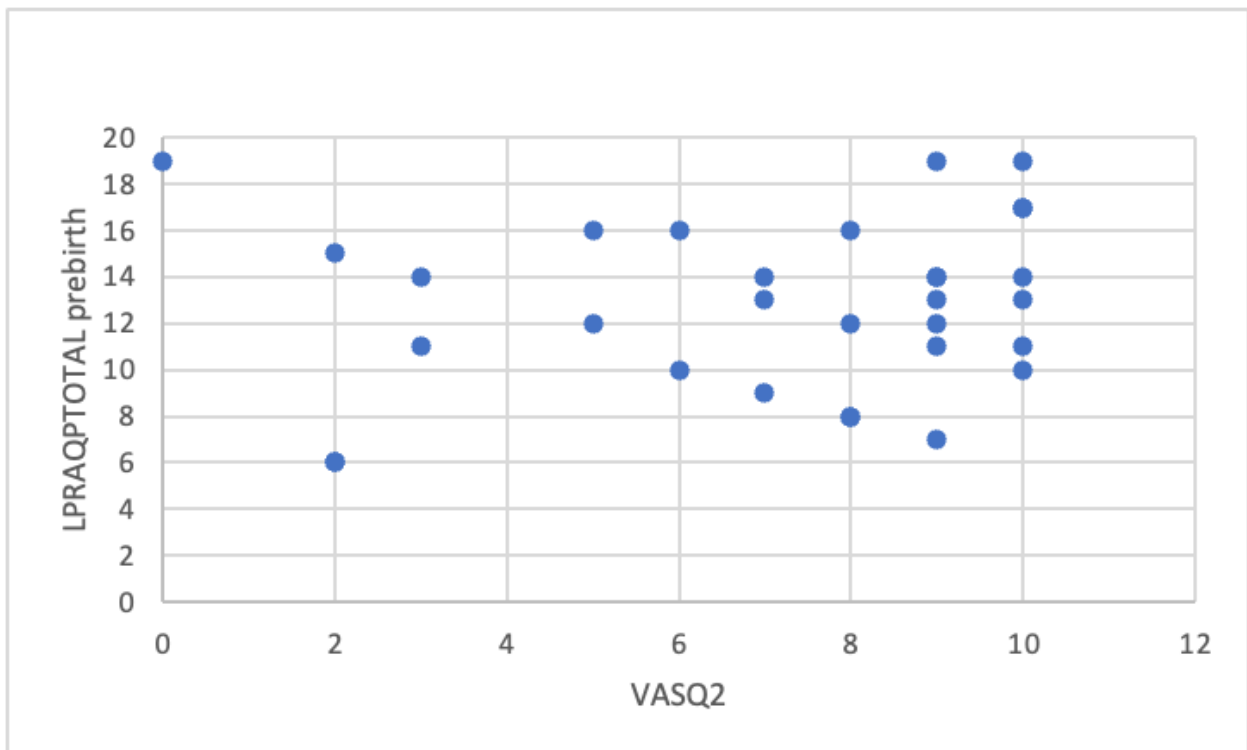


Figure 3-3. Scatter plot for 31 participants showing correlation between LPRAQ-p mean scores and pain during labor on the VAS.

As a secondary analysis, multiple one-way ANOVAs were carried out to identify differences amongst the delivery type, labor preparation, prior activity level, prior birth, and pain medication groups.

The first one-way ANOVA was used to determine the differences in the PCS, LPRAQ-p, EPDS, and VAS between participants who were not induced (n=7), were induced (n=17), or who had a cesarean section (n=7). The results of the one-way ANOVA determined that there was a difference in at least one of the delivery type groups. Since the equal variance assumption was met and the sample sizes of the groups were very different, the post-hoc test chosen was Hochberg's GT2. The post-hoc test showed that there was a significant difference amongst the mean PCS scores in the non-induced group (8.0, N=7) and the cesarean section group (2.6, N=7); ($F_{2,28}=3.496$, $p=0.044$). This appears to be an incidental finding, as it is unlikely that having lower pain catastrophizing could be associated with an emergency event requiring surgery for delivery. No other findings were significant amongst these groups.

| | | N | Mean | Std. Deviation |
|---------|---------------|----|------|----------------|
| PCS | 1 - Regular | 7 | 8.0* | 2.7 |
| | 2 - Induced | 17 | 4.7 | 4.7 |
| | 3 - C-Section | 7 | 2.6 | 2.4 |
| | Total | 31 | 4.9 | 4.2 |
| LPRAQ-p | 1 - Regular | 7 | 11.7 | 3.2 |
| | 2 - Induced | 17 | 13.1 | 3.6 |
| | 3 - C-Section | 7 | 13.0 | 4.5 |
| | Total | 31 | 12.8 | 3.7 |

| | | | | |
|-------|---------------|----|-----|-----|
| EPDS1 | 1 - Regular | 7 | 5.3 | 4.2 |
| | 2 - Induced | 17 | 5.2 | 2.8 |
| | 3 - C-Section | 7 | 6.7 | 2.0 |
| | Total | 31 | 5.6 | 3.0 |
| VASQ1 | 1 - Regular | 7 | .4 | .8 |
| | 2 - Induced | 17 | .8 | 1.6 |
| | 3 - C-Section | 7 | 1.0 | 1.4 |
| | Total | 31 | .8 | 1.4 |
| VASQ2 | 1 - Regular | 7 | 7.9 | 2.0 |
| | 2 - Induced | 17 | 7.0 | 3.3 |
| | 3 - C-Section | 7 | 6.6 | 3.2 |
| | Total | 31 | 7.1 | 2.9 |
| EPDS2 | 1 - Regular | 7 | 5.1 | 3.4 |
| | 2 - Induced | 17 | 6.7 | 3.3 |
| | 3 - C-Section | 7 | 7.9 | 2.7 |
| | Total | 31 | 6.6 | 3.2 |

Table 3. Mean questionnaire scores for labor type: regular group (1), induced group (2) and Cesarean-section group (3). PCS and LPRAQ-p were measured in prenatal women. EPDS was measured before (EPDS1) and after delivery (EPDS2). VAS for pain was measured during (VAS2) and after delivery (VAS1) indicates a significantly higher PCS score in the non-induced group compared to the induced and Cesarean group.*

The second one-way ANOVA was done to determine the differences in the questionnaires amongst the various activity level groups (not active, n=3; somewhat active, n=9; moderately active, n=11; very active, n=8). Since the equal variance assumption was met and the sample sizes of the groups were very different, the post-hoc test chosen was Hochberg's GT2. The moderately active and very active had significantly higher pain during labor (VAS2) ($F_{3,27}=4.1$, $p=0.015$). The very active group mean VAS2 score was 8.0 (N=8) and the moderately active group mean score was 7.9 (N=11), compared to the not active group mean of 2.3 (n=3) and the somewhat active group mean of 6.9 (n=9).

| | | N | Mean | Std. Deviation |
|---------|-----------------------|----|------|----------------|
| PCS | 0 - Not active | 3 | 4.7 | .6 |
| | 1 - Somewhat Active | 9 | 5.6 | 4.6 |
| | 2 - Moderately Active | 11 | 5.0 | 4.6 |
| | 3 - Very Active | 8 | 4.3 | 4.6 |
| | Total | 31 | 4.9 | 4.2 |
| LPRAQ-p | 0 - Not active | 3 | 13.7 | 6.8 |
| | 1 - Somewhat Active | 9 | 11.9 | 3.4 |
| | 2 - Moderately Active | 11 | 13.2 | 4.2 |
| | 3 - Very Active | 8 | 12.9 | 2.4 |
| | Total | 31 | 12.8 | 3.7 |
| EPDS1 | 0 - Not active | 3 | 5.0 | 1.7 |
| | 1 - Somewhat Active | 9 | 5.1 | 3.6 |
| | 2 - Moderately Active | 11 | 5.6 | 2.7 |
| | 3 - Very Active | 8 | 6.4 | 3.2 |
| | Total | 31 | 5.6 | 3.0 |
| VASQ1 | 0 - Not active | 3 | .3 | .6 |
| | 1 - Somewhat Active | 9 | .6 | 1.3 |
| | 2 - Moderately Active | 11 | 1.2 | 1.8 |
| | 3 - Very Active | 8 | .6 | 1.1 |
| | Total | 31 | .8 | 1.4 |
| VASQ2 | 0 - Not active | 3 | 2.3 | 2.5 |
| | 1 - Somewhat Active | 9 | 6.9 | 2.5 |
| | 2 - Moderately Active | 11 | 7.9* | 2.5 |
| | 3 - Very Active | 8 | 8.0* | 2.7 |
| | Total | 31 | 7.1 | 2.9 |
| EPDS2 | 0 - Not active | 3 | 4.7 | 3.2 |
| | 1 - Somewhat Active | 9 | 5.9 | 3.4 |
| | 2 - Moderately Active | 11 | 6.4 | 2.2 |
| | 3 - Very Active | 8 | 8.4 | 3.9 |

Total 31 6.6 3.2

*Table 4. Mean questionnaire scores for prior activity groups: not active (0), somewhat active (1), moderately active (2) and very active (3). PCS and LPRAQ-p were measured in prenatal women. EPDS was measured before (EPDS1) and after delivery (EPDS2). VAS for pain was measured during (VAS2) and after delivery (VAS1). * indicates a significantly higher VAS2 score for the moderately active and the very active groups, compared to the not active and the somewhat active groups.*

The third one-way ANOVA was done to determine the differences in the questionnaires amongst the nulliparous (n=19) and multiparous (n=12) groups. There was a statistically significant difference in the EPDS amongst the multiparous and nulliparous groups ($F_{1,29}=7.737$, $p=0.009$). The EPDS2 mean scores for the nulliparous group were 7.7 (N=19) and were 4.8 (N=12) for the multiparous.

| | | N | Mean | Std. Deviation |
|---------|-----------------|----|------|----------------|
| PCS | 0 - Nulliparous | 19 | 4.9 | 4.7 |
| | 1 - Multiparous | 12 | 5.0 | 3.5 |
| | Total | 31 | 4.9 | 4.2 |
| LPRAQ-p | 0 - Nulliparous | 19 | 12.6 | 3.6 |
| | 1 - Multiparous | 12 | 13.0 | 4.0 |
| | Total | 31 | 12.8 | 3.7 |
| EPDS1 | 0 - Nulliparous | 19 | 6.0 | 2.9 |
| | 1 - Multiparous | 12 | 4.9 | 3.0 |
| | Total | 31 | 5.6 | 3.0 |
| VASQ1 | 0 - Nulliparous | 19 | 1.1 | 1.6 |
| | 1 - Multiparous | 12 | .3 | .7 |
| | Total | 31 | .8 | 1.3 |
| VASQ2 | 0 - Nulliparous | 19 | 7.6 | 2.6 |
| | 1 - Multiparous | 12 | 6.2 | 3.4 |
| | Total | 31 | 7.1 | 2.9 |
| EPDS2 | 0 - Nulliparous | 19 | 7.7* | 3.1 |
| | 1 - Multiparous | 12 | 4.8* | 2.7 |
| | Total | 31 | 6.6 | 3.2 |

Table 5. Mean questionnaire scores for prior births: nulliparous =0, multiparous =1. PCS and LPRAQ-p were measured in prenatal women. EPDS was measured before (EPDS1) and after delivery (EPDS2). VAS for pain was measured during (VAS2) and after delivery (VAS1). * indicates a significant difference in the EPDS2 scores amongst nulliparous and multiparous women.

The fourth one-way ANOVA was done to determine the differences in the questionnaires amongst the group that prepared for labor (n=24) and the group that did not (n=7). The mean LPRAQ-p scores were significantly higher in the no labor preparation group compared to the labor preparation group ($F_{1,29}=5.375$, $p=0.028$). Mean LPRAQ-p in the no preparation group was 15.4 (N=7) and for the preparation group was 12.0 (N=24).

| | | N | Mean | Std. Deviation |
|---------|--------------------|----|-------|----------------|
| PCS | 0 - No preparation | 7 | 6.7 | 3.5 |
| | 1 - Preparation | 24 | 4.4 | 4.3 |
| | Total | 31 | 4.9 | 4.2 |
| LPRAQ-p | 0 - No preparation | 7 | 15.4* | 2.8 |
| | 1 - Preparation | 24 | 12.0 | 3.6 |
| | Total | 31 | 12.8 | 3.7 |
| EPDS1 | 0 - No preparation | 7 | 5.6 | 3.3 |
| | 1 - Preparation | 24 | 5.6 | 2.9 |
| | Total | 31 | 5.6 | 3.0 |
| VASQ1 | 0 - No preparation | 7 | .4 | .8 |
| | 1 - Preparation | 24 | .9 | 1.5 |
| | Total | 31 | .8 | 1.4 |
| VASQ2 | 0 - No preparation | 7 | 6.1 | 3.9 |
| | 1 - Preparation | 24 | 7.4 | 2.7 |
| | Total | 31 | 7.1 | 2.9 |
| EPDS2 | 0 - No preparation | 7 | 5.4 | 2.3 |
| | 1 - Preparation | 24 | 6.9 | 3.4 |
| | Total | 31 | 6.6 | 3.2 |

Table 6. Mean questionnaire scores for labor preparation: no preparation (0), preparation (1). PCS and LPRAQ-p were measured in prenatal women. EPDS was measured before (EPDS1) and after delivery (EPDS2). VAS for pain was measured during (VAS2) and after delivery (VAS1). * indicates a significantly higher LPRAQ-p in the no preparation group compared to the preparation group.

