Universitat de Lleida

The influence of a web-based biopsychosocial pain education intervention on pain, disability, and pain cognition in patients with chronic low back pain in primary care: a mixed methods approach

Fran Valenzuela Pascual

http://hdl.handle.net/10803/666274

Universitat de Lleida

TESI DOCTORAL

The influence of a web-based biopsychosocial pain education intervention on pain, disability, and pain cognition in patients with chronic low back pain in primary care: a mixed methods approach

Fran Valenzuela Pascual

Memòria presentada per optar al grau de Doctor per la Universitat de Lleida

Programa de Doctorat en Salut

Director
Dr. Fidel Molina Luque
Dr. Jorge Soler González
Dr. Francesc Corbi Soler

Tutor
Dr. Joan Blanco Blanco

2018

Acknowledgements

Although a doctoral thesis is mostly a scientific process, it is not less true that inspiration is also an important part of it. This thesis, my thesis, would not have existed without the inspiration of two great people to whom I will always be grateful.

Their presence was not physical during this long and difficult time. They were not with me in person, but their essence is behind each and every one of the words that make up this work. This thesis is dedicated to two good people, my brother Oscar and my dear friend, my Australian brother, Pete Gaal. I can speak about one of them in the present tense. Sadly, speaking of the other one requires the past tense.

My brother Oscar, my little big brother, left us suddenly, without warning, as this thesis started to take form. Despite not having a higher education I have known few people who displayed the same natural and wild intelligence that defined Oscar. Oscar was a noble and easygoing person, stubborn and therefore persevering. Should he have pursued his education he would be, no doubt about it, the one writing a thesis. I believe he will be very proud of his older brother, no matter where he is. Thanks to you Oscar, in moments of weakness, almost defeat because your perseverance in life inspired me to keep walking. Oscar, this thesis is also yours.

Fortunately I am able to thank you in present tense, my dear friend Pete, although I am afraid this project has been a real obstacle that has not allowed me to enjoy you and your family, my family. Anyway, today I am what I am thanks to you. Crossing my path with yours was a defining moment in my life, a time full of challenge and life changes. You were the first one to make me curious about the nature of pain. Without you this thesis would not exist.

You have been a positive influence in my life, taking a greater role as a leader than as merely a boss albeit an exemplary one. A teacher, a guide we all wanted to learn from and emulate. You made me realize of the knowledge I had and the wisdom I lacked. More importantly, you taught me to look for that knowledge by myself. You were my friend then and you are my friend now despite the geography between us. Pete, a faithful and loving person but at the same time straight and judicious. You taught me to be humble and a better person. Not only did you share your friendship with me, but you let me be part of your family. Thank you Gaal family! Thanks Pete! This thesis is also yours.

And to all of you who have been by my side during this time of academic endeavors, thank you! This thesis is also yours.

Agradecimientos

Aunque una tesis doctoral mayoritariamente es un proceso científico, no es menos cierto que la inspiración es también parte importante de la misma. Esta tesis, mi tesis, no habría existido sin la inspiración de dos grandes personas, a las que siempre estaré agradecido.

Su presencia todo este tiempo, un tiempo largo y difícil, no fue física. No estuvieron a mi lado en persona, pero su esencia está detrás de cada una de las palabras que componen este trabajo. Esta tesis está dedicada a dos buenas personas, mi hermano Oscar y mi querido amigo, mi hermano australiano, Pete Gaal. De uno puedo aún hablar en presente, del otro tristemente solo en pasado.

Mi hermano Oscar, mi pequeño gran hermano, nos dejó de golpe, sin avisar, mientras empezaba esta tesis. Aunque no tenía estudios superiores, pocas personas he conocido que tuvieran la inteligencia natural y salvaje que definía a Oscar. Persona noble y campechana, tozuda y por lo tanto perseverante. De haber seguido sus estudios, no tengo la menor duda de que Oscar podría haber estado en mi lugar en este momento. Además, sé que esté donde esté, estará muy orgulloso de su hermano mayor. Gracias a ti Oscar, en los momentos de flaqueza, de casi derrota, tu perseverancia me inspiró a seguir caminando. Oscar, esta tesis también es tuya.

Mi querido Pete, por suerte a ti sí que te podré dar las gracias en persona, aunque esta tesis ha sido un verdadero obstáculo que no me ha dejado disfrutar de ti y tu familia, mi familia. De todas maneras, hoy soy lo que soy gracias a ti. Hay varios momentos de inflexión, de cambios en mi vida, y uno de los más importantes es el momento en que nuestros caminos se cruzaron. Tú fuiste de los primeros en despertar una conciencia crítica en mí. Fuiste el primero en hacerme pensar y preguntarme qué era el dolor. Es un hecho que de no haberte conocido esta tesis no existiría. Has representado varios papeles en mi vida. Has sido ese jefe ejemplar, más líder que jefe, al que todos nos gustaría tener y al que a mí siempre me gustaría emular. Has sido un maestro, haciéndome pensar sobre cada una de las cosas que sabía y sobre las que no sabía y, sin presionar, hiciste que yo mismo buscara respuestas. Has sido y eres ese amigo que, aunque en la distancia, sigues estando a mi lado. Pete, australiano fiel y cariñoso pero a la vez recto y juicioso. Me enseñaste a ser humilde y mejor persona. No solo compartiste tu amistad conmigo sino que me dejaste ser parte de tu familia. ¡Gracias familia Gaal! ¡Gracias Pete! Esta tesis también es tuya.

A todos y todas que habéis estado a mi lado todo este tiempo, ¡gracias! Esta tesis también es vuestra.

Abstract

Background: Low back pain is the most frequently reported musculoskeletal problem worldwide. Up to 90 % of patients with low back pain have not received a clear explanation for the source and origin of their pain. These individuals commonly receive a diagnosis of non-specific low back pain.

Patient education is a way to provide information and advice aimed at changing patients' cognition and knowledge about their chronic state through the reduction of fear of anticipatory outcomes and the resumption of normal activities. Information technology and the expedited communication processes associated with this technology can be used to deliver healthcare information to patients. Hence, this technology and its ability to deliver life-changing information has grown as a powerful and alternative health promotion tool.

Several studies have suggested that web-based educational interventions can change and improve the knowledge of patients with chronic pain and have a positive impact on their attitudes and behaviors.

Objective: The aim of this project was to explore patients' beliefs regarding the origin and meaning of their chronic low back pain in order to develop and evaluate a web-based biopsychosocial pain education intervention using different educational formats and gamification techniques.

Methods/design: This study had a mixed-method sequential exploratory design. A total of 48 patients suffering from chronic low back pain between 20–65 years of age who were receiving treatment in a primary care setting in Spain, took part in this study. For the qualitative phase of the study, 16 subjects (8 men and 8 women) were contacted by their family physician and invited to participate in a personal semi-structured interview. The quantitative phase was structured as a parallel randomized controlled trial. The duration of the intervention was 15 days. 48 subjects were randomly allocated using a simple random sample technique.

Randomization and allocation to trial group were carried out by a central computer system. Patients and researchers were blinded to group assignment. The intervention group (*n* = 26) had access to a specific website where they were given information related to their chronic low back pain. This information was provided in different formats. All of this material was based on the information obtained in the qualitative phase. The control group (*n* = 22) followed conventional care provided by their family physician. The main outcome variable was pain intensity measured using a 0-100 visual analogue scale. Secondary outcome measures were fear-avoidance beliefs, kinesiophobia, and disability.

Results: A per-protocol analysis was carried out (*n* = 44) using a two-way mixed factorial analysis of variance. There was no statistically significant interaction between treatment and time for pain intensity (F[1, 42] = 0.847, *p* = 0.36). There was a statistically significant interaction between time and treatment for fear-avoidance beliefs (F[1, 42] = 4.516, *p* = 0.04), kinesiophobia (F[1, 42] = 5.388, *p* = 0.02), and disability (F[1, 42] = 4.379, *p* = 0.04) with more favorable results for the experimental group. In the short term, there was a statistically significant difference on disability favoring the experimental group (MD -4.1; CI 95% -7.53 to -0.68; η^2 = 0.11; *p* = 0.02), representing a medium effect size. No statistically significant differences were found in pain intensity, fear-avoidance beliefs and kinesiophobia between groups.

Conclusions: The patients have expressed the need to better understand their pain, which implies that health professionals should be more didactic in the management of patients with chronic low back pain.

A web-based biopsychosocial pain education intervention for patients with chronic low back pain proved to be more beneficial than conventional care provided by family physicians in primary care on disability, although this result could be more related to the greater disability scores post-test in the control group rather than with the improvement obtained in the experimental group.

Trial registration: ClinicalTrials.gov NCT02369120 Date: 02/20/2015.

Keywords: Low back pain, Patient education, Educational technology, Pain neurophysiology, Gamification

Resumen

Antecedentes: el dolor lumbar es el problema musculoesquelético más frecuente en todo el mundo. Hasta un 90% de pacientes con dolor lumbar no han recibido una explicación clara sobre la fuente y el origen de su dolor. Estas personas comúnmente reciben un diagnóstico de dolor lumbar inespecífico.

La educación del paciente es una manera de dar información y consejo con el objetivo de modificar la cognición y el conocimiento que estos pacientes tienen sobre su estado crónico. Esto se consigue a través de la reducción del miedo anticipatorio a las consecuencias y la reanudación de las actividades normales. Las tecnologías de la información y los procesos de comunicación asociados se pueden utilizar como un medio para dar información médica a los pacientes. Por lo tanto, estas tecnologías y su capacidad para proporcionar información vital ha crecido como una herramienta poderosa y alternativa para la promoción de la salud.

Varios estudios han sugerido que las intervenciones educativas basadas en la web pueden cambiar y mejorar el conocimiento de los pacientes crónicos y tener un impacto positivo en las actitudes y conductas de estos pacientes.

Objetivo: El objetivo de este proyecto fue explorar las creencias de los pacientes con respecto al origen y significado de su dolor lumbar crónico para desarrollar y evaluar una intervención educativa biopsicosocial sobre el dolor en la web utilizando diferentes formatos educativos y técnicas de gamificación.

Métodos/diseño: Este estudio usó un diseño mixto exploratorio secuencial. Un total de 48 pacientes con dolor lumbar crónico entre 20 y 65 años de edad que estaban recibiendo tratamiento en atención primaria participaron en este estudio. Para la fase cualitativa del estudio, 16 sujetos (8 hombres y 8 mujeres) fueron contactados por su médico de familia e invitados a participar en una entrevista personal semiestructurada. La fase cuantitativa se estructuró como un ensayo controlado aleatorio paralelo. La duración de la intervención fue de 15 días. 48

sujetos fueron asignados al azar utilizando una técnica de muestreo aleatoria simple. La aleatorización y la asignación a los grupos se llevaron a cabo mediante un sistema informático central. Los pacientes e investigadores fueron cegados a la asignación de grupo. El grupo de intervención (*n* = 26) tuvo acceso a un sitio web donde se les proporcionó información relacionada con su dolor lumbar crónico. Esta información fue proporcionada en diferentes formatos. Todo este material se basó en la información obtenida en la fase cualitativa. El grupo control (*n* = 22) siguió el tratamiento convencional proporcionado por su médico de familia. La variable de resultado principal del estudio fue la intensidad del dolor medida mediante una escala visual analógica de 0-100. Las variables de resultado secundarias fueron las creencias de miedo-evitación, la kinesiofobia y la discapacidad.

Resultados: Se realizó un análisis por protocolo (n = 44), usando un análisis factorial mixto. La interacción entre tratamiento y tiempo para la intensidad del dolor fue estadísticamente no significativa (F[1, 42] = 0.847, *p* = 0.36). La interacción entre tratamiento y tiempo fue estadísticamente significativa para las variables de resultado creencias de miedo-evitación (F[1, 42] = 4.516, *p* = 0.04), kinesiofobia (F[1, 42] = 5.388, *p* = 0.02) y discapacidad (F[1, 42] = 4.379, *p* = 0.04), con resultados más favorables al grupo experimental. A corto plazo el grupo experimental mostró unas diferencias estadísticamente significativas a su favor en la discapacidad (MD -4.1; CI 95% -7.53 to -0.68; $\eta^2 = 0.11$; *p* = 0.02), representando un tamaño del efecto medio. No se encontraron diferencias estadísticamente significativas en la intensidad del dolor, las creencias de miedo-evitación y la kinesiofobia entre los grupos.

Conclusiones: Los pacientes han expresado la necesidad de comprender mejor su dolor, lo que implica que los profesionales de la salud deberían ser más didácticos en el tratamiento de los pacientes con dolor lumbar crónico.

Una intervención educativa biopsicosocial sobre el dolor en la web para pacientes con dolor lumbar crónico demostró ser más beneficiosa que el tratamiento convencional proporcionado por los médicos de familia en atención primaria sobre la discapacidad, aunque este resultado podría estar más relacionado con los puntajes de discapacidad más elevados mostrados por el grupo control post-test que con la mejora obtenida por el grupo experimental.

Registro del estudio: ClinicalTrials.gov NCT02369120 Fecha: 02/20/2015.

Palabras clave: Dolor lumbar, Educación del paciente, Tecnologías para la educación, Neurofisiología del dolor, Gamificación

Resum

Antecedents: el dolor lumbar és el problema musculoesquelètic més freqüent a tot el món. Fins a un 90% de pacients amb dolor lumbar no han rebut una explicació clara sobre la font i l'origen del seu dolor. Aquestes persones normalment reben un diagnòstic de dolor lumbar inespecífic.

L'educació del pacient és una manera de donar informació i consell amb l'objectiu de modificar la cognició i el coneixement que aquests pacients tenen sobre el seu estat crònic. Això s'aconsegueix a través de la reducció de la por anticipatòria a les conseqüències i la represa de les activitats normals. Les tecnologies de la informació i els processos de comunicació associats es poden utilitzar com un mitjà per donar informació mèdica als pacients. Per tant, aquestes tecnologies i la seva capacitat per proporcionar informació vital ha crescut com una eina poderosa i alternativa per a la promoció de la salut.

Diversos estudis han suggerit que les intervencions educatives basades en la web poden canviar i millorar el coneixement dels pacients crònics i tenir un impacte positiu en les actituds i conductes d'aquests pacients.

Objectiu: L'objectiu d'aquest projecte va ser explorar les creences dels pacients respecte a l'origen i significat del seu dolor lumbar crònic per desenvolupar i avaluar una intervenció educativa biopsicosocial sobre el dolor al web utilitzant diferents formats educatius i tècniques de gamificació.

Mètodes/disseny: Aquest estudi va fer servir un disseny mixt exploratori seqüencial. Un total de 48 pacients amb dolor lumbar crònic entre 20 i 65 anys d'edat que estaven rebent tractament en atenció primària van participar en aquest estudi. Per a la fase qualitativa de l'estudi, 16 subjectes (8 homes i 8 dones) van ser contactats pel seu metge de família i convidats a participar en una entrevista personal semiestructurada. La fase quantitativa es va estructurar com un assaig controlat aleatori paral·lel. La durada de la intervenció va ser de 15 dies. 48

subjectes van ser assignats a l'atzar utilitzant una tècnica de mostreig aleatòria simple. L'aleatorització i l'assignació als grups es van dur a terme mitjançant un sistema informàtic central. Els pacients i investigadors van ser cegats a l'assignació de grup. El grup d'intervenció (n = 26) va tenir accés a un lloc web on se'ls va proporcionar informació relacionada amb el seu dolor lumbar crònic. Aquesta informació va ser proporcionada en diferents formats. Tot aquest material es va basar en la informació obtinguda en la fase qualitativa. El grup control (n = 22) va seguir el tractament convencional proporcionat pel seu metge de família. La variable de resultat principal de l'estudi va ser la intensitat del dolor mesurada mitjançant una escala visual analògica de 0-100. Les variables de resultat secundàries van ser les creences de por-evitació, la kinesiofòbia i la discapacitat.

Resultats: Es va realitzar una anàlisi per intenció de tractar (n = 44), usant una anàlisi factorial mixt. La interacció entre tractament i temps per a la intensitat del dolor va ser estadísticament no significatiu (F[1, 42] = 0.847, p = 0.36). La interacció entre tractament i temps va ser estadísticament significatiu per a les variables de resultat creences de por-evitació (F[1, 42] = 4.516, p = 0.04), kinesiofòbia (F[1, 42] = 5.388, p = 0.02) i discapacitat (F[1, 42] = 4.379, p = 0.04), amb resultats més favorables al grup experimental. A curt termini el grup experimental va mostrar unes diferències estadísticament significatives a favor seu en la discapacitat (MD -4.1; CI 95% -7.53 to -0.68; η^2 = 0.11; p = 0.02), representant una mida de l'efecte mitjà. No es van trobar diferències estadísticament significatives en la intensitat del dolor, les creences de por-evitació i la kinesiofòbia entre els grups.

Conclusions: Els pacients han expressat la necessitat de comprendre millor el seu dolor, el que implica que els professionals de la salut haurien de ser més didàctics en el tractament dels pacients amb dolor lumbar crònic.

Una intervenció educativa biopsicosocial sobre el dolor al web per a pacients amb dolor lumbar crònic va demostrar ser més beneficiosa que el tractament convencional proporcionat pels metges de família en atenció primària sobre la discapacitat, encara que aquest resultat podria estar més relacionat amb les puntuacions de discapacitat més elevades mostrades pel grup control post-test que amb la millora obtinguda pel grup experimental.

Registre de l'estudi: ClinicalTrials.gov NCT02369120 Data: 02/20/2015.

Paraules clau: Dolor lumbar, Educació del pacient, Tecnologies per a l'educació, Neurofisiologia del dolor, Gamificació

Table of contents

List of Tables

List of Figures

List of abbreviations

LBP: Low Back Pain

CLBP: Chronic Low Back Pain

DALYs: Disability-Adjusted Life Years

SR: Systematic Review

CGL: Clinical Practice Guidelines for the Pathology of the Lumbar Spine in Adults

NSAID: Nonsteroidal Anti-Inflammatory Drug

EGL: European Guidelines for the Treatment of Non-specific Chronic Low Back Pain

RCT: Randomized Controlled Trial

SMD: Standardized Mean Difference

CI: Confidence Interval

GA: Graded Activity

MD: Mean Difference

PNE: Pain Neuroscience Education

PAG: Periaqueductal Gray

RVM: Rostral Ventromedial Medulla

PI: Principal Investigator

BPE: Biopsychosocial Pain Education

MCID: Minimal Clinically Important Difference

VAS: Visual Analogue Scale

FABQ: Fear-Avoidance Beliefs Questionnaire

TSK: Tampa Scale for Kinesiophobia

RMQ: Roland–Morris Questionnaire

SD: Standard Deviation

SE: Standard Error

$\boldsymbol{\eta^2}$: Eta Squared

MC: Mean Change

CBT: Cognitive and Behavioral Therapy

Chapter 1: Theoretical framework

1.1 Low Back Pain in Context: Epidemiology and Definition

Low back pain (LBP) is the most reported musculoskeletal problem and one of the most common in the world (1–7). However, there is no consensus about the definition of LBP. Some authors have proposed that LBP is a pain localized below the 12th rib, on the upper side of the inferior gluteal folds, and that it could be accompanied by pain down the leg or not (2,4). In regards to its duration, LBP can be defined as acute (less than 6 weeks of duration), subacute (between 6 weeks and 3 months of duration), and chronic (longer than 3 months) (4). Almost everybody, regardless of age and gender, will have one or more episodes of LBP and some will develop chronic pain. There is not total agreement on the prevalence of LBP, although the figures range from 4 to 33% at some point in time (2,8), from 8 to 82.5% within a one-year time frame (4), and between 4 and 14% will develop chronic low back pain (CLBP) (9). In Spain, the prevalence of CLBP in adults over 20 years old was 7.7% in 2000 (10), and it was 19.9% in 2006 for adults over 15 years old (11). The prevalence by gender between the years 2016 and 2017 was 14.7% in men and 22.1% in women (12). At a more local level, the data in Catalonia (Spain) for the year 2015 were comparatively higher, standing at 18.7% in men and 30.1% in women (13).

LBP has major economic implications to our society in terms of direct and indirect costs (2–4,14,15). Worldwide, LBP causes more disability than any other particular condition when measured in years lived with disability. In terms of overall burden, measured in disability-adjusted life years (DALYs), LBP globally ranked sixth out of 291 conditions studied in 2010 (16), contributing a total of 83 million DALYs (17). In a different study published in 2012, the cost of healthcare per patient per year for LBP was estimated to be 1095€ in Germany and in 1431€ in France (18).

In addition, LBP has become an important work-related problem because of the loss of working days, which in turn has serious implications on productivity. For example, in the United States, the mean lost productivity time is 5.2 hours per week (4), whereas in Spain, there was a mean of 21.95 lost work days between 1993 and 1997 (19), which equals to 2.53 hours per week. In terms of DALYs, in

2010, it was estimated that LBP resulting from ergonomic exposures at work caused 21.7 million DALYs, 62% of which were in males (20).

Pain is a multifactorial experience associated with psychological and emotional factors that play important roles in the transition from acute to chronic states (21–23). Cessation of the healing process would result in the restoration of the insulted tissue. However, psychological factors can lead to changes in pain processing that could result in a chronic pain state (24). Although LBP usually improves within weeks, in some people pain and disability remain chronic (25). Moreover, up to 90% of patients with LBP have no clear explanation about the source and origin of their pain, which receives the name non-specific LBP (4).

Pain neuroscience has made substantial advances in the understanding of pain and found that changes in the central nervous system can be a contributor to CLBP. One of these mechanisms is central sensitization (26). This mechanism is defined as the increase in excitability of the neurons in the dorsal horn of the spinal cord as the result of a nociceptive input, which can be maintained over time even after the nociceptive input has decreased or ended (27,28). This implies that pain is not just on the periphery but rather is in the central nervous system. As a result of the latter, some authors have started to point out that treatments should be more brain-centered rather than peripherally oriented (26).

LBP has very important implications and consequences in other aspects of life. In fact, the impaired functionality and altered sleep of patients with LBP have a significantly negative impact on their lives, affecting their families as well as their social lives, resulting in emotional problems and disruption of their lives (2,4,8). Even though LBP is an important and recurrent problem nowadays, treatments addressing this problem have modest clinical benefits, in addition to low adherence (14,15).

1.2 CLBP: Treatments

In this section, we do not intend to analyze exhaustively the wide variety of possible treatments available for chronic low back pain but rather expose the most used options to demonstrate the complexity of this painful musculoskeletal syndrome. In addition, we must bear in mind that a large number of treatments that address this problem have modest clinical benefits and low adherence (14,15).

All the information used in this section has been extracted mostly from systematic reviews (SR), which are the “lens through which evidence should be viewed and applied” (29). We conducted the search in Pubmed and Cochrane, including only SRs published in the last 5 years (2014–2018).

1.2.1 Pharmacological treatments

The “Clinical practice guidelines for the pathology of the lumbar spine in adults” (CGL) of the Catalan Health Institute recommends that for the pharmacological approach of CLBP, during acute episodes, the same recommendations should be used as those given for the treatment of the acute low back pain. This guide bases its recommendations on the efficacy found in different studies, suggesting the use of paracetamol, nonsteroidal anti-inflammatory drugs (NSAID), and strong opioids (such as morphine or fentanyl) to provide adequate analgesia (30). On the other hand, the “European guidelines for the treatment of non-specific chronic low back pain” (EGL) recommend the use of NSAIDs and weak opioids (such as codeine) for a short time period for pain relief. This same guide also recommends the use of noradrenergic or noradrenergic-serotonergic antidepressants, muscle relaxants, and capsicum plasters for the treatment of pain but does not recommend the use of gabapentin (31). In line with this, Koes et al. (32) state that the first option and most-recommended drug by clinical practice guidelines is paracetamol, followed by NSAIDs.

Next, we will summarize the evidence found in different SRs published in the last 5 years on the effectiveness of the pharmacology used for the treatment of CLBP (Table 1).

1.2.1.1 Paracetamol

Although paracetamol is one of the most prescribed pharmacological substances in the treatment of CLBP, it has been shown that it is not better than a placebo in people with acute LBP and that it is not possible to make any recommendation about its use in CLBP because of the lack of scientific evidence found (33,34).

1.2.1.2 NSAIDs

As mentioned before, NSAIDs are one of the most recommended substances for the management of CLBP. Enthoven et al. (35) conducted a SR, concluding that when only the results of randomized controlled trials (RCT) with a low risk of bias are taken into account, there are no significant differences between NSAIDs and placebo. The same authors state that there are no clear results regarding the effectiveness of NSAIDs compared to other substances, nor on which NSAID is more effective than the others. However, Chung et al. (36), based on the results of their review, support the use of COX-2 NSAIDs as first-line drugs for the treatment of chronic non-specific LBP.

1.2.1.3 Opioids

Regarding opioid substances, the different SRs that have addressed their use in the treatment of CLBP suggest that in patients who tolerate this medication, its analgesic effect is modest in the short term but that this effect, within the recommended doses, is not clinically important (34,37). In fact, when these doses are increased, even in patients who initially tolerate it well, the effect produced in the short and medium term is still small and not clinically relevant. Moreover, in the latter case, in which the recommended doses are increased, many patients do not tolerate it and stop taking the drug since it does not produce any analgesic effect (37).

On the other hand, when comparing the use of opioids with other substances such as NSAIDs or antidepressants, the results show no significant differences. If opioids are compared to placebo, the results show short-term efficacy at the level of pain and function, but this evidence is of low to moderate quality (38). The different SRs discourage its prolonged use, since there are no RCT studies supporting its effectiveness and safety in the long-term treatment of CLBP (37,38). However, in the review by Chung et al. (36), it is concluded that the effects of opioids, including partial agonists, are statistically relevant with respect to pain reduction. Regarding this last review, the results should be considered with caution since the meta-analysis is on the effects of opioids in general; although it has been done using an analysis of random effects, none of the pooled studies have overlapping confidence intervals. Furthermore, the result of the calculation of the heterogeneity of the included studies is considerable (I^2 = 100%, $p < 0.00001$) (39). Some researchers suggest that when extreme heterogeneity occurs, the authors of the SR should refrain from performing meta-analysis of that variable (40).

1.2.1.4 Muscle relaxants

The SR by Abdel Shaheed et al. (41) on the efficacy and tolerability of muscle relaxants for the treatment of LBP included 15 RCTs with a total of 3362 subjects. These authors concluded that there is no evidence to support the use of benzodiazepines in the treatment of LBP. In fact, the bibliography does not recommend the prolonged use of these substances for LBP treatment. Their efficacy in the treatment of CLBP is unknown because of the few studies found and their low methodological quality. On the other hand, the methodological quality of this review has been questioned by Durg (42), which makes us take the results mentioned above with caution.

1.2.1.5 Gabapentinoids

The evidence concerning the use of gabapentinoids for CLBP is limited, concluding that the side effects derived from its use added to its lack of efficacy and that the associated costs do not justify their prescription in this type of patient (43).

1.2.1.6 Duloxetine

Finally, duloxetine, which is an inhibitor of the reabsorption of both serotonin and norepinephrine, is a drug that has its mechanism of action in the descending inhibitory pathways that are part of the nociceptive modulation exerted in the spinal cord. Cawston et al. (44) carried out a SR comparing the effects of duloxetine against other drugs, concluding that there was no difference in the efficacy of any of them. Nevertheless, in a more recent SR, Chou et al. (34) found that duloxetine was effective for the treatment of CLBP, although these effects were modest. Finally, Wielage et al. (45) suggested that, compared to other first-line drugs used for CLBP, duloxetine can be an effective and economical alternative.

Table 1: Pharmacological treatments for CLBP

DRUG	RESULTS
PARACETAMOL	• Not possible to make any recommendation due to the little evidence found (33,34).
NSAIDs	• No clear results regarding its effectiveness compared to other substances (35). • COX-2 as first-line drug (36).
OPIOIDS	• Modest effect not clinically important (34,37). • No significant differences compared to NSAIDs or antidepressants (38). • Compared to placebo, low to moderate evidence for a short-term efficacy on pain and function (38).
MUSCLE RELAXANTS	• Unknown efficacy. Few studies of low methodological quality (41).
GABAPENTINOIDS	• Lack of efficacy + side effects (43).
DULOXETINE	• No efficacy (44). • Effective but with a modest effect (34). • Effective and economical compared to other first-line drugs (45).

1.2.2 Non-pharmacological treatments

Next, we summarize the evidence found in different SRs published in the last 5 years on the effectiveness of the non-pharmacological treatments used for CLBP. There are two main groups of treatments within the non-pharmacological framework, exercise therapies and manual therapies.

1.2.2.1 Exercise therapy

In the existing literature, there are several types of therapeutic exercises used in the treatment of CLBP. Both the CGL and the EGL do not recommend any type of therapeutic exercise in a specific way (30,31). For example, the CGL does not recommend therapeutic exercise during the first week of treatment, although from that moment onwards, it advises patients to perform both aerobic and specific exercises two or three times a week in order to avoid recurrences and return to daily activities (30). In contrast, the EGL recommends supervised therapeutic exercise as a first-line treatment, without specifying whether it should be performed after the first week or not (31).

The types of exercise therapies included are strength/resistance, cardiorespiratory, graded activity/graded exposure, Pilates, yoga, proprioceptive training, and motor control (Table 2). The outcomes considered relevant for this section are pain, function, and quality of life.

1.2.2.1.1 Strength/resistance and cardiorespiratory

Only one meta-analysis analyzed the effectiveness of strength/resistance and cardiorespiratory therapeutic exercises. Searle et al. (46) included 39 studies in their meta-analysis to determine the type of therapeutic exercise most effective in reducing pain in patients with CLBP. When therapeutic exercise was compared to a control group, which included different types of treatments (usual activities, general practitioner care, electrotherapies, and manipulative therapies), pain significantly decreased ($p < 0.01$), although the effect size (47) was small (standardized mean difference [SMD] –0.32; confidence interval [CI] 95% –0.44 to –0.19). The same authors carried out an analysis by subgroups to assess which therapeutic exercise was the most effective. For the strength/resistance exercises, the results were again significant ($p < 0.01$) with a medium effect size (SMD –0.50; CI 95% –0.77 to –0.24). Finally, the authors concluded that strength/resistance exercises produced a benefit in the treatment of CLBP compared to other types of interventions, although this benefit proved to have a medium clinical relevance (effect size). In addition, they concluded that cardiorespiratory-type exercises and combined exercise programs (strength, endurance, stretching, and aerobic exercises) proved to be ineffective.

1.2.2.1.2 Graded activity and graded exposure

Both the graded activity (GA) and the graded exposure take into account cognitive and behavioral aspects to increase and improve activity tolerance. The difference between the types of exercises is that in graded exposure a hierarchical list of activities that produce fear in the patient is generated. The therapist and patient then approach the irrational beliefs and thoughts that lead to that fear, which diminishes the anxiety generated by carrying out these activities. In contrast, the graded activity uses operant conditioning to reinforce correct behavior. In addition, the graded activity is not focused on the pain but on the ability of the patient to increase his/her activity (48).

Only the meta-analysis of Lopez-de-Uralde-Villanueva et al. (49) analyzed this type of therapeutic exercise. The meta-analysis included nine studies comparing graded activity, graded exposure, or conventional treatment (medical and physiotherapy). The results showed that GA compared to other types of exercises did not obtain statistically significant results in the reduction of disability in the short ($p = 0.37$), intermediate ($p = 0.41$) and long term ($p = 0.22$). Furthermore, statistically significant results were not found in terms of quality of life and pain intensity in the short, intermediate, and long term.

When compared to a control group, the GA obtained significant results on disability only in the short ($p = 0.02$) and long term ($p < 0.0001$); although the effect size is small in the short term (SMD –0.3; CI 95% –0.55 to –0.05) and medium in the long term (SMD –0.53; CI 95% –0.79 to –0.27). Regarding pain intensity, the results were statistically not significant.

Graded exposure proved to be more effective than GA for disability in the short term ($p = 0.048$), with a small effect size (SMD 0.39; CI 95% 0.003 to 0.78), and for catastrophization in the short term ($p = 0.02$), with a small effect size (SMD 0.48; CI 95% 0.09 to 0.87). There were no statistically significant results in terms of pain intensity.

We conclude that there is some evidence that GA compared to a control group has a small effect on improving disability in the short term and a medium effect in the long term, but this benefit is unlikely to be clinically important. On the other hand, there is also some evidence that graded exposure is more effective than GA in reducing disability and catastrophizing in the short term, although the improvement may be clinically irrelevant.

1.2.2.1.3 Pilates

The Pilates method is a type of therapeutic exercise that takes into account both the body and the mind and includes exercises such as stretching and strengthening with the aim of developing a mental and physical strength (50).

Four SRs were found, of which two were meta-analyses (51,52) and one was narrative synthesis (53). The SR by Patti et al. (54) has not been taken into account because of its low methodological quality, as it mixes different types of studies (SRs, RCTs, and case-control studies) and does not assess the methodological quality of the included studies. On the other hand, it is interesting to note that the other three reviews found include seven of the same studies. Miyamoto et al. (51) and Yamato et al. (52) performed a meta-analysis, but although they used a large number of the same studies, Wells et al. (53) argued that they did not carry out the meta-analysis because the high heterogeneity of the included studies.

The reviews of Wells et al. (53) and Miyamoto et al. (51) obtained similar results, agreeing that the Pilates method is better than minimal intervention or usual care in the short term for pain and disability. However, compared to other types of exercises, the Pilates method obtained the same results in terms of reduction of pain in the short term. Nevertheless, the review of Yamato et al. (52), which is the most current of the three reviews (2016), criticizes the methodological quality of the studies found, stating that they are not high quality. Even so, these same authors agree with the two previous reviews in that the Pilates method is better than a minimal intervention in the short term for pain (mean difference [MD] –14.05; CI 95% –18.91 to –9.19; $p < 0.001$) and disability (MD –7.95; CI 95% –13.23 to –2.67, $p = 0.003$). However, they emphasize that the quality of the evidence is low to moderate and that the effect sizes are mostly medium. Furthermore, regarding the results of the comparison between the Pilates method and other types of exercises, the same authors found statistical significance that in the intermediate term there was a small effect in favor of other type of exercises in terms of function (MD –3.60; CI 95% –7.00 to –0.20), although the level of evidence is low.

Therefore, we conclude that the effectiveness of the Pilates method, in terms of pain and disability in the short and intermediate-term, is of low to medium quality.

1.2.2.1.4 Yoga

Yoga is a type of exercise that pursues the body–mind union to achieve a healthy state through the use of breathing exercises, pleasant postures, and meditation (55). Only one SR with meta-analysis examined the use of yoga for the treatment of CLBP. The review of Wieland et al. (56) included 12 trials (1080 participants).

When Wieland et al. (56) compared yoga to non-exercise control, the results in terms of function were significant in the short term ($p = 0.00070$) as well as in the intermediate ($p = 0.000076$) and long term ($p = 0.015$). However, the effect sizes were all small, in the short (SMD –0.45; CI 95% –0.71 to –0.19), intermediate (SMD –0.44, CI 95% –0.66 to –0.22), and long term (SMD -0.26; CI 95% -0.46 to -0.05). Regarding pain, there were only significant results in the short ($p = 0.034$)

and intermediate term (p = 0.0059), but their effect sizes did not reach the minimum clinically important change (15 on the 0–100 pain scale), in the short (MD –10.83; CI 95% –20.85 to –0.81) and intermediate term (MD –7.81; CI 95% –13.37 to –2.25).

When yoga was compared to exercise, the results were not significant in terms of function. For the pain outcome, only one study was included with clinically and statistically significant results, but it had a very low-quality evidence.

When yoga plus exercise was compared to exercise alone, the results were not significant for either function or pain.

In conclusion, there is low to moderate evidence that there is a small improvement in function when comparing yoga versus non-exercise controls in the short and medium term. When comparing yoga versus exercise or yoga plus exercise versus exercise alone there is no clear evidence that there are differences in favor of yoga.

1.2.2.1.5 Proprioceptive training

Proprioceptive training aims to improve sensorimotor function, using somatosensory signals such as proprioceptive or tactile stimuli without the help of other information modalities such as vision (57).

Only one SR was found that analyzed proprioceptive training. McCaskey et al. (58) carried out a SR on the effects of proprioceptive exercise on pain and function in people with LBP. The authors included 12 studies (1165 participants). It is important to note that only one of the included studies had a low risk of bias. The rest of the studies had at least two items considered to be at high risk of bias, and two of them had six or more items at high risk of bias.