The final one-way ANOVA was done to determine the differences in the questionnaires amongst the group that received analgesic medication (n=26) and the group that did not (n=5). There was a statistical difference between the LPRAQ-p in the group that received analgesic medication and the one that did not. The mean LPRAQ-p score in the pain medication group was 13.4 ± 3.6 (N=26) and was 9.80 ± 3.033 in the no pain medication group (N=5). These were statistically different according to a one-way ANOVA ($F_{1,29}=4.312$, $p=0.047$).

| | | N | Mean | Std. Deviation |
|---------|-------------------|----|-------|----------------|
| PCS | 0 - No medication | 5 | 8.2 | 2.8 |
| | 1 - Medication | 26 | 4.3 | 4.2 |
| | Total | 31 | 4.9 | 4.2 |
| LPRAQ-p | 0 - No medication | 5 | 9.8* | 3.0 |
| | 1 - Medication | 26 | 13.4* | 3.6 |
| | Total | 31 | 12.8 | 3.7 |
| EPDS1 | 0 - No medication | 5 | 5.4 | 3.7 |
| | 1 - Medication | 26 | 5.6 | 2.9 |
| | Total | 31 | 5.6 | 3.0 |
| VASQ1 | 0 - No medication | 5 | .4 | .9 |
| | 1 - Medication | 26 | .9 | 1.5 |
| | Total | 31 | .8 | 1.4 |
| VASQ2 | 0 - No medication | 5 | 8.8 | .8 |
| | 1 - Medication | 26 | 6.8 | 3.1 |
| | Total | 31 | 7.1 | 2.9 |
| EPDS2 | 0 - No medication | 5 | 6.0 | 3.2 |
| | 1 - Medication | 26 | 6.7 | 3.3 |
| | Total | 31 | 6.6 | 3.2 |

Table 7. Mean questionnaire scores for analgesic medication groups: no medication (0); medication (1). PCS and LPRAQ-p were measured in prenatal women. EPDS was measured before (EPDS1) and after

*delivery (EPDS2). VAS for pain was measured during (VAS2) and after delivery (VAS1). * indicates significantly different scores on the LPRAQ-p amongst the women receiving analgesic medication and those who did not.*

3.6 Discussion

None of our three hypotheses were supported in this study. Our hypotheses were: (1) catastrophizing will be associated with more pain, and a higher score on the LPRAQ-p scale during pregnancy would predict a high amount of pain felt during delivery; (2) the PCS scale and the LPRAQ-p scale are correlated; (3) the LPRAQ-p questionnaire and the EPDS are correlated. As stated in the results, PCS was not correlated with the VAS or the LPRAQ-p. In addition, the LPRAQ-p was not correlated to the EPDS or to the VAS.

Pain catastrophizing was not related to labor pain or postpartum pain in this study. Despite PCS scores being extremely low in our study, pain during labor was still quite high, with a mean of 7.1 on the VAS. This is interesting because normally PCS scores are associated with higher pain (Sullivan et al., 1995; Weissman-Fogel et al., 2008), but this was not the case here. It may be worth investigating that PCS scores may be different in a pregnant population than a non-pregnant population. It has been suggested that pregnancy associated pain may be different from traditional pain, because there is a known end-date to the condition and results in becoming a mother (Olsson et al., 2012). One recent study showed that PCS scores in a pregnant population were lower than a non-pregnant, nulliparous population (Bartholomew et al., 2024; Clark et al., 2022). Catastrophizing in pregnancy may be influenced by fear of giving birth, worry about the baby, and these thoughts may be altered depending on prenatal care, such as an appointment with a doctor (Olsson et al., 2012). One study measuring associations between PCS and lumbopelvic pain determined that PCS scores can fluctuate over time if taken at different stages of pregnancy and postpartum, with as many as 1 in 3 women having significantly different PCS results (Olsson et al., 2012). It is possible that this fluctuation in catastrophizing is associated with different events occurring during pregnancy, such as a

concern about the fetus, and this catastrophizing may decrease if the worries are put to rest after an ultrasound confirming that everything is fine (Olsson et al., 2012). There have been some previous studies done measuring catastrophizing as a predictor for labor pain, but they had different study designs than ours, and this may be a reason for the differences in our results compared to previous research. One previous study measuring the association between the PCS and labor pain instructed participants to answer the questions of the PCS in relation to how they thought their pain would be during labor (before entering labor), not in relation to pain they were actually in (Flink et al., 2009). The Flink et al. study also did not exclude women with prior history of mental illness, such as depression or anxiety. They did determine that pain catastrophizing about labor was associated with higher pain during birth (Flink et al., 2009). Our study asked women to answer the PCS in relation to pain that they were currently in related to pregnancy, though being in pain at the time of the first meeting was not a requirement. The fact that the measurement was not related to thoughts about labor pain and that our study excluded women with a history of mental illness may explain why the PCS scores for this group of 31 women is extremely low with a mean of 4.9. Another study was done measuring PCS while women were in active labor, before receiving analgesia (Ferber et al., 2005). They showed that PCS scores were correlated to pain during labor and two days postpartum measured on the VAS (Ferber et al., 2005). It can be assumed that women would be in pain while in active labor prior to analgesia, once again demonstrating a very different study design than ours. The PCS was also not correlated with the LPRAQ-p in our study. It is possible that this is due in part to the two scales measuring different constructs. The PCS has three components: rumination, magnification, and helplessness (Sullivan et al., 1995). The LPRAQ-p has two subcategories: women's perception and social environment (Hulsbosch et al., 2020). The two questionnaires are measuring different fundamental concepts, perhaps indicating why they were not correlated in our study.

In this present study, the LPRAQ-p was not correlated to the EPDS in either the prenatal period or the postpartum period. In the pilot study done during the validation of the LPRAQ-p, the EPDS was correlated with the LPRAQ-p, but it was noted that the Tilburg Pregnancy Distress Scale (TPDS) was a better predictor for requesting epidural analgesia than the EPDS (Hulsbosch et al., 2020). The TPDS measures symptoms of worrying about pregnancy and delivery (Hulsbosch et al., 2020). When a multiple linear regression was done including pregnancy distress symptoms, depression symptoms were not associated with increased demand for epidural analgesia (Hulsbosch et al., 2020). The EPDS was administered at 32 weeks of gestation and was not repeated in the postpartum period as it was in our study (Hulsbosch et al., 2020). This aforementioned study is the only other study done comparing the EPDS to the LPRAQ-p.

In our study, PCS scores were not correlated with EPDS scores, however this is aligned with other research. In a previous study measuring the association between PCS scores and probable postpartum depression, high PCS ≥ 25 was not associated with probable postpartum depression (Zeng et al., 2020). There was a link found between these two variables when high PCS scores were also associated with breakthrough pain during the epidural analgesia as well as a low body mass index (BMI) (Zeng et al., 2020). The mean PCS scores in our study was very low (4.94, n=31). The Zeng et al. comparable study divided the participants into a group with PCS score below 25 and greater than or equal to 25 (Zeng et al., 2020). There was no exclusion of women with prior mental health problems from participating, which may have contributed to having a group with such high scores ≥ 25 (Zeng et al., 2020). Nonetheless, when simply comparing the PCS ≥ 25 to the EPDS, there was no association, which is consistent with our results. To our knowledge, the study by Zeng et al. is the only other study comparing these two questionnaires to each other.

There were some notable results amongst the secondary analyzed data that may be of interest. The postpartum EPDS scores were higher in the no prior birth group compared to the

multiparous group. This is consistent with prior studies. A study looking to identify risk factors in postpartum depression identified that primiparous women had a significantly higher risk for postpartum depression, with higher EPDS scores (Dubey et al., 2021). Potential postpartum depression in primiparous women is supported by another study which showed that 1503 primiparous mothers had significantly higher self-reported postpartum depressive symptoms than their 1487 multiparous counterparts (Martínez-Galiano et al., 2019). The Martínez-Galiano et al. study did not however use the EPDS to identify postpartum depression. Multiparous mothers report less discomfort and have more experience with a newborn, which may contribute to lower rates of postpartum depression (Dubey et al., 2021; Martínez-Galiano et al., 2019). Something else of interest is that the LPRAQ-p scores were significantly higher in the group that received analgesia and the group that did not. The result of higher LPRAQ-p scores in the analgesia group aligns well with what the author had stated in the pilot study for the development of the LPRAQ-p (Hulsbosch et al., 2020). Currently, our study is the first study that has used the LPRAQ-p in this way since its validation. The only other study to use the LPRAQ-p to date measured if there was an association between the LPRAQ-p and intention to receive medication, but not whether it was ultimately administered (Kuipers & Van Beeck, 2022).

Another interesting finding was that the mean LPRAQ-p scores were higher in the no labor preparation group compared to the labor preparation group. While the no preparation group only had seven subjects, this is still of interest as it could indicate that preparing for labor may result in less requests for analgesic medication. Two previous studies showed that prenatal mindfulness and hypnosis did not impact the actual use of epidural analgesia (Duncan et al., 2017; Madden et al., 2016). The Duncan et al. and Madden et al. studies did not take the LPRAQ-p into consideration. It should be noted that there are two subscales in the LPRAQ-p: women's perception and social environment (Hulsbosch et al., 2020). It is possible that the reason that previous studies analyzing labor preparation on epidural use focused too much on

the women's perception and not enough on the social environment. It could be interesting to use the LPRAQ-p and then separate women into the two subscale groups and provide labor preparation intended towards the subscales and see if there would then be a difference in the use of analgesic medication during labor.

There were some other less notable significant findings upon secondary analysis. The prenatal EPDS was correlated with the postpartum pain measured on the VAS. The mean postpartum pain score was however quite low, measuring 0.8/10. This is of low clinical relevance as the VAS measures pain from 0-10, so being able to predict a negligible amount of pain is not of great value to a clinician. The prenatal EPDS and the postpartum EPDS were also correlated in this study, which is consistent with previous research (Hung et al., 2014; Silva et al., 2018). Labor pain in our study was higher in the very active and moderately active groups. This is not consistent with other studies (Carrascosa et al., 2021; Ghandali et al., 2021). Physical activity during pregnancy can contribute to decreased time in active labor and vaginal deliveries with less interventions, which should contribute to less pain (Haakstad & Bø, 2020). It is possible that the sample size amongst the different activity level groups were too small to accurately predict if there would be an effect on pain.

3.7 Limitations

One possible limitation to consider was the timing of the measurement of pain with the VAS. The labor pain was measured as a delayed recall during the second meeting. The fact that women had to recall pain that may have occurred over a month ago may have led to an inaccurate representation of their pain. We do feel as though this impact was minimal since the pain measured was still high, but it should be noted nonetheless. Furthermore, it was not specified whether the measurement of labor pain be in relation to pain before or after potentially receiving analgesia. The mean VAS2 scores for the group that received analgesia was 6.8 and

was 8.8 for the group not receiving analgesia. It is possible that there would have been a higher pain experience for the analgesic medication group if it would have been specified to state the level of pain prior to administration of analgesic drugs.

Another limitation with our study was the fact that the PCS, LPRAQ-p, and prenatal EPDS was taken at any time during the third trimester, which could have impacted the results, instead of having a fixed recording week of gestation.

3.8 Conclusion

The three original hypotheses of this study were not supported by the results. Catastrophizing was not associated with pain, the LPRAQ-p, or potential postpartum depression. It is possible that the PCS may not be the best tool for pregnancy as previously thought, potentially because women do not view pregnancy as an injury and know the end date. The PCS and LPRAQ-p are also different constructs. The EPDS was also not associated with the LPRAQ-p. The EPDS may not be the optimal tool to use in the prenatal period to determine potential use of epidural analgesia, or pain experience during labor. A secondary analysis showed that higher scores on the LPRAQ-p were associated with use of epidural analgesia, and that EPDS were higher amongst nulliparous than multiparous women.

Future studies with larger sample sizes would be helpful to continue research in this area. It would also be helpful to evaluate differences amongst nulliparous and multiparous women, as well as women receiving epidural analgesia and those who do not.

Chapter 4: General Discussion

This thesis explored two topics that were of great interest to me personally. Having worked with high level athletes for several years, I have witnessed the amount of medication that athletes take prophylactically and when injured. What has always been so striking to me is the lack of knowledge of what they are choosing to put into their bodies, and the ignorance regarding possible side effects to these medications. I found the topic of our systematic review interesting because of the knowledge I could bring to my athletes. The results of the review were even more interesting: that topical medications were more effective than oral when compared to a placebo. Being able to help my athletes make more informed choices of what to use for their injuries was very rewarding for me.

The second part of this thesis was exploring a topic that is my other greatest passion: women's health. I have been treating women throughout their pregnancies and in the postpartum period for the better part of the last decade. There is one recurrent theme among the majority of the women I treat in this population: lack of education at various stages of the prenatal and postpartum period, and lack of resources if there is a problem. This encouraged me to do research in this area, with hopes of being able to find links between maternal outcomes to better serve women before a problem arises.