On the other hand, the authors did not specify whether the results were significant or not, and the p value was not shown. Furthermore, and contrary to what is suggested in the Cochrane Handbook for SRs of Interventions Version 5.1.0 (39), when the authors of this review analyzed the continuous outcomes, they did not specify the limits, the units, or the direction of the scales used, nor did they mention what would be the minimally important difference. In fact, although they presented the SMD in some cases, it should have been accompanied by the rule of thumb to help the reader understand the results. For all the above mentioned, it is difficult to interpret the results of this review.

Even so, the conclusions of the authors are that there is low-quality evidence that proprioceptive training does not add any benefit to conventional physiotherapy and that it is inferior to educational interventions.

1.2.2.1.6 Motor control

Motor control training focuses on activation of the deep trunk muscles to regain their control and coordination. Through the pre-activation of these muscles, the goal is to perform tasks and complex movements that integrate the overall activation of the trunk muscles (59,60).

It is important to highlight that the term "motor control" encompasses other types of similar treatments that use different names such as "core stability" or "coordination/stabilization exercises," even though they are the same concept (61).

Four SRs were found with meta-analysis that addressed the treatment of CLBP using motor control training (46,59,62,63). Gomes-Neto et al. (63) included 11 studies (895 subjects) comparing stabilization exercises with general exercise (strengthening and/or stretching exercises) or manual therapy. The results were significant in terms of pain intensity, with the stabilization exercises being more effective than the general exercises (SMD –1.03; CI 95% –1.79 to –0.27). This result is considered clinically relevant. When comparing stabilization exercises to manual therapy, the results were not significant. Regarding disability, there was a significant improvement in the group of stabilization exercises compared to general exercise (SMD –5.41; CI 95% –8.34 to –2.49), but the results were again not significant when compared with manual therapy. Regarding the function, the results were all non-significant.

It should be noted that the studies included in this review did not provide enough information to perform a detailed analysis on the risk of bias. In addition, most of these studies did not provide specific information regarding random generation and concealment allocation, which decreases their internal validity by increasing the risk of bias.

Searle et al. (46) (12 studies, 1,343 subjects) obtained similar results, with the coordination/stabilization exercises showing a significant effect compared to the control group (SMD –0.47; CI 95%: –0.77 to –0.18, $p < 0.01$). However, these results must be taken with caution as the studies included in the meta-analysis had a high heterogeneity ($I^2 = 83.2\%$). Therefore the statistical analysis should not have been performed, as suggested by some authors (39,64).

Saragiotto et al. (59) analyzed 29 trials (2,431 subjects), obtaining the following results. When motor control training was compared to other exercises, there was low-quality evidence that there is a significant, not clinically important, effect that motor control training reduces pain in the short term (MD –7.53; CI 95% –10.54 to –4.52; $p < 0.001$). For the intermediate- and long-term follow up, the results are not significant. When assessing disability, the results show that there is low-quality evidence that there is a small but not clinically important effect on improving disability at short-term follow up (MD –4.82; CI 95% –6.95 to –2.68; $p < 0.001$). For the intermediate- and long-term follow up, the results are not significant. For function, there is moderate quality evidence that there is a significant small but not clinically important effect for improving function at short-term follow up (MD 7.29; CI 95% 1.53 to 13.04, $p = 0.01$). For the intermediate- and long-term follow up, the results are not significant. For the outcome quality of life (physical component), all the results are not significant.

When motor control training is compared to manual therapy, the results for pain, disability, and function, are all not significant.

When motor control training is compared to minimal intervention, there is moderate quality evidence that there is a clinically important effect that motor control training reduces pain with a medium effect size in the short term (MD –10.01; CI 95% –15.67 to –4.35; $p < 0.001$) and long-term follow up (MD –12.97; CI 95% –18.51 to –7.42; $p < 0.001$). There is low-quality evidence for a clinically important effect that motor control training reduces pain in the intermediate term, with a medium effect size (MD –12.61; CI 95% –20.53 to –4.69; $p = 0.002$). For the outcome disability, there is very low-quality evidence that there is a small but not clinically important significant effect on motor control for improving disability at short-term follow up (MD –8.63; CI 95% –14.78 to –2.47; $p < 0.01$). There is moderate-quality evidence that there is a significant, but not clinically important, effect favoring motor control training at intermediate (MD –5.47; CI 95% –9.17 to –1.77; $p = 0.004$) and long-term follow up (MD –5.96; CI 95% –9.81 to –2.11; $p = 0.002$), with small effect sizes. Results for function come from only one trial showing low-quality evidence that motor control training significantly improves function with a clinically important medium effect size at short term (MD 1.10; CI 95% 0.36 to 1.8; $p = 0.004$), intermediate-term (MD 1.00; CI 95% 0.16 to 1.84; $p = 0.02$), and long-term follow up (MD 1.50; CI 95% 0.68 to 2.32; $p < 0.001$).

When motor control training is compared to a combination of exercises and electrophysical agents, there is low-quality evidence that there is a clinically important effect that motor control training significantly reduces pain at short-term follow up, with a large effect size (MD –30.18; CI 95% –35.32 to –25.05; $p < 0.001$). Data for intermediate term were not pooled because of the high heterogeneity of the included trials. For disability, there is only one trial with results of very low quality. There is only one study assessing the quality of life and it shows no significant results for the mental component. For the physical component, there is low-quality evidence that there is a small but not clinically important significant effect favoring motor control training at short-term (MD 8.40; CI 95% 2.68 to 14.12; $p < 0.01$) and intermediate-term follow up (MD 8.0; CI 95% 2.25 to 13.75; $p < 0.01$).

Smith et al. (62) investigated the effectiveness of stabilization exercises compared to any other form of exercise. They included 22 studies (2,258 participants) for the pain outcome, and 24 studies (2,359 participants) for the disability outcome. This SR shows significant benefit for stabilization exercises at short- (MD –7.93; CI 95% –11.74 to –4.12), medium- (MD –6.10; CI 95% –10.54 to –1.65) and long-term follow up (MD –6.39; CI 95% –10.14 to –2.65). However, these results are not clinically important. The results for disability show a significant benefit for stabilization exercises at short- (MD –3.61; CI 95% –6.53 to –0.70) and long-term follow up (MD –3.92; CI 95% –7.25 to –0.59) but they are not clinically important.

In conclusion, there is minimal evidence that motor control training provides better outcomes than other types of exercise. In fact, Saragiotto et al. (59) suggest that patients and therapists should choose the type of therapeutic exercise for the treatment of CLBP by taking into account aspects such as cost and safety. In line with this, Smith et al. (62) do not recommend motor control training over other types of therapeutic exercises.

Table 2: Exercise therapies for CLBP

THERAPY	RESULTS
STRENGTH/RESISTANCE	• Beneficial compared to other types of interventions with a medium effect size (46).
CARDIORESPIRATORY	• Ineffective (46).
GRADED ACTIVITY	• Not significant results compared to other types of exercises or a control group (49).
GRADED EXPOSURE	• Not significant results compared to graded activity (49).
PILATES	• Better than minimal intervention or usual care in the short term (51–53). • Same results than other types of exercise in the short term (51–53). • Quality of the evidence is low to moderate (51).
YOGA	• When comparing yoga versus exercise or yoga plus exercise versus exercise alone there is no clear evidence that there are differences in favor of yoga (56).
PROPRIOCEPTIVE TRAINING	• There is low-quality evidence that proprioceptive training does not add any benefit to conventional physiotherapy, and that it is inferior to educational interventions (58).
MOTOR CONTROL	• There is minimal evidence that motor control training provides better outcomes than other types of exercise (46,59,62,63). • Motor control exercises show a tendency to worsen results in fear-avoidance (62).

1.2.2.2 Manual therapy

Next, we summarize the evidence found in different SRs published in the last 5 years on the effectiveness of manual therapy for the treatment of CLBP (Table 3). Manual therapy can be defined as any type of movement applied by a healthcare professional on the joints or any other body structure (65). The types of manual therapies included are therapeutic massage, muscle energy technique, manipulation and mobilization.

1.2.2.2.1 Therapeutic massage

According to Vickers and Zollman (2), therapeutic massage is defined as "the manipulation of the soft tissue of whole body areas to bring about generalized improvements in health, such as relaxation or improved sleep, or specific physical benefits, such as relief of muscular aches and pains." Both the CGL and the EGL do not recommend its use as a treatment for CLBP (30,31).

Only one SR was found. Furlan et al. (67) evaluated the effects of therapeutic massage for CLBP, including 25 studies with a total of 3,096 subjects. Although the number of studies and subjects included was high, the quality of the evidence found was "low" or "very low," so no definitive conclusions can be drawn as the authors themselves claimed to have very little confidence in their results. Among the reasons given by the review authors to justify their low confidence in the results are the small sizes of the studies and their methodical flaws.

1.2.2.2.2 Muscle energy

Greenman (68) defined muscle energy technique as the "procedure that involves the voluntary contraction of patient muscle in a precisely controlled direction, at varying levels of intensity, against a distinctly executed counterforce applied by the operator.'' None of the two guides used as a reference (CGL, EGL) mention this type of manual treatment (30,31).

Franke et al. (69) carried out a SR on the effectiveness of the muscle energy technique for the treatment of non-specific LBP, where they included 12 RCTs with a total of 500 subjects. As in the therapeutic massage, the quality of the evidence was poor, so the results are unreliable. Among the reasons that justify this poor quality of the evidence, the review authors mention the high risk of bias and the small sizes of the included studies.

1.2.2.2.3 Manipulation and mobilization

Rubinstein et al. (70) define manipulation as the use of a “high velocity impulse or thrust applied to a synovial joint over a short amplitude at or near the end of the passive or physiologic range of motion, which is often accompanied by an audible ‘crack’.” The same authors define mobilization as the “use [of] low-grade velocity, small or large amplitude passive movement techniques within the patient’s range of motion and control.” (70).

Only the review by Coulter et al. (71) analyzed the effectiveness of manipulation and mobilization for the treatment of CLBP. In this review, nine studies with a total of 1,176 subjects were included. According to the review authors “There is moderate-quality evidence that manipulation and mobilization are likely to reduce pain and improve function for patients with chronic low back pain; manipulation appears to produce a larger effect than mobilization.” However, these results should be qualified. The results of the analysis of the effect of the manipulations compared to active therapies in the short term (1 month) were (SMD −0.43; CI 95% −0.86 to 0.00; p = 0.05, I^2 = 79%). This result, although statistically significant, shows a small effect size. Even more, the CI is very wide and includes the no effect (0.00) and large effect (–0.86), which generates imprecision in the result. In addition, the heterogeneity of the study is high, causing inconsistency in the results (72). Regarding the effects of manipulation at 3 and 6 months, the results are (SMD −0.68; CI 95% −1.14 to −0.23; I^2 = 73.7%) and (SMD −0.72; CI 95% −0.99 to −0.45; I^2 = 0%) respectively. The results at 3 months improve slightly compared to those at 1 month; however, there is still a high heterogeneity and the CIs remain wide. Although the results at 6 months are ostensibly better, it can be doubted that this improvement is only due to the effect of the manipulations because, according to some authors, this improvement may be due to the natural history of the patient and other nonspecific factors (73).

Regarding the mobilizations, the results are smaller in the short term (SMD −0.20, CI 95% −0.35 to −0.04; p = 0.01; I^2 = 0%). Although they are statistically significant, the effect size goes from “not substantial” to small. The intermediate and long-term results were not significant.

In terms of disability, the results were very similar. Short-term manipulations compared to active comparators (SMD −0.86; CI 95% −1.27 to −0.45; p < 0.0001, I^2 = 46%) showed a CI that was again very wide with an effect size between small and large. The results at 6 months (SMD −0.71; CI 95% −0.98 to −0.44; I^2 = 0%) were better, but as with the pain outcome, this improvement could be attributed to the natural history of the patient and other nonspecific factors (73). Mobilizations did not obtain significant results for this outcome.

In short, we conclude that the results showed wide CIs and, in some cases, a high heterogeneity. It can also be stated that manipulations obtained better results than mobilizations.

1.2.2.2.4 Back School

The Back School originates in Sweden, where Zachrisson-Forsell in 1969 introduced the concept of “Swedish back school” (74). Currently, there is no unique content that defines the Back School and its duration may vary depending on the different existing types. As a general rule, the content of the Back School could be defined as information delivered to the patient about aspects such as the anatomy of the back, biomechanics, optimal posture, ergonomics, and back exercises (75).

The CGL considers that the Back School can be beneficial if performed in the occupational setting for the treatment of CLBP in patients who have not yet returned to their normal activities after 6 weeks or when there is a clear need to carry out a rehabilitation (30). Nevertheless, the EGL does not recommend the Back School for the treatment of CLBP when it is intended to obtain a long-term benefit. The EGL recommends the Back School for the short term only if the information provided is evidence-based (31).

Next, we summarize the evidence found in SRs published in the last 5 years on the effectiveness of Back School for the treatment of CLBP.

Only one Cochrane SR was found. The objective of Parreira et al. (76) was to determine the effect of Back School on pain and disability for adults with chronic non-specific LBP. They included 30 studies with a total of 4,105 subjects, comparing Back School with no treatment, medical care, passive physiotherapy, and exercises. The quality of the evidence ranged from very low to low. Furthermore, the results showed no difference in favor of Back School, so its effectiveness for the treatment of CLBP is uncertain.

1.2.2.3 Patient education

Patient education could be a way to provide information and advice aimed at changing patients' cognition and knowledge about their chronic state to reduce fear of serious outcomes and allow the resumption of normal activities (77–79). The use of pain neurophysiology as an educational intervention (PNE) has been proven to be effective in pain-related problems other than CLBP (80–82). Other types of patient education include interventions specifically focused on ergonomy and exercises based on anatomic and biomechanical models. In contrast, the neurophysiology of pain as a type of educational intervention for chronic pain patients focuses on describing the mechanisms of peripheral and central processing of the nociceptive signal and explaining how this transmission is modulated by brain processing and influenced by psychosocial factors. Thus, patients learn that the meaning of their pain may not always be related to the tissue damage of painful structures (82).

The CGL considers that the education based on lumbar symptoms is effective and should be provided individually. This education should focus on the symptoms of the lower back and how the patient should take care of his/her back (30). On the other hand, the EGL does not specify the most effective type of education but recommends that it must be of short duration and provided by a physiotherapist or a physician, with the aim of returning to normal activities and reducing sickness absence and disability associated with CLBP (31).

Next, we summarize the evidence found in SRs published in the last 5 years on the effectiveness of patient education for the treatment of CLBP.

Two SRs were found. The SR of Ainpradub et al. (83) compared education programs to no education program. Only two studies (208 subjects) assessed pain intensity and disability in the intermediate-term for CLBP. The results were not statistically significant for both outcomes. In addition, the results had substantial levels of heterogeneity (>85%). One possible explanation for these levels of heterogeneity is the type of pain education provided in each study. Pires et al. (84) used a PNE, whereas Sahin et al. (85) used a Back School program. In fact, the study of Pires et al. (84) showed better reductions on pain (MD –17.80) compared to the study of Sahin et al. (MD –0.71). However, because of the low quality of the evidence, no firm conclusions can be made.

The SR of Tegner et al. (86) evaluated the effect of PNE for patients with CLBP compared to placebo, no treatment, waiting list or other control interventions, and pharmacological treatments. They also compared individual PNE versus PNE in groups. They included 5 studies (212 subjects) reporting on pain at short term, and three studies (116 subjects) at 3 months follow up. The effect of PNE on disability was measured in seven studies (313 subjects) for the short term and four studies (170 subjects) at 3 months follow up.

The review authors concluded that there was moderate evidence that PNE has a small to moderate effect on pain for CLBP patients at short term. There was low evidence of small to moderate effect on disability at short term and small to moderate effect on pain and disability at 3 months follow up. No firm conclusions could be made on the superiority of any format of PNE (intensive one-on-one, small group tutorial or large group seminars lasting up to three hours) (86).

In conclusion, we may establish that patient education has better evidence and better results when the PNE is used.

Table 3: Manual therapies for CLBP

THERAPY	RESULTS
THERAPEUTIC MASSAGE	• No definite conclusions. The quality of the evidence is low or very low. Studies of small size with methodological flaws (67).
MUSCLE ENERGY	• Unreliable results. Poor quality of evidence. Studies of small size with high risk of bias (69).
MANIPULATION/ MOBILIZATION	• Manipulations obtain better results than mobilizations (71). • Unreliable results. Results with wide CIs and high heterogeneity (imprecision and inconsistency) (71).
BACK SCHOOL	• Uncertain results. Low to very low quality of evidence (76).
PATIENT EDUCATION	• Better results and better evidence with the pain neurophysiology education (83,86).

1.3 The biopsychosocial model and the neurophysiology of pain

In this chapter, we do not intend to make an in-depth analysis of the biopsychosocial model but rather to explain it briefly, justifying why this model is the reference model in this doctoral thesis, and the importance and relevance of the neurophysiology of pain as an explanation and justification of this model.

Nowadays and since the end of the last century, the health sciences field has been divided between two models of illness, the biomedical and the biopsychosocial.

Both models have positives and negatives, detractors and followers; we will present both models to later present arguments both for and against.

The **biomedical** model has been the dominant model during the last century, and has its origin in Virchow's claim that cellular abnormalities are the cause of all disease (87). As Engel stated (88): “The biomedical model embraces both reductionism, the philosophic view that complex phenomena are ultimately derived from a single primary principle, and mind–body dualism, the doctrine that separates the mental from the somatic.” This reductionist view of disease goes hand in hand with a set of beliefs (87):

- All illness has a single underlying cause
- Disease (pathology) is always the single cause
- Removal of the disease will result in return to health
- The patient has no responsibility for his illness
- The patient is a passive element in the recovery process

It must be remembered that the biomedical model has been effective in the diagnosis and treatment of many diseases. However, there are many other diseases for which this model has proven to be ineffective because it has not found the only underlying cause and the biomedical treatments proposed have been shown to be ineffective (89).

The fact that the biomedical model defines disease solely on the basis of biological indicators has the consequence that, in some cases, there are people who obtain positive laboratory results and are told they need to follow a treatment even though they feel well. Other people feel sick yet do not receive positive results in the laboratory and are told they are healthy, not sick, which generates a great contradiction (88). A clear example of the latter is the degenerative lesion of the intervertebral disc. On the one hand, we find people whose disc degeneration can be the cause of their pain and disability, so they are diseased; and on the other hand, we have people with the same disc degeneration but who are asymptomatic.

This shows that the pathoanatomical and pathophysiological connection between disc degeneration and pain and disability is unclear (89).

The **biopsychosocial** model emerged as an alternative to the biomedical model. Engel was the first person to endorse the idea that disease is a complex synthesis of aspects related to the body (bio), the mind (psycho), and the social environment, coining the term "biopsychosocial" (88). The biopsychosocial concept was developed as a model of disease in response to the missing dimensions of the biomedical model (90).

The biopsychosocial model can be applied to pain in addition to disease, giving rise to the biopsychosocial model of pain. Each person experiences pain in a peculiar and subjective way, and that experience is influenced by the psychological and socioeconomic aspects that in turn influence and modulate both the person's painful response and his/her disability (91). In fact, authors such as Gatchel et al. (92) or Waddell (93) recognize the utility of the biopsychosocial model of pain. Thanks to this model, we can better understand the processes that lead to spinal pain and its subsequent disability, and in this way, adapt the assessment and treatment to this model (94). So spinal pain can only be understood and treated on the premises of the biopsychosocial model (93).

Although the biopsychosocial model has been apparently useful in the management of pain, some authors question its efficacy. Weiner (89) underlines the fact that in the biopsychosocial model, too much attention has been paid to psychosocial aspects, giving less importance to the bio aspect, which is not entirely correct considering the subsequent pathology is not clearly defined. In line with this, other authors ask themselves what we should treat when dealing with the psychosocial aspects of pain and at what level their treatment should be directed, also taking into account that a realistic prediction of the results cannot be made (95). Moreover, although it is evident that the role of each of the three aspects that makes up the biopsychosocial model must be taken into account to understand the etiology and prognosis of low back pain, this interaction has not been addressed in the same way in the fields of research and clinical practice (96). Nevertheless, other authors argue that the problem of the biopsychosocial model is not its content but its application, the restrictive way in which it has been understood and used (96), and in the fact that sometimes health professionals are not properly educated and trained in this model (92,97). This is the case with primary care, where the established system limits opportunities to apply the biopsychosocial model, so that the psychosocial aspects of the pain experience remain unaddressed (96).

In fact, advances in the knowledge of the neurophysiology of pain have helped increase understanding of the relationship and interaction between the biological and psychosocial aspects within the pain experience of the person. It can be stated that there are biological effects derived from the psychological processing, and these biological effects, in turn, have consequences on the psychosocial context of the person (98). The latter help us understand the importance of the brain in all biopsychosocial aspects. Without knowing or understanding the human brain, it is difficult for us to apply the biopsychosocial model in a complete way. For example, a person's beliefs and evaluation processes may make an individual decide to ignore the pain and continue with his/her job and social life, whereas another may decide that his/her pain is disabling and needs to leave work temporarily, assuming the sick role (22). It is therefore necessary to know the mechanisms through which brain processing influences the response at the body level. The way in which the brain perceives a situation or a problem influences its response, so that this response is not influenced only by tissue damage or dysfunction. Additionally, alterations in the processing of the central nervous system can be the cause of the maintenance of pain and the increased sensitivity in tissues where there is no evidence of injury or in tissues that have been injured but have completely healed (99). Research in neuroscience has helped us to understand the different dimensions of pain, such as the affective and sensory, and has shown how emotions cannot be separated from the pain experience (100), which gives a scientific justification that pain is biopsychosocial in essence.

Furthermore, the International Association for the Study of Pain defines pain as (101): “An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” This definition of pain takes into account the three aspects that comprise the biopsychosocial model. Still, some authors think this definition should be revised (102,103).

As previously mentioned, the neurophysiology of pain has helped us understand and justify the biopsychosocial model at a clinical and research level. Perhaps, the important thing about applying the biopsychosocial model is that we teach patients with chronic pain all the knowledge we have learned about the neurophysiology of pain. Gifford said in 1998 (99): “Education about pain that includes the modification of commonly held ‘abnormal structure/mechanics’ related beliefs about pain is seen as vital to successful rehabilitation and outcome.” In agreement with this, other authors have used the neurophysiology of pain as an educational intervention with the aim of increasing patients' knowledge about their pain, resulting in a reduction in catastrophic thoughts, pain, and disability in the short term. These same authors state that information on the biology of pain is the means to justify the utility of the biopsychosocial model in the rehabilitation of patients with chronic pain (104).

It is shown above that the biopsychosocial model has not been used correctly and completely. Until the biopsychosocial model is fully accepted and integrated into the field of research and clinical practice, we cannot make a true assessment of its usefulness (96). Finally, going back to the contribution of Engel in the development and implementation of the biopsychosocial model and taking into account all the criticisms that may arise from its use, the words of Carrió et al (105) are interesting: “His biopsychosocial model was a call to change our way of understanding the patient and to expand the domain of medical knowledge to address the needs of each patient. It is perhaps the transformation of the way illness, suffering, and healing are viewed that may be Engel’s most durable contribution.”

1.4 Neurophysiology of pain: pain modulation

As already explained above, the International Association for the Study of Pain definition of pain is a definition that takes into account the biopsychosocial aspects of pain. To understand pain from a biopsychosocial model, it is necessary to know and understand the neurophysiology of pain.

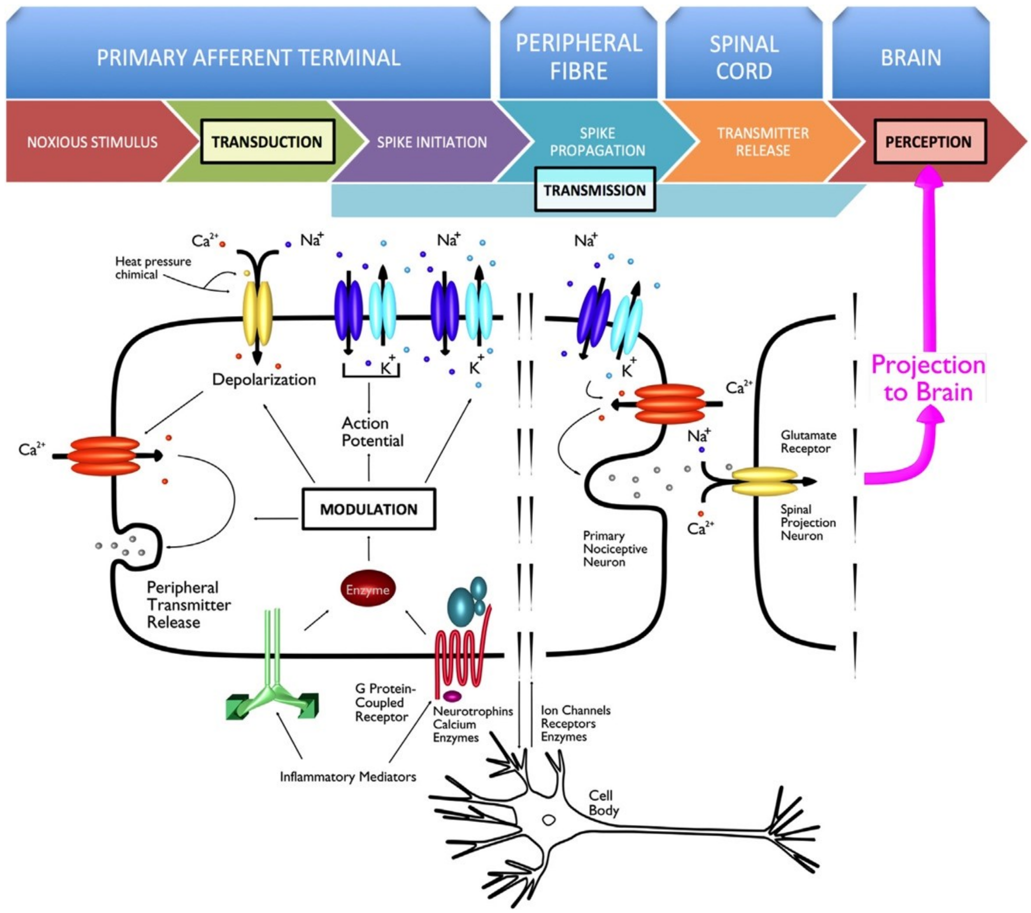
The neurophysiology of pain is a process covering the entire human body, from the periphery by detecting stimuli via the peripheral nervous system and more specifically through nociception (although nociception is also transmitted through the central nervous system) to the brain through the transmission and reception of stimuli by the central nervous system (106,107).

The neurophysiology of pain is a very broad field to cover in a doctoral thesis. As such, in this section, we will focus only on the modulation of pain, as it is the part of the neurophysiology of pain we believe can best help patients understand their chronic pain. It is for this reason that in this section we will not address aspects that are also relevant in pain such as nociception in the peripheral system and will focus mainly on the central nervous system.

Nociception is the neuronal mechanism by which a stimulus potentially harmful to the organism is detected by the individual without the need for being aware of said stimulus (108). This implies that when we perceive a potentially harmful stimulus in the periphery, first the stimulus is detected and then it is transmitted to the brain. This is what we call the phases of nociception (Figure 1) (109): transduction, transmission, modulation, and perception.

Figure 1: Phases of nociception, from noxious stimuli to brain perception.

From Mertens et al. (110).



As mentioned earlier, we will focus on the modulation phase. Modulation is a key aspect of the processing of noxious stimuli. These noxious stimuli can be modulated by up- or down-regulation at any stage of the nociceptive pathway, from the periphery through the central nervous system to higher brain centers (108). The modulation of pain is exerted in two ways, facilitating the nociceptive stimulation or inhibiting it. In addition, this ability of our body to modulate pain is vital for our survival (111). It is clear that ultimately the stimulus reaches the brain and therefore the brain is paramount in the processes of perception and modulation of pain. In short, pain is in the brain (108,111,112).

There are different parts of the brain related to nociception (Table 4) and in particular to the modulation of pain that have been identified by neuroimaging studies (Figure 2). In addition, it is important to bear in mind that although all these brain areas interact to give rise to the pain experience of the individual, they are also involved in other brain processes such as cognition, emotion, motivation, and sensations. Moreover, these interactions through the descending pain modulatory system are a clear example of the relation between pain experience and aspects related to emotions and motivation (113).

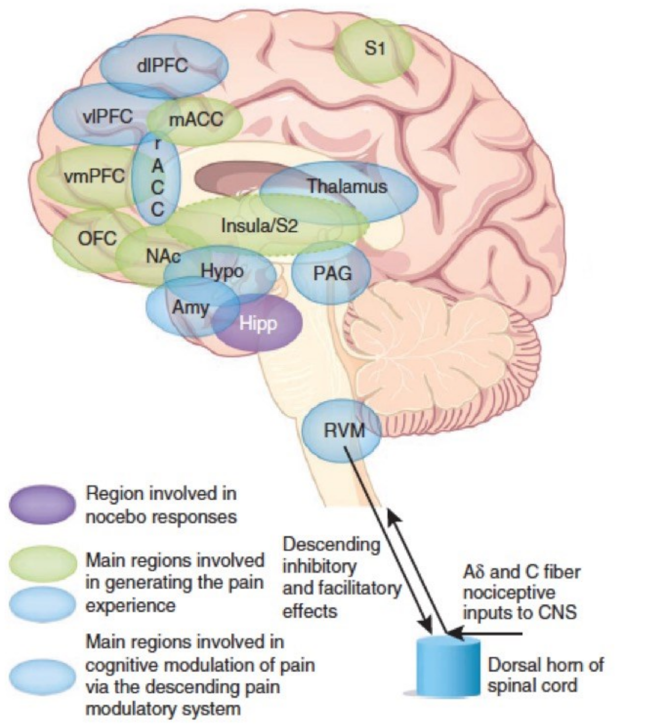
Table 4: Parts of the brain related to nociception (113,114).

BRAIN REGION ACTIVATED BY NOCICEPTION	FUNCTION
Primary somatosensory cortex and Secondary somatosensory cortex	• Sensory features of pain: quality, location, and duration
Anterior cingulate cortex (limbic system)	• Emotional and motivational responses • Affective and contextual aspect of pain
Prefrontal cortex (limbic system)	• Emotional and motivational responses • Affective and contextual aspect of pain
Insula	• Sensory features of pain: quality, location, and duration
Amygdala (limbic system)	• Emotional and motivational responses • Affective and contextual aspect of pain
Thalamus	• Pain modulation
Cerebellum	• Pain modulation
Mesolimbic (limbic system)	• Reward circuit • Emotional and motivational responses • Affective and contextual aspect of pain

Regarding the areas of the brain related to the modulation of pain, these are (115):

- Frontal lobe
- Anterior cingulate cortex
- Insula
- Amygdala
- Hypothalamus
- The periaqueductal gray (PAG)
- Nucleus cuneiformis
- Rostral ventromedial medulla

Figure 2: Brain regions involved in the pain experience. From Tracey (112).



We will divide the modulation of pain into different sections for better understanding. We have ascending and descending modulation, and in turn, this can be inhibitory (inhibits the nociceptive stimulus) or facilitatory (facilitates the nociceptive stimulus). It is important to mention that in patients with chronic pain, the modulation of pain, either ascending or descending, is altered, causing an increased and sustained state of the pain experience (115).

1.4.1 Ascending pain modulation

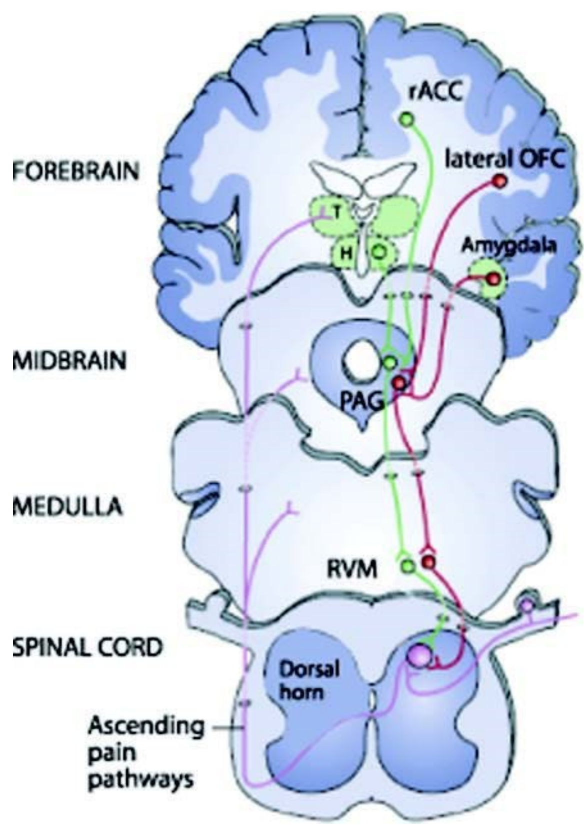
The ascending pain modulation system is also called "ascending nociceptive control" and has been described and researched since the 1990s (111,116). This system exerts its function through the spino-striato-rostral ventral medulla pain modulation pathway (117), through a pain-induced analgesia that occurs in the nucleus accumbens via opioid- and dopamine-dependent mechanisms (111).

Tambeli et al. (117) showed in a study with animals that the activation of the ascending nociceptive control through a peripheral nociceptive stimulus elicited a defensive response in life-threatening situations. Tobaldini et al. (111) proposed that threatening situations accompanied by acute intense pain activate the system of upward modulation, which in turn interacts with the descending modulation so that the latter exerts its modulating pain function when necessary and in the most optimal way depending on the situation. It has been shown that the connection path between the ascending modulation and the descending modulation is made through the parabrachial complex. When the nociceptive stimulus occurs, it ascends to the parabrachial complex, which connects to the rostral ventromedial medulla (RVM) from where the descending modulation is activated (118).

1.4.2 Descending pain modulation

The most important and known pathway in the descending pain modulation is that which forms the PAG-RVM (Figure 3). Cortical and subcortical structures in the brain connect with the PAG in the midbrain, which in turn projects to the RVM in the medulla. Finally, the RVM projects to the spinal cord, where it performs its control over the nociceptive system, modulating its transmission (111,113,119). Importantly, before higher-order processing is conducted, this descending modulation interacts with the nociceptive input in the spinal cord to allow an appropriate modulation of this input resulting in the suitable pain experience for an individual in a particular situation (115,120). Depending on the situation, this modulation could result in the facilitation or inhibition of the nociceptive input. The inhibitory effect of the descending modulation has the ability to block the nociceptive stimulus conveyed from the periphery in the spinal cord, which translates into a hyposensitivity or the absence of pain (121). The latter is of vital importance to humans because it lets us override pain in order to cope with critical situations and at the same time is the basis of placebo-induced analgesia (121). Moreover, a decrease in the inhibitory function of the descending modulation could be one of the causes of dysfunctional pain type syndrome (122).

Figure 3: Descending pain modulatory pathways. From Villemure and Schweinhardt (123).



Noradrenergic neurons are key in the descending inhibition. Interestingly, neither the PAG nor the RVM have noradrenergic neurons. Therefore, these two regions exert their inhibitory effect through a connection with noradrenergic nuclei, like the locus coeruleus. Ultimately, these nuclei block the nociceptive transmission in the spinal cord through the pre- or postsynaptic activation of α2-adrenergic receptors (120).

1.4.3 The RVM

The RVM is considered the last relay in the descending modulatory pain system before ending on the spinal cord. This last connection signals from two differentiated zones in the RVM, the nucleus raphe magnus and the nucleus reticulareis gigantocellularis, through the descending projections of the dorsolateral funiculus to the spinal cord, where it produces synaptic connections with the nociceptive afferent neurons, both primary and secondary (120,124). The RVM exerts its pain-modulating function both by inhibiting and facilitating the nociceptive stimulus (118,121,124).

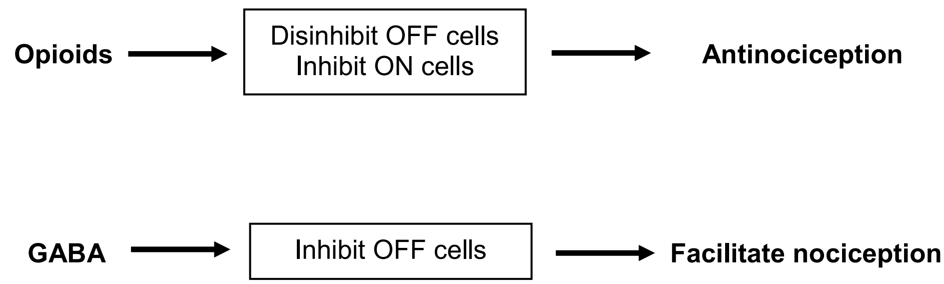
In the RVM, two different types of cell populations coexist (118,120,122):

ON cells: pronociceptive function, facilitate nociception
OFF cells: inhibit nociception, produce analgesia

These two types of cells are recruited from higher centers in a differentiated way to produce their effects, whether inhibitory or facilitatory, in a direct or indirect way. Thus, the activation of the RVM modulates nociception, influencing the pain experience of the individual (124,125).

Although many things need to be clarified about the functioning of the RVM, we know that the release of certain substances produces the inhibition or disinhibition of the different cell populations of the RVM (118,120,122,126) (Figure 4).

Figure 4: Cell populations in the RVM (118,120,122,126)**.**



In situations in which the nociceptive stimulus is maintained over time, there is an increase in both the ON and OFF cells (121,127). However, in pathological situations, such as chronic pain, there is an increase in the facilitating effects on nociception due to neuroplastic changes in the RVM. These facilitating effects on the spinal cord result in an amplification of pain (121). According to the latter, it has also been shown that the RVM has a direct relation with what we call central sensitization. This mechanism is defined as an increase in excitability of the neurons in the dorsal horn of the spinal cord as the result of a nociceptive input that can be maintained over time even after the nociceptive input has decreased or ended (27,28).

1.4.4 The PAG

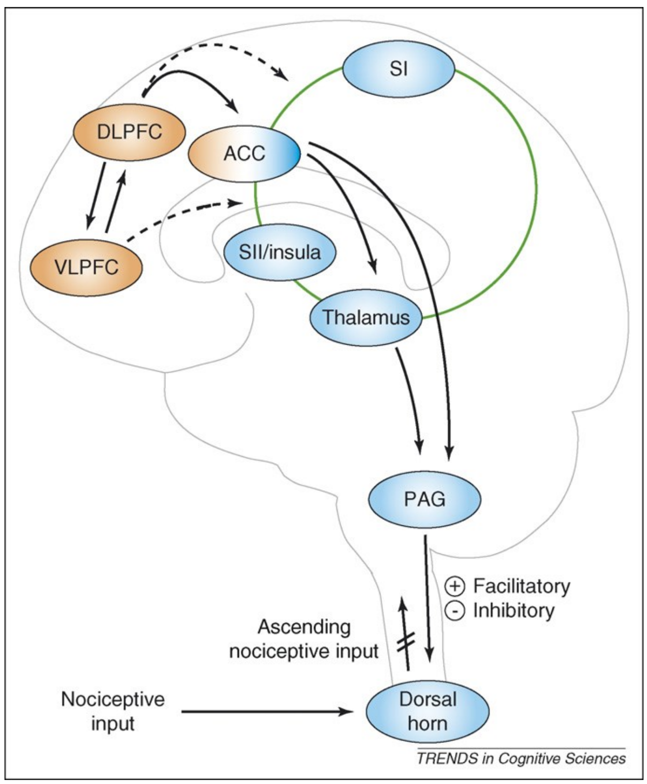
The PAG was the first recognized structure to exert its influence on the pain-analgesia relation. Specifically, the ventrolateral area of the PAG is related to the modulation of pain. The PAG exerts its anti-nociceptive function by activating the endogenous opioid system (113,115,126,128). In fact, several neurotransmitters, for example, those related to the activation of cannabinoid receptors (CB1), have been identified as activators of the modulatory pain system in the PAG. The anti-nociceptive effect of PAG is performed in the spinal cord, although not directly. Firstly, the PAG sends projections to the RVM, and the latter connects to the spinal cord. Thus, the activation of the PAG through the RVM exerts a blocking effect on peripheral nociceptive inputs in the spinal cord, preventing nociception from reaching higher centers (113,126,128).

Similar to the RVM, although less studied, the PAG contains a population of ON and OFF cells that act in a manner similar to that in the RVM in the face of nociceptive stimuli (129).

1.4.5 Cognitive and emotional factors related to the descending pain modulation

In the pain experience, the cognitive and emotional aspects are intertwined and interact to generate a response from the descending modulatory pain system (Figure 4).

Figure 5: Possible pathways of cognitive pain modulation. From Wiech et al. (130).



Regarding the cognitive aspects related to the descending modulation of pain, we find:

Attention: Attention involves increasing physiological and behavioral responses to a situation that we consider relevant. If we do not consider the situation important, our response is decreased. For example, pain always attracts our attention and it is difficult to ignore (130). The evidence has shown us that when we are distracted from pain, different brain areas are activated related to sensory, cognitive, and affective aspects of pain (131). In addition, there is a network related to attention that is part of the descending pain modulation, comprising the prefrontal cortex, anterior cingulate cortex, and PAG. When we are distracted from pain, we activate the descending modulation, releasing opioid-mediated analgesia in the spinal cord and thus decreasing the nociceptive effect (123,125,130).

- Expectation: The expectation of an event causes the organism to adjust at a sensory, cognitive, and motor level to obtain a response at the neuronal and behavioral levels. In the pain experience, the expectation that the pain intensity will be high means that once the pain occurs, it can be of high intensity. Thus, prior knowledge about a given stimulus influences our response (130). In fact, if an individual has the expectation that the pain he feels will diminish thanks to the action of a drug or a treatment, that positive expectation activates some mechanisms in the prefrontal area of the brain that release opioids in the PAG, causing the blockade of the nociceptive stimulus in the spinal cord (115,132). Therefore, a positive expectation towards pain activates the descending modulatory pain system, inhibiting nociception.

- Reappraisal: When pain is seen as a warning signal, it is perceived as a threat. The degree of threat with which an individual perceives the pain depends on his beliefs and his resources to face it. So if the individual considers that he has enough resources to deal with the pain, the pain becomes perceived as controllable and therefore less threatening (130). All this indicates that one of the possible mechanisms underlying this process is that when an individual goes from perceiving the pain as threatening to perceiving it as controllable, it is because he has carried out a reappraisal process and modified the meaning that he gave to the pain (130,133). Thus,

> the reappraisal processes can modify the perception of having control over the pain and could therefore positively influence the person´s pain experience. It should be noted that the these processes have a direct relation with prefrontal areas of the brain that depend on both the context and the personality trait of the individual (130).

In the same way that nociception and pain serve to protect the human body from possible injuries by limiting the behavior of the individual, negative moods limit our behavior to avoid exposure to danger (134).

Our emotional state directly influences pain modulation processes. In many cases, the emotional state interacts with cognitive factors, such as attention. It is easier for us to pay more attention to pain if we are under a negative emotional state, such as when we are worried or fearful (123,135). For example, negative emotions can activate the descending modulatory system in a facilitating way, thus increasing the nociceptive signals that reach the brain from the spinal cord, influencing the pain experience (123). Moreover, there is growing evidence that relates negative moods with chronic pain (134,136,137).

In fact, there is evidence from brain imaging studies that there are many similarities between mood disorders and chronic pain. These two health conditions share brain circuits of the limbic area (134). As indicated above, emotional states influence cognitive functions related to pain. This occurs because of the interaction between the amygdala and the prefrontal cortex, which influences the processes related to the pain experience, such as decision making, risk assessment, reward, or avoidance (120,138). The amygdala is a relevant brain area involved in the cerebral mechanisms related to emotions, stress, or anxiety and is part of the descending pain modulatory system. In addition, it is believed that the amygdala contributes remarkably in responses such as fear and anxiety related to the pain experience (120). Instead, the prefrontal cortex is a brain area related mainly to aspects such as cognition, motivation, emotion, and complex motor activity related to social interactions (139). Another example of this interaction between emotions and cognition is catastrophizing. Sullivan et al. (140) define catastrophizing as "a set of negative emotional and cognitive processes". This implies that an individual with catastrophic thinking magnifies the symptoms related to his pain, in addition to causing pain rumination and feeling helpless and pessimistic about his pain-related outcomes (115,141). Therefore, the pain experience may be influenced by the construct of catastrophizing, causing in the individual a modification of the attention and anticipation of pain as well as an increase in the emotional response to pain (115).

As a conclusion to this section, it is important to highlight that the variations in the pain experience we see among patients with chronic pain are determined by the context and meaning that each person gives to his pain. As explained above, this is due to the descending modulation of pain, influenced by cognitive and emotional factors, modifying the nociceptive stimulus. So the brain, and especially the brain areas related to the descending modulation, have implications not only in the pain experience but also in the cognitive and emotional processes of the individual (114). In short, pain is always in the brain and is defined as not only a sensory experience but also a cognitive and affective one (112).

1.5 Information and communication technologies in healthcare

The use of information and communication technologies in healthcare, seen as a way to transmit information to patients, is growing and becoming a powerful tool (142). In any case, the Internet is one of these new technologies that are part of our day to day. Thanks to the wide range of possibilities offered by the Internet, it is considered by different authors as an important platform from which to show information based on evidence in an interactive and high-quality format (143–148).

The data about Internet use in different countries are enlightening. For example, in 2008 in the United States, 74% of adults used the Internet, but the most relevant fact is that of that percentage, 61% used the Internet with the intention of seeking information related to health (149). In line with this, 86% of the Finnish population between the ages of 16 and 74 uses the Internet and of these, 68% do so in relation to health (150). Numerous studies have shown that websites can change and improve the knowledge of patients with chronic pain, and more importantly, have a positive impact on their attitudes and behavior (144,151–156), especially for physical and mental health problems (148,157–160). Bender et al. (161), in a SR of RCTs for the treatment of pain over the Internet, concluded that Internet-based interventions are promising for the treatment of pain, although more studies with better methodological designs are needed. More recently, Garg et al. (162) conducted a different SR to determine which web-based interventions aimed at CLBP were of benefit to patients. The review authors included nine RCTs with a total of 1,796 subjects, concluding that only online cognitive behavioral therapy showed some promising benefits in reducing catastrophization.

This shows the Internet is a tool widely used by our society for health-related information. However, there is a question that needs answered: could the use of the Internet replace the patient–therapist relationship? The studies of Keulers et al. (163) and Heikkinen et al. (164) partly answer this question. In an RCT by Keulers et al. (163) with 113 subjects with carpal tunnel syndrome, the authors compared the patient–computer interaction with that of patient–physician as tools to educate patients about their health problem. The conclusion of the study was favorable to the patient–computer interaction, in that patients who used the computer as a learning tool achieved higher levels of knowledge about their pathology than patients who were instructed by their physician. On a similar study, Heikkinen et al. (164) conducted an RCT with 147 patients having ambulatory orthopedic surgery, showing that patients receiving Internet-based education improved significantly their knowledge regarding their health and care compared to a face-to-face education group.

Primary care is one of the fields where information technology could be integrated as an adjunct to help primary care physicians with the delivery of treatments, especially to patients with chronic pain. In fact, the literature shows examples where research has been conducted in primary care. Jones et al. (165) conducted an RCT on the use of health information technology for the development of a primary-care-based shared decision-making process to educate patients with cardiovascular problems, showing that this type of intervention is well-accepted by patients and that they can be integrated in the primary routine care. Mohr et al. (166) surveyed 658 primary care patients to examine their level of interest in face-to-face, telephone, and internet treatments. They concluded that the Internet could be a good way to overcome barriers related to time constraints in primary care.

On the other hand, computers or the Internet used as instruments for the education of patients should have a series of characteristics that make them useful for all types of people. These tools should be easy to use; integrate different educational formats such as audio, video, or text; be suitable for people with low levels of literacy; and be translated into different languages that make them accessible to populations at risk such as migrants (167). In addition, design of these technological learning tools should take into account factors or characteristics that increase patient participation. As stated by Schubart et al. (149), it is important to know what characteristics make a technological tool appealing to patients, thus increasing their participation. In line with this, some authors propose that when evaluating the effectiveness of the programs used in patient education, certain characteristics of the program should be taken into consideration, such as how many times the patient has seen or used the program and how these technologies have been integrated into the general treatment program of these patients (167). Camerini et al. (168) suggest that patients should be included in the preliminary development of the programs used in their education. The same authors evaluated the effectiveness of an educational intervention on the Internet for patients with fibromyalgia, concluding that the participation of patients in the design of the intervention as well as the use of personalized content increased the overall effectiveness of the intervention.

There are other aspects related to the technological advances that could be useful when developing educational interventions in health, making them more effective. Some of these advances are not new to the public but have rarely been used with patients. One of these technological tools is video games. For example, Pessoa et al. (169) evaluated the different ways in which the Nintendo Wii platform had been used in different pathologies to improve the physical and cognitive aspects of patients. These authors concluded that when the Nintendo Wii, seen as a platform that generates virtual reality, is adapted to the characteristics and health conditions of the patients, it increases their participation and improves their motivation. In addition, using the Nintendo to perform therapeutic exercises prevents the patient from traveling to a hospital or rehabilitation center as both the exercises and their monitoring can be done from a distance.

Other therapeutic applications of video games have been seen in patients with mental and eating disorders. Claes et al. (170), in addition to using a video game in patients with eating disorders, used a system to monitor facial expression to measure the patients' emotions. Using this system, the authors demonstrated that patients' emotions could be trained using a video game. In a different study, video games were used as a complementary therapy in patients with mental disorders. The authors designed, developed, and evaluated a videogame with the aim of improving the attitude and emotional behavior of these patients. The results showed that, in the short term, patients with mental disorders managed to generate new and better coping strategies when facing negative emotions generated by stressful situations, thanks to the use of video games. (171).

Finally, gamification is one the strategies used in video games that could be useful in health management and more specifically in educational interventions. Gamification is the use of game design elements and game mechanics in non-game contexts resulting in an increase in the person's motivation and participation (172–174). In a study with patients affected by rheumatoid arthritis, the authors used an educational web platform where they assessed the effect of gamification and social support. The results of the study showed that the use of gamification in an educational intervention improved the patients' physical activity levels and their empowerment and decreased the use of health services (172).

1.6 Rationale of the study, study hypothesis, and objectives

Thirteen studies (84,175,184–186,176–183) examining the effect of PNE on physical performance, pain cognition, and disability in CLBP reported positive effects. However, there was a lack of standardization of the type of information provided. Nine studies provided detailed information concerning the PNE (84,175,177–182,184,186), whereas only six studies specifically addressed the description of the different components of the PNE (84,177–179,181,182). Although promising, there is no strong evidence for the use of PNE for CLBP patients because the quality of the evidence has been rated from very low to moderate by some authors due to the lack of good RCTs (77,86,187). Moreover, the authors in all of the studies developed the PNE without exploring the needs of CLBP patients. Therefore, the rationale of this thesis is to develop an educational tool focused on the beliefs and knowledge of patients regarding their CLBP. This rationale justifies the use of a mixed methodology wherein the author explores the patients' beliefs and knowledge about their pain for the subsequent development of an educational patient-centered tool. The authors understand that an educative tool must take into account the patients' thoughts and beliefs.

Therefore, the main objective of this thesis is to explore patients' beliefs concerning the origin and meaning of their CLBP with the goal of developing and testing a web-based biopsychosocial pain education (BPE) tool using different educational formats and gamification techniques.

The research question to be answered is:

- Is a web-based biopsychosocial pain education intervention for chronic low back pain more effective than conventional care in decreasing pain, fear-avoidance beliefs, kinesiophobia, and disability in primary care?

The hypothesis being tested is:

- A web-based biopsychosocial pain education intervention for chronic low back pain changes cognition about the origin and meaning of pain, with the outcome of pain reduction, fear-avoidance beliefs reduction, kinesiophobia reduction, and disability reduction compared to conventional care in primary care.

There are specific objectives for each phase of the study.

Phase 1 (QUAL)

- To explore patients' beliefs concerning the origin and meaning of chronic low back pain.

Phase 2 (Connecting procedure)

- To construct and develop a web-based biopsychosocial pain education intervention using the QUAL results.

Phase 3 (QUAN)

- Primary outcome. To evaluate the effect of a web-based biopsychosocial pain education intervention for chronic low back pain based on pain intensity compared to conventional care.

- Secondary outcomes. To assess the effect of a web-based biopsychosocial pain education intervention for chronic low back pain compared to conventional care on the following:

    - Fear-avoidance beliefs
    - Kinesiophobia
    - Disability

Chapter 2: Methodology

2.1 Study design

To answer the research question, the authors have used a mixed-method sequential exploratory design. The purpose of mixed methodology is not to replace qualitative or quantitative methodologies but to use the strengths of both while reducing their weaknesses (188). Specifically, the sequential exploratory design includes an initial qualitative phase followed by a quantitative phase, with the aim of developing an educational tool (189,190). In this thesis, it is proposed that both phases (qualitative and quantitative) must have the same relevance (QUAL-QUAN) for the development of the educational tool and that the development of the study must be conducted in three stages (191):

- Qualitative data collection through semi-structured personal interviews followed by thematic analysis.
- Construction of the educational tool with the results obtained in the previous step (topics or emerging categories).
- Analysis of the effectiveness of the educational tool using an RCT design.

The use of a mixed-method design is justified in this study because the integration of both methodologies (QUAL-QUAN) occurs when the data from the qualitative phase contribute to the construction of the educational tool (190).

2.2 Subjects

We performed the recruitment process independently in each phase of the study, although in both phases this recruitment process took place in the same primary care centers in the city of Lleida through family physicians. The inclusion and exclusion criteria were also common to both phases. Prior to the beginning of the first phase, the principal investigator (PI) of this study presented the project to the medical and nursing staff in each of the primary care centers involved in the study to ask for their cooperation.

2.2.1 Inclusion criteria

- History of CLBP longer than 3 months
- Patients between 20 and 65 years of age
- Able to read, speak, and understand Spanish or Catalan
- Access to the Internet, a computer or laptop, and e-mail address
- Accept and sign the informed consent form (appendices 1, and 2)

2.2.2 Exclusion criteria

Any red flag condition (192):

- Onset age <20 or >55 years
- Non-mechanical pain (unrelated to time or activity)
- Thoracic pain
- Previous history of carcinoma, steroid use, or HIV infection
- Feeling unwell
- Weight loss
- Widespread neurological symptoms
- Structural spinal deformity

2.3 Qualitative phase

For this phase, we used a generic qualitative approach. Generic qualitative research is defined as a type of qualitative methodology that is not guided by any philosophical current of those traditionally used in qualitative methodology, such as phenomenology, ethnography, and grounded theory (193). In addition, within the generic qualitative method in this phase, we used the descriptive qualitative subcategory. We chose the latter for its usefulness in mixed methodologies, as when it is conducted before the quantitative phase it provides relevant information for the development of the quantitative study (194).

2.3.1 *Subjects*

In this phase, family physicians recruited their respective patients. Once the physician made the diagnosis of CLBP and ensured that the individual met the inclusion and exclusion criteria, the physician informed the patient about this project and invited him/her to contact the author by telephone. If the person agreed to participate in the study, we scheduled the interview, which took place in the Faculty of Nursing and Physiotherapy of the University of Lleida.

2.3.2 Sample size

A purposive sample was used to achieve a representative number of patients with CLBP (195). To ensure the discursive significance of the results, we included a similar number of subjects from different age groups (9 patients between 20–50, and 7 patients between 51–65 years old), genders (8 males and 8 females), and educational levels in the sample (from general certificate education to a Master´s degree). Sixteen patients were included in this phase (Table 5).

Table 5: Participants´ demographics (n = 16).

	Participant ID code	Educational level*	Gender	Age
Patients personal interview	Patient 1	HNC	Male	46
	Patient 2	Primary Education	Female	61
	Patient 3	VET	Female	54
	Patient 4	Bachelor´s Degree	Female	64
	Patient 5	HNC	Female	50
	Patient 6	VET	Male	32
	Patient 7	Bachelor´s Degree	Male	52
	Patient 8	GCE	Male	47
	Patient 9	University Degree	Male	44
	Patient 10	VET	Male	38
	Patient 11	Primary Education	Female	60
	Patient 12	HNC	Male	50
	Patient 13	Primary Education	Female	50
	Patient 14	University Degree	Female	56
	Patient 15	Master´s Degree	Male	34
	Patient 16	Primary Education	Female	62

*HNC: Certificate of Higher Education. VET: Vocational Education and Training. GCE: General Certificate of Education.

2.3.3 Data collection

To meet the qualitative objective of this study, the authors used semi-structured personal interviews because they are useful for obtaining a better and wider understanding of the issues related to the chronic problem from the patient's perspective (196–198). The semi-structured individual interviews were conducted in 2015 individually by the PI of the study, in Spanish or Catalan (the mother tongue of the interviewer and interviewees), audio-recorded with the interviewee’s informed written consent, and later transcribed verbatim. Interviews took place in the Faculty of Nursing and Physiotherapy of the University of Lleida to generate a neutral and comfortable setting different from the primary care consultation (199). The PI used an interview guide (see appendix 3) produced after reviewing the literature and based on the experience and knowledge of the authors. Interviews were analyzed using inductive thematic analysis to identify common patterns across the interviews and generate potential themes to use in the development of the educational tool (200,201). To ensure the rigor and reliability of the material, an independent coder also analyzed the interviews (201).

2.3.4 Analysis

A thematic analysis of the transcripts was conducted using the ATLAS-ti 7 software to help with the coding process (202). First, the transcriptions were read to identify meaning units (i.e., sentences or paragraphs with the same meaning), which were open-coded with emergent codes, summarizing the content and expressing a higher level of abstraction. Second, to identify patterns across the texts, we combined and compared the resultant codes. Finally, we grouped codes under four themes.

2.3.5 Results

After analyzing the interviews, we identified four themes related to the patients' beliefs about their LBP. They are as follows: (1) structural/physiological alterations on the lower back as the cause of pain, (2) influence of the patient´s psychosocial aspects on the origin and maintenance of pain, (3) physical activity as a pain modulator, and (4) unawareness of the causes of their pain. We provide the most representative quotes throughout this section in italics.

Theme 1: Structural/physiological alterations on the lower back as the cause of pain

Pain is an alarm

The patients in this study have the perception that pain is an alarm, that when they feel pain something is wrong.

> *"Well, pain is a warning, it is telling me something is not working properly, I understand, […] but I have something that is not right and that's why I feel pain."* (Patient 9)

> *"Yes, it is a kind of alarm that tells you, beware!"* (Patient 12)

In addition, the pain indicates the anatomical location of the problem. The patients are clear that the place where it hurts is where the structure that generates their pain is located.

> *"I have the problem in my back. I notice it!"* (Patient 11)

In fact, as pain is an alarm, when the patients feel pain it means that they have done something they should not, and that has caused their pain.

> *"Sometimes the pain tells me what I should not have done and not the other way around."* (Patient 3)

For the patients, when the pain is different than usual, either because it increases in intensity or the type of pain is different, it stands for alarm, although it does not have any other accompanying symptom. This alarm forces the patient to seek medical consultation.

> *"This time I went to the doctor. Because it was not only the lower back pain but it was already coming down my left leg, and especially here, on the left side, I had a strong pain and I said "here there is something", and I thought to go to the physician in case it was something serious."* (Patient 15)

<u>The structural/physiological alterations that the patients identify as the origin of their lower back pain</u>

The patients in this study identify a series of structures or physiological alterations that they link to the cause of their pain. The patients always try to make sense of their pain by identifying a specific area or structure, or by identifying a physiological alteration. There are several structures identified in this study as the origin of LBP. These include the vertebrae.

> *"Let's see, if my vertebrae are like this, it is very clear that there must be some nerve that must be pinched or so."* (Patient 4)

The intervertebral disc.

> *"Well, on the back we have the discs. Inside the discs we have a kind of liquid that acts as a pad. When you lose what is inside, that can be lost, then*

the discs are touched, they rub against each other and that is what causes the pain." (Patient 10)

The muscles.

"I think in the end it hurts because ... I think it may be because I do not have a lumbar muscle or because it is atrophied." (Patient 15)

The nerve.

"I thought my back was locked up, a pinch or some nerve. As the prick came to my foot, I thought it was from the back." (Patient 9)

In addition to the anatomical structures of the lower back, patients blame physiological changes, such as degenerative processes and inflammation, for their pain.

"Man, let's see, there's a degeneration, right? In this area, I think that's all and little else." (Patient 3)

"I have as processes [...] as inflammatory outbreaks. I do not know why and that day I am as sore. Everything hurts, and that lasts a day, two days …" (Patient 3)

Moreover, the patients identify situations in which they believe structural damage has occurred in the lower back, and therefore, that is the source of their pain. These situations are bad postures at work or the performance of a bad gesture.

"For example, in the last four or five years I have been lifting weight. I was working on carrying drinks to bars. [...] With the barrels of beer, it's incredible! I got hurt." (Patient 10)

"Yes, at first I thought it was a wrong movement […] I thought it was a bad move, as everyone thinks, and that's it." (Patient 12)

<u>Age influences the structures that cause pain</u>

The patients link age, the fact of aging, with having more episodes of low back pain. In addition, they think that age negatively influences the structures of the lower back, causing them more pain.

> *"What happens is that I had episodes before […] I guess now with age, they have become more acute."* (Patient 3)

> *"It is not the same when you are 30 years old than 50. You already begin to notice the physical wear. I suppose that also because of the stiffness I feel more pain."* (Patient 1)

Theme 2: Influence of the patient´s psychosocial aspects on the origin and maintenance of pain

<u>The relationship between stress and the patient's pain experience</u>

The patients recognize that stressful life situations make them feel more pain, that their pain increases in intensity, and that they need to ask for sick leave.

> *"There was a time when I was a little more annoyed and I also had other things, other stress problems and things that still gave me more back pain and I was on sick leave for the first time."* (Patient 9)

There are situations in which the patients recognize that they do more than they should, which produces a stress situation that can also be aggravated by the fact of not asking for help when needed. This situation forces the patient to overexert, which ends up affecting their pain.

> *"Work, children, my mother, wanting to do everything by myself. In the sense that I did not want to bother my husband with my mother's situation. All these pile of things. I made a brutal effort and my body was not ready because it was already tense and then, I succumbed."* (Patient 11)

The patients are able to recognize specific situations in their life that cause them stress. Moreover, this stress does not only cause back pain; the patients also relate it to other pains.

> *"It had never hurt me, but […], I have a son who has given me a lot of problems, […] and I said that this was going to splatter on me one day. I started with many stomach pains, […] I noticed that my back started to ache, everything together."* (Patient 2)

> *"I believe that when you have concerns and when, […] You have other things on your mind that worry you more, that stress you out. My body realizes it, reacts. I always suffer from neck pain or back pain. Neck pain when I was younger and now more back pain."* (Patient 9)

On the one hand, although patients recognize that stress influences their pain, this is always secondary to the structural cause. Patients understand that stress may be behind their lower back pain.

> *"The nerves are worse then. When a person is nervous I think it affects him [...] it is one of those things that we do not value but I believe that nerves accumulate in your body´s weakest part and attack you."* (Patient 11)

On the other hand, it is easier for the patients to attribute the origin of their pain to a structural cause. That structural cause is a recognized alarm signal, and stress is not.

> *"When it hurts, I think the hernia is clenching me. My back is warning me, the nerve is warning me […] and it gets tense and hurts me."* (Patient 11)

<u>The recognition that emotions are part of the patient's pain experience</u>

The patients recognize that emotions can be part of the pain experience. Sometimes they identify a specific situation in which they had more pain than usual and, at the same time, they identify an emotional situation and relate it to the increase in their back pain.

“Everyone talks about very long pain processes over time. But because you start to have problems, that is, traumatological or such, and emotional problems.” (Patient 14)

On some occasions, the patients construct a story in which the emotional situation is at the origin of their lower back pain. They accept that they can "somaticize", that emotions can cause back pain.

“And right now, I do think that that time, that outbreak (of lumbar pain) so strong, I was somaticizing a very painful process for me, a lot.” (Patient 3)

The patients make a relationship between the "psychic" and emotions. In addition, they realize that there is a direct connection between improving in the "psychic" and improving their back pain.

“Everything has gone together, and sometimes the pain was better and so the psychic thing.” (Patient 14)

Theme 3: Physical activity as a pain modulator

<u>Physical activity as the cause and origin of LBP</u>

The patients link the origin of their LBP to physical activity. Specifically, they connect performing some type of exercise, such as abdominal work, with the start of their LBP. The patients relate the gym, and more specifically the use of gym equipment, with the onset of their pain.

“I started going to a gym and I think I screwed up there (the back) /…/ I didn't have anything better to do than abs with the equipment.” (Patient 16)

“I know that the sciatica came as a result of having started doing bodybuilding with equipment.” (Patient 1)

In addition, patients think part of the problem of doing physical activity in a gym is that, in their situation, they should do it under supervision. However, sometimes gyms do not have staff qualified to meet the needs of this type of client with lower back pain.

> *"But, I say that if someone taught me how to do some exercises to strengthen this area or something…"* (Patient 16)

> *"Let's see, I cannot blame a person but I think that a lot of staff that they have in gyms is not prepared. /…/ Prepared to have a person who needs more specific attention."* (Patient 1)

<u>Physical activity as a solution to pain</u>

The patients recognize that when they go to the gym they feel better. They relate the lack of physical activity and a sedentary life with their episodes of lumbar pain.

> *"Yes, in that aspect I find that I felt much better. /…/ And it was as a result of leaving the gym when the (back) pain started, not very strong but I noticed it."* (Patient 13)

> *"In the afternoon I am able to go to the gym and do elliptical and other equipment and I tell you, I notice that the movement is good for me. When I'm more static I'm worse, I feel like I'm getting cold and it's not going well."* (Patient 3)

The patients also recognize that they do not need to go to the gym to feel good, and that doing a less intense activity produces relaxation.

> *"Now I'm going to walk. I walk one hour each day. Also, it relaxes me walking."* (Patient 16)

<u>Variation in physical activity levels causes or increases pain</u>

On the one hand, the patients believe that excessive physical activity, having performed physical activity with great intensity and frequency, is the culprit of their

LBP. In fact, they identify being an athlete as the cause of structural changes in their lower back.

> *"Well, I do a lot of cycling and after a race I started to notice my leg as half numb. A pinch up here (lumbar) and I went to the doctor and ... I stopped cycling and that helped the pain to go away but when I return to do some physical activity ... It hurts again."* (Patient 6)

> *"Sportsmen are famous for being more injured than others, so there is a dehydration that makes the spring between vertebrae not work well enough."* (Patient 9)

On the other hand, the patients also believe that decreasing the amount of physical activity they used to perform has caused the discomfort to return. They have certainty that doing physical activity improves their LBP.

> *"When I stopped doing that physical activity with such intensity or with such periodicity then, I started to have discomfort (lumbar) once in a while, which I always reduced with exercise. If I exercised in the morning, in two weeks I had it (the pain) under control."* (Patient 9)

<u>The belief that not doing physical activity and having a sedentary life is related to pain.</u>

The patients realize that having had a sedentary life, not having performed physical activity, is the cause of their LBP. They recognize that during their lives they did not take into account the physical aspect, and that if they went back in time, they would be more physically active, with the belief that it would help them to solve their LBP.

> *"I do not like to look for culprits; I like to look for solutions; but in this case, if I had to find a culprit, that would be myself. /…/ For not having done physical activity for example."* (Patient 7)

“For my lower back pain (if I went back in time) I would not stop doing exercise. /…/ What I would do would be to try to take care of my fitness.” (Patient 9)

However, the patients sometimes wonder whether they should do physical activity or not, since both options can cause pain. This can confuse them when it comes to wanting to do physical activity to avoid a sedentary lifestyle.

“I think it's since I do not move so much (that I have lower back pain). Let's see, I'm not walking. I avoid walking or walking as little as possible for two reasons, one because I get tired and another because I suddenly get back pain.” (Patient 2)

Theme 4: Lack of awareness of the causes of their pain

<u>The search for a cause that justifies the pain</u>

The patients need to know the cause of their pain, what is causing their LBP. They usually contemplate several options, but in the absence of a cause that stands out from the others, they feel confused.

“It is that I do not know if it is ... It can be a little degenerative or because the physical efforts of, of wrong physical efforts. I do not know.” (Patient 10)

“If I was lying down for a few minutes I had the pain, but now I feel the pain when I'm lying down, when I'm sitting. I do not know. For example, last week I had three tremendous days (of pain).” (Patient 8)

In addition, if the patients do not identify a cause that justifies their LBP, they end up blaming themselves. They justify themselves by thinking they must be doing something wrong, and that is the cause of their pain.

“I spend many hours standing painting and now it's worse. /…/ I have to sit down. I have to find resources to move. I don't know. It may be a number of

circumstance or I may be doing something wrong and I do not know it." (Patient 9)

Lack of awareness generates fear and catastrophic thoughts

Pain causes anguish to patients. Perhaps not knowing the cause of their LBP makes them afraid of the pain.

"When I get up at seven in the morning, I'm afraid to get up. /…/ I'm scared of having back pain. God knows where this pain comes from! But it's always on the back." (Patient 5)

Moreover, not knowing the cause of their pain causes catastrophic thoughts in patients. The patients are in a continuous process of rumination about the cause of their pain and that desperation to find an answer leads them to consider catastrophic scenarios.

"I do not know what to think. /…/ It is such a strong pain that I thought I could have bone cancer. /…/ I prefer to move otherwise it hurts more. That is, when the pain radiates, I do not know what to think. I have no idea, it can be nervous." (Patient 2)

2.4 Connection phase

In a mixed-methods sequential exploratory design, the connection phase is one in which the results of the qualitative phase are used to develop the quantitative phase. In this study, the qualitative results serve to develop the educational material implemented in a website for testing in the quantitative phase of the study using an RCT. It is through the connection phase that this methodology is justified. If there were no such connection between the qualitative phase and the quantitative phase, if there were no link between both, then we would not be discussing a mixed methodology but two completely differentiated studies in which we would use the qualitative and quantitative methodologies separately.

2.4.1 Development of the educational material

Once the qualitative phase of the study was finished, its results were the starting point for the completion of the connection phase, where the educational materials were developed. In this section, we describe the connection between the results of the qualitative phase and the content of the different videos of the educational material. In addition to the videos, we developed written material to help the patients understand the content of the videos.

In this section, materials developed in other studies using the PNE, such as Moseley's and others (84,175,184,185,176–183), could have been used, but as far as we are aware, none of the studies using an educational intervention based on the neurophysiology of pain carried out a previous study exploring the needs of CLBP patients, nor did they justify the specific content of the neurophysiology of pain. Therefore, one of the objectives in this section was to develop educational materials based on the neurophysiology of pain taking into account the themes derived from the interviews. However, we have to admit that although we based the choice of content about the neurophysiology of pain on these interviews, it is ultimately the authors of this thesis making the choice. It may be that with the same information extracted from the qualitative phase, other authors would have chosen different content about the neurophysiology of pain. Even so, in the sections related to the contents of each video, we justify why that content, based on the neurophysiology of pain, is the most appropriate to explain the different themes extracted from the interviews with patients.

Explaining the neurophysiology of pain to patients in this study has two main objectives, and both are based on scientific evidence: first, to correct the patients' misbeliefs about their CLBP, and second, to reaffirm their correct beliefs.

2.4.2 Content of the educational material

The themes and subthemes that emerged from the qualitative phase formed the basis of the content of the educational material. We found 4 themes and 11 sub-themes (Table 6). To address these results, we decided to develop five videos. Our first intention was to make more videos of shorter duration. However, because of the complexity of the topics to be explained, we decided to address them in five longer videos (203). Nevertheless, these same videos were later broken down into shorter videos for the implementation of gamification in the development of the website. We will explain the latter in the quantitative phase.

Table 6: Themes and subthemes.

THEMES	Structural/physiological alterations on the lower back as the cause of pain	Influence of the patient´s psychosocial aspects on the origin and maintenance of pain	Physical activity as a pain modulator	Unawareness of the causes of their pain
SUBTHEMES	Pain is an alarm	The relationship between stress and the patient's pain experience	Physical activity as the cause and origin of LBP	The search for a cause that justifies the pain
	The structural/physiological alterations that the patients identify as the origin of their lower back pain	The recognition that emotions are part of the patient's pain experience	Physical activity as a solution to pain	Unawareness generates fear and catastrophic thoughts
	Age influences the structures that cause pain		Variation in physical activity levels causes or increases pain	
			The belief that not doing physical activity, having a sedentary life, is related to pain.	

2.4.3 Making the videos

For the technical realization of the videos, we had the help of the "Center Integral de FP ILERNA" of Lleida, through the INNOVA collaboration program. This collaborative program has the objective of vocational education and training for students to learn through the realization of projects developed by private companies. In this case, we signed a collaboration document with ILERNA for the realization of the videos by the students of the audiovisual training module, with the technical supervision of the coordinator of that module. Once we developed the contents of the different videos, we passed to them a "script" format for making the videos. This script was supervised by ILERNA's coordinator, so in most cases, the scripts were modified to aid the technical aspect. Modification of the scripts did not alter in any way the content of the educational material. The different modifications carried out throughout the process of making the videos had to do with visually explaining the content of the educational material.

Once the scripts were ready, the students of ILERNA went on to make the audiovisual content. We decided to create the videos in two formats, 2D and 3D. These two formats used together would make it easier for patients to understand the content. During this process, our mission was ensuring that the videos reflected the content we developed in a reliable way (Table 7).