This thesis was not met without obstacles. The definition of an athlete was a major challenge that we discovered while writing the systematic review. Many articles would state that they were evaluating pain measurements for athletic injuries, but the research would be carried out on a non-athletic population. Other studies would state that the participants were athletes,

but really they were just active people. We truly wanted to conduct the meta-analysis on an exclusive athlete population, since athletes have many differences from the regular population.

With regards to the maternal outcomes portion, we faced a different set of challenges. We originally wanted to have the least amount of confounding variables possible, but we quickly realized that that would be difficult. The prenatal and postpartum period can vary so much from woman to woman, and truly no two people have an identical birth story. It would be extremely hard to have a more uniform population when discussing childbirth. The labor experience can vary so much: from a vaginal and unassisted birth, induction, and cesarean section, to differently analgesic medications being used. Even if two women had a similar labor experience, their postpartum care can vary so much - whether they have support at home from family members or others, whether they are breastfeeding or not, if they are able to remain on maternity leave at a full salary, to name a few differences. This period is inherently different for different women, so even if there would have been a more uniform study population, perhaps the results would not have been able to be extrapolated to a larger population. Ultimately, we conducted the analyses with all the different types of participants, and there are still interesting findings that came out of this. For a medical practitioner involved in the prenatal care of women, knowing that the catastrophizing may not impact their pain, but that the LPRAQ-p is a high predictor for use of epidural, are some examples of ways in which we can help women when they are feeling uninformed and scared.

Both of these topics, while seemingly different to most people, helped me tremendously in my personal practice, both as an Athletic Therapist and an Osteopath. I am grateful to have been supported and had the opportunity to take part in this research.

Declarations

Ethics Approval, Consent to participate, and dissemination

The proposed project was approved by the Research Ethics Unit of Concordia University in Montreal, Quebec.

Participants were allocated an individual trial number, which was used to code the data and maintain confidentiality of patient information. Written informed consent to participate in the study was obtained from each participant. The findings from this study were disseminated in a variety of ways, including abstract, posters, publications in peer-reviewed journals and presentations at conferences.

Consent for publication

Written informed consent to publish the data of this study was obtained from each participant.

Competing Interests

The authors declare that they have no competing interests

Funding

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Availability of Data and Materials

Not Applicable

Author's Contributions

Conception and design: SN, GD
Drafting the manuscript: SN, GD
Critical revision of the manuscript
Final Approval of the manuscript: SN, GD