Table 7: Themes and videos.

THEMES	Structural and physiological alterations on the lower back as the cause of pain	Influence of the patient´s psychosocial aspects on the origin and maintenance of pain	Physical activity as a pain modulator	Unawarenes s of the causes of their pain
VIDEOS	VIDEO 2 VIDEO 3	VIDEO 2 VIDEO 4	VIDEO 2 VIDEO 5	VIDEO 2 VIDEO 4

This phase began in October 2016 and ended with the delivery of the five videos in April 2017. The titles of the five educational videos are as follows (click on each video to watch it):

- Video 1: Presentation
- Video 2: Pain modulation
- Video 3: Origin and causes of CLBP
- Video 4: What is the relationship between pain and stress?
- Video 5: What is the relationship between pain and physical activity?

2.4.4 Development of the content of video 1: Presentation

This first video is a presentation of the website and explains the composition of the multidisciplinary team behind this thesis, which includes the doctoral student, the thesis supervisors, the thesis tutor, and the staff of the Higher Polytechnic School responsible for the development and maintenance of the website. This team comprises people from different specialties such as medicine, sociology, physiotherapy, nursing, physical activity and sports, physics, and computer science. In the video, we tell the patient the objective of the website, which is to explain everything related to his/her LBP in an understandable way.

At the content level, the only relevant aspect in relation to pain is the fact that at the end of the video, the patient's pain is validated:

> "There is no better help for a patient with chronic pain than to be able to understand what is happening to him, so that in this way he can make the best possible decision about how to face it and what steps to follow to get out of this vicious circle of chronic pain. For us, the most important thing is to show him that we understand his pain, that his pain is ALWAYS REAL."

Validation is understood as a process in which the thoughts and feelings of one person are understood and legitimized by the person who listens (204). In this study, we intend to validate the patient’s pain, considering that in many cases patients with CLBP believe that others see their pain as something unreal (205,206). In fact, if the person does not feel validated or understood by their interlocutor, it may increase his levels of physiological and emotional distress (207). Moreover, validating the patient's pain has positive effects on an emotional level, and increases their adherence to treatment, something very important when treating patients (208). Therefore, it is important when developing interventions in patients with chronic pain to validate their pain by telling them their pain is always real.

We have taken this validation process into account not only in this video but also in the development of the rest of the videos.

After watching the video, the patient can choose one of the statements from the list found right below the video. Each statement links to another video with the answer (Table 8).

Table 8: Video 1 statements and links to the explanation.

STATEMENTS	LINK TO
• Pain is always in the brain and it is the brain that decides whether to feel pain or not. • Chronic pain is no longer an alarm that indicates injury to the lower back. • The way in which our brain perceives what is happening will directly influence the way in which we move and interact with our environment.	Video 2: Pain modulation
• If I have chronic low back pain, it means that something is wrong in the lower back. • If I have a herniated disc, it is normal to have chronic low back pain. • If I had an imaging test, we would surely know why it hurts. • Having a degenerative or muscular problem in the lower back justifies the fact that I have chronic low back pain. • Sometimes I think my pain is not real because nobody can tell me why it hurts.	Video 3: Origin and causes of CLBP
• Although sometimes I feel stressed, stress has nothing to do with my low back pain. • It is normal to be afraid to perform certain activities or movements because they reproduce my low back pain and that makes it worse. • I am worried that my low back pain will get worse and may not be able to care for myself in the future.	Video 4: What is the relationship between pain and stress?
• As I have low back pain, it is better to lead a sedentary life. • Although I like it, I do not do physical activity for fear of worsening my low back pain. • I do not think that doing physical activity helps me to improve my low back pain.	Video 5: What is the relationship between pain and physical activity?

2.4.5 Development of the content of video 2: Pain modulation

Unlike other studies where similar interventions based on the PNE were evaluated, the basis of this video focuses on the modulation of pain. For example, the study by Moseley et al. (179) used a PNE focusing “solely on the nervous system.” The relevant topics of their PNE were “The nervous system,” “Synapses,” and “Plasticity of the Nervous system.” Thus, that educational material focused mainly on aspects of nociception and on central mechanisms such as “central sensitization.”

On the contrary, when explaining chronic pain to a patient, we consider it relevant to give more importance to the central nervous system and, above all, to the descending pain modulation. In addition, to help patients better understand these processes, we used the model of fear-avoidance developed by Lethem in 1983 (209). There are several reasons for using this model. Evidence has shown that patients with CLBP have high levels of fear-avoidance beliefs, and that fear of injury and pain avoidance cause disability in these patients (210,211). In addition, patients with CLBP have developed an altered interpretation of their chronic pain based on their assumption of further tissue damage and catastrophic consequences rather than evidence (212,213). When compared with patients with acute LBP whose levels of fear-avoidance beliefs decrease over time through the healing process, patients with CLBP exhibit persistent elevated levels of fear-avoidance beliefs that remain unchanged (210,214,215). Therefore, the objective of this video is to modify the meaning that patients give to their pain. We show through the video that pain is in the brain, that the pain has stopped being an alarm meaning that something is going wrong in the periphery. In short, to modify the patients’ misbeliefs with regard to the meaning they give to their pain.

The main message in this video is that "**pain is in the brain.**" To justify this statement, we used the neurophysiology of pain and more specifically the descending modulation to show the patient that it is really the brain that makes the decision whether to feel pain or not. To make that decision, the brain takes into account not only the sensitive aspects centered in the periphery but mainly the cognitive and emotional aspects that are part of the individual. Other main concepts developed in this video are shown in Table 9.

Table 9: Main concepts developed in video 2.

CONCEPTS	REFERENCES
• Pain is in the brain	(108,111,112)
• All stimuli travel to the brain	(106,107,109)
• Different parts of the brain work together to produce an answer to a given stimulus	(113,114)
• The response of the brain is influenced by personality traits, cognition and emotions	(114,115,135–139,120,123,125,130–134)
• The brain can either facilitate or inhibit the noxious stimulus through the descending modulation	(28,108,124–126,128,111,115–121)

The information contained in the video comes from the Chapter 1.4 "Neurophysiology of pain: pain modulation" of this thesis. For the development of the video script, the language was adapted for a better understanding by patients. In addition, the use of 2D and 3D images helps to understand the message. For this video, we have not produced any written material for the patients since we believe the message we intend to transmit is easily understandable through the animations and explanations of the video.

After watching the video, the patient can choose one of the statements from the list found right below the video. Each statement links to another video with the explanation (Table 10).

Table 10: Video 2 statements and links to the explanation.

STATEMENTS	LINK TO
• Pain is always in the brain and it is the brain that decides whether to feel pain or not. • Chronic pain is no longer an alarm that indicates injury to the lower back. • The way in which our brain perceives what is happening will directly influence the way in which we move and interact with our environment.	Video 3: Origin and causes of CLBP

2.4.5.1 Script for video 2

During the day, we perform many activities that include the movement of the lower back. They are every day, normal movements that do not take the tissues to the maximum stretch or travel. They are safe lumbar movements that usually do not cause us pain. So, why do we have chronic low back pain? We could also ask ourselves why sometimes the same movement one day hurts and another day does not. Most of the time, we blame the muscles, the vertebra, the nerves, or perhaps the degenerative processes, or a herniated disc. However, are these structures the real culprits of our chronic low back pain?

To begin with, we start from the basis that pain is multifactorial, not dependent on a single factor but on several. Also, pain is in the brain, always! (Concept 1) Which means that our brain, depending on the mood state, the personality type, our current situation, and the meaning we give to that situation, will decide if we feel pain or do not feel it (Concept 1,4). The latter can be understood by taking into account situations we have seen in the news or even experienced ourselves. These are situations in which because of an accident or as the result of a war injury, the person suffered serious injuries, yet did not feel pain and was able to rescue the members of his family from a burning car or from badly wounded companions in a combat situation. What does that mean? Something as simple as the brain, depending on many factors, decides whether you feel pain or not (Concept 1,4).

But let's go back to our video. Manuel performs activities that are normal. Your lower back moves or is in flexion but the stimulus that reaches the brain is very small, enough for you to know that your lower back is moving. But how does this process work?

During the activities or movements that Manuel has been doing, the stimulus that occurs in the lower back is very small. To simplify it, we will call it stimulus 3. This stimulus 3 travels through the nerve endings to the spinal cord. There, stimulus 3 can be combined with what we call the descending modulation (Concept 2,3).

To understand the descending modulation, we have to see the brain not as a single part but as the union of different parts with different tasks that come together to produce an answer. These different areas related to the pain experience are as follows (Concept 3):

- The somatosensory area: related to the location, quality, and duration of the stimulus.
- The prefrontal area and the limbic system: related to emotions and motivational responses, the affective and contextual aspect of the pain experience.
- The motor cortex: related to the motor response, to the movement.

Returning to the spinal cord, if our brain does not perceive the situation as dangerous for our lower back, in the sense that we are not afraid of the movement and therefore do not generate a stress response, stimulus 3 (and here is where you should see a 0 arriving at the spinal cord) will not be affected by this descending modulation, and will arrive intact to the brain (Concept 2). So the brain will respond only to stimulus 3, which will allow Manuel to perform the movement without pain. But what happens if, for example, the movement that we are going to do causes us fear, either because we have noticed a slight discomfort or because we have already had previous situations in which we have been "locked" and have developed a fear of almost any movement that involves our lower back? Let's see again our protagonist doing the same movement as before. Manuel, in this case, is a person who has had back pain before, so the lower back is an area that worries him. Any movement involving his back generates a certain fear. Moreover, if he can, he avoids the movement.

But let's see what happens in your brain. The limbic zone, due to the fear produced by the movement, begins to generate a response, which in turn is combined with the response of the prefrontal cortex (Concept 3), since Manuel thinks that any movement involving his lower back is dangerous. In the end, the response of all these areas is combined to generate a high stimulus, say stimulus 16, which will descend through the descending modulation. This stimulus 16 will combine in the spinal cord with stimulus 3, which is the stimulus resulting from the movement in the lumbar area. Finally, the resulting stimulus will be a 19, which is the stimulus that will reach the brain, so Manuel’s brain, in this situation, will not respond to a stimulus 3, as in the previous situation, but will respond to a stimulus 19, and as a result it will make him feel pain (Concept 1,2,3).

In this way, we have seen how the same situation, depending on how our brain interprets and processes what is happening, may or may not generate a painful response, regardless of whether there is joint degeneration, herniated discs, or muscular problems (Concept 1,4,5). This should make us think that our low back pain is probably no longer due to a hernia or a degenerative process of the tissues but to the way in which our brain perceives and interprets everything that surrounds our lower back. Then, in this case, the pain is no longer an alarm indicating that we have injured the lower back. The way in which our brain perceives what is happening will directly influence the way in which we move and interact with our environment (Concept 1,4,5).

2.4.6 Development of the content of video 3: Origin and causes of CLBP

Video 3 is related to the theme "The structural/physiological alterations on the lower back as the cause of pain" (see Table 7). Thus, the main objective of this video is to dismantle the misbelief that CLBP has a single origin and a single cause that derives from the structural and physiological alterations of the lower back. As previously mentioned, these types of misbeliefs cause fear-avoidance and over time can result in disability. In addition, to reinforce the main message, other topics are addressed, such as the excess of unnecessary imaging tests, or the little correlation between the findings in the imaging tests and the LBP. In addition, messages from the first two videos like "Pain is in the Brain" or the validation of pain are repeated to reinforce them. Other main concepts developed on this video are in Table 11. For this video, we have produced written material to help the patients to understand the message we intend to transmit (appendix 4).

Table 11: Main concepts developed in video 3.

CONCEPTS	REFERENCES
• Pain is in the brain.	(108,111,112)
• Pain is always real (validation).	(204–208)
• Pain is multifactorial.	(216,217)
• Imaging tests for CLBP are only needed when there is suspicion of a serious pathology.	(31,32,218)
• Only a small percentage of patients with CLBP develop functional limitations with age.	(219)
• The response of the brain is influenced by personality traits, cognition, and emotions.	(114,115,135–139,120,123,125,130–134)
• Lumbar disc abnormalities do not predict the development of LBP and are part of the aging process.	(219–222)

After watching the video, the patient can choose one of the statements from the list found right below the video. Each statement links to another video with the explanation (Table 12).

Table 12: Video 3 statements and links to the explanation.

STATEMENTS	LINK TO
• If I have chronic low back pain, it means that something is wrong in the lower back. • If I have a herniated disc, it is normal to have chronic low back pain. • Having a degenerative or muscular problem in the lower back justifies the fact that I have chronic low back pain.	Video 3.1
• If I had an imaging test, we would surely know why it hurts.	Video 3.2
• Sometimes I think my pain is not real because nobody can tell me why it hurts.	Video 3.3

2.4.6.1 Script for video 3

Hello, I'm Professor Valenzuela and in this video we want to show you what the scientific evidence tells us about many of the beliefs we have about the origin and causes of chronic low back pain.

Since we were little, we have been taught that pain is an alarm and that, therefore, when your back hurts, something goes wrong in your back. But, is it always like this?

When we are hit with something or make excessive movements that affect our muscles and tissues, we can feel pain and in this case the pain is an alarm. Our brain generates pain during the time necessary for our body to avoid repeating the action that generated the injury and allow it to be repaired. It generally takes about 6 weeks as all tissues tend to have healed by that time. It is what we call acute pain and it is in this situation that the brain generates pain to protect the area that is recovering (Concept 1). But what happens when the pain lasts longer than established? Is this pain still an alarm? Has it failed to fulfill that protective function?

Certainly, when our back hurts we would like to look inside, to have an imaging test done like an MRI, to see the scope of our problem and to know the reason for our lower back pain. What better than seeing directly what causes my back pain! On the other hand, scientific studies tell us that if we have a minor injury, we do not need to perform an imaging test because we usually know the reason for our pain and it will progressively heal with the help of health professionals (Concept 4). Unfortunately, most of the time patients with CLBP have no evidence of having sustained an injury; there is no evidence of any clear and precise injury mechanism. The pain started in a progressive way without an apparent reason and that is when we began to worry about the origin of our low back pain. Sometimes we even demand to have an imaging test, an X-ray or a MRI scan, to find the cause of our pain. Then, the question is, if pain is no longer an alarm, is what we see in the imaging test the cause of our low back pain?

When you ask someone with chronic low back pain about the cause of his pain, he may give you different answers, but they all have in common that the reason for the pain is always in the lower back:

- It's because of the vertebrae
- I have a herniated disc
- It is muscular
- It is a pinched nerve
- It is a degenerative problem
- I have swelling in my lower back
- I'm sure I have one leg longer than the other

As you can see, there are many answers, but are any of these answers correct? Is there only one cause of chronic low back pain?

First of all, I would like you to ask yourself the following: if my back pain is really caused by something structural, be it the vertebra, the herniated disc, the muscle, or rheumatism, why are there some days that it hurts me and others it does not? Why the same movement or the same activity some days hurts more, others less, and others does not hurt at all? It would seem logical to think that if I have an alteration in the lower back, such as a herniated disc, I always have the hernia, however, some days it hurts, some others it does not, right?

If you look at these two X-rays of the lower back, you can see that one has more alterations than the other. Does not it seem more logical to think that the person on the right has more pain than the one on the left because his lower back has more degenerative problems? However, it turns out that the one with more lumbar degeneration "experiences" less pain and this is something that often happens, although it seems hard to believe.

After what we have just explained, I'm sure you are thinking: “so if it's not because of the problems I have in my back, why do I have chronic low back pain then?” Interestingly, the scientific evidence tells us that pain is **MULTIFACTORIAL** and **BIOPSYCHOSOCIAL**. Multifactorial refers to the fact that there is not a single factor that justifies the pain but many that cause and maintain this pain (Concepts 3,6).

The biopsychosocial aspect of pain refers to the fact that these factors that generate or maintain our pain are (Concepts 3,6)**:**

- Neurobiological. How our brain and nervous system respond to different situations, releasing small substances such as hormones and neurotransmitters that influence our behavior. This causes some people to have pain whereas others in the same situation do not.

- Cognitive. How our brain perceives and interprets what happens around us. Each of our brains can interpret the same situation in different ways. For example, what for some can be a half-full glass, for others is a half-empty glass.

- Social or environmental. How what surrounds us, work, family, society, and so on could cause us stress or happiness, which directly affects us as individuals.

We give you an example: These are Manoli and Jordi. Both are the same age and work in an office. Both were diagnosed a few years ago with a herniated disc accompanied by degenerative problems in the vertebrae. However, only Manoli has chronic low back pain. Instead, Jordi, with the same diagnosis, leads a normal life. What happened so that only one of them developed chronic pain?

A few years ago, Manoli started to feel more low back pain than usual and began to worry. She had an MRI scan and was diagnosed with a herniated disc and an early stage of joint degeneration in the vertebrae. The herniated disc and vertebral degeneration seemed to be very serious things to Manoli. In fact, she thinks that her lower back pain started after lifting some boxes in her house, although she is not sure. Ever since, she has avoided lifting things or movements that involve her lower back because she is afraid that her "problems" in the lower back will worsen and will reproduce the pain. For her, the pain means that the herniated disc is getting worse and if she keeps going this way, she will end up undergoing surgery or, even worse, in a wheelchair. She has also stopped doing sports and activities that she likes. This lack of activity has also impacted her muscles and joints. She does not feel the same.

Something similar happened to Jordi. He began to feel low back pain and had some medical tests done and was diagnosed the same as Manoli. However, a health professional explained to Jordi that a herniated disc and joint degeneration are a normal occurrence in humans. In fact, most of us have had an episode of low back pain and many of us have herniated discs without pain. So Jordi, after a few days of relative rest, returned to performing physical activity supervised by his physiotherapist. Little by little, he realized that although he had some "small problems in his lower back" he could continue to do the activities that he liked, progressively and under the supervision of his physiotherapist.

In this example, we can see that Manoli's interpretation of her lower back pain generates an attitude of overprotection. Then her brain responds by feeling pain, to protect her lower back, although it is NOT NECESSARY, because after a few weeks, the tissues have healed and there is no alarm anymore (Concepts 1,6,7). On the other hand, Jordi's brain interprets what happens in his lower back as normal, so he does not need to "overprotect" it and with time, he no longer feels pain with activity (Concepts 1,6,7).

Let's go back to the scientific evidence. Recent studies tell us that all these alterations that we can observe in the imaging tests, such as those of Manoli and Jordi, are part of the natural aging process; remember that the aging process starts at the age of 20. In addition, these alterations increase as we get older. But what is more important is that the scientific evidence tells us there is no connection between these structural alterations in the lower back and the development of chronic pain (Concept 7). Manoli did not know this since no one told her.

An interesting fact to know is that between 50 and 80% of the world population has suffered an episode of low back pain in their lives. However, not everyone develops chronic pain, although they have similar diagnoses, such as in the case of Jodi and Manoli (Concept 7). Surely most of us would think that as we get older and these alterations increase in our lower back, we should feel more pain and have more functional limitations. It turns out scientific studies tell us only 7% of older people with low back pain have functional limitations. Only 7%! Did you think it was more? Well, it is not! (Concept 5)

Another interesting fact that science has found is that **PAIN IS IN THE BRAIN** (Concept 1). But do not worry, this does not mean that you are making it up, or that your back pain is not real. It is the opposite, **PAIN IS ALWAYS REAL!** It is the human brain that makes the decision whether to feel pain or not (Concepts 1,2). And more importantly, our brain makes this decision by taking into account factors such as our previous experience, the social and environmental aspects that surround us, and above all, the way our brain interprets what is happening (Concepts 1,3,6).

As you have seen in the previous example, Manoli's brain interpreted that what she had in her lower back was something "dangerous" and that, therefore, she should protect her back. Her brain generated pain, thus stopping Manoli from performing any activity that involved her lower back. On the other hand, Jordi's brain perceived what was happening in the lower back as something normal and consequently did not generate pain and allowed Jordi to carry on with his normal life.

I understand that everything you have seen and been told in this video may break somehow with what you knew about your pain. We do not pretend you can now go swim or run a marathon, but we would like you to reappraise your beliefs and attitudes toward your lower back pain. Remember that the scientific evidence has shown us that having a herniated disc, a degenerative process in the vertebrae, or any alteration at the muscular or nerve level is normal in human beings. It is part of our aging process and, more importantly, it does not have to cause chronic pain, as there are many people with the same diagnosis and they continue having active and normal, pain-free lives (Concepts 5,7).

Remember: **PAIN IS ALWAYS IN THE BRAIN AND IT DEPENDS ON HOW OUR BRAIN INTERPRETS WHAT IS HAPPENING!** (Concepts 1,2,6)

2.4.7 Development of the content of video 4: What is the relationship between pain and stress?

This video responds to the themes “The influence of the patient´s psychosocial aspects on the origin and maintenance of pain” and “The unawareness of the causes of their pain”. More specifically, the subthemes derived from the first theme refer to the connection of stress and emotions with pain. Patients in the qualitative phase recognize that stress and emotions may have something to do with their low back pain, but they are not sure what the connection is between them. Furthermore, patients also recognize that they need to know the cause of their pain, otherwise they feel confused and afraid of their pain. Therefore, this video aims to explain the connection between stress/emotions and pain, so that the patient can understand the importance that these two factors have in the origin and maintenance of their LBP. In addition, messages from the first videos like "Pain is in the Brain" or the validation of pain are repeated to reinforce them. Other main concepts developed on this video are in Table 13. For this video, we have produced written material to help the patients to understand the message we intend to transmit (appendix 5).

Table 13: Main concepts developed in video 4.

CONCEPTS	REFERENCES
• Pain is in the brain.	(108,111,112)
• Pain is always real (validation).	(204–208)
• Pain itself can be a stress factor.	(223)
• Stress and negative emotions influence the pain experience.	(98,137,224–227)
• Stress promotes inflammation, which is involved in the origin of other diseases.	(228–230)
• Reducing stress and changing negative emotions and misbeliefs improve pain and quality of life.	(223,231–235)

After watching the video, the patient can choose one of the statements from the list found right below the video. Each statement links to another video with the explanation (Table 14).

Table 14: Video 4 statements and links to the explanation.

STATEMENTS	LINK TO
• Although sometimes I feel stressed, stress has nothing to do with my low back pain.	Video 4.1
• It is normal to be afraid to perform certain activities or movements because they reproduce my low back pain and that makes it worse.	Video 4.2
• I am worried that my low back pain will get worse and may not be able to care for myself in the future.	Video 4.3

2.4.7.1 Script for video 4

In this video, we want to show you how our low back pain is affected by stress and fear. To understand these processes, it is important to remember that pain is always in the brain and that, therefore, this pain is always real (Concepts 1,2).

Surely, more than once you have related your stress to your pain and you have seen how during stressful situations you may have more pain than usual. Or, for example, you have anticipated your low back pain before performing a movement or activity; you may even have identified some movement, posture, or activity that you hardly perform for fear of triggering your low back pain. Is there a relation or connection between your low back pain and stress and/or fear?

There are two types of stress that can influence pain. On the one hand, low back pain itself can be a factor that causes us stress. On the other hand, we have environmental or psychosocial factors related to our work, or the lack of it, family, friends, emotions, money, and so on; all these factors can also generate stress. So stress, regardless of its source, can cause our back pain to start or to hurt more than usual (Concepts 3,4).

It is important to keep in mind that many times we are not aware we are suffering from stress, but still stress continues to act within our body. Now, let's see what happens inside our body when that back pain or those environmental situations cause us stress that remains over time.

When we suffer from stress, specific areas of our bodies are activated, including the amygdala and the hypothalamic–pituitary–adrenal axis. The amygdala, when activated, sends a signal to the hypothalamus that secretes a hormone called CRH. This hormone activates the pituitary gland, which in turn releases another hormone called ACTH. Finally, the hormone ACTH activates the adrenal glands and they release cortisol. Cortisol has very important functions in our body. It helps us to wake up in the morning and gives us energy if we are in a compromised situation and have to flee. In addition, it is a powerful anti-inflammatory that keeps the pro-inflammatory substances that are released in situations of stress at bay. The problem comes when we suffer from chronic stress, because then our body exhausts the cortisol and there is a situation of imbalance in which the pro-inflammatory substances exceed the anti-inflammatory substances. This may produce pain or increase the pain that we already had, without having injured our lower back (Concept 4).

The scientific evidence tells us that stress-induced inflammatory processes are involved in different diseases such as osteoporosis, fibromyalgia, low back pain, and sciatica. In fact, when pro-inflammatory substances are released near the nerve roots of the lower back, you may experience sciatic pain without nerve injury (Concepts 4,5).

Let's see a practical example of what we just explained.

Manuel is 50 years old and has had recurrent low back pain for the last 5 years. Each time he has an episode of low back pain, his brain, specifically the prefrontal cortex, gives a meaning to the pain (Concept 1). Manuel thinks that if he continues like this he will end up in a wheelchair and need surgery. In addition, all this makes him think he will have to leave his job and stop practicing his favorite sport.

Evidently, these thoughts make him feel scared about the future consequences of his low back pain. In addition, fear causes stress, and as we have seen previously, stress activates the amygdala, resulting in the activation of the HPA axis and the release of cortisol. If this situation is maintained over time, our body runs out of cortisol. As a consequence, we will have more pain, as there will be more inflammatory substances in the body (Concept 4). In addition, this fear or concern for our pain and its consequences causes us anxiety, resulting in an increase in the perception of pain and a decrease in the movements and activities that involve our lower back.

It is important to understand that sometimes the cause of our back pain is not only in the back itself; it has to do with situations such as stress, fear, anxiety, or concerns (Concept 4). These factors produce changes at the neurophysiological level that result in the onset of pain or in an increase in the perception of pain (Concept 4).

After watching this video, we would like you to think about the things you have stopped doing because of your low back pain and analyze the extent to which stress, whether caused by your low back pain or by different factors related to your life, may be involved with your current situation (Concepts 3,4).

Reducing stress, concerns, and eliminating the catastrophic and incorrect thoughts about our low back pain will help us to improve our situation, with the result of being more active and having a better quality of life (Concept 6).

2.4.8 Development of the content of video 5: What is the relationship between pain and physical activity?

This video refers to the theme “Physical activity as a pain modulator” The aim of this video is to show patients that keeping active is better than being sedentary. It is also important to teach patients that there are different types of physical activity and that even walking can be healthy. In addition, this video shows that therapeutic exercise is a good tool to fight low back pain thanks to its therapeutic properties. Other main concepts developed on this video are shown in Table 15. For this video, we have produced written material to help the patients to understand the message we intend to transmit (appendix 6).

Table 15: Main concepts developed in video 5.

CONCEPTS	REFERENCES
• Sedentary life is unhealthy.	(236–241)
• Sedentary life worsens the pain experience.	(242–244)
• Moderate exercise is also healthy.	(245–247)
• Therapeutic exercise improves the pain experience.	(236,243,256–260,248–255)
• Therapeutic exercise should be supervised by a health professional.	(259)

After watching the video, the patient can choose one of the statements from the list found right below the video. Each statement links to another video with the explanation (Table 16).

Table 16: Video 5 statements and links to the explanation.

STATEMENTS	LINK TO
• As I have low back pain, it is better to lead a sedentary life.	Video 5.1
• Although I like it, I do not do physical activity for fear of worsening my low back pain.	Video 5.2
• I do not think that doing physical activity helps me to improve my low back pain.	Video 5.3

2.4.8.1 Script for Video 5

In this video, we want to show you how physical activity positively influences and helps in the treatment of chronic pain. We will also talk about the negative aspects derived from maintaining a sedentary life.

More or less, we all know that having a sedentary life is not good, but to what extent is it not good? Both the World Health Organization and the Centers for Disease Control and Prevention warn us that leading a life with insufficient physical activity is a major risk factor worldwide for developing non-communicable diseases, such as cancer and diabetes, as the cause of death (Concept 1). In addition, living a sedentary life, apart from not reducing the symptoms of our chronic pain, increases the sensation of pain, increases the possibilities of generating disability, makes us lose strength, reduces our range of movement, and limits us physically as our abilities diminish (Concept 2). On the other hand, physical activity produces a series of beneficial effects, not only on our general health but also on our pain experience (Concept 4):

- Reduces the possibility of generating disability
- Decreases the severity and intensity of pain
- Decreases the duration of sick leave

- Reduces the risk of getting sick
- Improves cognition
- Improves our mood
- Improves sleep
- Positively influences our quality of life
- Decreases the risk of developing hypertension
- Reduces the risk of developing cardiovascular diseases
- Decreases the risk of developing type 2 diabetes
- Decreases the risk of developing osteoporosis
- Reduces the risk of developing obesity
- Improves our Independence

As you have seen, the list of benefits of including physical activity in our daily lives is long and important. In fact, scientific evidence is growing in favor of the use of physical activity as a treatment of chronic diseases thanks to the aforementioned benefits.

Although it is clear that physical activity is beneficial in the treatment of chronic pain, very few health professionals prescribe it and, therefore, very few patients with chronic low back pain include it in their daily routines. Some of the reasons for not using physical activity as a treatment for chronic pain are the lack of information or the existence of misbeliefs, such as believing that rest is better than staying active. Some patients even argue they do not have time for physical activity or they fear that their pain will get worse. In addition, they do not know what physical activity to perform or what health professional should guide and supervise their physical activity.

First, it is important to keep in mind that physical activity can be of low and high intensity. Therefore, running or swimming is considered physical activity as well as going for a walk (Concept 3). Many people who suffer from chronic pain lead a sedentary life because of their pain. Most likely, they are people who previously practiced some type of physical activity but stopped because of their pain. It is very normal when suffering from chronic pain to be afraid of physical activity, but is that fear based on something real? Or, is it simply due to a lack of information?

The scientific evidence tells us that physical activity is generally safe and that the possible and few side effects are temporary and can be avoided by providing the patient the right information and performing physical activity progressively (Concept 5). Moreover, the scientific evidence tells us physical activity is effective in the prevention of low back pain and that, especially in those patients who lead a sedentary life and want to start being more active, a low-intensity physical activity also produces benefits (Concept 4). So, you can see that physical activity is beneficial and safe even for those suffering from chronic low back pain.

Now, let's see how physical activity helps our body fight chronic pain and improve our health.

When we perform physical activity, even if it is of low intensity such as walking at a fast pace, a series of changes occur within our body that affect our general health and our lower back pain in particular. Performing physical activity, even if it is of low intensity (Concept 3):

- Increases the heart rate
- Increases blood pressure
- Increases muscular activity

These mechanisms in turn produce three very important effects for our body (Concept 4):

- Analgesic effect
- Decreased activity in the pathways that trigger pain
- Anti-inflammatory effect

Now we will see how each of these effects helps our body improve our chronic low back pain.

The analgesic effect occurs when our body, during physical activity, releases substances such as B-endorphins and growth factors. The pituitary and the hypothalamus release B-endorphins. When the pituitary releases them, there is a peripheral effect in our organism, but when they are released by the hypothalamus, there is an effect at the central level. In this way, when the body releases B-endorphins, there is a decrease in pain, which is not bad, right? Instead, growth factors help the body to repair the tissues that may be damaged, which in turn also produces an analgesic effect (Concept 4).

When we perform physical activity, our body releases different substances such as endocannabinoids, catecholamines, and nitric oxide. These elements force our nervous system to release fewer excitatory products both peripherally and at the level of the spinal cord, so it reduces the possibility for our brain to generate pain (Concept 4).

Finally, but not least, physical activity produces a very important anti-inflammatory effect. This anti-inflammatory effect is generated by different mechanisms. On the one hand, physical activity causes our body to release a substance called IL-6, which in turn releases products with an anti-inflammatory effect in the body (IL-10, IL-1RA). In addition, physical activity reduces the mass of visceral fat in the body, which is very important as it decreases the release of inflammatory substances called adipokines. On the other hand, physical activity produces an acute stress effect that is beneficial for our body because there is a release of corticosteroids, which are substances with a very important anti-inflammatory effect (Concept 4).

Doing physical activity helps us fight against our chronic low back pain and helps us have a healthier life, with a better quality (Concept 4).

It is important to keep in mind that the intention of this video is not for you to go for a run right now, as it would surely be counterproductive because your body is not yet ready for it. Our intention is that you have enough information to start doing very low intensity physical activity, that you get back the active life you put aside because of your low back pain. You can start by simply incorporating walking into your daily routine and, little by little, increasing the speed of the step. Doing this frequently will produce the effects we have seen before. Finally, if you want to increase your physical activity, as this will help you even more in the future, we advise you to do the following:

- First, visit your physician to verify that your general health is adequate.
- Second, go to your physiotherapist, as he/she is the appropriate health professional when it comes to prescribing and supervising physical activity in people with health problems. This is what we call therapeutic exercise.

Finally, and returning to the scientific evidence, the effects of therapeutic exercise are more beneficial when it is tailored to the person, progresses slowly, and is carried out under the supervision of the appropriate health professional (Concept 5).

So, walking is healthy!

2.4.9 Website development

Staff of the Higher Polytechnic School of the University of Lleida assisted in the development of the website and the educational material. The website was developed using Drupal as a content management system. Some modules of the management system, including those related to questionnaires and video tutorials, were modified and adapted to the needs of the project. This helped to increase the versatility of the platform, resulting in better utilization of the majority of the systems (i.e., the registration modules by adding security for the available data using Advanced Encryption Standard, synchronization, and mass mailing modules).

Therefore, the platform was developed to provide patients with customized tasks that allow them to use the metaphor of the journey (the narrative as a dynamic of the game) to feel that they manage their own path, which would change negative perceptions into positive perceptions about certain actions. At all times, the patients were able to choose among different information sources, such as videos about the origin of chronic pain, 3D representations of different neurophysiological processes, and FAQs. Additionally, the patients were able to contact a specialist in the neurophysiology of pain by email. Furthermore, to reinforce patients' motivation and participation, we implemented gamification techniques (defined as elements forming part of the design of video games but used in a different context) (172–174).

2.5 Quantitative phase

2.5.1 Study design

For the development of the quantitative phase of the study, the authors used a double-blind RCT with a parallel group design. This RCT followed the recommendations indicated by the Consolidated Standards of Reporting Trials statement (CONSORT) for the development of the structure and the performance of the study (261). The authors chose the RCT design because it is the gold standard for answering research questions related to the effectiveness of an intervention (262).

2.5.2 Subjects

The recruitment process started after the end of the qualitative phase and the development of the educational tool. For the selection of the sample and obtaining the necessary authorization to carry out the study, the authors contacted the management of the primary care services of the city of Lleida. Once the authorization was obtained, a specialist technician extracted a list of patients diagnosed with CLBP from the primary care ECAP database. The primary care physicians established the diagnosis. The list contained the data of 550 patients diagnosed with CLBP by their family physicians, with an age between 20 and 65 years and of both sexes. All were attended to in the primary care centers of the city of Lleida in the last year.

The authors carried out a simple random probabilistic sampling to obtain the sample. The PI of the study, assisted by two students in the last year of studying for a degree in physiotherapy, a student of the Master´s degree in research and a PhD student in health, contacted the patients by telephone. The patients who agreed to participate in the study by telephone met with the researchers at the Faculty of Nursing and Physiotherapy of the University of Lleida, where they were informed of the study and signed the informed consent form. Prior to the signing of the informed consent form, a member of the research team ensured that the patient met the inclusion and exclusion criteria established in this study (see Chapter 2.2 Subjects).

2.5.3 Randomization

The researchers used a simple randomization technique. An external researcher from the Higher Polytechnic School of the University of Lleida generated the randomization assignment using a computer random number generator, the STATS® program (263), and kept the assignments on a computer assigned for this study that was inaccessible to the rest of the staff.

This same external researcher, using the randomized assignment, generated 60 codes, 30 for each group. These codes were composed of a letter and six numbers (U201727). Neither the participants nor the investigators responsible for enrolling the patients of this RCT could foresee the assignment because of the central allocation used for the purposes of this study.

2.5.4 Sample size

The sample size calculation was based on the study of Ryan et al. (180) and the SR of Clarke et al. (187). From these studies and taking into account other similar studies such as Ostelo and colleagues (264) and Furlan et al. (265), we assumed that pain measurement in the patients at baseline would be approximately 50 ± 18 on a scale of 0 to 100. We assumed that a reduction of 30% (15 points) in the pain scale would be sufficient to be considered clinically relevant.

Accepting an alpha risk of 0.05 and a beta risk of 0.15 (statistical power of 85%), using a one-sided test, and assuming a 10% withdrawal rate, it was necessary to include 24 subjects in each group to detect a difference greater than or equal to 15 units (assuming a baseline distribution of 50 ± 18). We assumed there would be a 10% withdrawal rate. We used the sample-size calculator GRANMO version 7.12.