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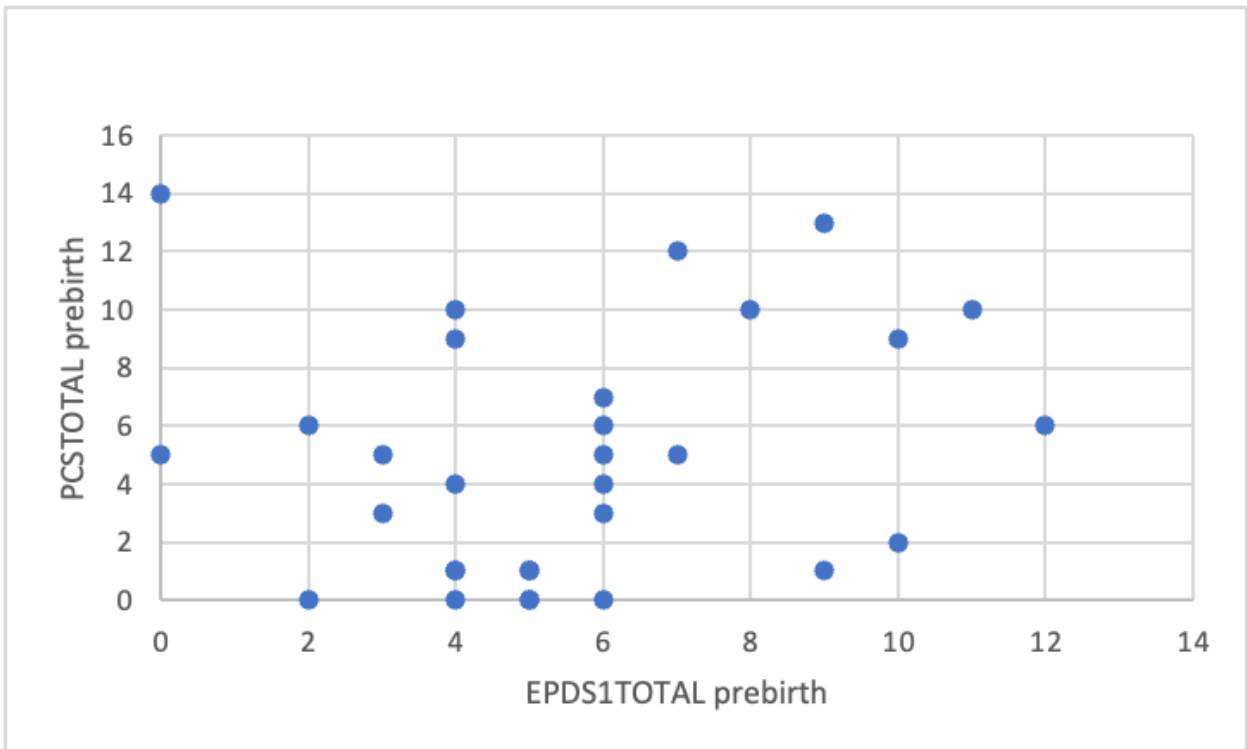
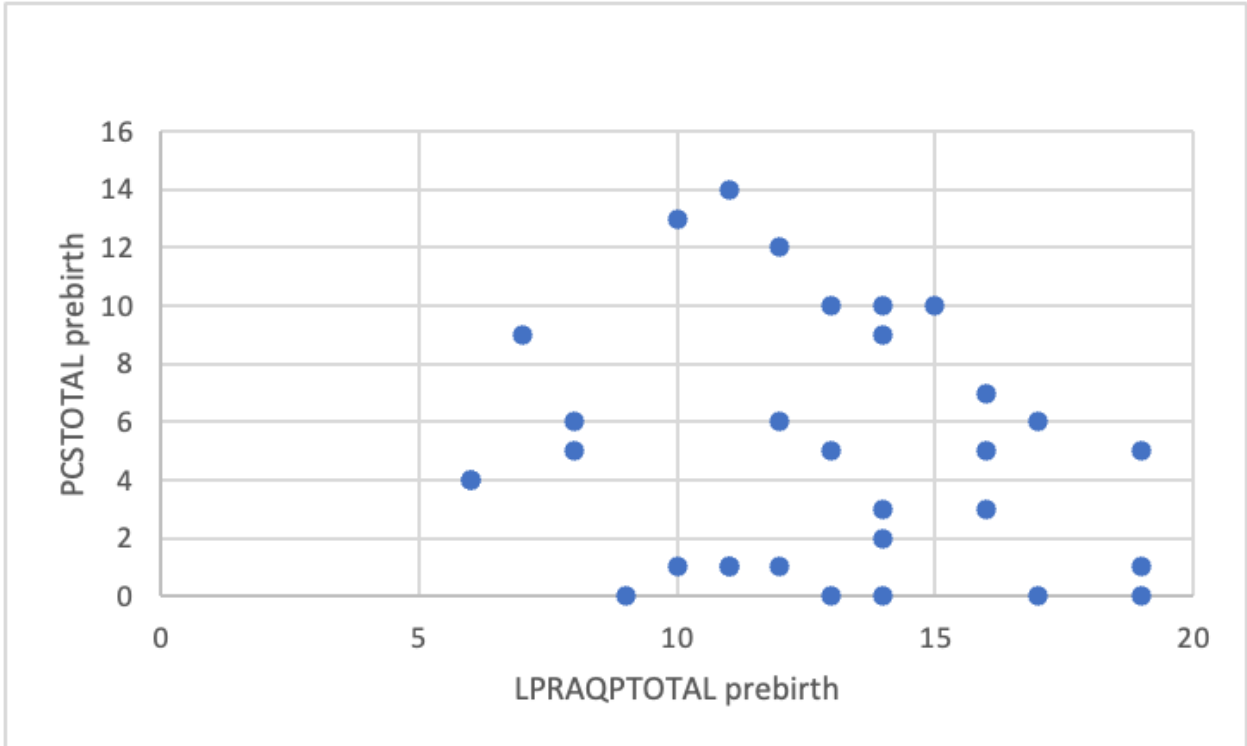
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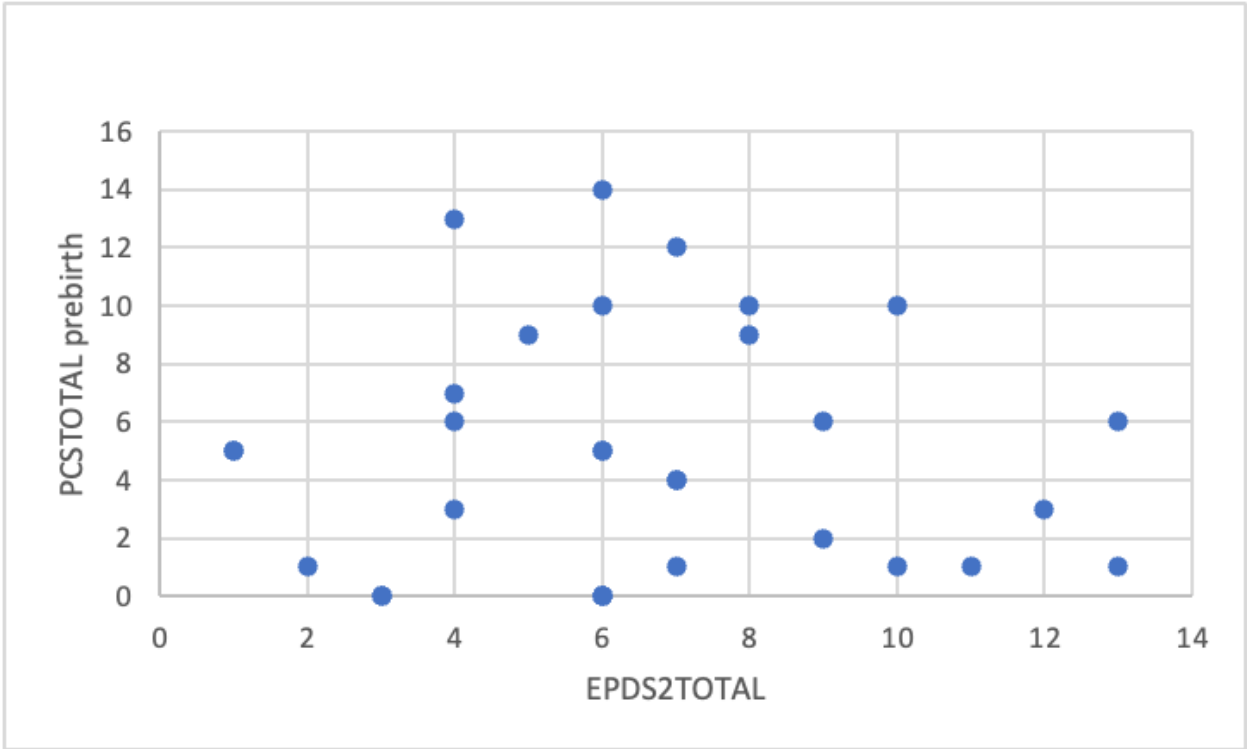
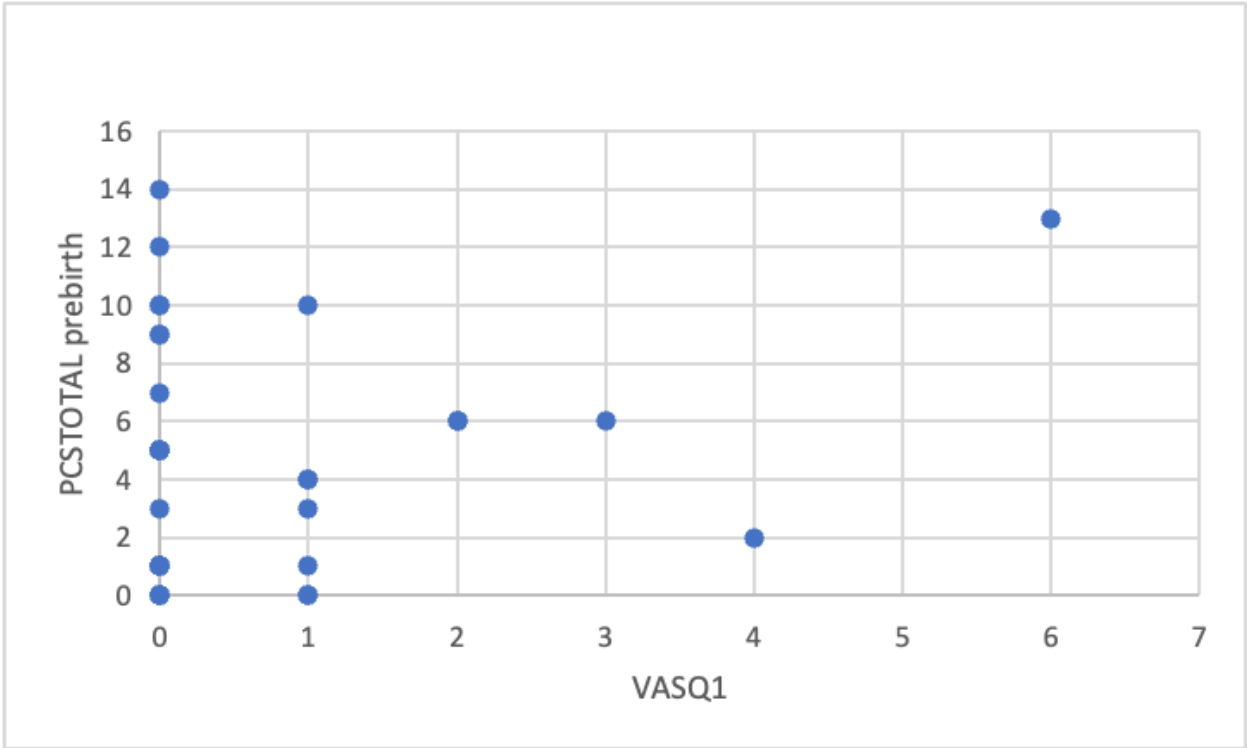
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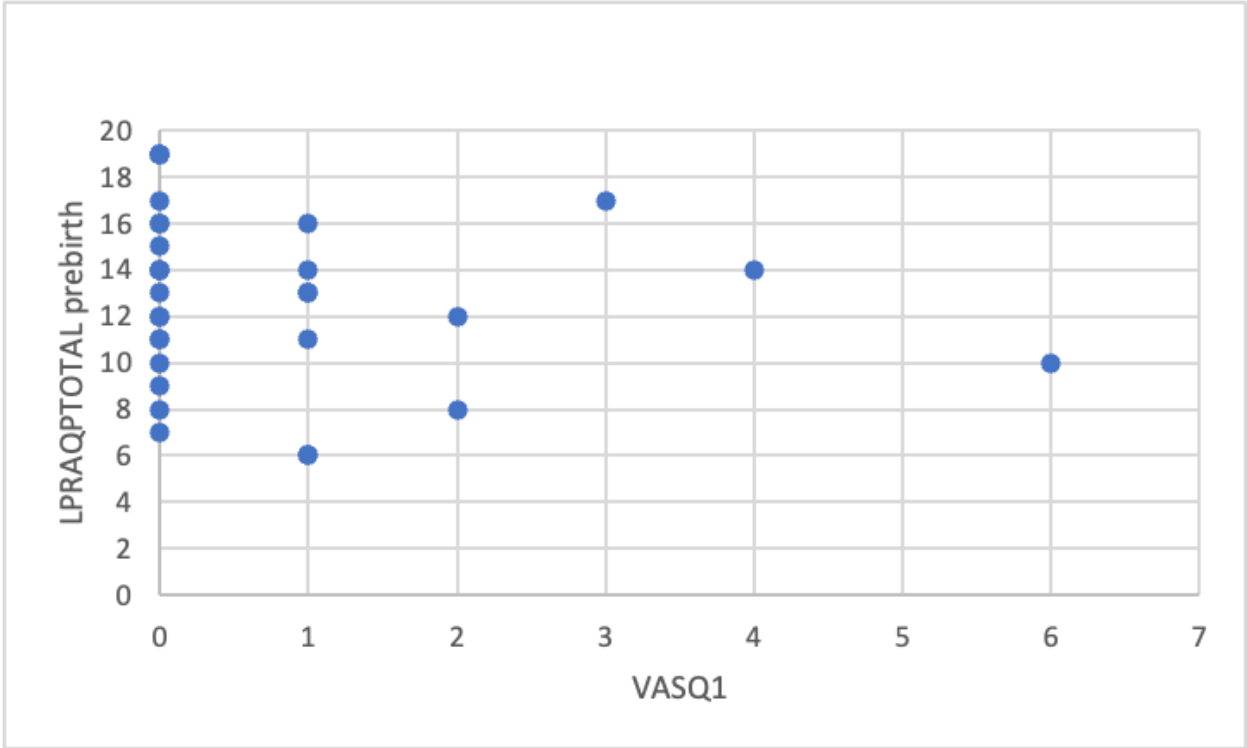
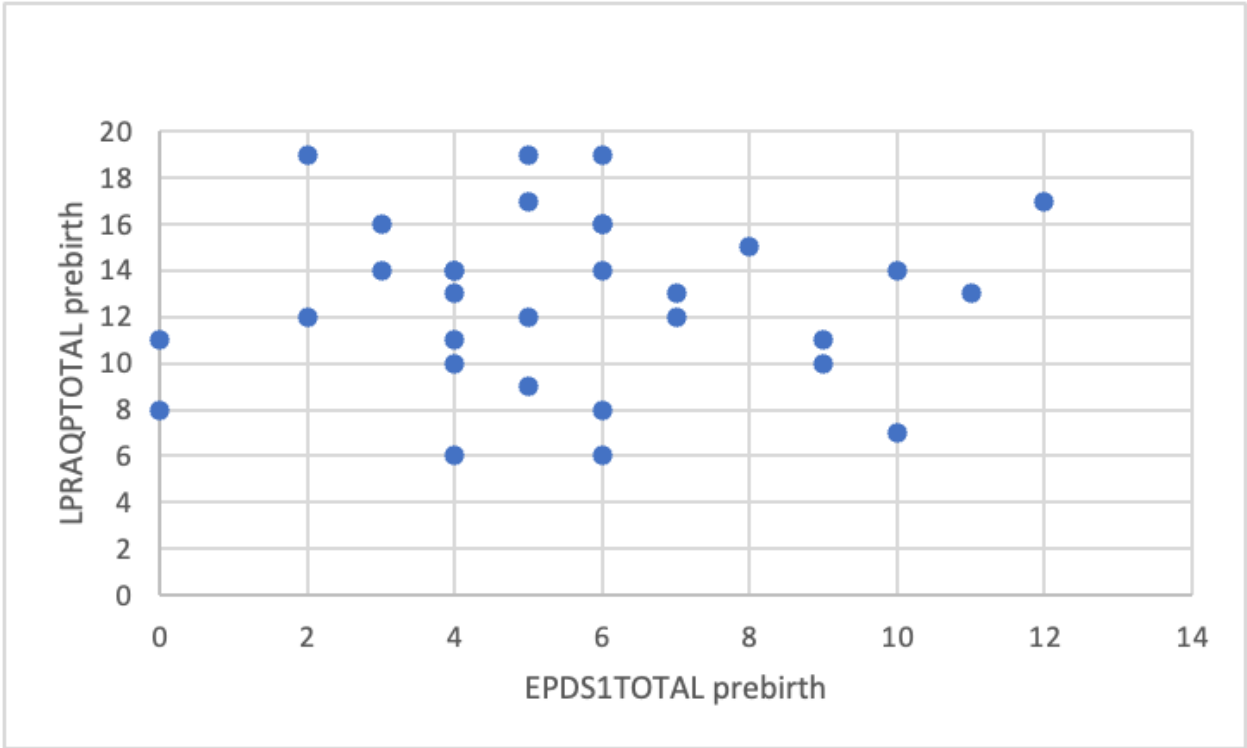
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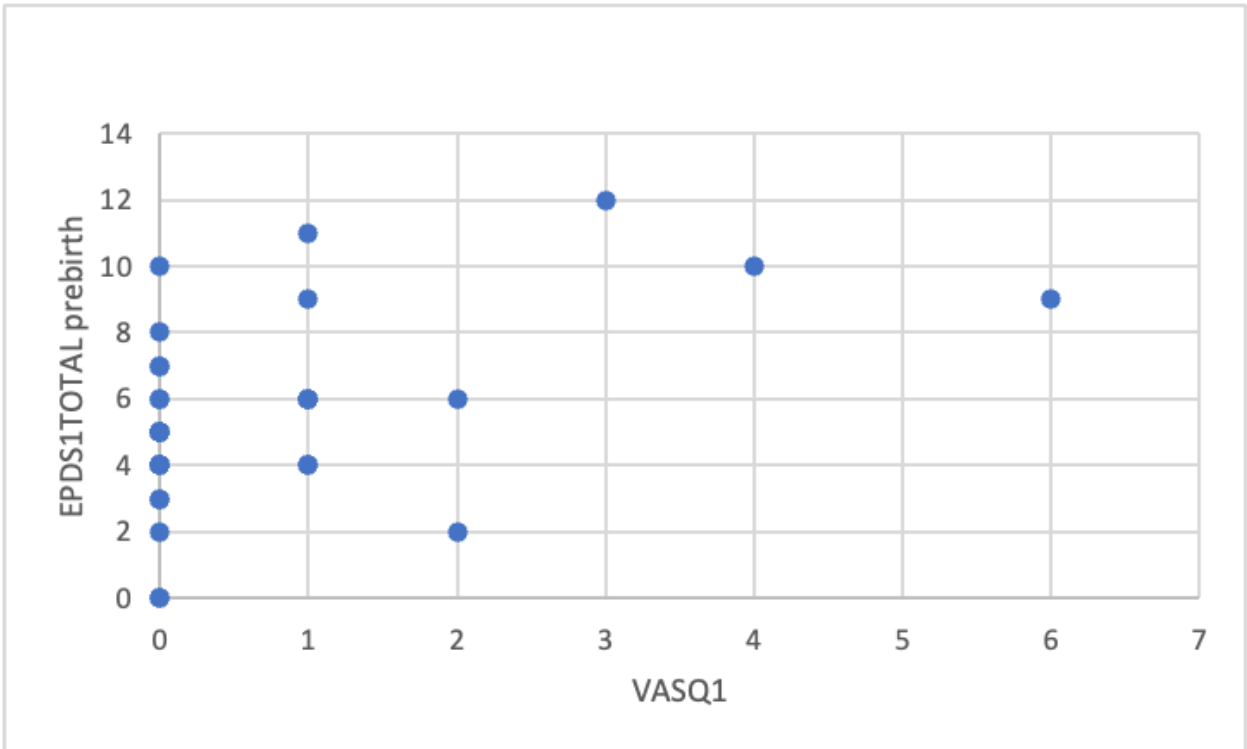
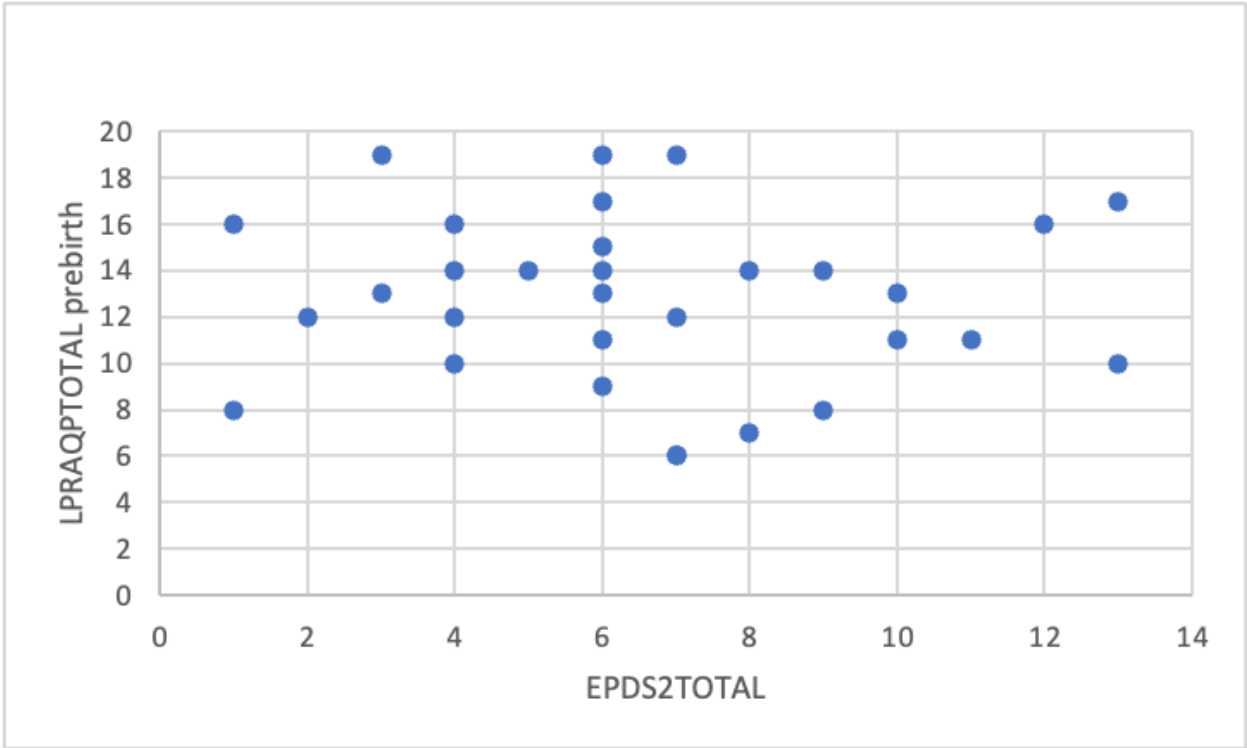
Appendix