2.5.5 Intervention

Once individuals signed the consent form, they were given a code and the website address to access the web platform at any time from any computer or laptop until the end of the trial. After logging into the website, patients were asked to fill in the questionnaires and the demographics information.

Once this step was completed, subjects from the control group saw a message on the screen asking them to follow the conventional treatment provided by their family physician in primary care, and they were reminded that in 2 weeks they had to re-access the website to again fill in the questionnaires. Subjects from the experimental group saw a different message requesting them to access the "educational tool" on the homepage of the website. Only subjects from the experimental group had access to the educational tool using their personal password.

2.5.5.1 Experimental group

Patients in the experimental group were asked not to take any medication or seek additional treatments during the study period, except to see their family physician if they needed to. Once the patient assigned to the experimental group accessed the website using his/her personal password delivered after he/she signed the informed consent, he/she saw a screen with general information about the website (Figure 6). In this same screen, the patient was able to choose different sections. The sections directly related to this research are:

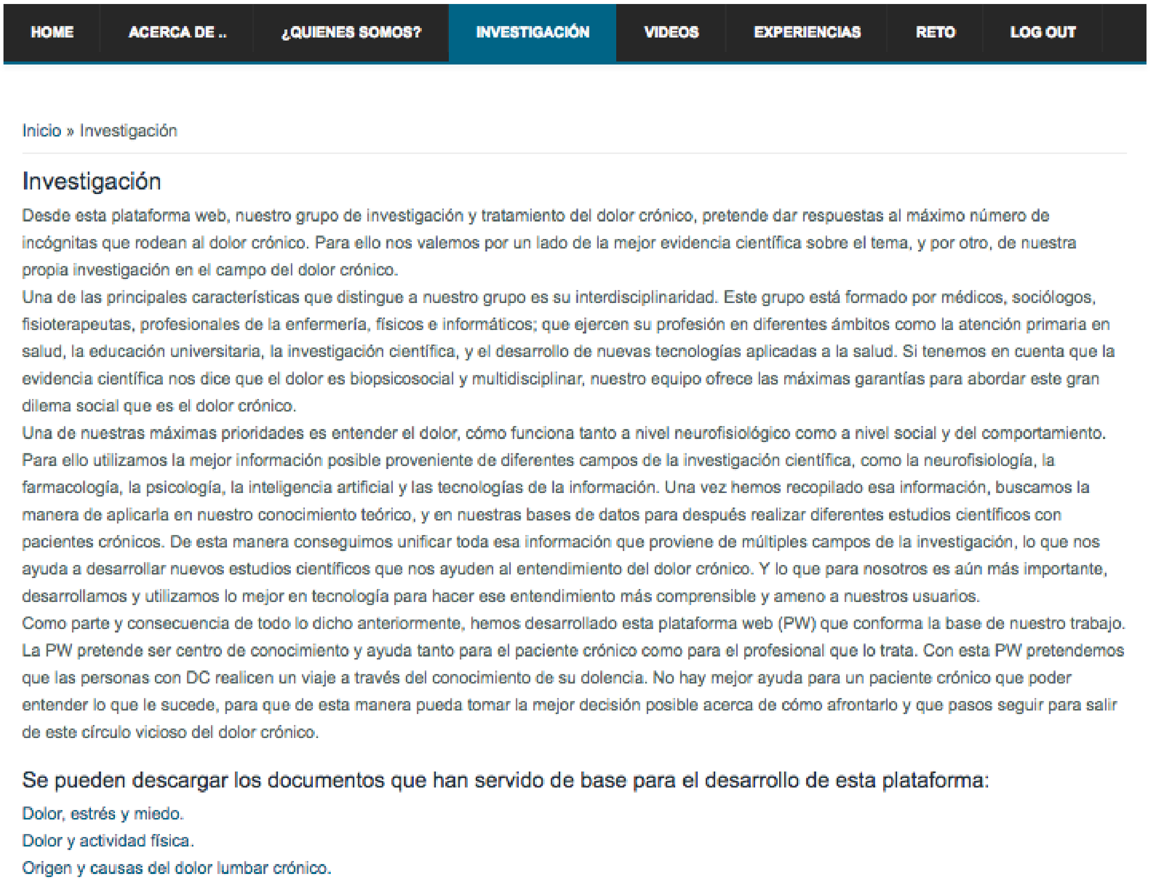
- Research
- Videos
- Experiences
- Challenge

Figure 6: Initial screen

2.5.5.1.1 Research

In this section (Figure 7), the objectives of the website are explained to the patient, and the multidisciplinary nature of the components of the research team is also mentioned. Finally, at the end of this section, patients can click on the titles of videos 3, 4, and 5, and download in PDF format the documents that have served as the basis for developing these videos (see appendices 4, 5, and 6). Hence, the patient not only had access to the educational materials developed in the connection phase in video format but also had the same information in PDF format. This PDF format was developed only for videos 3, 4, and 5 because they were thought to have the most difficult content for patients to understand, and the written format could help them understand these contents.

Figure 7: Objectives of the website



HOME | ACERCA DE .. | ¿QUIENES SOMOS? | INVESTIGACIÓN | VIDEOS | EXPERIENCIAS | RETO | LOG OUT

Inicio » Investigación

Investigación

Desde esta plataforma web, nuestro grupo de investigación y tratamiento del dolor crónico, pretende dar respuestas al máximo número de incógnitas que rodean al dolor crónico. Para ello nos valemos por un lado de la mejor evidencia científica sobre el tema, y por otro, de nuestra propia investigación en el campo del dolor crónico.

Una de las principales características que distingue a nuestro grupo es su interdisciplinaridad. Este grupo está formado por médicos, sociólogos, fisioterapeutas, profesionales de la enfermería, físicos e informáticos; que ejercen su profesión en diferentes ámbitos como la atención primaria en salud, la educación universitaria, la investigación científica, y el desarrollo de nuevas tecnologías aplicadas a la salud. Si tenemos en cuenta que la evidencia científica nos dice que el dolor es biopsicosocial y multidisciplinar, nuestro equipo ofrece las máximas garantías para abordar este gran dilema social que es el dolor crónico.

Una de nuestras máximas prioridades es entender el dolor, cómo funciona tanto a nivel neurofisiológico como a nivel social y del comportamiento. Para ello utilizamos la mejor información posible proveniente de diferentes campos de la investigación científica, como la neurofisiología, la farmacología, la psicología, la inteligencia artificial y las tecnologías de la información. Una vez hemos recopilado esa información, buscamos la manera de aplicarla en nuestro conocimiento teórico, y en nuestras bases de datos para después realizar diferentes estudios científicos con pacientes crónicos. De esta manera conseguimos unificar toda esa información que proviene de múltiples campos de la investigación, lo que nos ayuda a desarrollar nuevos estudios científicos que nos ayuden al entendimiento del dolor crónico. Y lo que para nosotros es aún más importante, desarrollamos y utilizamos lo mejor en tecnología para hacer ese entendimiento más comprensible y ameno a nuestros usuarios.

Como parte y consecuencia de todo lo dicho anteriormente, hemos desarrollado esta plataforma web (PW) que conforma la base de nuestro trabajo. La PW pretende ser centro de conocimiento y ayuda tanto para el paciente crónico como para el profesional que lo trata. Con esta PW pretendemos que las personas con DC realicen un viaje a través del conocimiento de su dolencia. No hay mejor ayuda para un paciente crónico que poder entender lo que le sucede, para que de esta manera pueda tomar la mejor decisión posible acerca de cómo afrontarlo y que pasos seguir para salir de este círculo vicioso del dolor crónico.

Se pueden descargar los documentos que han servido de base para el desarrollo de esta plataforma:

Dolor, estrés y miedo.
Dolor y actividad física.
Origen y causas del dolor lumbar crónico.

2.5.5.1.2 Videos

This section gave access to the educational material (videos and PDFs) developed in the connection phase (Figure 8). When the patient selected the “Videos section” he/she first watched video 1 “Presentation”. Once the patient visualized the video, a series of statements appeared on the screen and the patient was asked to select the statement related to his back pain with which he most agrees (Figure 9). At the same time, in the lower part of the screen, the patient could send any question to the computer technician in charge of maintaining the website. In the event the question was related to the content of the video or was related to the patient´s pain, the same computer technician referred the question to the pain expert, which was the doctoral student (Figure 10). Each statement was related to a different aspect of low back pain and was a hyperlink that led to a specific video connected to that statement. For example, if the patient had clicked on the statement "If I have chronic low back pain, something is wrong with my lower back", a new screen would have appeared with video 3 “Origin and causes of CLBP.” In this way, we used gamification through personalized tasks that allowed the patient to use the metaphor of the trip (the narrative as a game dynamic) to feel that he/she was managing his/her own path. In addition, once the patient saw the video to which he/she was directed, a series of statements related specifically to that video appeared to verify that he/she understood its content. For example, if the patient had watched video 3 "Origin and causes of CLBP", he/she would have seen a series of statements related to the content of that video (Figure 11). In this way, the patient would have been re-sent to a specific video related to the selected statement. To see the relationship between the videos and their respective statements, see Table 8, Table 10, Table 12, Table 14, and Table 16.

Figure 8: Screen video 1

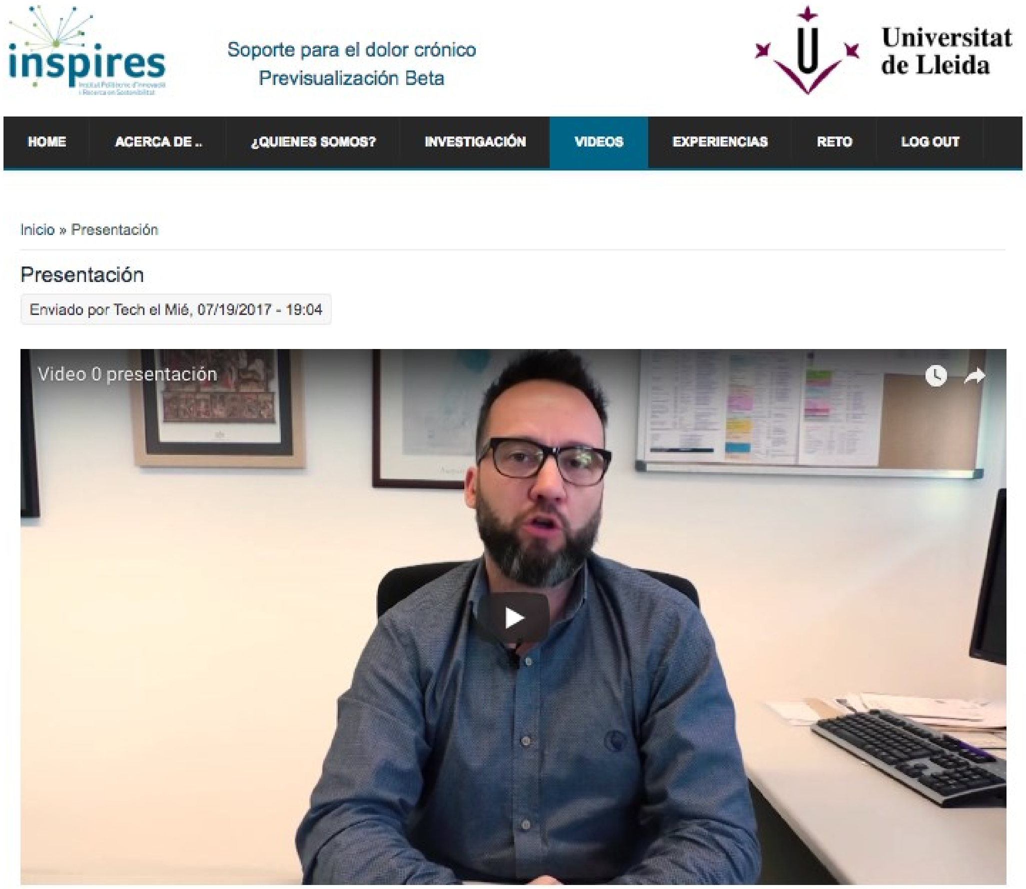


Figure 9: Screen video 2

Después de ver el vídeo, marca la frase con la que estés más de acuerdo:

El dolor siempre está en el cerebro y es el cerebro el que decide si sentir dolor o no.
El dolor crónico ha dejado de ser una alarma que indique lesión en la zona lumbar.
La manera en la que nuestro cerebro perciba lo que está sucediendo influirá directamente en la manera en la que nos movemos e interactuamos con nuestro entorno
Si tengo dolor crónico lumbar es que algo está mal en la zona lumbar.
Si tengo una hernia discal es normal que tenga dolor crónico
Si me hicieran una prueba por imagen, seguramente sabríamos por qué me duele.
Tener un problema degenerativo o muscular en la zona lumbar justifica el hecho de que tenga dolor crónico.
A veces creo que mi dolor no es real ya que nadie me sabe decir el motivo de mi problema lumbar.
Aunque a veces me siento estresad@, el estrés no tiene nada que ver con mi dolor lumbar.
Es normal que tenga miedo a realizar ciertas actividades o movimientos ya que me reproducen el dolor lumbar y eso hará que empeore.
Estoy preocupad@ de que mi dolor lumbar vaya a más y no pueda valerme en el futuro.
Como tengo dolor lumbar es mejor que lleve una vida sedentaria.
Aunque me gusta, no realizo actividad física por miedo a empeorar mi dolor lumbar.
No creo que realizar actividad física ayude a mejorar mi dolor lumbar.

Figure 10: Questions screen

Figure 11: Statements video 3

Después de ver el vídeo, marca la frase con la que estés más de acuerdo:

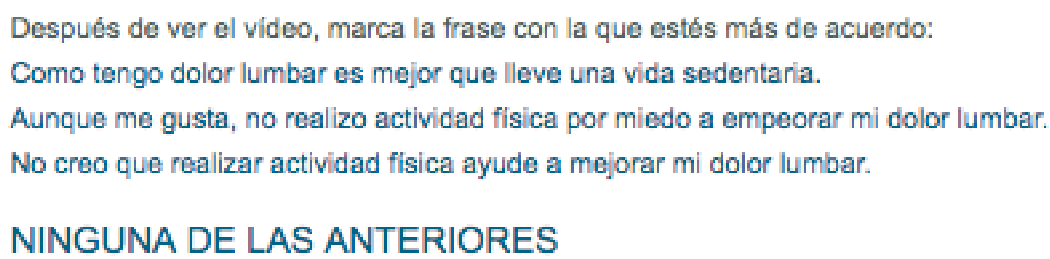
Si tengo dolor crónico lumbar es que algo está mal en la zona lumbar.
Si tengo una hernia discal es normal que tenga dolor crónico.
Si me hicieran una prueba por imagen, seguramente sabríamos por qué me duele.
Tener un problema degenerativo o muscular en la zona lumbar justifica el hecho de que tenga dolor crónico.
A veces creo que mi dolor no es real ya que nadie me sabe decir el motivo de mi problema lumbar.

NINGUNA DE LAS ANTERIORES

Specific cuts were made of videos 3, 4, and 5, so that 9 short videos were generated to respond in a more specific way to the statements selected by the patients after watching videos 3, 4, and 5.

Therefore, patients assigned to the experimental group could not directly access a specific video based on the topic of preference (stress, physical activity, etc.). The only way to access each of the main videos was by first visualizing video 1 "presentation" and then selecting the statement that best corresponded to the LBP of the patient. If the patient did not select any of the statements corresponding to the last video watched, he could select "NONE OF THE ABOVE" and go back and choose any of the statements related to the previous video (Figure 12). Therefore, each patient of the experimental group watched the videos in a certain order that depended on the statement selected at each moment. Thus, each patient followed a different and personalized path thanks to the gamification of the website.

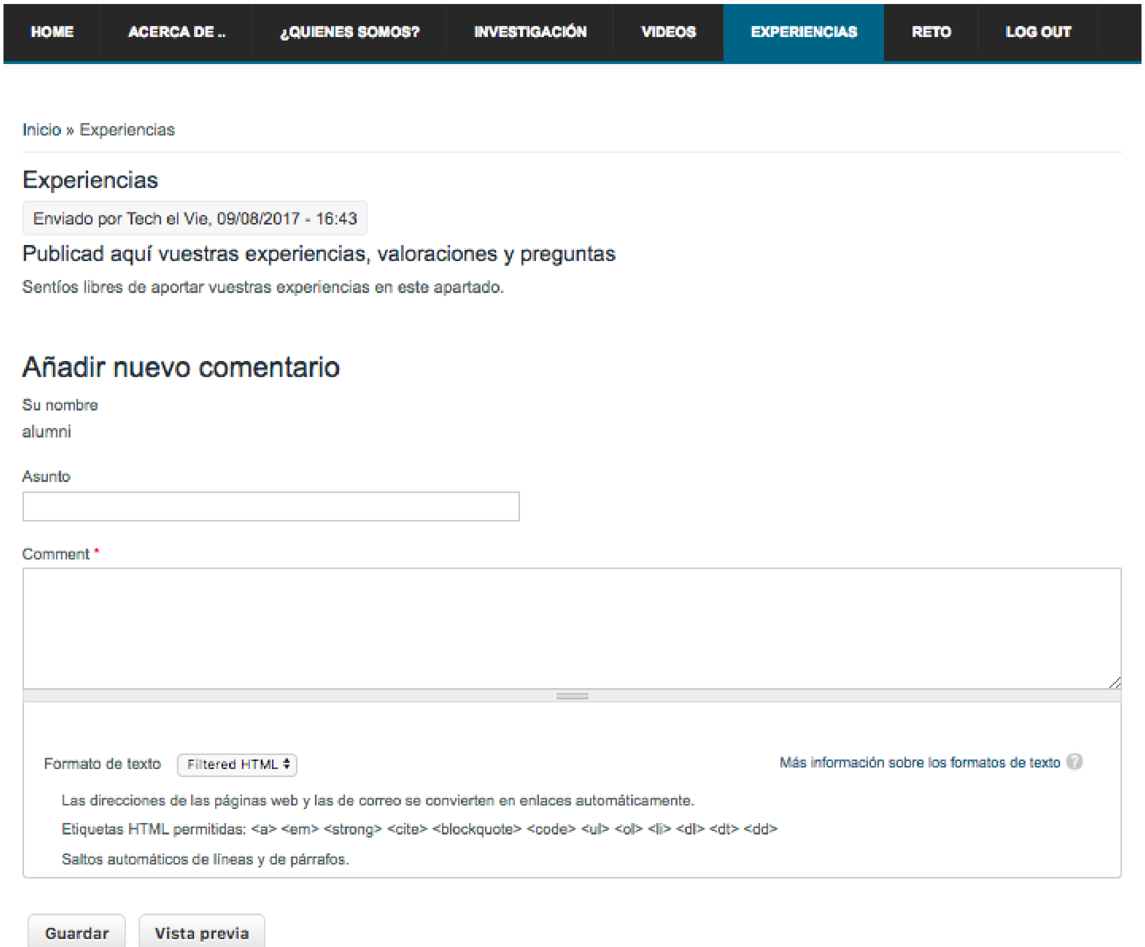
Figure 12: "None of the above"



2.5.5.1.3 Experiences

In this section, any patient could post his/her pain experience to be seen by other patients with the aim of helping each other (Figure 13).

Figure 13: Adding experiences



2.5.5.1.4 Challenge

The challenge is a gamification format where the game is used as a dynamic learning tool. In this case, the challenge served to reinforce and insist on some of the messages contained in the videos for the patient to retain them.

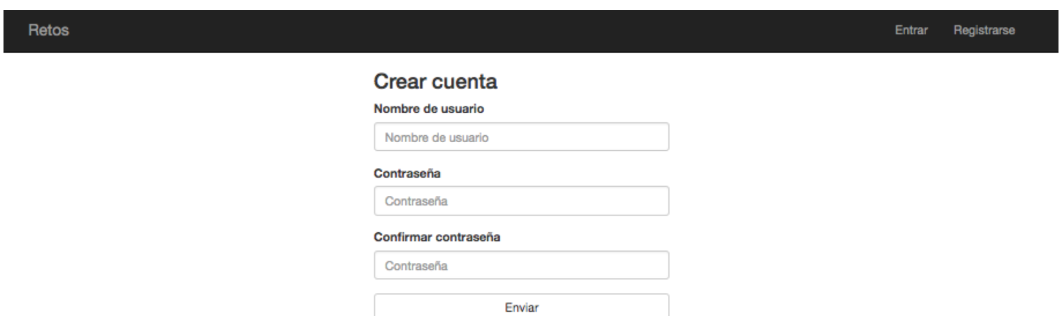
The challenge was the Hangman game and was developed by a student of the last course of the computer engineering degree as part of his final degree project. The original idea of the challenge came from the doctoral student and the engineering student was responsible for the technical development.

The challenge was accessed from the website by clicking on the top right (Figure 14). The first time a patient accessed the site, they were asked to enter a nickname and a password that may be the same as that provided when they entered the study (Figure 15). Once they accessed the challenge, the patient was asked to drag each of the images found on the left and place them in the boxes on the right. The images were as follows:

- Walk 15 minutes
- Climb stairs
- Lift weight
- Ride a bicycle
- Walk 30 minutes

Figure 14: Challenge

Figure 15: Log in to the challenge



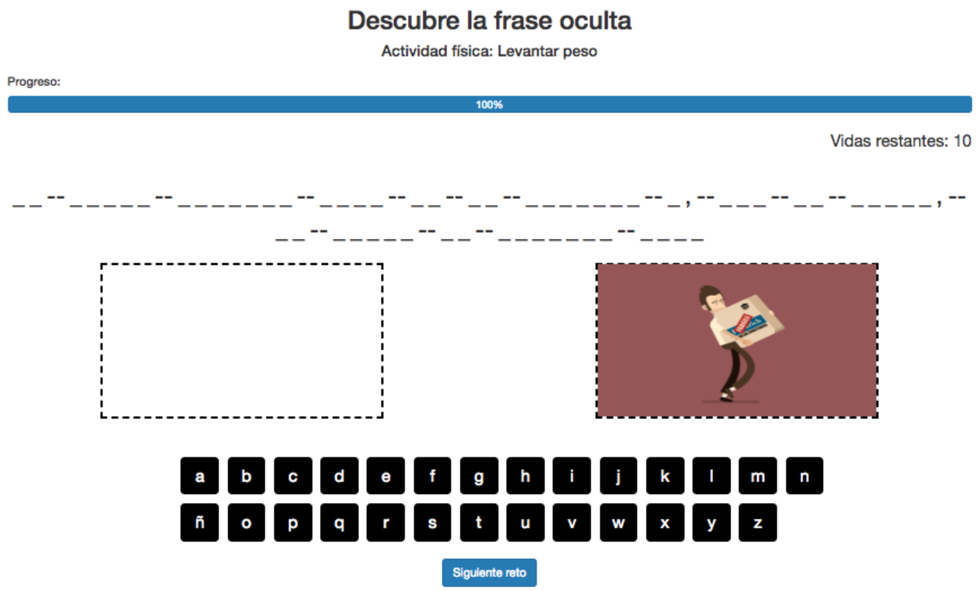
Each box on the right had a phrase on its bottom. These phrases were as follows:

- It is impossible for me to do it because of my pain.
- I could do it but I would have a lot of pain.
- I can do it but with difficulty.
- I can do it but I would have some discomfort.
- I think I can do it although I would have some discomfort.

The boxes were ordered according to the ability of the patient to perform the activities of the images taking into account their lower back pain. Once the patient had placed each image in a box, he had to select "I'm ready". At that moment, a new screen appeared with the image that the patient selected as "it is impossible for me to do it because of my pain". On the left of the screen there was the box where the hangman was outlined as the patient failed the challenge. The patient had to select the letters from the keyboard at the bottom of the screen. As the patient selected the right words, the phrase appeared at the top of the screen (Figure 16). In addition, on the upper side there was a progress bar where the patient was able to visualize his/her evolution. The patient had three lives to discover the hidden phrase. There were four hidden phrases:

- Pain is always in the brain and, therefore, pain is always real.
- There is no relationship between these alterations in the lower back and the development of chronic pain.
- The inflammatory processes derived from the stress are involved in different ailments such as low back pain and sciatica.
- Performing physical activity produces a series of beneficial effects, not only on our general health but also on our pain.

Figure 16: The hidden phrase



As can be observed, the four phrases were related to the messages contained in the educational material. Once the patient had successfully completed the challenge, he/she was awarded a diploma as "Pain Expert" (Figure 17).

As mentioned above, the aim of the challenge was to reinforce to the patient the important messages derived from the educational material through a game dynamic.

Figure 17: Pain expert certificate

2.5.5.2 Control group

Patients in the control group were asked to follow the conventional treatment provided by their family physician in primary care. They were also asked not to seek any additional treatments during the study period, except to see their family physician in case they needed to. The treatment followed by the control group was based on the CGL (30).

2.5.6 Study variables

For all the variables used in this study, whenever possible, the minimal clinically important difference (MCID) to be detected was established. Jaeschke et al. (266) defined MCID as ‘‘the smallest difference in a score of a domain of interest that patients perceive to be beneficial and would mandate, in the absence of troublesome side effects and excessive costs, a change in the patients' management.” Therefore, the MCID serves to establish if the differences detected between groups should be considered clinically important.

2.5.6.1 Pain intensity

2.5.6.1.1 Visual Analogue Scale (VAS)

This scale was developed by Huskinson in 1976 as a method to measure pain intensity (267). VAS is an easy, simple, and reproducible tool that can be used by the same patient on multiple occasions. The scale consists of a 10 cm line with a description on both extremes. “No pain” is on the far left and “worst pain ever” is on the far right of the scale. For some authors, VAS is the most sensitive measurement in clinical pain research (267–269). The MCID for the VAS has been established in 2 points (when scoring range goes from 0-10), or 15 points (when scoring range goes from 0-100) (264,270) (appendix 7).

2.5.6.2 Cognition

2.5.6.2.1 Fear-Avoidance Beliefs Questionnaire (FABQ)

The FABQ is a self-reported questionnaire that consists of 16 items about the beliefs of LBP patients regarding the influence physical activity and work have on their pain. Each item can be scored from 0 (totally disagree) to 6 (totally agree) (271,272). The total score ranges from 0 to 96, with a higher value meaning a higher degree of fear-avoidance beliefs. The FABQ is divided into two subscales. The work subscale reflects fear-avoidance beliefs about work. The physical subscale reflects fear-avoidance beliefs about physical activities (271–273). The Spanish version of the FABQ has demonstrated good reliability (273). To the best of our knowledge, the MCID for the FABQ has not yet been established (appendix 8).

2.5.6.2.2 Tampa Scale for Kinesiophobia (TSK-11)

The TSK was originally developed by Kori et al. and Miller et al. in the early 1990s (274). The original scale consisted of 17 items, each one provided with a 4-point Likert scale (275). This scale is widely used in pain medicine to assess pain-related fear. In 2005, the original TSK scale was revised by Woby et al. (274), concluding that a shortened version (TSK-11) still had similar psychometric properties but with the advantage of brevity. The total score of the TSK-11 ranges from 11 to 44 points, with higher scores indicating greater fear of re-injury due to movement (276). The Spanish format has 11 items, with each score ranging from 1 (totally disagree) to 4 (totally agree). The Spanish version of the TSK is easy to use, reliable and valid (272). The MCID for the TSK-11 has been estimated at 5.9 points (274,276) (appendix 9).

2.5.6.3 Disability

2.5.6.3.1 Roland–Morris Questionnaire (RMQ)

This is a self-reported questionnaire developed by Roland and Morris in 1983 (277) for assessing function and disability. It is an easy instrument for patients with scores ranging from 0 (no disability) to 24 (severe disabled). Each positive answer equals 1 point. A change of 4 or more points is considered clinically important (180,278). The RMQ is reliable, valid, and adequate to assess disability in patients with LBP (180,278,279), and the Spanish version has been successfully validated (279) (appendix 10).

2.5.7 Data collection

The intervention lasted for two weeks, and variables were measured pre- and post-test. The researchers based the duration of the intervention on the study of Keulers et al. (163), which had similar characteristics. An external researcher from the Higher Polytechnic School of the University of Lleida was responsible for the maintenance of the website. Moreover, this external researcher kept the data from the subjects and the questionnaires used in this study in the laptop employed for this study. Once individuals signed the consent form, they were given a password to access the web platform at any time from any computer or laptop until the end of the trial. After logging into the website, patients were asked to fill in the questionnaires. Once this step was completed, subjects from the control group saw a message on the screen asking them to follow the conventional treatment provided by their family physician in primary care, and they were reminded that in 2 weeks' time they had to re-access the website to again fill in the questionnaires.

This treatment was based on the CGL (30). Subjects from the experimental group saw a different message requesting them to access the “educational tool” on the homepage of the website. Only subjects from the experimental group had access to the educational tool using their personal password.

Both groups of subjects received two email messages during the study (one 2 days before the end of the study and the second one the last day) to remind them to fill out the questionnaires again. Ultimately, patients who after the deadline had not responded to the questionnaires were contacted by phone and sent WhatsApp messages reminding them to fill out the questionnaires.

2.5.8 Statistical analysis

The statistical analysis was carried out with the statistical program SPSS v24, using the per-protocol analysis with an alpha of 0.05. Quantitative variables were described using the mean, standard deviation (SD), standard error (SE), median, and interquartile range. Sociodemographic baseline characteristics were compared between groups by using the student t-test for continuous data and chi-square tests of independence for categorical data.

To answer the research question the authors used the two-way mixed ANOVA to determine whether there was an interaction effect between the two independent variables, treatment (control and experimental) and time (pre- and post-test). Data was assessed for outliers, normality and homogeneity of variances. As the study sample was less than 50 subjects, the Shapiro–Wilk normality test was used. To determine the homogeneity of variances the authors used the Levene´s test of equality of variances. The data was transformed if it was not normally distributed or the variances did not have homogeneity.

The MD with 95% CI was calculated to analyze continuous outcomes. We used the partial Eta Squared (η^2) as a measure of the effect size. We considered a partial $\eta^2 > 0.009$ as a small effect size, partial $\eta^2 > 0.058$ as a medium effect size, and partial $\eta^2 > 0.137$ as a large effect size (47,280).

2.6 Ethical issues

This thesis follows the Declaration of Helsinki and the “Guidelines for Good Clinical Practice” (CPMP/ICH/135/95) and has been approved by the Ethical Committee of Clinical Research in Primary Care IDIAP in Catalonia, Spain (P14/138) (appendix 11).

Because the intervention does not involve any physical activity/intervention, it was not expected to have any physical side effects. Patients in the experimental group were advised to contact their family physician if they experienced any physical problems or worsening of their condition. Additionally, all subjects in the experimental group were able to contact the author through the website, the email address, or by telephone. All patients volunteered to participate in the study. Before signing the informed consent forms, the patients were informed about the characteristics and objectives of the study and were given the opportunity to ask any kind of question related to the study.

Chapter 3: Results

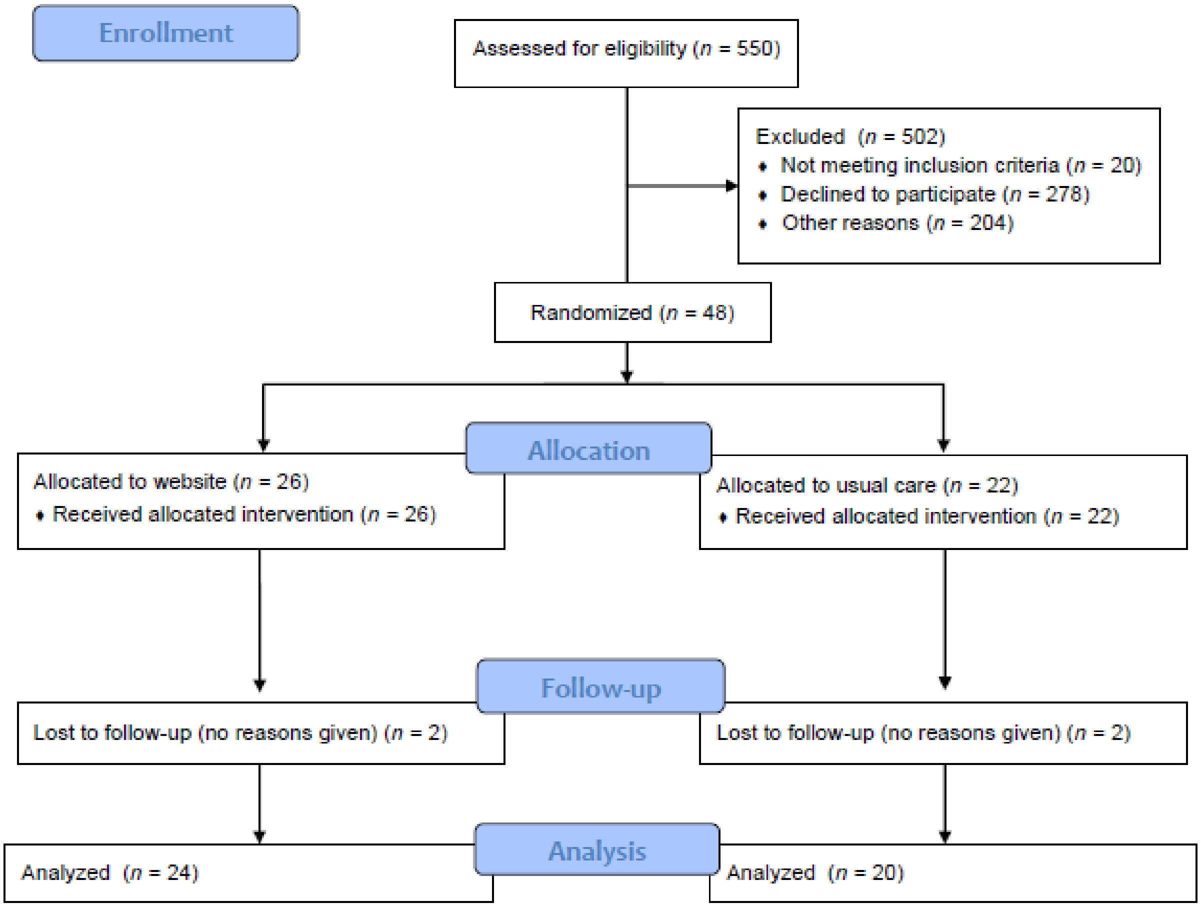
3.1 Recruitment and flow of participants

The patient recruitment period was carried out for one year, between September 2017 and September 2018. For the recruitment, and after receiving authorization from the board of the primary care in Lleida, the authors used a patient list extracted by an expert technician from the ECAP database of the primary care service of Lleida. The list contained the data of 550 patients diagnosed with CLBP by their family physicians, with ages between 20 and 65 years and including both genders. All visited the primary care centers of the city of Lleida within the last year.

The PI of this project, supported by volunteer staff of the Faculty of Nursing and Physiotherapy of the University of Lleida, made telephone calls to contact patients who were offered the opportunity to participate voluntarily in the study. Of the total of 550 patients, 20 were excluded because they did not meet the inclusion criteria, one was excluded because of the red flags described in the exclusion criteria, 278 declined to participate in the study, and 203 did not answer the telephone calls. Finally, 48 patients volunteered to participate in the study.

After the randomization process of the 48 patients in the sample, the researchers assigned 26 patients to the experimental group and 22 to the control group, as described in Section 2.5.3 Randomization. Of the 48 patients, 44 completed the pre- and post-test questionnaires and only four patients (8%) did not complete the post-test questionnaires, two from the experimental group and two from the control group. Three days before the end of the intervention, an email was sent to all the participants, reminding them that they had to complete the questionnaires again. One week after the end of the intervention, the authors tried to locate the patients who had not yet completed the questionnaires by telephone, email, and WhatsApp message to remind them do so. Four did not answer, so the reasons they did not complete the post-test questionnaires are unknown (see Figure 18).

Figure 18: Flow diagram



3.2 Baseline characteristics

The baseline characteristics were collected from the 48 patients who formed the sample before being randomized, and are presented in Table 17. At the baseline, there were no statistically significant differences between the two groups for any of the sociodemographic variables. The mean ages of patients in both groups were similar, 47.04 (SD 11.11) years in the experimental group and 45.73 (SD 8.81) in the control group.

Of the 48 patients in the sample, 28 were women (58%) and 20 were men (42%). At the group level, the proportion of women in the experimental group was 61% and that of the control group was 54%. As for men, their percentage was 46% in the control group and 39% in the experimental group.

At the work level, 11 patients (23%) of the total sample were out of work because of their LBP, and 4 (8%) received a disability allowance. In the experimental group, 19% of patients were out of work and 8% received a disability allowance. In the control group 27% of patients were out of work and 9% receiving a disability allowance.

At the medication level, 33 patients (69%) took medication for their CLBP, whereas 15 patients (31%) were not taking medications at the time of the intervention. In the control group, 72% of the patients took medication for their CLBP, whereas in the experimental group, it was 65% of patients.

Finally, 26 patients (54%) claimed to have sleep disturbances. In the experimental group, 46% of patients claimed to have sleep disturbances, whereas in the control group it was 63%.

Table 17: Baseline characteristics

		Experimental group (*n* = 26)	Control group (*n* = 22)	Total (*n* = 48)	*p* value
Age*		47.04 (11.11)	45.73 (8.81)	46.85 (9.62)	0.462[b]
Gender	Female	16 (61)	12 (54)	28 (58)	0.624[a]
	Male	10 (39)	10 (46)	20 (42)	
Working status	Active	21 (81)	16 (73)	37 (77)	0.546[a]
	Not active	5 (19)	6 (27)	11 (23)	
Disability benefit	Yes	2 (8)	2 (9)	4 (8)	0.861[a]
	No	24 (92)	20 (91)	44 (92)	
Medication	Yes	17 (65)	16 (72)	33 (69)	0.584[a]
	No	9 (35)	6 (28)	15 (31)	
Sleep disturbances	Yes	12 (46)	14 (63)	26 (54)	0.226[a]
	No	14 (54)	8 (37)	22 (46)	

Values are numbers (%) unless stated otherwise. *Mean (SD). [a]Analysed by the chi-squared test. [b]Analysed by the student t-test.

3.3 Primary outcome

The main outcome in this study was pain intensity measured with a 0–100 VAS scale. The measurements were taken on-line pre- and post-test, before the start of the intervention and after 15 days (Table 18, Table 19, and Table 20).

3.3.1 Pain intensity

A two-way mixed ANOVA was conducted to examine the effects of time and treatment on pain intensity. Outliers were assessed by inspection of a boxplot, normality was assessed using Shapiro-Wilk's normality test for each cell of the design and homogeneity of variances was assessed by Levene's test. There were outliers, pain intensity was not normally distributed ($p < 0.05$) and there was homogeneity of variances. Therefore, transformation of data for all groups was carried out using a square root transformation, creating a new variable. This new variable was assessed again and was normally distributed ($p > 0.05$) and there was homogeneity of variances ($p > 0.05$).

There was no statistically significant interaction between treatment and time on pain intensity, $F(1, 42) = 0.847$, $p = 0.363$, partial $\eta^2 = 0.020$. Therefore, an analysis of the main effect was performed for treatment and time. The main effect for treatment was not statistically significant, $F(1, 42) = 1.055$, $p = 0.310$, partial $\eta^2 = 0.025$. The main effect for time was not statistically significant, $F(1, 42) = 1.037$, $p = 0.314$, partial $\eta^2 = 0.024$.

3.4 Secondary outcomes

There were three secondary outcomes: fear-avoidance beliefs, kinesiophobia, and disability (Table 18, Table 19, and Table 20).

3.4.1 Fear-avoidance beliefs (FABQ)

A two-way mixed ANOVA was conducted to examine the effects of time and treatment on fear-avoidance beliefs. There were outliers, residuals were normally distributed ($p > 0.05$) and there was homogeneity of variances ($p > 0.05$). Outliers were included in the analysis and the results were compared with the results of the two-way ANOVA without the outliers showing that the results were essentially the same.

The interaction effect between time and treatment on fear-avoidance beliefs was statistically significant, $F(1, 42) = 4.516$, $p = 0.040$, partial $\eta^2 = 0.097$. Therefore, an analysis of the simple main effects was performed for treatment and time. All pairwise comparisons were run where reported 95% confidence intervals and p-values are Bonferroni-adjusted. For the experimental group, fear-avoidance beliefs were statistically significantly reduced at post-test compared to pre-test (MC -10.95; CI 95% –20.76 to -1.15; $\eta 2 = 0.18$; $p = 0.03$). For the control group, fear-avoidance beliefs were not statistically significantly reduced at post-test compared to pre-test (MC 2.35; CI 95% –5.65 to 10.35; $\eta 2 = 0.19$; $p = 0.54$).

3.4.2 Kinesiophobia (TSK-11)

A two-way mixed ANOVA was conducted to examine the effects of time and treatment on kinesiophobia. There were no outliers, residuals were normally distributed ($p > 0.05$) and there was homogeneity of variances ($p > 0.05$).

The interaction effect between time and treatment on kinesiophobia was statistically significant, $F(1, 42) = 5.388$, $p = 0.02$, partial $\eta^2 = 0.114$. Therefore, an analysis of simple main effects was performed for treatment and time. All pairwise comparisons were run where reported 95% confidence intervals and p-values are Bonferroni-adjusted. For the control group, kinesiophobia was not statistically significantly different between pre-test and post-test (MC 0.95; CI 95% –1.22 to 3.12; $\eta^2 = 0.18$; $p = 0.37$), but for the experimental group, kinesiophobia was statistically significantly reduced at post-test compared to pre-test (MC -1.91; CI 95% –3.41 to -0.42; $\eta^2 = 0.23$; $p = 0.014$).

3.4.3 Disability (RMQ)

A two-way mixed ANOVA was conducted to examine the effects of time and treatment on disability. There were outliers, disability was not normally distributed ($p < 0.05$) and there was homogeneity of variances ($p > 0.05$). Therefore, transformation of data for all groups was carried out using a square root transformation, creating a new variable. This new variable was assessed again and was normally distributed ($p > 0.05$).

The interaction effect between time and treatment on disability was statistically significant, $F(1, 42) = 4.379$, $p = 0.04$, partial $\eta^2 = 0.09$. Therefore, an analysis of simple main effects was performed for treatment and time. All pairwise comparisons were run where reported 95% confidence intervals and p-values are Bonferroni-adjusted. Disability was statistically significantly lower in the experimental group post-test compared to the control group (MD -4.1; CI 95% -7.53 to -0.68; $\eta^2 = 0.11$; $p = 0.023$). This represents a medium effect size. For the experimental group, disability was not statistically significantly different between pre-test and post-test (MC -0.16; CI 95% –0.52 to 0.19; $\eta^2 = 0.03$; $p = 0.35$), but for the control group disability was statistically significantly greater at post-test compared to pre-test (MC 0.33; CI 95% 0.01 to 0.65; $\eta^2 = 0.19$; $p = 0.044$).

Table 18: Results control group

Control group (n = 20)			
Outcome	**Pre**	**Post**	**p value**
	Mean (SD)	Mean (SD)	
VAS (O-100)	44.5 (15.38)	52.5 (20.74)	0.226
FABQ (0-96)	42.3 (17.81)	44.65 (18.21)	0.546
TSK-11 (11-44)	28.3 (6.62)	29.25 (7.08)	0.372
RMQ (0-24)	7 (3.71)	9.4 (6.31)	**0.044***

*Statistically significant difference. VAS: Visual Analogue Scale. FABQ: Fear-Avoidance Beliefs Questionnaire. TSK-11: Tampa Scale for Kinesiophobia. RMQ: Roland-Morris Questionnaire

Table 19: Results experimental group

Experimental group (n = 24)			
Outcome	**Pre**	**Post**	**p value**
	Mean (SD)	Mean (SD)	
VAS (0-100)	43.54 (23.88)	44.58 (27.85)	0.940
FABQ (0-96)	47.7 (24.62)	36.75 (21.55)	**0.030***
TSK-11 (11-44)	29.58 (6.12)	27.66 (6.67)	**0.014***
RMQ (0-24)	6.25 (5.04)	5.29 (4.95)	0.356

*Statistically significant difference. VAS: Visual Analogue Scale. FABQ: Fear-Avoidance Beliefs Questionnaire. TSK-11: Tampa Scale for Kinesiophobia. RMQ: Roland-Morris Questionnaire

Table 20: Comparative results

Results (n = 44)					
Outcome	**Group**	**Mean change** (SE)	**Mean difference (95% CI)**	**Effect size**	***p* value**
VAS (0-100)	Control (20)	8.00 (5.05)	-7.91 (-23.12 to 7.29)	0.036	0.217
	Experimental (24)	1.04 (4.61)			
FABQ (0-96)	Control (20)	2.35 (4.62)	-7.9 (-20.18 to 4.38)	0.039	0.202
	Experimental (24)	-10.95 (4.22)			
TSK-11 (11-44)	Control (20)	0.95 (0.91)	-1.5 (-5.77 to 2.61)	0.014	0.450
	Experimental (24)	-1.91 (0.83)			
RMQ (0-24)	Control (20)	2.40 (1.01)	-4.1 (-7.53 to -0.68)	0.117	**0.023***
	Experimental (24)	-0.95 (0.92)			

*Statistically significant difference. VAS: Visual Analogue Scale. FABQ: Fear-Avoidance Beliefs Questionnaire. TSK-11: Tampa Scale for Kinesiophobia. RMQ: Roland-Morris Questionnaire. Partial eta squared, F and p-values were calculated using a two-way mixed ANOVA.

Chapter 4: Discussion

4.1 Overview of the main study findings

The primary outcome of this study was to evaluate the effects of a biopsychosocial pain education (BPE) web-based intervention for chronic low back pain based on pain intensity compared to conventional care, with the secondary outcomes of fear-avoidance beliefs, kinesiophobia, and disability. The results of this study suggest that the use of a BPE intervention based on a web platform offers not statistically significant results on pain intensity, fear-avoidance beliefs, and kinesiophobia when compared with the conventional treatment provided by the primary care physician. However, the use of a BPE intervention based on a web platform offers statistically significant results on disability when compared with the conventional treatment provided by the primary care physician (see Table 20).

4.2 Comparison with other studies

The comparison of the results of our study with other similar studies is difficult considering that this is the first study with CLBP in which a PNE was developed using a web platform for implementation instead of the traditional face-to-face educational interventions (in one-on-one or group formats).

Even so, the authors have tried to compare the results extracted in this research with other studies that address the same topic (all of them RCTs), for which they have been divided into two groups. A first group of studies that used the PNE as an independent variable in patients with CLBP (Table 21), and a second group of studies that used web-based educational interventions for CLBP (Table 22).

It is important to point out, as already mentioned above, that none of the RCTs based on the PNE used a mixed-methods design nor did they use a website for implementation. Related to the effectiveness of the PNE, Tegner et al. (86) conducted a SR comparing the PNE to placebo, no treatment, waiting list, or other control interventions, and pharmacological treatments and concluded that there was moderate evidence that PNE had a small to moderate effect on pain in the short term; low evidence of a small to moderate effect on disability in the short term; and of small to moderate effect of pain and disability at a 3 month follow-up.

Regarding web-based studies, none were based on the PNE. However, three of these studies (281–283) used cognitive and behavioral therapy (CBT) as the educational intervention. Several SRs have addressed this issue recently. Bender et al. (161) concluded that Internet-based interventions were promising for the treatment of pain. Garg et al. (162) conducted a different SR to determine which web-based interventions were of benefit to CLBP patients, concluding that only online CBT showed some promising benefits in reducing catastrophization.

Regarding the duration of the interventions, it is important to note that only the studies by Moseley et al. (179) and Téllez-García et al. (185) had a duration that was similar (2 and 3 weeks, respectively) than that in our study. The study by Moseley et al. (179) was carried out in Australia and had a sample of 58 subjects that were divided into two groups. The experimental group received individual educational sessions on neurophysiology of pain, whereas the control group received education based on the anatomy and physiology of the lumbar spine in addition to ergonomics advice, principles of stretching, and fitness training. The duration of the intervention was 2 weeks. Téllez-García et al. (185) carried out their study in Spain with a sample of 12 subjects. The authors randomized the patients into two groups. One group had trigger point dry needling alone, and the other group had the same intervention combined with neuroscience education. The intervention had a duration of 3 weeks.

Taking into account the number of subjects in our study (n = 48), only the studies by Téllez-García et al. (185), Wälti et al. (184), and Ryan et al. (180), had a lower sample. Wälti et al. (184) carried out a study in Switzerland with a sample of 28 subjects who were randomized into two groups. The control group received the usual physiotherapy treatment while the experimental group received a multimodal treatment consisting of patient education on the neurophysiology of pain combined with sensory and motor retraining. The study by Ryan et al. (180) was performed in Scotland with a sample of 38 subjects randomized into two groups. One group received pain biology education whereas the other was given the same patient education plus exercise classes. The duration of the intervention was 6 weeks.

Finally, the study by Téllez-García et al. (185), explained above, had a sample of 12 subjects. This small sample number may reflect the complexity of recruiting patients for studies, as occurred in our study in primary care.

4.2.1 Pain intensity

In our study, both the experimental group (MC 1.04) and the control group (MC 8) showed not statistically significant differences (p = 0.217) (see Table 20).

These results are in contrast to the literature, as in all the comparable studies (see Table 21 and Table 22) the experimental group always obtained a decrease in pain intensity. However, this improvement in the experimental group was only significant in five of the studies that used the PNE (84,175,180,184,186), and in only one of the web-based studies (153). If we analyze the studies that obtained significant results in pain intensity, it is observed that the studies of Bodes et al. (186), Pires et al. (84), Wälti et al. (184), and Moseley (175) did not use the PNE in isolation but used it in combination with another intervention. For example, the study by Bodes et al. (186) conducted in Spain with a sample of 56 subjects compared a control group (MC 3) that performed therapeutic exercise with an experimental group (MC -5.2) that had the same intervention combined with PNE. The study by Pires et al. (84) compared aquatic exercise alone (MC -14.8) with the same aquatic exercise plus PNE (MC -22.8). This study was carried out in Portugal with a sample of 62 subjects and a duration of 6 weeks. Wälti et al. (184) used an experimental group (MC -2.14) in which a multimodal treatment was applied, which included a PNE, compared to usual physiotherapy (MC -0.69). Similarly, Moseley (175) used the PNE along with physiotherapy (MC -2.9) and compared it to a group in which they only received physiotherapy treatment (MC -1.4). This study was carried out in Australia with a sample of 57 subjects and a duration of the intervention of four weeks. However, only the study by Ryan et al. (180) used the PNE in isolation (MC -30.9), obtaining significant results in pain intensity (p = 0.025), compared to the group in which the PNE plus exercise was performed (MC -4.2).

In reference to the web-based studies, only the study by Lorig et al. (153) obtained significant results (p = 0.002). The number of subjects in this study (n = 580) was much higher than the rest of web-based studies in which the improvement in pain intensity was not significant (281–286). Lorig et al. (153) carried out their study in the United States, where they randomized the study subjects into two groups, a control group (MC -1.02) that received a subscription to a non-health-related magazine of their choice and an experimental group (MC -1.50) that consisted of a closed, moderated, e-mail discussion group. Subjects in the experimental group also received a book and a videotape about back pain.

It could be argued that 2 weeks of an educational intervention is not enough to significantly change pain intensity. In fact, in the study by Téllez-García (185) where the intervention lasted 3 weeks, the results obtained with respect to pain intensity were not statistically significant, which could indicate that the duration of the intervention should be longer than 3 weeks. However, the results of these authors have to be taken with caution as the authors did not calculate the sample size. Considering the results of these authors and accepting an alpha risk of 0.05 in a two-sided test with 6 subjects in each group, the statistical power is 10% for the variable pain intensity. The latter suggests that the authors could have committed a type 1-β error, and their results may have been significant if the sample were increased (287).

On the other hand, and taking into account the characteristics of the studies that obtained significant results in this variable, it could be argued that the PNE could obtain better results on pain intensity if used in conjunction with another intervention different from exercise and that using a web platform is not a differentiating factor.

4.2.2 Fear-avoidance beliefs

In our study, both the experimental group (MC -10.95) and the control group (MC 2.35) showed not statistically significant differences (p = 0.202) (see Table 20). However, the within-subjects analysis showed that for the experimental group fear-avoidance beliefs were statistically significantly reduced at post-test compared to pre-test (MC -10.95; $p = 0.030$) (see Table 19).

Of all the studies analyzed in this discussion, only three of them evaluated the fear-avoidance beliefs. The study by Wälti et al. (184) using the PNE combined with sensory and motor training obtained results similar to those of our study ($p > 0.05$). Related to the web-based interventions, the study by Chiauzzi et al. (283), conducted in the United States with a sample of 228 subjects, the experimental group that used a website based on CBT and self-management principles also obtained not statistically significant differences ($p > 0.05$) compared to the control group that used a back-pain guide. However, the study by Carpenter et al. (281), conducted in the United States with a sample of 141 subjects, the experimental group (MC -1.1) that had an online CBT intervention showed significantly better results ($p < 0.001$) compared to the wait-list control group (MC -0.9) on the FABQ-physical activity. On the FABQ-work not statistically significant differences were found.

Therefore, only the online CBT intervention showed statistically significant results when compared to a wait-list control group. When the CBT intervention was compared to a more robust control group (back-pain guide) the results obtained were not statistically significant.

4.2.3 Kinesiophobia

In our study, both the experimental group (MC -1.91) and the control group (MC 0.95) showed not statistically significant differences ($p = 0.450$) (see Table 20). However, kinesiophobia in the experimental group was statistically significantly reduced at post-test compared to pre-test (MC -1.91; $p = 0.014$), although this difference was not clinically relevant (see Table 19).

These results are similar to those obtained by other studies in which the PNE was used (84,180), although only the studies of Bodes et al. ($p < 0.001$) (186) and Téllez-García et al. ($p < 0.05$) (185) obtained a statistically significant improvement.

What is more difficult to find out is the reason why the studies by Bodes et al. (186) and by Téllez-García et al. (185) obtained statistically significant differences in comparison to the rest of the studies. It could be argued that the fact of combining the PNE with therapeutic exercise (MC -12.6) (186) or trigger point-dry needling (MC -17.7) (185) would offer better comparative results. However, the studies by Pires et al. (84) and Ryan et al. (180) also combine PNE with aquatic exercise (MC -1.7) and exercise classes (MC -3.9), respectively, and their comparative results were not statistically significant.

Having a small sample is not a differentiating fact because the study by Téllez-García et al. (185) has the smallest sample (*n* = 12) of all the studies analyzed in this discussion, and still obtained the same significant results as the study by Bodes et al. (186) with a sample of 56 subjects. In fact, the study by Téllez-García et al. (185) is the one that showed the most improvement (MC -12.7) compared to the study by Bodes et al. (186) (MC -8.5). These two studies differ from our study in the type of sample, which could explain the different results. Bodes et al. (186) obtained their sample from private clinics, whereas the sample of Téllez-García et al. (185) was formed by patients who had been referred to physiotherapy by their physician. However, the sample from our study came from a list of patients with CLBP from primary care. Although the sample of the two previous studies could be said to be of patients actively seeking a solution to their pain, the patient in our sample is a patient who, perhaps because of his/her low disability score at baseline, does not have the immediate need to alleviate his/her pain nor the expectation of improvement that the subjects of the other two studies could have. The pre-test mean kinesiophobia scores in our study for the control and experimental group were 28.30 (SD 6.62) and 29.58 (SD 6.12) respectively. However, in the study by Téllez-García et al. (185) the pre-test mean scores for the kinesiophobia outcome were 43.3 (SD 5.9) for the control group, and 41.5 (SD 6.2) for the experimental group. These higher scores may represent a troublesome ceiling effect (288). In addition, a possible positive expectation toward pain may have activated the descending modulatory pain system, inhibiting nociception (115,132), which may explain in part the difference of results with our study.

Regarding the use of web-based educational interventions, none of the studies found assessed kinesiophobia. These data are relevant if one takes into account that this study and four of those that use the PNE assessed kinesiophobia (84,180,185,186). In line with Gregg et al. (289), it could be argued that the assessment of kinesiophobia using the TSK does not offer any predictive value on the response of patients with LBP to treatment. However, and as suggested by Lüning et al. (290), the use of TSK to classify patients according to their levels of kinesiophobia can be a very useful tool in the treatment of patients with CLBP to identify those patients with high levels of kinesiophobia with the aim of reducing their activity limitations.

4.2.4 Disability

In our study, disability was statistically significantly lower in the experimental group post-test compared to the control group (MC -4.1; $p = 0.023$) (see Table 20). In addition, disability in the control group was statistically significantly greater at post-test compared to pre-test (MC 2.4; $p = 0.044$) (see Table 18). It could be argued that the statistically significant difference found between groups in our study were more related to the greater disability scores post-test in the control group rather than with the improvement obtained in the experimental group.

Findings from the current study are consistent with the results obtained by the PNE studies of Bodes et al. (186) ($p < 0.001$), Moseley et al. (179) ($p < 0.05$), and Moseley (175) ($p < 0.025$). Regarding the educational material used in the studies of Bodes et al. (186), Moseley et al. (179), and Moseley (175), the first two authors used a similar material. Bodes et al. (186) used the book *Explain Pain* to develop their PNE. On the other hand, Moseley et al. (179) based their PNE on specific aspects of pain neurophysiology such as "the nervous system," "synapses," and the "plasticity of the nervous system." Moseley (175) did not specify the educational material of his PNE. In contrast, our study used a mixed methodology to develop the educational material. For this purpose, semi-structured personal interviews were performed on 16 patients with CLBP to identify their chronic low back pain beliefs about the origin and meaning of pain. Hence, to develop our educational material, we took the patient into account as suggested by Camerini et al. (168) (see Table 6).

Regarding the studies that used a web-based educational intervention other than the PNE, our results on disability were similar. For example, Moessner et al. (285) carried out a study in Germany with a sample of 75 subjects and a duration of 15 weeks. These authors compared an experimental group that used an Internet-based aftercare intervention following multidisciplinary therapy for back pain, with a control group that received treatment as usual (not specified). The experimental group obtained statistically significant better results than the control group. In addition, the results of this study have to be taken with caution as only 34 of the 75 subjects in the sample completed all three assessments, which represents a 45% loss, producing an attrition bias (291). However, in our study, we lost only 4 subjects (8%), which indicates that the adherence was greater in our study, perhaps due to the gamification of the website, as some authors have posed (172–174).

Similar to our study, the study by Lorig et al. (153) obtained results with statistically significant differences in favor of the experimental group. Compared to the other similar studies, the significant differences obtained by Lorig et al. (153) could have been due to the different educational materials used (email discussion, book, and a videotape) or to the large sample used (n = 580), much higher than the rest of the studies.

Our results are also similar to the study by Krein et al. (284). This study was carried out in the United States with a sample of 229 subjects and a duration of 12 months. The authors compared an experimental group using a pedometer with access to a website that provided personal walking data, walking goals, feedback, and participation in e-community, with a control group also using a pedometer but without access to the website. The group with access to the website showed significantly better comparative results on disability than the group without access to the website in the medium term (6 months), but no difference was found in the long term (12 months). Although this experimental intervention was different from that of our study, using a website and promoting moderate physical activity among the patients in the study could be the reason they obtained good results. However, as mentioned above, these significant improvements were not sustained in the long term.

Another of the differentiating aspects between our study and the rest of web-based studies is that our website was gamified. The aim of this gamification was for each patient to adapt the educational content of the website to their own needs (see Chapter 2.4.9). The latter coincides with the results of the study by Allam et al. (172) on patients affected by rheumatoid arthritis. These authors concluded that the use of gamification in an educational intervention improved the patients' physical activity and their empowerment as well as decreased the use of health services.

Table 21: Comparison of the findings from the current study with previous work on PNE.

	Type of intervention/ duration of intervention	Outcomes from the current study			
		Pain intensity	Fear-avoidance beliefs	Kinesiophobia	Disability
Bodes et al. (2018) (*n* = 56)	PNE + therapeutic exercise/3 months	✖	N/A	✖	✔
Pires et al. (2015) (*n* = 62)	PNE + aquatic exercise/6 weeks	✖	N/A	✔	✖
Téllez-García et al. (2015) (*n* = 12)	PNE + trigger point-dry needling/3 weeks	✔	N/A	✖	✖
Wälti et al. (2015) (*n* = 28)	PNE + sensory training + motor training/12 weeks	✖	✔	N/A	✖
Ryan et al. (2010) (*n* = 38)	PNE alone vs. PNE + exercise/6 weeks	✖	N/A	✔	✖
Moseley et al. (2004) (*n* = 58)	PNE alone/2 weeks	N/A	N/A	N/A	✔
Moseley (2002) (*n* = 57)	PNE + physiotherapy/ 4 weeks	✖	N/A	N/A	✔

✖: Results different to the current study. ✔: Results similar to the current study. N/A: Outcome not assessed. PNE: pain neurophysiology education.

Table 22: Comparison of the findings from the current study with previous web-based interventions

	Type of intervention/ duration of intervention	Outcomes from the current study			
		Pain intensity	Fear-avoidance beliefs	Kinesiophobia	Disability
Krein et al. (2013) (*n* = 229)	Pedometer-based, Internet-mediated intervention/12 months	✔	N/A	N/A	✔
Moessner et al. (2012) (*n* = 75)	Internet-based intervention for aftercare following multidisciplinary pain treatment/15 weeks	✔	N/A	N/A	✔
Carpenter et al. (2012) (*n* = 141)	Online CBT intervention/6 weeks	N/A	✖	N/A	✖
Buhrman et al. (2011) (*n* = 54)	Web-based multimodal pain management program based on CBT/12 weeks	✔	N/A	N/A	N/A
Chiauzzi et al. (2010) (*n* = 228)	Back pain website based on CBT and chronic pain management principles/6 months	✔	✔	N/A	✖
Buhrman et al. (2004) (*n* = 56)	Web-based multimodal pain management program/8 weeks	✔	N/A	N/A	N/A
Lorig et al. (2002) (*n* = 580)	Moderated email discussion group; back pain help book; videotape/1 year	✖	N/A	N/A	✔

✖: Results different to the current study. ✔: Results similar to the current study.
N/A: Outcome not assessed. CBT: cognitive and behavioral therapy

4.3 Strengths and limitations of the study

Possibly the greatest strength of our study lies in the multidisciplinary nature of the team. This team comprises doctors, sociologists, physiotherapists, nurses, sports scientists, physicists, and computer specialists that practice their profession in different areas such as primary care, university education, scientific research, and the development of new technologies applied to health. It is important to address CLBP in a biopsychosocial way from multiple perspectives; this has been reflected in the development of both the educational materials and the website.

Another strength of this study is the use of a mixed methodological design. The mixed methodology takes into account both qualitative and quantitative methodology, needing and strengthening each other, and using the strengths of both while reducing their weaknesses (188). For the authors, it has been enriching to be able to carry out a qualitative methodology before the development of the educational materials and the website. Through individualized semi-structured interviews, it has been possible to observe what the patient thinks first hand and draw conclusions from it. In this investigation, it has been possible to examine the beliefs the patients have developed throughout their experience with CLBP, which has helped us understand their pain experience. The information the patients have provided has also been the fundamental pillar in the development of the educational materials. As a team, we have always questioned that, both in research and in the academic world, it is usually decided unilaterally what students or patients need to know. Therefore, this type of mixed methodology has helped develop a material that is made directly by the patient and for the patient.

Another aspect highlighted in this study is the different formats in which the educational materials have been developed. It is important that in subjects such as neurophysiology, which are already difficult to explain and sometimes understand, the educational material is simple and understandable. Therefore, from the beginning, we decided to develop different formats. For this intervention, five 3D videos were developed to explain the content of the educational material in a more visual manner. This was accompanied by reading material easily downloaded at home and complemented with more playful activities such as the “Challenge,” so that through the game, the messages sent to the patient could be reinforced.

Finally, using new technologies in the field of patient education is always a strong point. Gamification, understood as an element used to attract and motivate the patient (172–174), is a very relevant aspect of this intervention. Getting the patient to want to learn more about his lower back pain is not an easy task. Moreover, many times you can have the right information for the right patient, but that message does not reach the patient. Studies such as that of Parsons et al. (292) show the existence of different conflicts in the doctor–patient communication in primary care that hinder the achievement of positive results. New technologies may break these therapist–patient communication barriers and help the right message be received by the person who needs it. In the development of the website, it was possible to implement the gamification in a way that each patient was able to personalize his visit. The gamification made it possible for each patient who accessed the website to see the content differently, which for the authors is a way to make the educational intervention more attractive to the patient.

There are a number of limitations in this study that are important to highlight. Because of the type of probabilistic sampling used, in which patients were recruited through a list provided by primary care, the type of patient in our sample was a patient with very low levels of disability compared to the levels of disability of other similar studies. In fact, studies such as those of Wälti et al. (184) or Téllez-García et al. (185) had an inclusion criterion of 4 points or more in the RMQ. With these criteria, some of the patients in our sample would have been excluded from their studies. Therefore, if our study had a sample with higher levels of disability, perhaps we would have been obtained better results. It could be argued that there may be a floor effect in our results. However, only a 6.8% of our sample had an RMQ score of 0 at baseline. Roland and Fairbank (278) recommend the use of the RMQ in patients with relatively little disability as it may still discriminate low levels of disability when the scores are at minimum.

Another possible limitation is the duration of the intervention. Although initially, and based on the study by Keulers et al. (163), it was established that 15 days was a sufficient duration for the intervention, it might be necessary to increase this duration if we take into account the results obtained. It could be suggested that even if good results were obtained, they could have been better if the patients could have used the website for a longer period of time. Yet, we do not know how often and for how long our patients have accessed the website. Patients were allowed to view the website for 15 days, but they could have visited only once or several times. Therefore, increasing the time of use of the website by patients could help to consolidate what has been learned with the educational material and may improve the scores on the different outcomes. However, lengthening the intervention is not without risks. Patients are more likely to lose motivation, or to forget readings, and thus they distance themselves from the research.

Another criticizable point of our intervention is the lack of a robust control group. Some authors may question the usefulness of a control group based on the usual treatment by the family physician, as in some cases the treatment could consist of only advice or the prescription of a drug. On the other hand, taking into account that the patient of the experimental group had access to the website 24 hours a day for 15 days, it could be perceived that the patient in the control group received little attention. In addition, it must be considered that many times the information received by the patients from the health professional can further reinforce their misbeliefs regarding their CLBP (293,294).

Regarding the qualitative part of this study and the connection phase, one of the limitations could be the interpretation made by the authors of the content of the patients' interviews. The results of the qualitative part must be interpreted within the local context where they have been carried out. Even so, the qualitative material has been treated in the appropriate methodological manner and the credibility of the results is based on the triangulation of the information by the members of this study with different profiles and from different academic and professional fields (295). Although the choice of content about the neurophysiology of pain is based on these interviews, the authors of this study made the last choice. It may be that with the same information extracted from the qualitative phase, other authors would have chosen different content about the neurophysiology of pain. However, this content has already been justified in the connection section (see Chapter 2.4 Connection phase).

4.4 Implications for practice

One of the reasons we carried out this project was to incorporate the education of the patient with CLBP into primary care. There is a lot of evidence to suggest that patients with CLBP do not receive enough information from the health professional about their pain, and that this information could help them better understand their situation (296–298). Many times, it is the physiotherapist in primary care who spends the longest time with this type of patient and is therefore the health professional who may be better able to educate the patient with CLBP. Physiotherapy has driven this project and begun to lead the field of patient education for musculoskeletal pain in our country. Even so, the current situation in our community is that physiotherapists are almost nonexistent in primary care. Not only that, but in the few communities where there are physiotherapists in primary care in Spain, they are subject to the diagnosis and referral by the family physician. In other words, in the primary care of our country there are hardly any physiotherapists and there is no direct access to physiotherapy (299). Something that the authors want to claim, especially when researchers such as Ojha et al. (300) in 2014 already concluded in their SR that “Physical therapy by way of direct access may contain healthcare costs and promote high-quality healthcare. Third-party payers should consider paying for physical therapy by direct access to decrease healthcare costs and incentivize optimal patient outcomes.”

Regarding the practical implications, the next step would be to improve the intervention in the aspects mentioned in the limitations. In addition, it would be important to start the implementation of the web platform developed in this study in some of the primary care centers of the city of Lleida. If we take into account the large number of patients with CLBP who visit these primary care centers daily, the treatment provided by the family physician could be supplemented by using the website of this study.

4.5 Implications for future research

This study is part of a project carried out from the Faculty of Nursing and Physiotherapy of the University of Lleida. This is a multidisciplinary project, led by physiotherapists with the collaboration of other health professions such as nursing and medicine, where chronic pain is investigated from different approaches. Fundamentally, there are two pillars on which our research is based. The first would be the BPE directed not only to patients but also to health professionals and students of different degrees in health sciences, and a second field of research focused on the investigation on motor control.

In reference to the research on motor control, a first doctoral thesis has already been published by Rubí-Carnacea (301). This research, completed in 2017, showed that the motor control training group was more effective than the control group in the treatment of patients with CLBP in primary care.

Regarding BPE, this study is the first investigation that has been carried out in our Faculty, having already published the protocol (302) (appendix 12). In addition, and as a consequence of the results obtained in the qualitative part of this study, a qualitative parallel study was performed addressing the perceptions and beliefs of both patients and primary care health professionals on CLBP. This study is currently under review in the journal *Pain Medicine*. Preliminary findings of this study were presented with the number PTH172 at the 16th World Congress on Pain, held at the Pacifico Convention Center in Yokohama, Japan, 2016 (appendix 13).

Taking into account the above, the future lines derived from this study are:

- The use of the BPE by primary care health professionals: this research has already begun as part of a doctoral thesis. At this moment the authors are in the development phase of the educational material and the website. The objective of this research is to assess whether the primary care professionals receiving the BPE have fewer fear-avoidance beliefs and catastrophic thoughts than the health professionals on a control group based on explanatory videos of the clinical practice guidelines of the Catalan Institute of Health. The study protocol is currently awaiting final response from the editor of the journal *BMC Family Practice*.

- The use of the website and the educational material developed in this study for the teaching of the neurophysiology of pain to students of health sciences degrees in our university. This project is in the development phase. The objective is to assess whether the gamified website can be effective for health science students to acquire the necessary knowledge to understand the neurophysiology of pain compared to traditional group teaching.

Chapter 5: Conclusions

5.1 Study conclusions

The patients:

1. relate the origin and significance of their chronic low back pain with the structural and physiological alterations located in their lumbar area.
2. feel that pain is always a warning signal but that other aspects such as stress and emotions may have some influence on their pain experience.
3. recognize that physical activity can be a modulator of their lower back pain, so physical activity can be both the origin of their pain and a possible solution.
4. tend to look for a cause that justifies their lower back pain and failure to find that cause causes them fear and catastrophic thoughts.
5. have expressed the need to better understand their pain, which implies that health professionals should be more didactic in the management of chronic low back pain patients.

A web-based biopsychosocial pain education intervention for chronic low back pain:

6. is not more effective than conventional care in improving pain intensity in the short-term.
7. is not more effective than conventional care in improving the fear-avoidance beliefs in the short-term.
8. is not more effective than conventional care in improving kinesiophobia in the short-term.
9. is effective in reducing disability in the short term compared to conventional care.

References

1. Deyo RA, Mirza SK, Martin BI. Back pain prevalence and visit rates: Estimates from U.S. national surveys, 2002. Spine (Phila Pa 1976). 2006;31(23):2724–7.

2. Woolf AD, Pfleger B. Burden of major musculoskeletal conditions. Bull World Health Organ. 2003;81(9):646–56.

3. Hoy D, Bain C, Williams G, March L, Brooks P, Blyth F, et al. A systematic review of the global prevalence of low back pain. Arthritis Rheum. 2012;64(6):2028–37.

4. Woolf AD, Erwin J, March L. The need to address the burden of musculoskeletal conditions. Best Pract Res Clin Rheumatol. 2012;26(2):183–224.

5. Johannes CB, Le TK, Zhou X, Johnston JA, Dworkin RH. The Prevalence of Chronic Pain in United States Adults: Results of an Internet-Based Survey. J Pain. 2010;11(11):1230–9.

6. Langley PC, Ruiz-Iban MA, Molina JT, De Andres J, Castellón JRG-E-E. The prevalence, correlates and treatment of pain in Spain. J Med Econ. 2011;14(3):367–80.

7. Langley PC. The prevalence, correlates and treatment of pain in the European Union. Curr Med Res Opin. 2011;27(2):463–80.

8. Carmona L, Ballina J, Gabriel R, Laffon A. The burden of musculoskeletal diseases in the general population of Spain: Results from a national survey. Ann Rheum Dis. 2001;60(11):1040–5.

9. Parthan A, Evans CJ, Le K. Chronic low back pain: Epidemiology, economic burden and patient-reported outcomes in the USA. Expert Rev Pharmacoeconomics Outcomes Res. 2006;6(3):359–69.

10. Carmona L, Peña Sagredo JL, Ortiz AAM, Mendiola AH, Carmona L, Peña

JL, et al. Impacto poblacional del dolor lumbar en España : resultados del estudio EPISER. Vol. 29, Revista Española de Reumatología. 2002. 471-478 p.