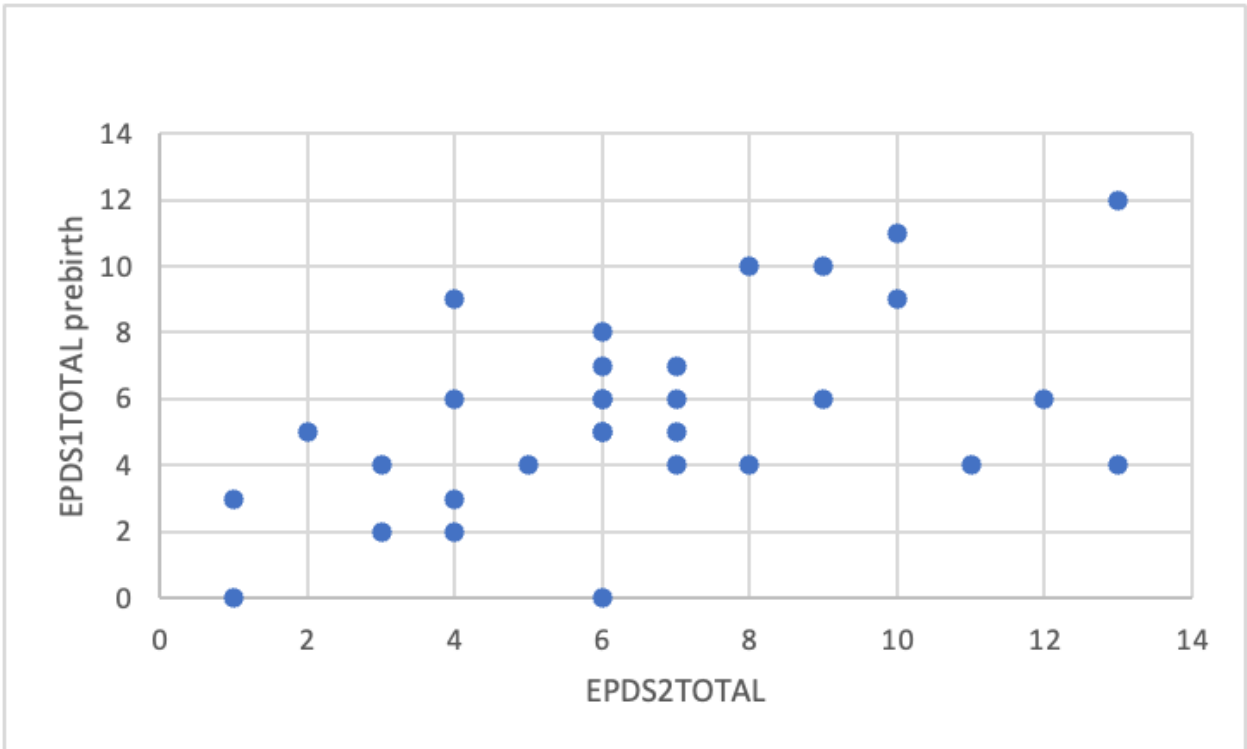
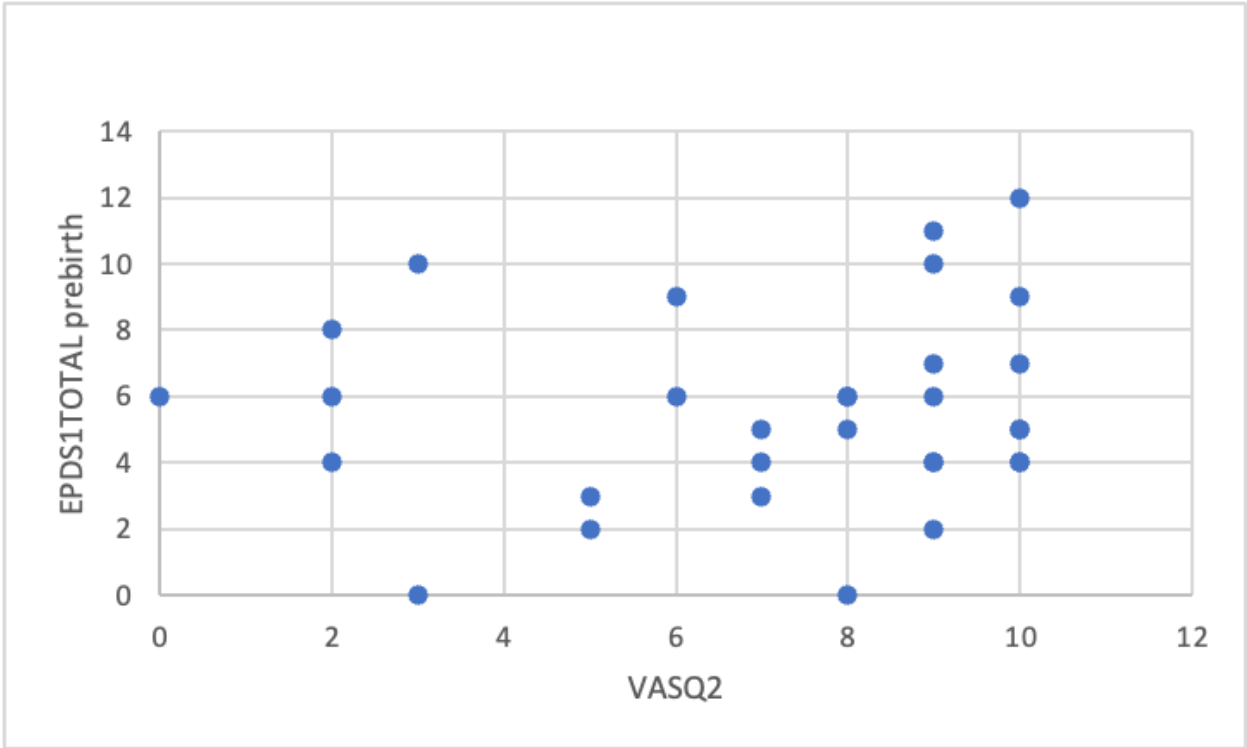
Scatter Plots

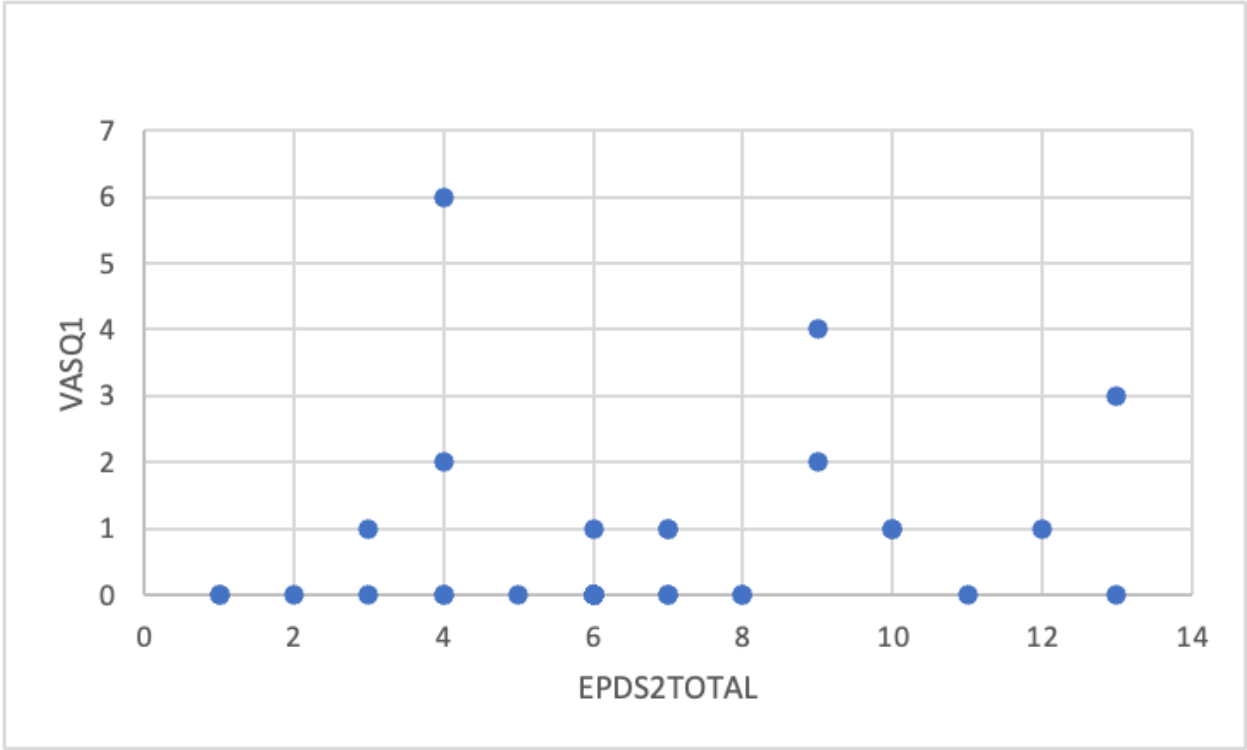
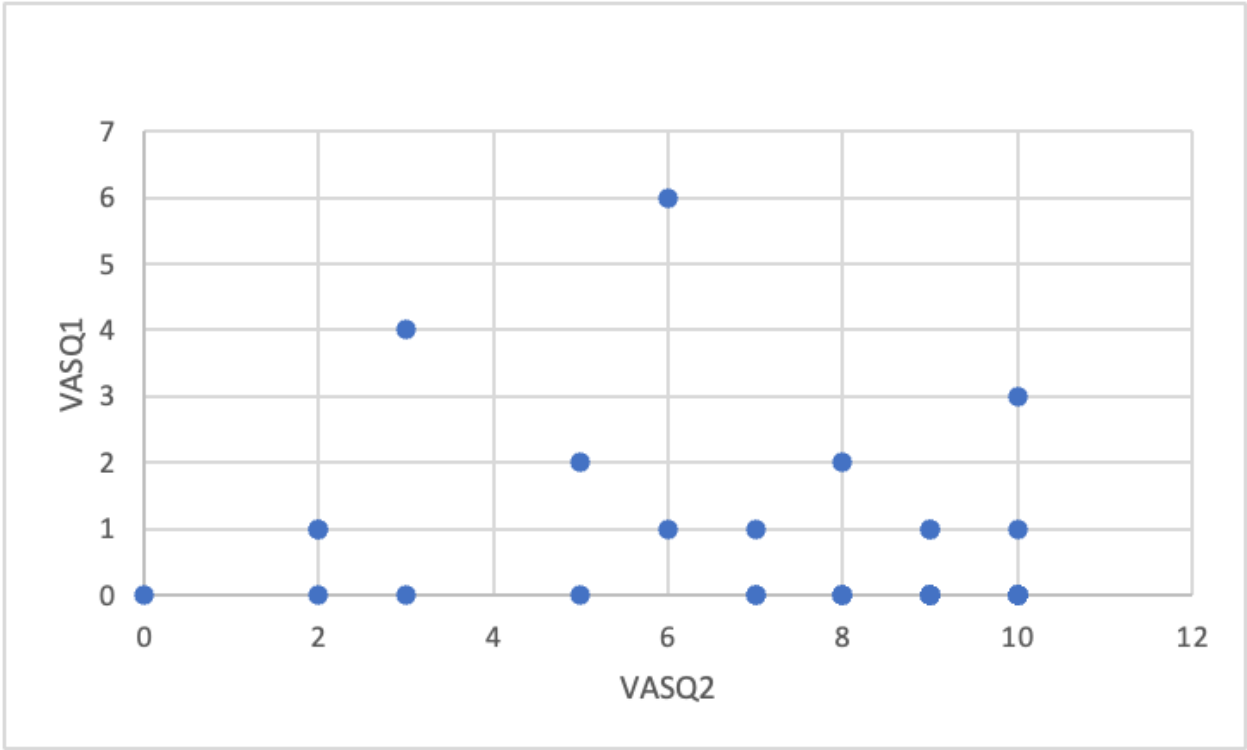