11. Fernández-de-las-Peñas C, Hernández-Barrera V, Alonso-Blanco C, Palacios-Ceña D, Carrasco-Garrido P, Jiménez-Sánchez S, et al. Prevalence of neck and low back pain in community-dwelling adults in Spain: a population-based national study. Spine (Phila Pa 1976). 2011;36(3):E213-9.

12. MInisterio de Sanidad Consumo y Bienestar Social. ENSE Encuesta Nacional de Salud España 2017 [Internet]. 2018 [cited 2018 Aug 15]. p. 1–12. Available from: http://www.msssi.gob.es/estadEstudios/estadisticas/encuestaNacional/encuestaNac2017/ENSE17_pres_web.pdf

13. Generalitat de Catalunya, Departament de Salut. Informe 2015 de l'Enquesta de salut de Catalunya (ESCA) [Internet]. Esca. 2016 [cited 2018 Aug 13]. p. 99. Available from: http://salutweb.gencat.cat/web/.content/home/el_departament/estadistiques_sanitaries/enquestes/esca_2015.pdf

14. Maetzel A, Li L. The economic burden of low back pain: A review of studies published between 1996 and 2001. Best Pract Res Clin Rheumatol. 2002;16(1):23–30.

15. Asche C V, Kirkness CS, McAdam-Marx C, Fritz JM. The societal costs of low back pain: Data published between 2001 and 2007. J Pain Palliat Care Pharmacother. 2007;21(4):25–33.

16. Hoy D, March L, Brooks P, Blyth F, Woolf A, Bain C, et al. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis. 2014;73(6):968–74.

17. Buchbinder R, Blyth FM, March LM, Brooks P, Woolf AD, Hoy DG. Placing the global burden of low back pain in context. Best Pract Res Clin Rheumatol. 2013;27(5):575–89.

18. Leadley RM, Armstrong N, Lee YC, Allen A, Kleijnen J. Chronic diseases in the European Union: the prevalence and health cost implications of chronic pain. J Pain Palliat Care Pharmacother. 2012;26(4):310–25.

19. González Viejo MA, Condón Huerta MJ. Disability from low back pain in Spain. Med Clin (Barc). 2000;114(13):491–2.

20. Driscoll T, Jacklyn G, Orchard J, Passmore E, Vos T, Freedman G, et al. The global burden of occupationally related low back pain: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis. 2014;73(6):975–81.

21. Lundberg M, Frennered K, Hägg O, Styf J. The Impact of Fear-Avoidance Model Variables on Disability in Patients With Specific or Nonspecific Chronic Low Back Pain. Spine (Phila Pa 1976). 2011;36(19):1547–53.

22. Turk DC, Okifuji A. Psychological factors in chronic pain: Evolution and revolution. J Consult Clin Psychol. 2002;70(3):678–90.

23. McCracken LM, Gross RT. Does anxiety affect coping with chronic pain? Clin J Pain. 1993;9(4):253–9.

24. Feizerfan A, Sheh G. Transition from acute to chronic pain. Contin Educ Anaesth Crit Care Pain. 2015;15(2):98–102.

25. Pengel LHM, Herbert RD, Maher CG, Refshauge KM. Acute low back pain: systematic review of its prognosis. BMJ. 2003;327(7410):323.

26. Nijs J, Apeldoorn A, Hallegraeff H, Clark J, Smeets R, Malfliet A, et al. Low back pain: guidelines for the clinical classification of predominant neuropathic, nociceptive, or central sensitization pain. Pain Physician. 2015;18(3):E333-46.

27. Woolf CJ. Central sensitization: Implications for the diagnosis and treatment of pain. Pain. 2011;152(Supplement):S2–15.

28. Lee MC, Zambreanu L, Menon DK, Tracey I. Identifying Brain Activity Specifically Related to the Maintenance and Perceptual Consequence of Central Sensitization in Humans. J Neurosci. 2008;28(45):11642–9.

29. Murad MH, Asi N, Alsawas M, Alahdab F. New evidence pyramid. Evid Based Med. 2016;21(4):125–7.

30. Bordas J, Forcada J, García J, Joaniquet F, Pellisé F, Mazeres O, et al. Patologia de la columna lumbar en l'adult. Guies de pràctica clínica i material docent. Institut Català de la Salut [Internet]. 2004 [cited 2015 Oct 8]. p. 1–49. Available from: http://ics.gencat.cat/web/.content/documents/assistencia/gpc/guia_lumbalgies.pdf

31. Airaksinen O, Brox JI, Cedraschi C, Hildebrandt J, Klaber-Moffett J, Kovacs F, et al. Chapter 4 European guidelines for the management of chronic nonspecific low back pain. Eur Spine J. 2006;15(S2):s192–300.

32. Koes BW, van Tulder M, Lin C-WC, Macedo LG, McAuley J, Maher C. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. Eur Spine J. 2010;19(12):2075–94.

33. Saragiotto BT, Machado GC, Ferreira ML, Pinheiro MB, Abdel Shaheed C, Maher CG. Paracetamol for low back pain. Cochrane Database Syst Rev. 2016;(6):No.: CD012230.

34. Chou R, Deyo R, Friedly J, Skelly A, Weimer M, Fu R, et al. Systemic Pharmacologic Therapies for Low Back Pain: A Systematic Review for an American College of Physicians Clinical Practice Guideline. Ann Intern Med. 2017;166(7):480.

35. Enthoven WT, Roelofs PD, Deyo RA, van Tulder MW, Koes BW. Non-

steroidal anti-inflammatory drugs for chronic low back pain. Cochrane Database Syst Rev. 2016;(2):69.

36. Chung J, Zeng Y, Tk W. Drug therapy for the treatment of chronic nonspecific low back pain: systematic review and meta-analysis. Pain Physician. 2013;16(6):685–704.

37. Abdel Shaheed C, Maher CG, Williams KA, Day R, McLachlan AJ. Efficacy, Tolerability, and Dose-Dependent Effects of Opioid Analgesics for Low Back Pain. JAMA Intern Med. 2016;176(7):958.

38. Chaparro LE, Furlan AD, Deshpande A, Mailis-Gagnon A, Atlas S, Turk DC. Opioids compared to placebo or other treatments for chronic low-back pain. Cochrane Database Syst Rev. 2013;8(8):CD004959.

39. Higgins J, Green S. Cochrane Handbook for Systematic Reviews of Interventions 5.1.0 [updated March 2011]. The Cochrane Collaboration, 2011; 2011.

40. Schroll JB, Moustgaard R, Gøtzsche PC. Dealing with substantial heterogeneity in Cochrane reviews. Cross-sectional study. BMC Med Res Methodol. 2011;11(1):22.

41. Abdel Shaheed C, Maher CG, Williams KA, McLachlan AJ. Efficacy and tolerability of muscle relaxants for low back pain: Systematic review and meta-analysis. Eur J Pain. 2017;21(2):228–37.

42. Durg S. Comment on Abdel Shaheed et al.: “Efficacy and tolerability of muscle relaxants for low back pain: Systematic review and meta-analysis.” Eur J Pain. 2017;21(2):397–9.

43. Shanthanna H, Gilron I, Rajarathinam M, AlAmri R, Kamath S, Thabane L, et al. Benefits and safety of gabapentinoids in chronic low back pain: A systematic review and meta-analysis of randomized controlled trials. PLOS Med. 2017;14(8):e1002369.

44. Cawston H, Davie A, Paget M-A, Skljarevski V, Happich M. Efficacy of duloxetine versus alternative oral therapies: an indirect comparison of randomised clinical trials in chronic low back pain. Eur Spine J. 2013;22(9):1996–2009.

45. Wielage R, Bansal M, Wilson K, Klein R, Happich M. Cost-Effectiveness of Duloxetine in Chronic Low Back Pain. Spine (Phila Pa 1976). 2013;38(11):936–46.

46. Searle A, Spink M, Ho A, Chuter V. Exercise interventions for the treatment of chronic low back pain: a systematic review and meta-analysis of randomised controlled trials. Clin Rehabil. 2015;29(12):1155–67.

47. Cohen J. Statistical power analysis for the behavioural sciences. Second. Lawrence Earlbaum Associates I, editor. New Jersey, USA; 1988. 567 p.

48. Macedo LG, Smeets RJEM, Maher CG, Latimer J, McAuley JH. Graded Activity and Graded Exposure for Persistent Nonspecific Low Back Pain: A Systematic Review. Phys Ther. 2010;90(6):860–79.

49. López-de-Uralde-Villanueva I, Muñoz-García D, Gil-Martínez A, Pardo-Montero J, Muñoz-Plata R, Angulo-Díaz-Parreño S, et al. A Systematic Review and Meta-Analysis on the Effectiveness of Graded Activity and Graded Exposure for Chronic Nonspecific Low Back Pain. Pain Med. 2016;17(1):172–88.

50. Muscolino JE, Cipriani S. Pilates and the "powerhouse"—I. J Bodyw Mov Ther. 2004;8(1):15–24.

51. Miyamoto GC, Costa LOP, Cabral CMN. Efficacy of the Pilates method for pain and disability in patients with chronic nonspecific low back pain: a systematic review with meta-analysis. Brazilian J Phys Ther. 2013;17(6):517–32.

52. Yamato TP, Maher CG, Saragiotto BT, Hancock MJ, Ostelo RWJG, Cabral

CMN, et al. Pilates for Low Back Pain. Spine (Phila Pa 1976). 2016;41(12):1013–21.

53. Wells C, Kolt GS, Marshall P, Hill B, Bialocerkowski A. The Effectiveness of Pilates Exercise in People with Chronic Low Back Pain: A Systematic Review. PLoS One. 2014;9(7):e100402.

54. Patti A, Bianco A, Paoli A, Messina G, Montalto MA, Bellafiore M, et al. Effects of Pilates exercise programs in people with chronic low back pain: a systematic review. Medicine (Baltimore). 2015;94(4):e383.

55. Wren AA, Wright MA, Carson JW, Keefe FJ. Yoga for persistent pain: New findings and directions for an ancient practice. Pain. 2011;152(3):477–80.

56. Wieland LS, Skoetz N, Pilkington K, Vempati R, D'Adamo CR, Berman BM. Yoga treatment for chronic non-specific low back pain. Cochrane Database Syst Rev. 2017;1:CD010671.

57. Aman JE, Elangovan N, Yeh I-L, Konczak J. The effectiveness of proprioceptive training for improving motor function: a systematic review. Front Hum Neurosci. 2014;8(January):1075.

58. McCaskey MA, Schuster-Amft C, Wirth B, de Bruin ED. Effects of proprioceptive exercises on pain and function in chronic neck- and low back pain rehabilitation: a systematic literature review. Physiotherapy. 2015;101:e969–70.

59. Saragiotto BT, Maher CG, Yamato TP, Costa LO, Menezes Costa LC, Ostelo RW, et al. Motor control exercise for chronic non-specific low-back pain. Cochrane Database Syst Rev. 2016;(1):CD012004.

60. O'Sullivan PB, Phyty GDM, Twomey LT, Allison GT. Evaluation of Specific Stabilizing Exercise in the Treatment of Chronic Low Back Pain With Radiologic Diagnosis of Spondylolysis or Spondylolisthesis. Spine (Phila Pa 1976). 1997;22(24):2959–67.

61. McNeill W. Core stability is a subset of motor control. J Bodyw Mov Ther. 2010;14(1):80–3.

62. Smith BE, Littlewood C, May S. An update of stabilisation exercises for low back pain: a systematic review with meta-analysis. BMC Musculoskelet Disord. 2014;15(1):416.

63. Gomes-Neto M, Lopes JM, Conceição CS, Araujo A, Brasileiro A, Sousa C, et al. Stabilization exercise compared to general exercises or manual therapy for the management of low back pain: A systematic review and meta-analysis. Phys Ther Sport. 2017;23:136–42.

64. Higgins JPT. Measuring inconsistency in meta-analyses. BMJ. 2003;327(7414):557–60.

65. Page MJ, Green S, McBain B, Surace SJ, Deitch J, Lyttle N, et al. Manual therapy and exercise for rotator cuff disease. Cochrane Database Syst Rev. 2016;(6).

66. Vickers A, Zollman C. ABC of complementary medicine: Massage therapies. BMJ. 1999;319(7219):1254–7.

67. Furlan AD, Giraldo M, Baskwill A, Irvin E, Imamura M. Massage for low-back pain. Cochrane Database Syst Rev. 2015;(9).

68. Greenman P. Principles of Manual Therapy. 2nd ed. Williams & Wilkins, editor. 1996.

69. Franke H, Fryer G, Ostelo RW, Kamper SJ. Muscle energy technique for non-specific low-back pain. Cochrane Database Syst Rev. 2015;2012(5).

70. Rubinstein SM, van Middelkoop M, Assendelft WJ, de Boer MR, van Tulder MW. Spinal manipulative therapy for chronic low-back pain. Cochrane Database Syst Rev. 2011;(2).

71. Coulter ID, Crawford C, Hurwitz EL, Vernon H, Khorsan R, Suttorp Booth M, et al. Manipulation and mobilization for treating chronic low back pain: a systematic review and meta-analysis. Spine J. 2018;18(5):866–79.

72. Balshem H, Helfand M, Schünemann HJ, Oxman AD, Kunz R, Brozek J, et al. GRADE guidelines: 3. Rating the quality of evidence. J Clin Epidemiol. 2011;64(4):401–6.

73. Menke JM. Do Manual Therapies Help Low Back Pain? A Comparative Effectiveness Meta-analysis. Spine (Phila Pa 1976). 2014;39(7):E463–72.

74. Zachrisson-Forsell M. The Swedish Back School. Physiotherapy. 1980;66(4):112–4.

75. Poquet N, Lin C-WC, Heymans MW, van Tulder MW, Esmail R, Koes BW, et al. Back schools for acute and subacute non-specific low-back pain. Cochrane Database Syst Rev. 2016;(4).

76. Parreira P, Heymans MW, van Tulder MW, Esmail R, Koes BW, Poquet N, et al. Back Schools for chronic non-specific low back pain. Cochrane Database Syst Rev. 2017;(8).

77. Engers AJ, Jellema P, Wensing M, van der Windt DA, Grol R, van Tulder MW. Individual patient education for low back pain. Cochrane Database Syst Rev. 2008;(1).

78. van den Borne HW. The patient from receiver of information to informed decision-maker. Patient Educ Couns. 1998;34(2):89–102.

79. Cherkin DC, Deyo RA, Street JH, Hunt M BW. Pitfalls of Patient Education: Limited Success of a Program for Back Pain in Primary Care. Spine (Phila Pa 1976). 1996;21(3):345–55.

80. Van Oosterwijck J, Nijs J, Meeus M, Truijen S, Craps J, Van den Keybus N, et al. Pain neurophysiology education improves cognitions, pain thresholds,

and movement performance in people with chronic whiplash: A pilot study. J Rehabil Res Dev. 2011;48(1):43.

81. Meeus M, Nijs J, Hamers V, Ickmans K, van Oosterwijcks J. The efficacy of patient education in whiplash associated disorders: A systematic review. Pain Physician. 2012;15(5):351–61.

82. Louw A, Diener I, Butler DS, Puentedura EJ. The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain. Arch Phys Med Rehabil. 2011;92(12):2041–56.

83. Ainpradub K, Sitthipornvorakul E, Janwantanakul P, van der Beek AJ. Effect of education on non-specific neck and low back pain: A meta-analysis of randomized controlled trials. Man Ther. 2016;22:31–41.

84. Pires D, Cruz EB, Caeiro C. Aquatic exercise and pain neurophysiology education versus aquatic exercise alone for patients with chronic low back pain: a randomized controlled trial. Clin Rehabil. 2015;29(6):538–47.

85. Sahin N, Albayrak I, Durmus B, Ugurlu H. Effectiveness of back school for treatment of pain and functional disability in patients with chronic low back pain: A randomized controlled trial. J Rehabil Med. 2011;43(3):224–9.

86. Tegner H, Frederiksen P, Esbensen BA, Juhl C. Neurophysiological Pain Education for Patients With Chronic Low Back Pain: A Systematic Review and Meta-Analysis. Clin J Pain. 2018;34(8):778–86.

87. Wade DT, Halligan PW. Do biomedical models of illness make for good healthcare systems? BMJ. 2004;329(7479):1398–401.

88. Engel GL. The need for a new medical model: a challenge for biomedicine. Science. 1977;196(4286):129–36.

89. Weiner BK. The biopsychosocial model and spine care. Spine (Phila Pa 1976). 2008;33(2):219–23.

90. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry. 1980;137(5):535–44.

91. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: Scientific advances and future directions. Psychol Bull. 2007;133(4):581–624.

92. Gatchel RJ, Turk DC. Criticisms of the Biopsychosocial Model in Spine Care. Spine (Phila Pa 1976). 2008;33(25):2831–6.

93. Waddell G. Preventing incapacity in people with musculoskeletal disorders. Br Med Bull. 2006;77–78(1):55–69.

94. Gatchel RJ, Kishino ND, Strezak A. The importance of outcome assessment in orthopaedics: an overview. In: Spivak JM (Jeffrey M, Connolly PJ, editors. Orthopaedic knowledge update Spine 3. American Academy of Orthopaedic Surgeons; 2006. p. 95–102.

95. Blyth FM, Macfarlane GJ, Nicholas MK. The contribution of psychosocial factors to the development of chronic pain: the key to better outcomes for patients? Pain. 2007;129(1–2):8–11.

96. Pincus T, Kent P, Bronfort G, Loisel P, Pransky G, Hartvigsen J. Twenty-Five Years With the Biopsychosocial Model of Low Back Pain—Is It Time to Celebrate? Spine (Phila Pa 1976). 2013;38(24):2118–23.

97. Margalit APA, Glick SM, Benbassat J, Cohen A, Katz M. Promoting a biopsychosocial orientation in family practice: effect of two teaching programs on the knowledge and attitudes of practising primary care physicians. Med Teach. 2005;27(7):613–8.

98. Campbell CM, Edwards RR. Mind–body interactions in pain: the neurophysiology of anxious and catastrophic pain-related thoughts. Transl Res. 2009;153(3):97–101.

99. Gifford L. Pain, the Tissues and the Nervous System: A conceptual model. Physiotherapy. 1998;84(1):27–36.

100. Lumley MA, Cohen JL, Borszcz GS, Cano A, Radcliffe AM, Porter LS, et al. Pain and emotion: a biopsychosocial review of recent research. J Clin Psychol. 2011;67(9):942–68.

101. Merskey H, Albe-Fessard D, Bonica J, Carmon A, Dubner R, Kerr F, et al. Pain terms: a list with definitions and notes on usage. Recommended by the IASP Subcommittee on Taxonomy. Pain. 1979;6(3):249.

102. Cohen M, Quintner J, van Rysewyk S. Reconsidering the International Association for the Study of Pain definition of pain. PAIN Reports. 2018;3(2):e634.

103. Treede R-D. The International Association for the Study of Pain definition of pain: as valid in 2018 as in 1979, but in need of regularly updated footnotes. Pain reports. 2018;3(2):e643.

104. Moseley GL, Butler DS. Fifteen Years of Explaining Pain: The Past, Present, and Future. J Pain. 2015;16(9):807–13.

105. Borrell-Carrió F, Suchman AL, Epstein RM. The biopsychosocial model 25 years later: principles, practice, and scientific inquiry. Ann Fam Med. 2004;2(6):576–82.

106. Sluka KA. Mechanisms and management of pain for the physical therapist. Seattle : IASP Press; 2009.

107. Strong J, Wall PD. Pain : a textbook for therapists. Edinburgh [etc.] : Churchill Livingstone; 2002.

108. Jaggar S. Overview of pain pathways. In: Holdcroft A, Jaggar S, editors. Core Topics in Pain. Cambridge: Cambridge University Press; 2005. p. 3–5.

109. De León-Casasola OA. Pain pathways and mechanisms of neuropathic pain. Rev Mex Anest. 2007;30(S1):133–8.

110. Mertens P, Blond S, David R, Rigoard P. Anatomy, physiology and neurobiology of the nociception: A focus on low back pain (part A). Neurochirurgie. Elsevier Masson SAS; 2015 Mar;61(S1):S22–34.

111. Tobaldini G, Sardi NF, Guilhen VA, Fischer L. Pain Inhibits Pain: an Ascending-Descending Pain Modulation Pathway Linking Mesolimbic and Classical Descending Mechanisms. Mol Neurobiol. 2018;1–14.

112. Tracey I. Getting the pain you expect: mechanisms of placebo, nocebo and reappraisal effects in humans. Nat Med. 2010;16(11):1277–83.

113. Ossipov MH, Morimura K, Porreca F. Descending pain modulation and chronification of pain. Curr Opin Support Palliat Care. 2014;8(2):143–51.

114. Bushnell MC, Čeko M, Low LA. Cognitive and emotional control of pain and its disruption in chronic pain. Nat Rev Neurosci. 2013;14(7):502–11.

115. Tracey I, Mantyh PW. The Cerebral Signature for Pain Perception and Its Modulation. Neuron. 2007;55(3):377–91.

116. Gear RW, Levine JD. Antinociception produced by an ascending spino-supraspinal pathway. J Neurosci. 1995;15(4):3154–61.

117. Tambeli CH, Fischer L, Monaliza SL, Menescal-de-Oliveira L, Parada CA. The functional role of ascending nociceptive control in defensive behavior. Brain Res. 2012;1464:24–9.

118. Roeder Z, Chen Q, Davis S, Carlson JD, Tupone D, Heinricher MM. The parabrachial complex links pain transmission to descending pain modulation. Pain. 2016;157(12):2697–708.

119. Fields HL, Anderson SD, Clanton CH, Basbaum AI. Nucleus raphe magnus:

a common mediator of opiate- and stimulus-produced analgesia. Trans Am Neurol Assoc. 1976;101:208–10.

120. Ossipov MH, Dussor GO, Porreca F. Central modulation of pain. J Clin Invest. 2010;120(11):3779–87.

121. Kuner R. Central mechanisms of pathological pain. Nat Med. 2010;16(11):1258–66.

122. Ossipov MH. The Perception and Endogenous Modulation of Pain. Scientifica (Cairo). 2012;2012:1–25.

123. Villemure C, Schweinhardt P. Supraspinal pain processing: Distinct roles of emotion and attention. Neuroscientist. 2010;16(3):276–84.

124. Heinricher MM, Fields HL. Central nervous system mechanisms of pain modulation. In: McMahon SB, Koltzenburg M, Tracey I, Turk D, editors. Wall and Melzack's Textbook of Pain. Elsevier/Saunders; 2013. p. 129–42.

125. Fields HL. Pain modulation: expectation, opioid analgesia and virtual pain. Prog Brain Res. 2000;122:245–53.

126. Lau BK, Vaughan CW. Descending modulation of pain: the GABA disinhibition hypothesis of analgesia. Curr Opin Neurobiol. 2014;29:159–64.

127. Heinricher MM, Tavares I, Leith JL, Lumb BM. Descending control of nociception: Specificity, recruitment and plasticity. Brain Res Rev. 2009;60(1):214–25.

128. Ossipov MH. Pain Pathways: Descending Modulation. In: Encyclopedia of Neuroscience. Elsevier; 2009. p. 393–9.

129. Heinricher M, Cheng Z, Fields H. Evidence for two classes of nociceptive modulating neurons in the periaqueductal gray. J Neurosci. 1987;7(1):271–8.

130. Wiech K, Ploner M, Tracey I. Neurocognitive aspects of pain perception.

Trends Cogn Sci. 2008;12(8):306–13.

131. Wiech K, Seymour B, Kalisch R, Enno Stephan K, Koltzenburg M, Driver J, et al. Modulation of pain processing in hyperalgesia by cognitive demand. Neuroimage. 2005;27(1):59–69.

132. Wager TD, Rilling JK, Smith EE, Sokolik A, Casey KL, Davidson RJ, et al. Placebo-induced changes in FMRI in the anticipation and experience of pain. Science. 2004;303(5661):1162–7.

133. Arntz A, Schmidt AJM. Perceived control and the experience of pain. In: Steptoe A, Appels A, editors. Stress, personal control and health. Oxford, England: John Wiley & Sons, Ltd; 1989. p. 131–62.

134. Baliki MN, Apkarian AV. Nociception, Pain, Negative Moods, and Behavior Selection. Neuron. 2015;87(3):474–91.

135. Keogh E, Ellery D, Hunt C, Hannent I. Selective attentional bias for pain-related stimuli amongst pain fearful individuals. Pain. 2001;91(1):91–100.

136. Vachon-Presseau E, Centeno MV, Ren W, Berger SE, Tétreault P, Ghantous M, et al. The Emotional Brain as a Predictor and Amplifier of Chronic Pain. J Dent Res. 2016;95(6):605–12.

137. Vachon-Presseau E, Roy M, Martel MO, Caron E, Marin MF, Chen J, et al. The stress model of chronic pain: Evidence from basal cortisol and hippocampal structure and function in humans. Brain. 2013;136(3):815–27.

138. Neugebauer V, Galhardo V, Maione S, Mackey SC. Forebrain pain mechanisms. Brain Res Rev. 2009;60(1):226–42.

139. Kuo M-F, Nitsche MA. Exploring prefrontal cortex functions in healthy humans by transcranial electrical stimulation. Neurosci Bull. 2015;31(2):198–206.

140. Sullivan MJL, Thorn B, Haythornthwaite JA, Keefe F, Martin M, Bradley LA, et al. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain. 2001;17(1):52–64.

141. Edwards RR, Bingham CO, Bathon J, Haythornthwaite JA. Catastrophizing and pain in arthritis, fibromyalgia, and other rheumatic diseases. Arthritis Rheum. 2006;55(2):325–32.

142. de Aguilera Moyano M, Pindado Pinado J. Nuevos enfoques en comunicación y salud: perspectivas de investigación. Comun Media Educ Res J. 2006;13(26):13–20.

143. Bayliss EA, Ellis JL, Steiner JF. Barriers to Self-Management and Quality-of-Life Outcomes in Seniors With Multimorbidities. Ann Fam Med. 2007;5(5):395–402.

144. Caiata Zufferey M, Schulz PJ. Potentialities and limits of Internet health communication in chronic care: results from a qualitative study. Soc Semiot. 2010;20(1):61–75.

145. Eysenbach G. The Impact of the Internet on Cancer Outcomes. CA Cancer J Clin. 2003;53(6):356–71.

146. Kerr C, Murray E, Stevenson F, Gore C, Nazareth I. Internet interventions for long-term conditions: patient and caregiver quality criteria. J Med Internet Res. 2006;8(3):e13.

147. Lasker JN, Sogolow ED, Sharim RR. The Role of an Online Community for People With a Rare Disease: Content Analysis of Messages Posted on a Primary Biliary Cirrhosis Mailinglist. J Med Internet Res. 2005;7(1):e10.

148. Rini C, Williams DA, Broderick JE, Keefe FJ. Meeting them where they are: Using the Internet to deliver behavioral medicine interventions for pain. Transl Behav Med. 2012;2(1):82–92.

149. Schubart JR, Stuckey HL, Ganeshamoorthy A, Sciamanna CN. Chronic health conditions and internet behavioral interventions: a review of factors to enhance user engagement. Comput Inform Nurs. 2011;29(2 Suppl):TC9-20.

150. Heikkinen K. A comparison of two educational interventions for the cognitive empowerment of ambulatory orthopaedic surgery patients. Annales Universitatis Turkuensis D 973. University of Turku; 2011.

151. Berman RLH, Iris MA, Bode R, Drengenberg C. The effectiveness of an online mind-body intervention for older adults with chronic pain. J Pain. 2009;10(1):68–79.

152. Høybye MT, Johansen C, Tjørnhøj-Thomsen T. Online interaction. Effects of storytelling in an internet breast cancer support group. Psychooncology. 2005;14(3):211–20.

153. Lorig KR, Laurent DD, Deyo RA, Marnell ME, Minor MA, Ritter PL. Can a Back Pain E-mail Discussion Group Improve Health Status and Lower Health Care Costs? Arch Intern Med. 2002;162(7):792.

154. Murray E, Burns J, See Tai S, Lai R, Nazareth I. Interactive Health Communication Applications for people with chronic disease. In: Murray E, editor. Cochrane Database of Systematic Reviews. Chichester, UK: John Wiley & Sons, Ltd; 2005.

155. Shigaki CL, Smarr KL, Yang Gong, Donovan-Hanson K, Siva C, Johnson RA, et al. Social interactions in an online self-management program for rheumatoid arthritis. Chronic Illn. 2008;4(4):239–46.

156. Weinert C, Cudney S, Hill WG. Rural women, technology, and self-management of chronic illness. Can J Nurs Res. 2009;40(3):114–34.

157. Andersson G, Bergström J, Holländare F, Carlbring P, Kaldo V, Ekselius L. Internet-based self-help for depression: randomised controlled trial. Br J Psychiatry. 2005;187(5):456–61.

158. Andersson G, Bergström J, Carlbring P, Lindefors N. The use of the Internet in the treatment of anxiety disorders. Curr Opin Psychiatry. 2005;18(1):73–7.

159. Glasgow RE, Boles SM, McKay HG, Feil EG, Barrera M. The D-Net diabetes self-management program: long-term implementation, outcomes, and generalization results. Prev Med (Baltim). 2003;36(4):410–9.

160. Krishna S, Francisco BD, Balas E a., Konig P, Graff GR, Madsen RW. Internet-Enabled Interactive Multimedia Asthma Education Program: A Randomized Trial. Pediatrics. 2003;111(3):503–10.

161. Bender JL, Radhakrishnan A, Diorio C, Englesakis M, Jadad AR. Can pain be managed through the Internet? A systematic review of randomized controlled trials. Pain. 2011;152(8):1740–50.

162. Garg S, Garg D, Turin TC, Chowdhury MFU. Web-Based Interventions for Chronic Back Pain: A Systematic Review. J Med Internet Res. 2016 Jul 26;18(7):e139.

163. Keulers BJ, Welters CFM, Spauwen PHM, Houpt P. Can face-to-face patient education be replaced by computer-based patient education? A randomised trial. Patient Educ Couns. 2007;67:176–82.

164. Heikkinen K, Helena LK, Taina N, Anne K, Sanna S. A comparison of two educational interventions for the cognitive empowerment of ambulatory orthopaedic surgery patients. Patient Educ Couns. 2008;73(2):272–9.

165. Jones JB, Bruce CA, Shah NR, Taylor WF, Stewart WF. Shared decision making: using health information technology to integrate patient choice into primary care. Transl Behav Med. 2011;1(1):123–33.

166. Mohr DC, Siddique J, Ho J, Duffecy J, Jin L, Fokuo JK. Interest in behavioral and psychological treatments delivered face-to-face, by telephone, and by internet. Ann Behav Med. 2010;40(1):89–98.

167. Fox MP. A systematic review of the literature reporting on studies that examined the impact of interactive, computer-based patient education programs. Patient Educ Couns. 2009;77(1):6–13.

168. Camerini L, Camerini AL, Schulz PJ. Do participation and personalization matter? A model-driven evaluation of an Internet-based patient education intervention for fibromyalgia patients. Patient Educ Couns. 2013;92(2):229–34.

169. Pessoa TM, Coutinho DS, Pereira VM, De Oliveira Ribeiro NP, Nardi AE, De Oliveira e Silva AC. The Nintendo Wii as a tool for neurocognitive rehabilitation, training and health promotion. Comput Human Behav. 2014;31(1):384–92.

170. Claes L, Jiménez-Murcia S, Santamaría JJ, Moussa MB, Sánchez I, Forcano L, et al. The facial and subjective emotional reaction in response to a video game designed to train emotional regulation (Playmancer). Eur Eat Disord Rev. 2012;20(6):484–9.

171. Fernández-Aranda F, Jiménez-Murcia S, Santamaría JJ, Gunnard K, Soto A, Kalapanidas E, et al. Video games as a complementary therapy tool in mental disorders: PlayMancer, a European multicentre study. J Ment Heal. 2012;21(4):364–74.

172. Allam A, Kostova Z, Nakamoto K, Schulz PJ. The Effect of Social Support Features and Gamification on a Web-Based Intervention for Rheumatoid Arthritis Patients: Randomized Controlled Trial. J Med Internet Res. 2015;17(1):e14.

173. Deterding S, Sicart M, Nacke L, O'Hara K, Dixon D. Gamification. using game-design elements in non-gaming contexts. In: Proceedings of the 2011 annual conference extended abstracts on Human factors in computing systems - CHI EA '11. New York, New York, USA: ACM Press; 2011. p. 2425.

174. Domínguez A, Saenz-de-Navarrete J, De-Marcos L, Fernández-Sanz L, Pagés C, Martínez-Herráiz J-J. Gamifying learning experiences: Practical implications and outcomes. Comput Educ. 2013;63:380–92.

175. Moseley L. Combined physiotherapy and education is efficacious for chronic low back pain. Aust J Physiother. 2002;48(4):297–302.

176. Moix, J., Cañellas, M., Osorio, C., Bel, X., Girvent, F., Martos A. Eficacia de un tratamiento interdisciplinar en pacientes con dolor lumbar. Dolor. 2003;18(3):149–57.

177. Moseley GL. Evidence for a direct relationship between cognitive and physical change during an education intervention in people with chronic low back pain. Eur J Pain. 2004;8(1):39–45.

178. Moseley GL. Widespread brain activity during an abdominal task markedly reduced after pain physiology education: fMRI evaluation of a single patient with chronic low back pain. Aust J Physiother. 2005;51(1):49–52.

179. Moseley GL, Nicholas MK, Hodges PW. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. Clin J Pain. 2004;20(5):324–30.

180. Ryan CG, Gray HG, Newton M, Granat MH. Pain biology education and exercise classes compared to pain biology education alone for individuals with chronic low back pain: a pilot randomised controlled trial. Man Ther. 2010;15(4):382–7.

181. Louw A, Puentedura EL, Mintken P. Use of an abbreviated neuroscience education approach in the treatment of chronic low back pain: A case report. Physiother Theory Pract. 2012;28(1):50–62.

182. Moseley GL. Joining Forces – Combining Cognition-Targeted Motor Control Training with Group or Individual Pain Physiology Education: A Successful Treatment For Chronic Low Back Pain. J Man Manip Ther. 2003;11(2):88–

94.

183. Moix J, Cañellas M, Girvent F, Martos A, Ortigosa L, Sánchez C, et al. Confirmación de la eficacia de un programa interdisciplinar en pacientes con dolor de espalda crónico. Rev Soc Esp Dolor. 2004;11(3):141–9.

184. Wälti P, Kool J, Luomajoki H. Short-term effect on pain and function of neurophysiological education and sensorimotor retraining compared to usual physiotherapy in patients with chronic or recurrent non-specific low back pain, a pilot randomized controlled trial. BMC Musculoskelet Disord. 2015;16(1):83.

185. Téllez-García M, De-la-Llave-Rincón AI, Salom-Moreno J, Palacios-Ceña M, Ortega-Santiago R, Fernández-de-las-Peñas C. Neuroscience education in addition to trigger point dry needling for the management of patients with mechanical chronic low back pain: A preliminary clinical trial. J Bodyw Mov Ther. 2015;19(3):464–72.

186. Bodes Pardo G, Lluch Girbés E, Roussel NA, Gallego Izquierdo T, Jiménez Penick V, Pecos Martín D. Pain Neurophysiology Education and Therapeutic Exercise for Patients With Chronic Low Back Pain: A Single-Blind Randomized Controlled Trial. Arch Phys Med Rehabil. 2018;99(2):338–47.

187. Clarke CL, Ryan CG, Martin DJ. Pain neurophysiology education for the management of individuals with chronic low back pain: systematic review and meta-analysis. Man Ther. 2011;16(6):544–9.

188. Johnson RB, Onwuegbuzie AJ. Mixed Methods Research : A Research Paradigm Whose Time Has Come. Educ Res. 2004;33(7):14–26.

189. Creswell JJ, Klassen ACA, Plano V, Smith KKC, Plano Clark V, Smith KKC. Best practices for mixed methods research in the health sciences [Internet]. National Institutes of Health. Bethesda (Maryland); 2011 [cited 2015 Jul 22]. Available from: http://obssr.od.nih.gov/mixed_methods_research

190. Fetters MD, Curry LA, Creswell JW. Achieving integration in mixed methods designs-principles and practices. Health Serv Res. 2013;48(6 Pt 2):2134–56.

191. Hernández R, Fernández C, Baptista M del P. Los métodos mixtos. In: Metodología de la investigación. 5th ed. México: Mc Graw Hill; 2010. p. 544–601.

192. Koes BW. Diagnosis and treatment of low back pain. BMJ. 2006;332(7555):1430–4.

193. Caelli K, Ray L, Mill J. 'Clear as Mud': Toward Greater Clarity in Generic Qualitative Research. Int J Qual Methods. 2003;2(2):1–13.