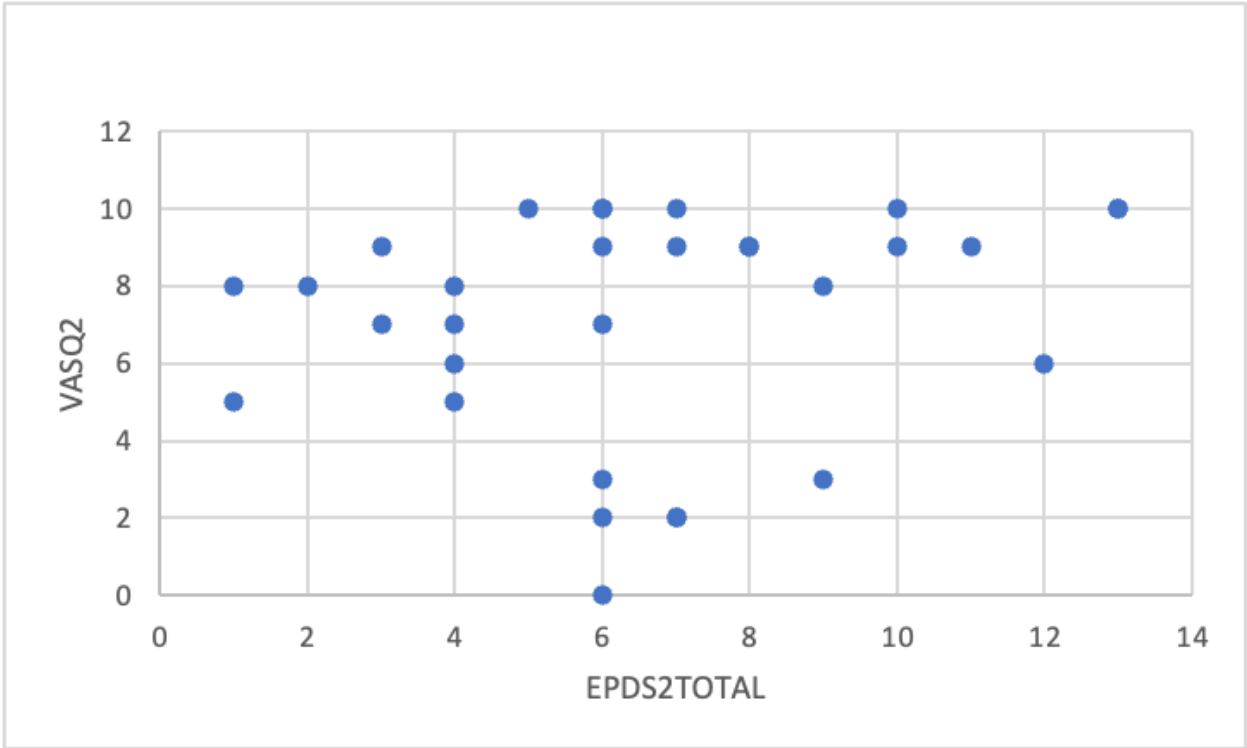












SPSS Analysis

| | Pearson Correlation | Sig. (2-tailed) | 95% Confidence Interval | |
|-----------------|------------------------|-----------------|-------------------------|-------|
| | | | Lower | Upper |
| PCS - LPRAQ-p | -.160 | .390 | -.487 | .206 |
| PCS - EPDS1 | .159 | .394 | -.207 | .486 |
| PCS - VASQ1 | .181 | .331 | -.186 | .503 |
| PCS - VASQ2 | -.187 | .313 | -.508 | .179 |
| PCS - EPDS2 | -.027 | .887 | -.377 | .331 |
| LPRAQ-p - EPDS1 | .043 | .818 | -.316 | .391 |
| LPRAQ-p - VASQ1 | -.135 | .470 | -.467 | .231 |
| LPRAQ-p - VASQ2 | .119 | .525 | -.246 | .454 |
| LPRAQ-p - EPDS2 | -.070 | .708 | -.414 | .292 |
| EPDS1 - VASQ1 | .474 | .007 | .144 | .709 |
| EPDS1 - VASQ2 | .112 | .548 | -.252 | .449 |
| EPDS1 - EPDS2 | .520 | .003 | .204 | .739 |
| VASQ1 - VASQ2 | -.174 | .348 | -.498 | .192 |
| VASQ1 - EPDS2 | .165 | .375 | -.201 | .491 |
| VASQ2 - EPDS2 | .190 | .305 | -.176 | .510 |

Confidence Intervals for Correlations

| | | N | Mean | Std. Deviation | Std. Error | 95% Confidence Interval for Mean | | Minimum | Maximum |
|---------|-------|----|-------|----------------|------------|----------------------------------|-------------|---------|---------|
| | | | | | | Lower Bound | Upper Bound | | |
| PCS | 1 | 7 | 8.00 | 2.708 | 1.024 | 5.50 | 10.50 | 5 | 12 |
| | 2 | 17 | 4.65 | 4.663 | 1.131 | 2.25 | 7.04 | 0 | 14 |
| | 3 | 7 | 2.57 | 2.370 | .896 | .38 | 4.76 | 0 | 7 |
| | Total | 31 | 4.94 | 4.211 | .756 | 3.39 | 6.48 | 0 | 14 |
| LPRAQ-p | 1 | 7 | 11.71 | 3.200 | 1.209 | 8.76 | 14.67 | 7 | 16 |
| | 2 | 17 | 13.12 | 3.638 | .882 | 11.25 | 14.99 | 6 | 19 |
| | 3 | 7 | 13.00 | 4.546 | 1.718 | 8.80 | 17.20 | 6 | 19 |
| | Total | 31 | 12.77 | 3.685 | .662 | 11.42 | 14.13 | 6 | 19 |
| EPDS1 | 1 | 7 | 5.29 | 4.152 | 1.569 | 1.45 | 9.13 | 0 | 11 |
| | 2 | 17 | 5.24 | 2.773 | .673 | 3.81 | 6.66 | 0 | 12 |
| | 3 | 7 | 6.71 | 1.976 | .747 | 4.89 | 8.54 | 5 | 10 |
| | Total | 31 | 5.58 | 2.953 | .530 | 4.50 | 6.66 | 0 | 12 |
| VASQ1 | 1 | 7 | .43 | .787 | .297 | -.30 | 1.16 | 0 | 2 |

| | | | | | | | | | |
|-------|-------|----|------|-------|-------|------|-------|---|----|
| | 2 | 17 | .82 | 1.590 | .386 | .01 | 1.64 | 0 | 6 |
| | 3 | 7 | 1.00 | 1.414 | .535 | -.31 | 2.31 | 0 | 4 |
| | Total | 31 | .77 | 1.383 | .248 | .27 | 1.28 | 0 | 6 |
| VASQ2 | 1 | 7 | 7.86 | 2.035 | .769 | 5.97 | 9.74 | 5 | 10 |
| | 2 | 17 | 7.00 | 3.260 | .791 | 5.32 | 8.68 | 0 | 10 |
| | 3 | 7 | 6.57 | 3.155 | 1.192 | 3.65 | 9.49 | 2 | 10 |
| | Total | 31 | 7.10 | 2.948 | .529 | 6.02 | 8.18 | 0 | 10 |
| EPDS2 | 1 | 7 | 5.14 | 3.436 | 1.299 | 1.96 | 8.32 | 1 | 10 |
| | 2 | 17 | 6.65 | 3.278 | .795 | 4.96 | 8.33 | 2 | 13 |
| | 3 | 7 | 7.86 | 2.673 | 1.010 | 5.39 | 10.33 | 4 | 12 |
| | Total | 31 | 6.58 | 3.223 | .579 | 5.40 | 7.76 | 1 | 13 |

Descriptive Statistics - Pregnancy Type Variable

| | | Sum of Squares | df | Mean Square | F | Sig. |
|---------|----------------|----------------|----|-------------|-------|------|
| PCS | Between Groups | 106.274 | 2 | 53.137 | 3.496 | .044 |
| | Within Groups | 425.597 | 28 | 15.200 | | |
| | Total | 531.871 | 30 | | | |
| LPRAQ-p | Between Groups | 10.226 | 2 | 5.113 | .360 | .701 |
| | Within Groups | 397.193 | 28 | 14.185 | | |
| | Total | 407.419 | 30 | | | |
| EPDS1 | Between Groups | 11.632 | 2 | 5.816 | .652 | .529 |
| | Within Groups | 249.916 | 28 | 8.926 | | |
| | Total | 261.548 | 30 | | | |
| VASQ1 | Between Groups | 1.234 | 2 | .617 | .308 | .738 |
| | Within Groups | 56.185 | 28 | 2.007 | | |
| | Total | 57.419 | 30 | | | |
| VASQ2 | Between Groups | 6.138 | 2 | 3.069 | .338 | .716 |
| | Within Groups | 254.571 | 28 | 9.092 | | |
| | Total | 260.710 | 30 | | | |
| EPDS2 | Between Groups | 25.952 | 2 | 12.976 | 1.272 | .296 |
| | Within Groups | 285.597 | 28 | 10.200 | | |
| | Total | 311.548 | 30 | | | |
| | Total | 311.548 | 30 | | | |

One-Way Anova - Pregnancy Type Variable

| | | Levene Statistic | df1 | df2 | Sig. |
|---------|--|---------------------|-----|--------|------|
| PCS | Based on Mean | 2.524 | 2 | 28 | .098 |
| | Based on Median | 2.237 | 2 | 28 | .126 |
| | Based on Median and with adjusted df | 2.237 | 2 | 23.755 | .129 |
| | Based on trimmed mean | 2.436 | 2 | 28 | .106 |
| LPRAQ-p | Based on Mean | .721 | 2 | 28 | .495 |
| | Based on Median | .572 | 2 | 28 | .571 |
| | Based on Median and with adjusted df | .572 | 2 | 27.022 | .571 |
| | Based on trimmed mean | .716 | 2 | 28 | .498 |
| EPDS1 | Based on Mean | 2.575 | 2 | 28 | .094 |
| | Based on Median | 1.847 | 2 | 28 | .176 |
| | Based on Median and with adjusted df | 1.847 | 2 | 24.838 | .179 |
| | Based on trimmed mean | 2.576 | 2 | 28 | .094 |
| VASQ1 | Based on Mean | .506 | 2 | 28 | .609 |
| | Based on Median | .245 | 2 | 28 | .784 |
| | Based on Median and with adjusted df | .245 | 2 | 23.158 | .785 |
| | Based on trimmed mean | .356 | 2 | 28 | .704 |
| VASQ2 | Based on Mean | .837 | 2 | 28 | .443 |
| | Based on Median | .581 | 2 | 28 | .566 |
| | Based on Median and with adjusted df | .581 | 2 | 26.459 | .566 |

| | | | | | |
|-------|--------------------------------------|------|---|--------|------|
| | Based on trimmed mean | .761 | 2 | 28 | .477 |
| EPDS2 | Based on Mean | .193 | 2 | 28 | .825 |
| | Based on Median | .197 | 2 | 28 | .822 |
| | Based on Median and with adjusted df | .197 | 2 | 26.374 | .822 |
| | Based on trimmed mean | .184 | 2 | 28 | .833 |

Homogeneity of Variances - Pregnancy Type Variable

| Dependent Variable | (I) Pregnancy Type variable | (J) Pregnancy Type variable | Mean Difference (I-J) | Std. Error | Sig. | 95% Confidence Interval | |
|--------------------|-----------------------------|-----------------------------|-----------------------|------------|-------|-------------------------|-------------|
| | | | | | | Lower Bound | Upper Bound |
| PCS | 1 | 2 | 3.353 | 1.751 | .181 | -1.08 | 7.79 |
| | | 3 | 5.429 | 2.084 | .042 | .15 | 10.71 |
| | 2 | 1 | -3.353 | 1.751 | .181 | -7.79 | 1.08 |
| | | 3 | 2.076 | 1.751 | .562 | -2.36 | 6.51 |
| | 3 | 1 | -5.429 | 2.084 | .042 | -10.71 | -.15 |
| | | 2 | -2.076 | 1.751 | .562 | -6.51 | 2.36 |
| LPRAQ-p | 1 | 2 | -1.403 | 1.691 | .792 | -5.69 | 2.88 |
| | | 3 | -1.286 | 2.013 | .891 | -6.38 | 3.81 |
| | 2 | 1 | 1.403 | 1.691 | .792 | -2.88 | 5.69 |
| | | 3 | .118 | 1.691 | 1.000 | -4.17 | 4.40 |
| | 3 | 1 | 1.286 | 2.013 | .891 | -3.81 | 6.38 |
| | | 2 | -.118 | 1.691 | 1.000 | -4.40 | 4.17 |
| EPDS1 | 1 | 2 | .050 | 1.342 | 1.000 | -3.35 | 3.45 |
| | | 3 | -1.429 | 1.597 | .753 | -5.47 | 2.62 |
| | 2 | 1 | -.050 | 1.342 | 1.000 | -3.45 | 3.35 |
| | | 3 | -1.479 | 1.342 | .617 | -4.88 | 1.92 |
| | 3 | 1 | 1.429 | 1.597 | .753 | -2.62 | 5.47 |
| | | 2 | 1.479 | 1.342 | .617 | -1.92 | 4.88 |
| VASQ1 | 1 | 2 | -.395 | .636 | .898 | -2.01 | 1.22 |
| | | 3 | -.571 | .757 | .834 | -2.49 | 1.35 |
| | 2 | 1 | .395 | .636 | .898 | -1.22 | 2.01 |
| | | 3 | -.176 | .636 | .989 | -1.79 | 1.43 |
| | 3 | 1 | .571 | .757 | .834 | -1.35 | 2.49 |
| | | 2 | .176 | .636 | .989 | -1.43 | 1.79 |
| VASQ2 | 1 | 2 | .857 | 1.354 | .893 | -2.57 | 4.29 |
| | | 3 | 1.286 | 1.612 | .810 | -2.80 | 5.37 |
| | 2 | 1 | -.857 | 1.354 | .893 | -4.29 | 2.57 |
| | | 3 | .429 | 1.354 | .984 | -3.00 | 3.86 |
| | 3 | 1 | -1.286 | 1.612 | .810 | -5.37 | 2.80 |

| | | | | | | | |
|-------|---|---|--------|-------|------|-------|------|
| EPDS2 | 1 | 2 | -429 | 1.354 | .984 | -3.86 | 3.00 |
| | | 2 | -1.504 | 1.434 | .653 | -5.14 | 2.13 |
| | | 3 | -2.714 | 1.707 | .319 | -7.04 | 1.61 |
| | 2 | 1 | 1.504 | 1.434 | .653 | -2.13 | 5.14 |
| | | 3 | -1.210 | 1.434 | .783 | -4.84 | 2.42 |
| | 3 | 1 | 2.714 | 1.707 | .319 | -1.61 | 7.04 |
| | | 2 | 1.210 | 1.434 | .783 | -2.42 | 4.84 |

Multiple Comparisons - Hochberg's GT2 Post Hoc Test - Pregnancy Type Variable

| | | N | Mean | Std. Deviation | Std. Error | 95% Confidence Interval for Mean | | Minimum | Maximum |
|---------|-------|----|-------|----------------|------------|----------------------------------|-------------|---------|---------|
| | | | | | | Lower Bound | Upper Bound | | |
| PCS | 0 | 7 | 6.71 | 3.546 | 1.340 | 3.44 | 9.99 | 0 | 10 |
| | 1 | 24 | 4.42 | 4.313 | .880 | 2.60 | 6.24 | 0 | 14 |
| | Total | 31 | 4.94 | 4.211 | .756 | 3.39 | 6.48 | 0 | 14 |
| LPRAQ-p | 0 | 7 | 15.43 | 2.760 | 1.043 | 12.88 | 17.98 | 12 | 19 |
| | 1 | 24 | 12.00 | 3.600 | .735 | 10.48 | 13.52 | 6 | 19 |
| | Total | 31 | 12.77 | 3.685 | .662 | 11.42 | 14.13 | 6 | 19 |
| EPDS1 | 0 | 7 | 5.57 | 3.259 | 1.232 | 2.56 | 8.59 | 2 | 11 |
| | 1 | 24 | 5.58 | 2.933 | .599 | 4.34 | 6.82 | 0 | 12 |
| | Total | 31 | 5.58 | 2.953 | .530 | 4.50 | 6.66 | 0 | 12 |
| VASQ1 | 0 | 7 | .43 | .787 | .297 | -.30 | 1.16 | 0 | 2 |
| | 1 | 24 | .88 | 1.513 | .309 | .24 | 1.51 | 0 | 6 |
| | Total | 31 | .77 | 1.383 | .248 | .27 | 1.28 | 0 | 6 |
| VASQ2 | 0 | 7 | 6.14 | 3.891 | 1.471 | 2.54 | 9.74 | 0 | 10 |
| | 1 | 24 | 7.38 | 2.651 | .541 | 6.26 | 8.49 | 2 | 10 |
| | Total | 31 | 7.10 | 2.948 | .529 | 6.02 | 8.18 | 0 | 10 |
| EPDS2 | 0 | 7 | 5.43 | 2.299 | .869 | 3.30 | 7.55 | 3 | 10 |
| | 1 | 24 | 6.92 | 3.412 | .697 | 5.48 | 8.36 | 1 | 13 |
| | Total | 31 | 6.58 | 3.223 | .579 | 5.40 | 7.76 | 1 | 13 |

Descriptives Statistics - Labor Preparation Variable

| | | Sum of Squares | df | Mean Square | F | Sig. |
|---------|----------------|----------------|----|-------------|-------|------|
| PCS | Between Groups | 28.609 | 1 | 28.609 | 1.649 | .209 |
| | Within Groups | 503.262 | 29 | 17.354 | | |
| | Total | 531.871 | 30 | | | |
| LPRAQ-p | Between Groups | 63.705 | 1 | 63.705 | 5.375 | .028 |
| | Within Groups | 343.714 | 29 | 11.852 | | |
| | Total | 407.419 | 30 | | | |
| EPDS1 | Between Groups | .001 | 1 | .001 | .000 | .993 |
| | Within Groups | 261.548 | 29 | 9.019 | | |
| | Total | 261.548 | 30 | | | |
| VASQ1 | Between Groups | 1.080 | 1 | 1.080 | .556 | .462 |
| | Within Groups | 56.339 | 29 | 1.943 | | |
| | Total | 57.419 | 30 | | | |
| VASQ2 | Between Groups | 8.228 | 1 | 8.228 | .945 | .339 |
| | Within Groups | 252.482 | 29 | 8.706 | | |
| | Total | 260.710 | 30 | | | |
| EPDS2 | Between Groups | 12.001 | 1 | 12.001 | 1.162 | .290 |
| | Within Groups | 299.548 | 29 | 10.329 | | |
| | Total | 311.548 | 30 | | | |

One-Way ANOVA - Labor Preparation Variable

| | | 95% Confidence Interval for Mean | | | | | | | |
|---------|-------|----------------------------------|-------|----------------|------------|-------------|-------------|---------|---------|
| | | N | Mean | Std. Deviation | Std. Error | Lower Bound | Upper Bound | Minimum | Maximum |
| PCS | 0 | 3 | 4.67 | .577 | .333 | 3.23 | 6.10 | 4 | 5 |
| | 1 | 9 | 5.56 | 4.586 | 1.529 | 2.03 | 9.08 | 0 | 14 |
| | 2 | 11 | 5.00 | 4.561 | 1.375 | 1.94 | 8.06 | 0 | 13 |
| | 3 | 8 | 4.25 | 4.590 | 1.623 | .41 | 8.09 | 0 | 12 |
| | Total | 31 | 4.94 | 4.211 | .756 | 3.39 | 6.48 | 0 | 14 |
| LPRAQ-p | 0 | 3 | 13.67 | 6.807 | 3.930 | -3.24 | 30.58 | 6 | 19 |
| | 1 | 9 | 11.89 | 3.371 | 1.124 | 9.30 | 14.48 | 7 | 16 |
| | 2 | 11 | 13.18 | 4.167 | 1.256 | 10.38 | 15.98 | 6 | 19 |
| | 3 | 8 | 12.88 | 2.357 | .833 | 10.90 | 14.85 | 10 | 17 |
| | Total | 31 | 12.77 | 3.685 | .662 | 11.42 | 14.13 | 6 | 19 |
| EPDS1 | 0 | 3 | 5.00 | 1.732 | 1.000 | .70 | 9.30 | 3 | 6 |
| | 1 | 9 | 5.11 | 3.586 | 1.195 | 2.35 | 7.87 | 0 | 10 |
| | 2 | 11 | 5.55 | 2.734 | .824 | 3.71 | 7.38 | 2 | 11 |
| | 3 | 8 | 6.38 | 3.159 | 1.117 | 3.73 | 9.02 | 3 | 12 |
| | Total | 31 | 5.58 | 2.953 | .530 | 4.50 | 6.66 | 0 | 12 |
| VASQ1 | 0 | 3 | .33 | .577 | .333 | -1.10 | 1.77 | 0 | 1 |
| | 1 | 9 | .56 | 1.333 | .444 | -.47 | 1.58 | 0 | 4 |
| | 2 | 11 | 1.18 | 1.779 | .536 | -.01 | 2.38 | 0 | 6 |
| | 3 | 8 | .63 | 1.061 | .375 | -.26 | 1.51 | 0 | 3 |
| | Total | 31 | .77 | 1.383 | .248 | .27 | 1.28 | 0 | 6 |
| VASQ2 | 0 | 3 | 2.33 | 2.517 | 1.453 | -3.92 | 8.58 | 0 | 5 |
| | 1 | 9 | 6.89 | 2.472 | .824 | 4.99 | 8.79 | 3 | 10 |
| | 2 | 11 | 7.91 | 2.548 | .768 | 6.20 | 9.62 | 2 | 10 |
| | 3 | 8 | 8.00 | 2.726 | .964 | 5.72 | 10.28 | 2 | 10 |
| | Total | 31 | 7.10 | 2.948 | .529 | 6.02 | 8.18 | 0 | 10 |
| EPDS2 | 0 | 3 | 4.67 | 3.215 | 1.856 | -3.32 | 12.65 | 1 | 7 |
| | 1 | 9 | 5.89 | 3.444 | 1.148 | 3.24 | 8.54 | 1 | 12 |
| | 2 | 11 | 6.36 | 2.157 | .650 | 4.91 | 7.81 | 3 | 10 |
| | 3 | 8 | 8.38 | 3.926 | 1.388 | 5.09 | 11.66 | 3 | 13 |

| | | | | | | | | |
|-------|----|------|-------|------|------|------|---|----|
| Total | 31 | 6.58 | 3.223 | .579 | 5.40 | 7.76 | 1 | 13 |
|-------|----|------|-------|------|------|------|---|----|

Descriptive Statistics - Prior Activity Level Variable. 0 = not active, 1 = somewhat active, 2 = moderately active, 3 = somewhat active.

| | | Levene Statistic | df1 | df2 | Sig. |
|---------|--|---------------------|-----|--------|------|
| PCS | Based on Mean | 1.916 | 3 | 27 | .151 |
| | Based on Median | 1.292 | 3 | 27 | .297 |
| | Based on Median and with adjusted df | 1.292 | 3 | 22.189 | .302 |
| | Based on trimmed mean | 1.850 | 3 | 27 | .162 |
| LPRAQ-p | Based on Mean | 1.879 | 3 | 27 | .157 |
| | Based on Median | .861 | 3 | 27 | .473 |
| | Based on Median and with adjusted df | .861 | 3 | 12.385 | .487 |
| | Based on trimmed mean | 1.817 | 3 | 27 | .168 |
| EPDS1 | Based on Mean | .511 | 3 | 27 | .678 |
| | Based on Median | .627 | 3 | 27 | .604 |
| | Based on Median and with adjusted df | .627 | 3 | 25.086 | .604 |
| | Based on trimmed mean | .523 | 3 | 27 | .670 |
| VASQ1 | Based on Mean | .521 | 3 | 27 | .672 |
| | Based on Median | .489 | 3 | 27 | .693 |
| | Based on Median and with adjusted df | .489 | 3 | 24.766 | .693 |
| | Based on trimmed mean | .365 | 3 | 27 | .779 |
| VASQ2 | Based on Mean | .016 | 3 | 27 | .997 |
| | Based on Median | .009 | 3 | 27 | .999 |

| | | | | | |
|-------|--------------------------------------|-------|---|--------|------|
| EPDS2 | Based on Median and with adjusted df | .009 | 3 | 26.248 | .999 |
| | Based on trimmed mean | .009 | 3 | 27 | .999 |
| | Based on Mean | 1.735 | 3 | 27 | .183 |
| | Based on Median | 1.546 | 3 | 27 | .225 |
| | Based on Median and with adjusted df | 1.546 | 3 | 21.759 | .231 |
| | Based on trimmed mean | 1.737 | 3 | 27 | .183 |

Test of Homogeneity of the Variances - Prior Activity Level

| | | Sum of Squares | df | Mean Square | F | Sig |
|---------|----------------|----------------|----|-------------|-------|------|
| PCS | Between Groups | 7.482 | 3 | 2.494 | .128 | .942 |
| | Within Groups | 524.389 | 27 | 19.422 | | |
| | Total | 531.871 | 30 | | | |
| LPRAQ-p | Between Groups | 11.352 | 3 | 3.784 | .258 | .855 |
| | Within Groups | 396.067 | 27 | 14.669 | | |
| | Total | 407.419 | 30 | | | |
| EPDS1 | Between Groups | 8.057 | 3 | 2.686 | .286 | .835 |
| | Within Groups | 253.491 | 27 | 9.389 | | |
| | Total | 261.548 | 30 | | | |
| VASQ1 | Between Groups | 3.019 | 3 | 1.006 | .499 | .686 |
| | Within Groups | 54.400 | 27 | 2.015 | | |
| | Total | 57.419 | 30 | | | |
| VASQ2 | Between Groups | 82.245 | 3 | 27.415 | 4.148 | .015 |
| | Within Groups | 178.465 | 27 | 6.610 | | |
| | Total | 260.710 | 30 | | | |
| EPDS2 | Between Groups | 41.572 | 3 | 13.857 | 1.386 | .268 |
| | Within Groups | 269.976 | 27 | 9.999 | | |
| | Total | 311.548 | 30 | | | |

One-Way ANOVA - Prior Activity Level Variable.

| Hochberg | | | | | | | |
|--------------------|--|--|-----------------------|------------|-------|-------------------------|-------------|
| Dependent Variable | (I) activity level variable not active =0, somewhat active =1, moderately active =2, very active = 3 | (J) activity level variable not active =0, somewhat active =1, moderately active =2, very active = 3 | Mean Difference (I-J) | Std. Error | Sig. | 95% Confidence Interval | |
| | | | | | | Lower Bound | Upper Bound |
| PCS | 0 | 1 | -.889 | 2.938 | 1.000 | -9.19 | 7.41 |
| | | 2 | -.333 | 2.870 | 1.000 | -8.44 | 7.78 |
| | | 3 | .417 | 2.984 | 1.000 | -8.01 | 8.85 |
| | 1 | 0 | .889 | 2.938 | 1.000 | -7.41 | 9.19 |
| | | 2 | .556 | 1.981 | 1.000 | -5.04 | 6.15 |
| | | 3 | 1.306 | 2.141 | .989 | -4.74 | 7.36 |
| | 2 | 0 | .333 | 2.870 | 1.000 | -7.78 | 8.44 |
| | | 1 | -.556 | 1.981 | 1.000 | -6.15 | 5.04 |
| | | 3 | .750 | 2.048 | .999 | -5.04 | 6.54 |
| | 3 | 0 | -.417 | 2.984 | 1.000 | -8.85 | 8.01 |
| | | 1 | -1.306 | 2.141 | .989 | -7.36 | 4.74 |
| | | 2 | -.750 | 2.048 | .999 | -6.54 | 5.04 |
| LPRAQ-p | 0 | 1 | 1.778 | 2.553 | .979 | -5.44 | 8.99 |
| | | 2 | .485 | 2.495 | 1.000 | -6.56 | 7.53 |
| | | 3 | .792 | 2.593 | 1.000 | -6.53 | 8.12 |
| | 1 | 0 | -1.778 | 2.553 | .979 | -8.99 | 5.44 |
| | | 2 | -1.293 | 1.721 | .970 | -6.16 | 3.57 |
| | | 3 | -.986 | 1.861 | .995 | -6.24 | 4.27 |
| | 2 | 0 | -.485 | 2.495 | 1.000 | -7.53 | 6.56 |
| | | 1 | 1.293 | 1.721 | .970 | -3.57 | 6.16 |
| | | 3 | .307 | 1.780 | 1.000 | -4.72 | 5.33 |
| | 3 | 0 | -.792 | 2.593 | 1.000 | -8.12 | 6.53 |

| | | | | | | | |
|-------|---|---|----------|-------|-------|--------|------|
| | | 1 | .986 | 1.861 | .995 | -4.27 | 6.24 |
| | | 2 | -.307 | 1.780 | 1.000 | -5.33 | 4.72 |
| EPDS1 | 0 | 1 | -.111 | 2.043 | 1.000 | -5.88 | 5.66 |
| | | 2 | -.545 | 1.996 | 1.000 | -6.18 | 5.09 |
| | | 3 | -1.375 | 2.074 | .984 | -7.24 | 4.49 |
| | 1 | 0 | .111 | 2.043 | 1.000 | -5.66 | 5.88 |
| | | 2 | -.434 | 1.377 | 1.000 | -4.33 | 3.46 |
| | | 3 | -1.264 | 1.489 | .947 | -5.47 | 2.94 |
| | 2 | 0 | .545 | 1.996 | 1.000 | -5.09 | 6.18 |
| | | 1 | .434 | 1.377 | 1.000 | -3.46 | 4.33 |
| | | 3 | -.830 | 1.424 | .992 | -4.85 | 3.19 |
| | 3 | 0 | 1.375 | 2.074 | .984 | -4.49 | 7.24 |
| | | 1 | 1.264 | 1.489 | .947 | -2.94 | 5.47 |
| | | 2 | .830 | 1.424 | .992 | -3.19 | 4.85 |
| VASQ1 | 0 | 1 | -.222 | .946 | 1.000 | -2.90 | 2.45 |
| | | 2 | -.848 | .925 | .925 | -3.46 | 1.76 |
| | | 3 | -.292 | .961 | 1.000 | -3.01 | 2.42 |
| | 1 | 0 | .222 | .946 | 1.000 | -2.45 | 2.90 |
| | | 2 | -.626 | .638 | .901 | -2.43 | 1.18 |
| | | 3 | -.069 | .690 | 1.000 | -2.02 | 1.88 |
| | 2 | 0 | .848 | .925 | .925 | -1.76 | 3.46 |
| | | 1 | .626 | .638 | .901 | -1.18 | 2.43 |
| | | 3 | .557 | .660 | .948 | -1.31 | 2.42 |
| | 3 | 0 | .292 | .961 | 1.000 | -2.42 | 3.01 |
| | | 1 | .069 | .690 | 1.000 | -1.88 | 2.02 |
| | | 2 | -.557 | .660 | .948 | -2.42 | 1.31 |
| VASQ2 | 0 | 1 | -4.556 | 1.714 | .073 | -9.40 | .29 |
| | | 2 | -5.576 * | 1.675 | .015 | -10.31 | -.84 |
| | | 3 | -5.667 * | 1.741 | .018 | -10.58 | -.75 |
| | 1 | 0 | 4.556 | 1.714 | .073 | -.29 | 9.40 |

| | | | | | | | |
|-------|---|---|---------|-------|-------|-------|-------|
| | | 2 | -1.020 | 1.156 | .937 | -4.28 | 2.24 |
| | | 3 | -1.111 | 1.249 | .935 | -4.64 | 2.42 |
| | 2 | 0 | 5.576 * | 1.675 | .015 | .84 | 10.31 |
| | | 1 | 1.020 | 1.156 | .937 | -2.24 | 4.28 |
| | | 3 | -.091 | 1.195 | 1.000 | -3.47 | 3.28 |
| | 3 | 0 | 5.667 * | 1.741 | .018 | .75 | 10.58 |
| | | 1 | 1.111 | 1.249 | .935 | -2.42 | 4.64 |
| | | 2 | .091 | 1.195 | 1.000 | -3.28 | 3.47 |
| EPDS2 | 0 | 1 | -1.222 | 2.108 | .992 | -7.18 | 4.73 |
| | | 2 | -1.697 | 2.060 | .954 | -7.52 | 4.12 |
| | | 3 | -3.708 | 2.141 | .430 | -9.76 | 2.34 |
| | 1 | 0 | 1.222 | 2.108 | .992 | -4.73 | 7.18 |
| | | 2 | -.475 | 1.421 | 1.000 | -4.49 | 3.54 |
| | | 3 | -2.486 | 1.537 | .505 | -6.83 | 1.85 |
| | 2 | 0 | 1.697 | 2.060 | .954 | -4.12 | 7.52 |
| | | 1 | .475 | 1.421 | 1.000 | -3.54 | 4.49 |
| | | 3 | -2.011 | 1.469 | .678 | -6.16 | 2.14 |
| | 3 | 0 | 3.708 | 2.141 | .430 | -2.34 | 9.76 |
| | | 1 | 2.486 | 1.537 | .505 | -1.85 | 6.83 |
| | | 2 | 2.011 | 1.469 | .678 | -2.14 | 6.16 |

*. The mean difference is significant at the 0.05 level.

Multiple Comparisons - Hochberg's GT2 - Prior Activity Level Variable

| | | N | Mean | Std. Deviation | Std. Error | 95% Confidence Interval for Mean | | Minimum | Maximum |
|---------|-------|----|-------|----------------|------------|----------------------------------|-------------|---------|---------|
| | | | | | | Lower Bound | Upper Bound | | |
| PCS | 0 | 19 | 4.89 | 4.701 | 1.078 | 2.63 | 7.16 | 0 | 14 |
| | 1 | 12 | 5.00 | 3.490 | 1.008 | 2.78 | 7.22 | 0 | 10 |
| | Total | 31 | 4.94 | 4.211 | .756 | 3.39 | 6.48 | 0 | 14 |
| LPRAQ-p | 0 | 19 | 12.63 | 3.562 | .817 | 10.91 | 14.35 | 6 | 19 |
| | 1 | 12 | 13.00 | 4.023 | 1.161 | 10.44 | 15.56 | 6 | 19 |
| | Total | 31 | 12.77 | 3.685 | .662 | 11.42 | 14.13 | 6 | 19 |
| EPDS1 | 0 | 19 | 6.00 | 2.925 | .671 | 4.59 | 7.41 | 0 | 12 |
| | 1 | 12 | 4.92 | 2.999 | .866 | 3.01 | 6.82 | 0 | 11 |
| | Total | 31 | 5.58 | 2.953 | .530 | 4.50 | 6.66 | 0 | 12 |
| VASQ1 | 0 | 19 | 1.05 | 1.649 | .378 | .26 | 1.85 | 0 | 6 |
| | 1 | 12 | .33 | .651 | .188 | -.08 | .75 | 0 | 2 |
| | Total | 31 | .77 | 1.383 | .248 | .27 | 1.28 | 0 | 6 |
| VASQ2 | 0 | 19 | 7.63 | 2.565 | .588 | 6.40 | 8.87 | 2 | 10 |
| | 1 | 12 | 6.25 | 3.415 | .986 | 4.08 | 8.42 | 0 | 10 |
| | Total | 31 | 7.10 | 2.948 | .529 | 6.02 | 8.18 | 0 | 10 |
| EPDS2 | 0 | 19 | 7.74 | 3.052 | .700 | 6.27 | 9.21 | 3 | 13 |
| | 1 | 12 | 4.75 | 2.667 | .770 | 3.06 | 6.44 | 1 | 10 |
| | Total | 31 | 6.58 | 3.223 | .579 | 5.40 | 7.76 | 1 | 13 |

Descriptive Statistics - Prior Birth Variable. 0 = nulliparous, 1 = multiparous

| | | Sum of Squares | df | Mean Square | F | Sig. |
|---------|----------------|----------------|----|-------------|-------|------|
| PCS | Between Groups | .081 | 1 | .081 | .004 | .947 |
| | Within Groups | 531.789 | 29 | 18.338 | | |
| | Total | 531.871 | 30 | | | |
| LPRAQ-p | Between Groups | .998 | 1 | .998 | .071 | .791 |
| | Within Groups | 406.421 | 29 | 14.015 | | |
| | Total | 407.419 | 30 | | | |
| EPDS1 | Between Groups | 8.632 | 1 | 8.632 | .990 | .328 |
| | Within Groups | 252.917 | 29 | 8.721 | | |
| | Total | 261.548 | 30 | | | |
| VASQ1 | Between Groups | 3.805 | 1 | 3.805 | 2.058 | .162 |
| | Within Groups | 53.614 | 29 | 1.849 | | |
| | Total | 57.419 | 30 | | | |
| VASQ2 | Between Groups | 14.039 | 1 | 14.039 | 1.650 | .209 |
| | Within Groups | 246.671 | 29 | 8.506 | | |
| | Total | 260.710 | 30 | | | |
| EPDS2 | Between Groups | 65.614 | 1 | 65.614 | 7.737 | .009 |
| | Within Groups | 245.934 | 29 | 8.480 | | |
| | Total | 311.548 | 30 | | | |

One-Way ANOVA - Prior Birth Variable

| | | N | Mean | Std. Deviation | Std. Error | 95% Confidence Interval for Mean | | Minimum | Maximum |
|---------|-------|----|-------|----------------|------------|----------------------------------|-------------|---------|---------|
| | | | | | | Lower Bound | Upper Bound | | |
| PCS | 0 | 5 | 8.20 | 2.775 | 1.241 | 4.75 | 11.65 | 5 | 12 |
| | 1 | 26 | 4.31 | 4.183 | .820 | 2.62 | 6.00 | 0 | 14 |
| | Total | 31 | 4.94 | 4.211 | .756 | 3.39 | 6.48 | 0 | 14 |
| LPRAQ-p | 0 | 5 | 9.80 | 3.033 | 1.356 | 6.03 | 13.57 | 7 | 14 |
| | 1 | 26 | 13.35 | 3.566 | .699 | 11.91 | 14.79 | 6 | 19 |
| | Total | 31 | 12.77 | 3.685 | .662 | 11.42 | 14.13 | 6 | 19 |
| EPDS1 | 0 | 5 | 5.40 | 3.715 | 1.661 | .79 | 10.01 | 0 | 10 |
| | 1 | 26 | 5.62 | 2.872 | .563 | 4.46 | 6.78 | 0 | 12 |
| | Total | 31 | 5.58 | 2.953 | .530 | 4.50 | 6.66 | 0 | 12 |
| VASQ1 | 0 | 5 | .40 | .894 | .400 | -.71 | 1.51 | 0 | 2 |
| | 1 | 26 | .85 | 1.461 | .287 | .26 | 1.44 | 0 | 6 |
| | Total | 31 | .77 | 1.383 | .248 | .27 | 1.28 | 0 | 6 |
| VASQ2 | 0 | 5 | 8.80 | .837 | .374 | 7.76 | 9.84 | 8 | 10 |
| | 1 | 26 | 6.77 | 3.102 | .608 | 5.52 | 8.02 | 0 | 10 |
| | Total | 31 | 7.10 | 2.948 | .529 | 6.02 | 8.18 | 0 | 10 |
| EPDS2 | 0 | 5 | 6.00 | 3.162 | 1.414 | 2.07 | 9.93 | 1 | 9 |
| | 1 | 26 | 6.69 | 3.284 | .644 | 5.37 | 8.02 | 1 | 13 |
| | Total | 31 | 6.58 | 3.223 | .579 | 5.40 | 7.76 | 1 | 13 |

Descriptive Statistics - Analgesic Medication Variable. 0 = No medication, 1 = received analgesic medication

| | | Sum of Squares | df | Mean Square | F | Sig. |
|---------|----------------|----------------|----|-------------|-------|------|
| PCS | Between Groups | 63.533 | 1 | 63.533 | 3.934 | .057 |
| | Within Groups | 468.338 | 29 | 16.150 | | |
| | Total | 531.871 | 30 | | | |
| LPRAQ-p | Between Groups | 52.735 | 1 | 52.735 | 4.312 | .047 |
| | Within Groups | 354.685 | 29 | 12.231 | | |
| | Total | 407.419 | 30 | | | |
| EPDS1 | Between Groups | .195 | 1 | .195 | .022 | .884 |
| | Within Groups | 261.354 | 29 | 9.012 | | |
| | Total | 261.548 | 30 | | | |
| VASQ1 | Between Groups | .835 | 1 | .835 | .428 | .518 |
| | Within Groups | 56.585 | 29 | 1.951 | | |
| | Total | 57.419 | 30 | | | |
| VASQ2 | Between Groups | 17.294 | 1 | 17.294 | 2.060 | .162 |
| | Within Groups | 243.415 | 29 | 8.394 | | |
| | Total | 260.710 | 30 | | | |
| EPDS2 | Between Groups | 2.010 | 1 | 2.010 | .188 | .668 |
| | Within Groups | 309.538 | 29 | 10.674 | | |
| | Total | 311.548 | 30 | | | |

One-Way Anova - Analgesic Medication Variable