194. Neergaard MA, Olesen F, Andersen RS, Sondergaard J. Qualitative description – the poor cousin of health research? BMC Med Res Methodol. 2009;9(1):52.

195. Teddlie C, Yu F. Mixed Methods Sampling: A Typology With Examples. J Mix Methods Res. 2007;1(1):77–100.

196. Briones-Vozmediano E, Vives-Cases C, Ronda-Pérez E, Gil-González D. Patients' and professionals' views on managing fibromyalgia. Pain Res Manag. 2013;18(1):19–24.

197. Pope C, Ziebland S, Mays N. Qualitative research in health care. Analysing qualitative data. BMJ. 2000;320(7227):114–6.

198. Fitzpatrick R, Boulton M. Qualitative methods for assessing health care. Qual Saf Heal Care. 1994;3(2):107–13.

199. Doody O, Noonan M. Preparing and conducting interviews to collect data. Nurse Res. 2013;20(5):28–32.

200. Braun V, Clarke V. Using thematic analysis in psychology. Qual Res Psychol. 2006;3(2):77–101.

201. Vaismoradi M, Turunen H, Bondas T. Content analysis and thematic analysis: Implications for conducting a qualitative descriptive study. Nurs Health Sci. 2013;15(3):398–405.

202. Graneheim U., Lundman B. Qualitative content analysis in nursing research: concepts, procedures and measures to achieve trustworthiness. Nurse Educ Today. 2004;24(2):105–12.

203. Nielsen J. How long do users stay on web pages? [Internet]. Nielsen Norman Group. 2011 [cited 2018 Aug 27]. Available from: https://www.nngroup.com/articles/how-long-do-users-stay-on-web-pages/

204. Edmond SN, Keefe FJ. Validating pain communication. Pain. 2015 Feb;156(2):215–9.

205. Reid M. An Assessment of Health Needs of Chronic Low Back Pain Patients from General Practice. J Health Psychol. 2004;9(3):451–62.

206. Holloway I, Sofaer-Bennett B, Walker J. The stigmatisation of people with chronic back pain. Disabil Rehabil. 2007;29(18):1456–64.

207. Greville-Harris M, Hempel R, Karl A, Dieppe P, Lynch TR. The Power of Invalidating Communication: Receiving Invalidating Feedback Predicts Threat-Related Emotional, Physiological, and Social Responses. J Soc Clin Psychol. 2016;35(6):471–93.

208. Linton SJ, Boersma K, Vangronsveld K, Fruzzetti A. Painfully reassuring? The effects of validation on emotions and adherence in a pain test. Eur J Pain. 2012;16(4):592–9.

209. Lethem J, Slade PD, Troup JD, Bentley G. Outline of a Fear-Avoidance Model of exaggerated pain perception--I. Behav Res Ther. 1983;21(4):401–8.

210. Rainville J, Smeets RJEM, Bendix T, Tveito TH, Poiraudeau S, Indahl AJ.

Fear-avoidance beliefs and pain avoidance in low back pain—translating research into clinical practice. Spine J. 2011;11(9):895–903.

211. Verbunt J a, Seelen H a, Vlaeyen JW, van der Heijden GJ, Knottnerus JA. Fear of injury and physical deconditioning in patients with chronic low back pain. Arch Phys Med Rehabil. 2003;84(8):1227–32.

212. Arntz A, Claassens L. The meaning of pain influences its experienced intensity. Pain. 2004;109(1):20–5.

213. Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain. 2000;85(3):317–32.

214. Grotle M, Vøllestad NK, Veierød MB, Brox JI. Fear-avoidance beliefs and distress in relation to disability in acute and chronic low back pain. Pain. 2004;112(3):343.

215. Newcomer KL, Shelerud R a, Vickers Douglas KS, Larson DR, Crawford BJ. Anxiety levels, fear-avoidance beliefs, and disability levels at baseline and at 1 year among subjects with acute and chronic low back pain. J Inj Funct Rehabil. 2010;2(6):514–20.

216. Moseley GL, Vlaeyen JWS. Beyond nociception: the imprecision hypothesis of chronic pain. Pain. 2015;156(1):35–8.

217. Amirdelfan K, McRoberts P, Deer TR. The differential diagnosis of low back pain: a primer on the evolving paradigm. Neuromodulation. 2014;17 Suppl 2(S2):11–7.

218. Dagenais S, Tricco AC, Haldeman S. Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. Spine J. 2010;10(6):514–29.

219. Rubin DI. Epidemiology and risk factors for spine pain. Neurol Clin. 2007;25(2):353–71.

220. Brinjikji W, Luetmer PH, Comstock B, Bresnahan BW, Chen LE, Deyo RA, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. AJNR Am J Neuroradiol. 2015;36(4):811–6.

221. Jarvik JG, Hollingworth W, Heagerty PJ, Haynor DR, Boyko EJ, Deyo RA. Three-year incidence of low back pain in an initially asymptomatic cohort: Clinical and imaging risk factors. Spine (Phila Pa 1976). 2005;30(13):1541–8.

222. Savage RA, Whitehouse GH, Roberts N. The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males. Eur Spine J. 1997;6(2):106–14.

223. Hannibal KE, Bishop MD. Chronic stress, cortisol dysfunction, and pain: a psychoneuroendocrine rationale for stress management in pain rehabilitation. Phys Ther. 2014;94(12):1816–25.

224. Johnson AC, Greenwood-Van Meerveld B. Stress-induced pain: a target for the development of novel therapeutics. J Pharmacol Exp Ther. 2014;351(2):327–35.

225. Colloca L, Benedetti F. Nocebo hyperalgesia: how anxiety is turned into pain. Curr Opin Anaesthesiol. 2007;20(5):435–9.

226. Kuehl LK, Michaux GP, Richter S, Schächinger H, Anton F. Increased basal mechanical pain sensitivity but decreased perceptual wind-up in a human model of relative hypocortisolism. Pain. 2010;149(3):539–46.

227. Choi JC, Chung MI, Lee YD. Modulation of pain sensation by stress-related testosterone and cortisol. Anaesthesia. 2012;67(10):1146–51.

228. Geiss A, Rohleder N, Kirschbaum C, Steinbach K, Bauer HW, Anton F. Predicting the failure of disc surgery by a hypofunctional HPA axis: evidence from a prospective study on patients undergoing disc surgery. Pain. 2005;114(1–2):104–17.

229. Finestone HM, Alfeeli A, Fisher WA. Stress-induced Physiologic Changes as a Basis for the Biopsychosocial Model of Chronic Musculoskeletal Pain. Clin J Pain. 2008;24(9):767–75.

230. Ortego G, Villafañe JH, Doménech-García V, Berjano P, Bertozzi L, Herrero P. Is there a relationship between psychological stress or anxiety and chronic nonspecific neck-arm pain in adults? A systematic review and meta-analysis. J Psychosom Res. 2016;90:70–81.

231. Henschke N, Ostelo RW, van Tulder MW, Vlaeyen JW, Morley S, Assendelft WJ, et al. Behavioural treatment for chronic low-back pain. Cochrane Database Syst Rev. 2010;(7):CD002014.

232. Chou R, Deyo R, Friedly J, Skelly A, Hashimoto R, Weimer M, et al. Nonpharmacologic Therapies for Low Back Pain: A Systematic Review for an American College of Physicians Clinical Practice Guideline. Ann Intern Med. 2017;166(7):493–505.

233. Cramer H, Haller H, Lauche R, Dobos G. Mindfulness-based stress reduction for low back pain. A systematic review. BMC Complement Altern Med. 2012;12(1):162.

234. Monticone M, Ferrante S, Rocca B, Baiardi P, Farra FD, Foti C. Effect of a long-lasting multidisciplinary program on disability and fear-avoidance behaviors in patients with chronic low back pain: results of a randomized controlled trial. Clin J Pain. 2013;29(11):929–38.

235. Grossman P, Niemann L, Schmidt S, Walach H. Mindfulness-based stress reduction and health benefits. A meta-analysis. J Psychosom Res. 2004;57(1):35–43.

236. Ambrose KR, Golightly YM. Physical exercise as non-pharmacological treatment of chronic pain: Why and when. Best Pract Res Clin Rheumatol. 2015;29(1):120–30.

237. Tremblay MS, Colley RC, Saunders TJ, Healy GN, Owen N. Physiological and health implications of a sedentary lifestyle. Appl Physiol Nutr Metab. 2010;35(6):725–40.

238. Caruso MV, Serra R, Perri P, Buffone G, Caliò FG, DE Franciscis S, et al. A computational evaluation of sedentary lifestyle effects on carotid hemodynamics and atherosclerotic events incidence. Acta Bioeng Biomech. 2017;19(3):42–52.

239. Blümel JE, Fica J, Chedraui P, Mezones-Holguín E, Zuñiga MC, Witis S, et al. Sedentary lifestyle in middle-aged women is associated with severe menopausal symptoms and obesity. Menopause. 2016;23(5):488–93.

240. Semanik PA, Lee J, Song J, Chang RW, Sohn M-W, Ehrlich-Jones LS, et al. Accelerometer-monitored sedentary behavior and observed physical function loss. Am J Public Health. 2015;105(3):560–6.

241. Lee J, Chang RW, Ehrlich-Jones L, Kwoh CK, Nevitt M, Semanik PA, et al. Sedentary Behavior and Physical Function: Objective Evidence From the Osteoarthritis Initiative. Arthritis Care Res (Hoboken). 2015;67(3):366–73.

242. Lin C-WC, McAuley JH, Macedo L, Barnett DC, Smeets RJ, Verbunt JA. Relationship between physical activity and disability in low back pain: a systematic review and meta-analysis. Pain. 2011;152(3):607–13.

243. Ellingson LD, Shields MR, Stegner AJ, Cook DB. Physical Activity, Sustained Sedentary Behavior, and Pain Modulation in Women With Fibromyalgia. J Pain. 2012;13(2):195–206.

244. Stefansdottir R, Gudmundsdottir SL. Sedentary behavior and musculoskeletal pain: a five-year longitudinal Icelandic study. Public Health. 2017;149:71–3.

245. Kaleth AS, Saha CK, Jensen MP, Slaven JE, Ang DC. Effect of moderate to vigorous physical activity on long-term clinical outcomes and pain severity in

fibromyalgia. Arthritis Care Res (Hoboken). 2013;65(8):1211–8.

246. Giannotti E, Koutsikos K, Pigatto M, Rampudda ME, Doria A, Masiero S. Medium-/Long-Term Effects of a Specific Exercise Protocol Combined with Patient Education on Spine Mobility, Chronic Fatigue, Pain, Aerobic Fitness and Level of Disability in Fibromyalgia. Biomed Res Int. 2014;2014:1–9.

247. Latorre PÁ, Santos MA, Heredia-Jiménez JM, Delgado-Fernández M, Soto VM, Mañas A, et al. Effect of a 24-week physical training programme (in water and on land) on pain, functional capacity, body composition and quality of life in women with fibromyalgia. Clin Exp Rheumatol. 2013;31(6 Suppl 79):S72-80.

248. Hagen KB, Dagfinrud H, Moe RH, Østerås N, Kjeken I, Grotle M, et al. Exercise therapy for bone and muscle health: an overview of systematic reviews. BMC Med. 2012;10(1):167.

249. Kroll HR. Exercise therapy for chronic pain. Phys Med Rehabil Clin N Am. 2015;26(2):263–81.

250. Naugle KM, Fillingim RB, Riley JL. A Meta-Analytic Review of the Hypoalgesic Effects of Exercise. J Pain. 2012;13(12):1139–50.

251. Nijs J, Kosek E, Van Oosterwijck J, Meeus M. Dysfunctional endogenous analgesia during exercise in patients with chronic pain: to exercise or not to exercise? Pain Physician. 2012;15(3 Suppl):ES205-13.

252. Cooper MA, Kluding PM, Wright DE. Emerging Relationships between Exercise, Sensory Nerves, and Neuropathic Pain. Front Neurosci. 2016;10(AUG):372.

253. Henchoz Y, Kai-Lik So A. Exercise and nonspecific low back pain: a literature review. Joint Bone Spine. 2008;75(5):533–9.

254. van Middelkoop M, Rubinstein SM, Verhagen AP, Ostelo RW, Koes BW, van

Tulder MW. Exercise therapy for chronic nonspecific low-back pain. Best Pract Res Clin Rheumatol. 2010;24(2):193–204.

255. Daenen L, Varkey E, Kellmann M, Nijs J. Exercise, not to exercise, or how to exercise in patients with chronic pain? Applying science to practice. Clin J Pain. 2015;31(2):108–14.

256. Hoffman MD, Shepanski MA, Mackenzie SP, Clifford PS. Experimentally induced pain perception is acutely reduced by aerobic exercise in people with chronic low back pain. J Rehabil Res Dev. 2005;42(2):183–90.

257. Hodges PW, Smeets RJ. Interaction between pain, movement, and physical activity: short-term benefits, long-term consequences, and targets for treatment. Clin J Pain. 2015;31(2):97–107.

258. Tantimonaco M, Ceci R, Sabatini S, Catani MV, Rossi A, Gasperi V, et al. Physical activity and the endocannabinoid system: an overview. Cell Mol Life Sci. 2014;71(14):2681–98.

259. Hayden JA, van Tulder MW, Tomlinson G. Systematic review: strategies for using exercise therapy to improve outcomes in chronic low back pain. Ann Intern Med. 2005;142(9):776–85.

260. Gleeson M, Bishop NC, Stensel DJ, Lindley MR, Mastana SS, Nimmo MA. The anti-inflammatory effects of exercise: mechanisms and implications for the prevention and treatment of disease. Nat Rev Immunol. 2011;11(9):607–15.

261. Moher D, Hopewell S, Schulz KF, Montori V, Gøtzsche PC, Devereaux PJ, et al. CONSORT 2010 explanation and elaboration: updated guidelines for reporting parallel group randomised trials. Int J Surg. 2012;10(1):28–55.

262. Begg C, Cho M, Eastwood S, Horton R, Moher D, Olkin I, et al. Improving the quality of reporting of randomized controlled trials. The CONSORT statement. JAMA. 1996;276(8):637–9.

263. Hernández Sampieri R, Fernández Collado C, Baptista Lucio P. Selección de la muestra. In: Metodología de la Investigación. 2010. p. 170–95.

264. Ostelo RWJG, Deyo RA, Stratford P, Waddell G, Croft P, Von Korff M, et al. Interpreting Change Scores for Pain and Functional Status in Low Back Pain. Spine (Phila Pa 1976). 2008;33(1):90–4.

265. Furlan AD, Pennick V, Bombardier C, van Tulder M. 2009 Updated Method Guidelines for Systematic Reviews in the Cochrane Back Review Group. Spine (Phila Pa 1976). 2009;34(18):1929–41.

266. Jaeschke R, Singer J, Guyatt GH. Measurement of health status. Control Clin Trials. 1989;10(4):407–15.

267. Serrano-Atero M, Caballero J, Cañas A, Gracía-Saura P, Serrano-Álvarez C, Prieto J. Valoración del dolor (I). Rev Soc Esp Dolor. 2002;9(2):94–108.

268. Serrano-Atero M, Caballero J, Cañas A, Gracía-Saura P, Serrano-Álvarez C, Prieto J. Valoración del dolor (II). Rev Soc Esp Dolor. 2002;9(2):109–21.

269. Younger J, McCue R, Mackey S. Pain Outcomes: A Brief Review of Instruments and Techniques. Curr Pain Headache Rep. 2009;13(1):39–43.

270. Dworkin RH, Turk DC, Wyrwich KW, Beaton D, Cleeland CS, Farrar JT, et al. Interpreting the Clinical Importance of Treatment Outcomes in Chronic Pain Clinical Trials: IMMPACT Recommendations. J Pain. 2008;9(2):105–21.

271. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain. 1993;52(2):157–68.

272. Gómez-Pérez L, López-Martínez AE, Ruiz-Párraga GT. Psychometric Properties of the Spanish Version of the Tampa Scale for Kinesiophobia (TSK). J Pain. 2011;12(4):425–35.

273. Kovacs FM, Muriel A, Medina JM, Abraira V, Sánchez MDC, Jaúregui JO. Psychometric characteristics of the Spanish version of the FAB questionnaire. Spine (Phila Pa 1976). 2006;31(1):104–10.

274. Woby SR, Roach NK, Urmston M, Watson PJ. Psychometric properties of the TSK-11: a shortened version of the Tampa Scale for Kinesiophobia. Pain. 2005;117(1–2):137–44.

275. Vlaeyen JW, Kole-Snijders a M, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain. 1995;62(3):363–72.

276. Hapidou EG, O'Brien MA, Pierrynowski MR, de Las Heras E, Patel M, Patla T. Fear and Avoidance of Movement in People with Chronic Pain: Psychometric Properties of the 11-Item Tampa Scale for Kinesiophobia (TSK-11). Physiother Can. 2012;64(3):235–41.

277. Roland M, Morris R. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. Spine (Phila Pa 1976). 1983;8(2):141–4.

278. Roland M, Fairbank J. The Roland-Morris Disability Questionnaire and the Oswestry Disability Questionnaire. Spine (Phila Pa 1976). 2000;25(24):3115–24.

279. Kovacs FMM, Llobera J, del Real MTG, Abraira V, Gestoso M, Fernandez C, et al. Validation of the Spanish version of the Roland-Morris Questionnaire. Spine (Phila Pa 1976). 2002;27(5):538–42.

280. Richardson JTE. Eta squared and partial eta squared as measures of effect size in educational research. Educ Res Rev. 2011;6(2):135–47.

281. Carpenter KM, Stoner SA, Mundt JM, Stoelb B. An online self-help CBT intervention for chronic lower back pain. Clin J Pain. 2012;28(1):14–22.

282. Buhrman M, Nilsson-Ihrfeldt E, Jannert M, Ström L, Andersson G. Guided internet-based cognitive behavioural treatment for chronic back pain reduces pain catastrophizing: a randomized controlled trial. J Rehabil Med. 2011;43(6):500–5.

283. Chiauzzi E, Pujol LA, Wood M, Bond K, Black R, Yiu E, et al. painACTION-back pain: a self-management website for people with chronic back pain. Pain Med. 2010;11(7):1044–58.

284. Krein SL, Kadri R, Hughes M, Kerr EA, Piette JD, Holleman R, et al. Pedometer-based internet-mediated intervention for adults with chronic low back pain: randomized controlled trial. J Med Internet Res. 2013;15(8):e181.

285. Moessner M, Schiltenwolf M, Neubauer E. Internet-based aftercare for patients with back pain-a pilot study. Telemed J E Health. 2012;18(6):413–9.

286. Buhrman M, Fältenhag S, Ström L, Andersson G. Controlled trial of Internet-based treatment with telephone support for chronic back pain. Pain. 2004;111(3):368–77.

287. Whitley E, Ball J. Statistics review 4: sample size calculations. Crit Care. 2002;6(4):335–41.

288. Lewis-Beck MS, Bryman A, Liao TF. Ceiling Effect. In: The SAGE Encyclopedia of Social Science Research Methods. 2455 Teller Road, Thousand Oaks California 91320 United States of America: Sage Publications, Inc.; 2004.

289. Jacobs GD, Pace-Schott EF, Stickgold R, Otto MW. Cognitive behavior therapy and pharmacotherapy for insomnia: a randomized controlled trial and direct comparison. Arch Intern Med. 2004;164(17):1888–96.

290. Lüning Bergsten C, Lundberg M, Lindberg P, Elfving B. Change in kinesiophobia and its relation to activity limitation after multidisciplinary rehabilitation in patients with chronic back pain. Disabil Rehabil.

2012;34(10):852–8.

291. Higgins JPT, Altman DG, Gotzsche PC, Juni P, Moher D, Oxman AD, et al. The Cochrane Collaboration’s tool for assessing risk of bias in randomised trials. BMJ. 2011 Oct 18;343(oct18 2):d5928–d5928.

292. Parsons S, Harding G, Breen A, Foster N, Pincus T, Vogel S, et al. The Influence of Patients´ and Primary Care Practitioners´ Beliefs and Expectations About Chronic Musculoskeletal Pain on the Process of Care. Clin J Pain. 2007;23(1):91–8.

293. Darlow B, Fullen BM, Dean S, Hurley D a, Baxter GD, Dowell A. The association between health care professional attitudes and beliefs and the attitudes and beliefs, clinical management, and outcomes of patients with low back pain: a systematic review. Eur J Pain. 2012;16(1):3–17.

294. Linton SJ, Vlaeyen J, Ostelo R. The Back Pain Beliefs of Health Care Providers: Are We Fear-Avoidant? J Occup Rehabil. 2002;12(4):223–32.

295. Kahlke RM. Generic qualitative approaches: Pitfalls and benefits of methodological mixology. Int J Qual Methods. 2014;13(1):37–52.

296. Laerum E, Indahl A, Skouen JS. What is “the good back-consultation”? A combined qualitative and quantitative study of chronic low back pain patients’ interaction with and perceptions of consultations with specialists. J Rehabil Med. 2006;38(4):255–62.

297. Nielsen M, Jull G, Hodges PW. Information needs of people with low back pain for an online resource: a qualitative study of consumer views. Disabil Rehabil. 2014;36(13):1085–91.

298. Verbeek J, Sengers M-J, Riemens L, Haafkens J. Patient Expectations of Treatment for Back Pain. Spine (Phila Pa 1976). 2004;29(20):2309–18.

299. Souto Camba S. Autonomía profesional y acceso directo en fisioterapia.

Fisioterapia. 2013;35(3):89–91.

300. Ojha HA, Snyder RS, Davenport TE. Direct access compared with referred physical therapy episodes of care: a systematic review. Phys Ther. 2014;94(1):14–30.

301. Rubí-Carnacea FJ. Efectivitat d’una intervenció fisioterapèutica basada en la pre-activació del transvers abdominal en pacients amb lumbàlgia inespecífica crònica en Atenció Primària. University of Lleida; 2017.

302. Valenzuela-Pascual F, Molina F, Corbi F, Blanco-Blanco J, Gil RM, Soler-Gonzalez J. The influence of a biopsychosocial educational internet-based intervention on pain, dysfunction, quality of life, and pain cognition in chronic low back pain patients in primary care: a mixed methods approach. BMC Med Inform Decis Mak. 2015;15(1):97.

Appendices

Appendix 1: Informed consent qualitative phase

CONSENTIMIENTO INFORMADO

Título del Estudio: Influencia de una intervención educativa biopsicosocial en el dolor, la disfunción, y las cogniciones del dolor, en pacientes con dolor lumbar crónico en atención primaria: Un enfoque de métodos mixtos.

Investigador: Francesc Valenzuela Pascual, profesor de la Facultad de Enfermería de la UdL. Fisioterapeuta col. Nº 2703

El profesor Valenzuela es un fisioterapeuta especializado en dolor lumbar crónico, que estudia la manera en la que los pacientes perciben este dolor y cómo influye en sus vidas diarias. Los resultados de este estudio nos permitirá construir una herramienta educativa online de la que se podrán beneficiar en un futuro los pacientes con dolor lumbar crónico.

El estudio y sus procedimientos han sido aprobados por el "Comitè Ètic d'Investigació Clínica de l'IDIAP Jordi Gol". El procedimiento del estudio implica que no habrá daños previsibles. El procedimiento consiste en una entrevista personal sobre el dolor lumbar crónico del paciente que será audiograbada. La participación en el estudio va a ocuparle, aproximadamente, 90 minutos.

Su participación en el estudio es voluntaria; no tiene ninguna obligación de participar. Tiene derecho a abandonar el estudio cuando quiera.

La información del estudio será codificada para que no pueda relacionarse con usted. Su identidad no se publicará durante la realización del estudio, ni una vez haya sido publicado. Toda la información del estudio será recopilada por el profesor Valenzuela, se mantendrá en un lugar seguro y no será compartida con nadie más sin su permiso.

He leído el formulario de consentimiento y voluntariamente consiento en participar en este estudio.

____

Firma del sujeto Fecha

He explicado el estudio al individuo arriba representado y he confirmado su comprensión para el consentimiento informado.

____

Firma del investigador Fecha

Appendix 2: Informed consent quantitative phase

CONSENTIMIENTO INFORMADO

Se les invita a participar en el siguiente estudio de investigación llevado a cabo por el equipo de investigación de la Facultad de Enfermería y Fisioterapia de la Universitat de Lleida.

Título del estudio: Influencia de una intervención educativa biopsicosocial en el dolor, la disfunción, y las cogniciones del dolor, en pacientes con dolor lumbar crónico en atención primaria: Un enfoque de métodos mixtos.

Investigador principal: Francesc Valenzuela Pascual, profesor de la Facultad de Enfermería de la UdL. Fisioterapeuta col. Nº 2703

Centro: Universitat de Lleida. Facultad de Enfermería y Fisioterapia
C/ Montserrat Roig, 2. 25198 Lleida
Telf. 973702457

PARTE I: Información del estudio

Introducción.

El profesor Valenzuela es un fisioterapeuta especializado en dolor lumbar crónico, que estudia la manera en la que los pacientes perciben este dolor y cómo influye en sus vidas diarias.

El estudio y sus procedimientos han sido aprobados por el "Comitè Ètic d'Investigació Clínica de l'IDIAP Jordi Gol". El procedimiento del estudio implica que no habrá daños previsibles. A continuación, va a recibir información y va a ser invitado a participar en dicha investigación. Antes de dar su consentimiento lea con atención esta hoja informativa. No tiene que responder hoy, puede consultar con el equipo de investigación u otros profesionales ajenos antes de tomar una decisión. Si tiene dudas, formule todas las preguntas que sean necesarias.

Participación voluntaria.

Su participación en esta investigación es totalmente voluntaria. Usted puede elegir si quiere o no participar. De aceptar participar, podrá abandonar el estudio en cualquier momento con total libertad, sin que ello suponga que usted recibirá una peor atención.

Descripción general del estudio.

Los pacientes serán asignados al azar a los grupos (intervención y control), por lo que, hasta que no se efectúe el sorteo no sabrá a que grupo será asignado. La probabilidad de que se le asigne uno u otro es del 50%.

Si participa en el estudio deberá seguir las instrucciones que se le indiquen en la página web. Una vez usted haya consentido participar en el estudio, tendrá acceso a la página web mediante un código que se le proporcionará en el momento que firme este consentimiento informado. En el momento de acceder, aparecerá un formulario de solicitud de datos personales y una serie de cuestionarios que tendrá que rellenar siguiendo las instrucciones que se le proporcionarán. Después de haber rellenado todos los cuestionarios por primera vez, aparecerá un mensaje en la pantalla donde se le explicarán los siguientes pasos a seguir. Pueden darse dos opciones, **solamente se le asignará una de ellas**:

1. Se le dará acceso a la plataforma web. Este acceso tendrá una duración de 15 días. Durante este tiempo usted se compromete a leer y seguir la información y las instrucciones que aparecerán en dicha plataforma web.

2. Se le pedirá que acuda a su médico de familia para que aplique el protocolo establecido por el ICS. En el caso de que su médico ya le haya prescrito recientemente un tratamiento, se le pedirá que continúe con él.

Independientemente del grupo al que haya sido asignado, se le pedirá que repita los cuestionarios dos semanas más tarde siguiendo el mismo procedimiento de acceso a la página web. A los 14-15 días recibirá un correo electrónico para recordarle que debe volver a rellenar los cuestionarios.

Beneficios y riesgos derivados de su participación en el estudio.

Los resultados obtenidos ayudarán a desarrollar una plataforma web basada en la neurofisiología del dolor para cambiar la cognición de los pacientes y, consecuentemente, reducir las creencias erróneas con respecto al dolor lumbar crónico.

Debido a que la intervención es de carácter educativo, no existe ningún riesgo derivado del estudio para usted.

Confidencialidad.

La gestión, la comunicación y la cesión de los datos de carácter personal de todos los participantes se ajustará a lo dispuesto en la Ley Orgánica 15/1999, del 13 de diciembre de protección de datos de carácter personal. De acuerdo a lo que se establece en dicha legislación, usted puede ejercer los derechos de acceso, modificación, oposición y cancelación de datos, para lo cual deberá dirigirse a la persona de contacto.

La información recogida durante la investigación será totalmente confidencial. Los datos estarán identificados mediante un código, de forma que nadie pueda asociar la información a su persona. Únicamente los miembros del equipo investigador/colaboradores podrán relacionar dichos datos.

El acceso a su información personal quedará restringido al personal del estudio y colaboradores, autoridades sanitarias, al Comité Ético de Investigación Clínica y personal autorizado por el investigador, cuando lo precisen para comprobar los datos y el procedimiento del estudio, pero siempre manteniendo la confidencialidad de los mismos de acuerdo a la legislación vigente.

Compensación económica.

La financiación de dicho estudio es gestionada por el investigador principal. Su participación en el estudio no le supondrá ningún gasto. La intervención educativa es gratuita.

Por otro lado, tampoco recibirá ninguna compensación económica ya que su participación no supone ningún riesgo para su salud.

Con quien contactar.

En caso de dudas o aparecer problemas o complicaciones durante la realización del estudio puede contactar con la persona responsable de la investigación.

Francesc Valenzuela Pascual, fisioterapeuta. Col. 2703
fvp1969@dif.udl.cat 692864540

Otra información relevante.

Cualquier información que surja durante la investigación y pueda afectar a su participación, le será comunicada por el investigador lo antes posible.

El investigador podrá excluir del estudio si lo considera oportuno a los participantes que no sigan el procedimiento establecido en el estudio. En el caso de ser excluido, usted recibirá una explicación adecuada del motivo que ha ocasionado su retirada del estudio.

Al firmar la hoja de consentimiento adjunta, se compromete a cumplir con los procedimientos del estudio que se le han expuesto.

Cuando acabe su participación no podrá seguir utilizando la plataforma web utilizada en el estudio.

PARTE II: Declaración del paciente.

Declaro que he sido informado/a por el investigador principal del estudio del objetivo del presente proyecto de investigación y que la participación no implica ninguna intervención física, por lo que no se esperan efectos secundarios y/o reacciones adversas; y que sé que, en cualquier momento puedo revocar mi consentimiento.

Estoy satisfecho/a de la información recibida, he podido formular todas las preguntas que he creído conveniente y me han aclarado todas las dudas planteadas.

En consecuencia, doy mi consentimiento.

Nombre:

Apellidos:

____

Firma del sujeto Fecha

He explicado el estudio al individuo arriba representado y he confirmado su comprensión para el consentimiento informado.

CÓDIGO:

____

Firma del investigador Fecha

Appendix 3: Interview guide qualitative phase

Interview guide

1	For how long have you had this low back pain?
2	Do you remember how did this pain begin?
3	What do you do every time you feel the pain?
4	Using your own words, could you tell me what have you been told about your pain?
5	What kind of treatments have you had?
6	Even though you have had different treatments, why do you think the pain is still there?
7	What does this pain mean for you? In other words, when it hurts, why do you think it hurts?
8	Do you think the health professionals who have treated you have understood your pain?
9	What activities or hobbies have you stopped doing since this low back pain started?
10	Why?
11	In reference to your back pain, if you could go back in time, what things would you change? What things would you do differently?
12	In what way your pain has affected your family, work, and social life?
13	What would you like to know about your pain?
14	If you were with the world expert on chronic low back pain, what would you ask him?

Appendix 4: Written material video 3

ORIGEN Y CAUSAS DEL DOLOR LUMBAR CRÓNICO

FRAN VALENZUELA PASCUAL

FACULTAD DE ENFERMERÍA Y FISIOTERAPIA,
UNIVERSIDAD DE LLEIDA.

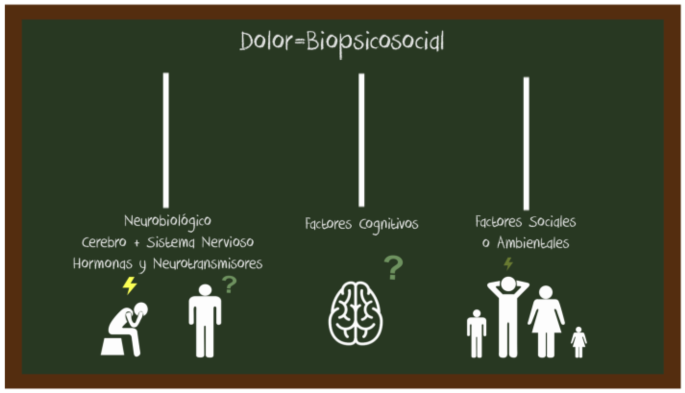
En este texto queremos mostrarle qué nos dice la evidencia científica sobre muchas de las creencias que tenemos sobre el origen y las causas del dolor lumbar crónico.

Desde pequeños nos han enseñado que el dolor es una alarma y que, por lo tanto, cuando duele la espalda es que algo va mal en la propia espalda. Pero esto, ¿es siempre así? Cuando nos damos un golpe o realizamos movimientos excesivos que afectan a nuestros músculos y tejidos, podemos sentir dolor y en este caso el dolor es una alarma. Nuestro cerebro genera dolor durante el tiempo necesario para que nuestro cuerpo evite repetir la acción que generó la lesión y permita repararla. Este tiempo está estipulado en unas 6 semanas. Es lo que llamamos dolor agudo y es en esta situación en la que el cerebro, para proteger a la zona que está recuperándose, genera el dolor.

Pero, ¿qué pasa cuando el dolor dura más tiempo de lo establecido? ¿Este dolor sigue siendo una alarma o bien ha dejado de cumplir esa función protectora? Seguramente, a la mayoría de nosotros cuando nos duele la espalda nos gustaría poder mirar dentro, que nos hicieran una prueba por imagen como una resonancia magnética, para ver el alcance de nuestro problema y conocer la razón de nuestro dolor lumbar. ¡Qué mejor que ver in situ lo que causa mi dolor lumbar! En cambio, los estudios científicos nos dicen que si nos hemos lesionado sin gravedad, no hace falta

Nuestro cerebro genera dolor durante el tiempo necesario para que nuestro cuerpo evite repetir la acción que generó la lesión y permita repararla. Este tiempo está estipulado en unas 6 semanas.

realizar una prueba por imagen ya que conocemos la razón de nuestro dolor y éste irá desapareciendo progresivamente con ayuda de los profesionales sanitarios. Por desgracia, en la mayoría de los casos de dolor lumbar crónico, no hay una evidencia de lesión, no ha existido un mecanismo lesional claro y preciso. El dolor empezó de una manera progresiva sin una razón aparente y es entonces cuando nos empezamos a preocupar por la causa de nuestro dolor lumbar. A veces incluso exigimos que nos hagan una prueba por imagen, una radiografía o una resonancia magnética, para encontrar la causa del dolor. Entonces surge una pregunta, si el dolor ha dejado de ser una alarma, ¿realmente lo que veamos en la resonancia magnética, en la prueba por imagen, será la causa de nuestro dolor lumbar?

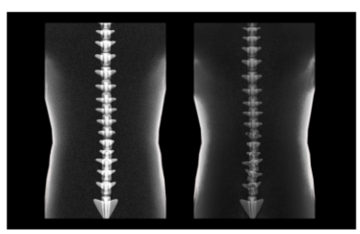


Cuando a una persona con dolor lumbar crónico le preguntas por la causa de su dolor puede darte diferentes respuestas pero todas ellas tienen en común que la razón del dolor está siempre en la zona lumbar:

- Es por culpa de las vértebras
- Tengo una hernia discal
- Es muscular
- Es un nervio que está pinzado
- Es un problema degenerativo
- Tengo la zona lumbar inflamada
- Seguro que tengo una pierna más larga que la otra

Como puede ver, las respuestas son múltiples pero, ¿es alguna de estas respuestas la correcta? ¿Hay una sola causa en el dolor lumbar crónico?
En primer lugar, me gustaría que se hiciera las siguientes preguntas: si realmente mi dolor lumbar está causado por algo estructural, ya sea la vértebra, la hernia discal, el músculo o el reuma, ¿por qué hay días que me duele y otros que no? ¿Por qué el mismo movimiento o la misma actividad unos días me duele más, otros menos y otros incluso no me duele? Parecería lógico pensar que si tengo una alteración en la zona lumbar, como una hernia discal, la tengo siempre y no unos días sí y otros no, ¿verdad?

Si se fijan en estas dos radiografías de la zona lumbar, podrá observar que una tiene más alteraciones que la otra, ¿verdad que parece más lógico pensar que la persona de la derecha tiene más dolor que la de la izquierda ya que su zona lumbar tiene más problemas degenerativos que la otra? Pero en cambio, resulta que el que tiene más degeneración lumbar "experimenta" menos dolor y esto es algo que ocurre a menudo aunque parezca difícil de creer.



Después de lo que acabamos de explicar, seguro que se preguntará: entonces sino es por culpa de los problemas que tengo en mi espalda ¿por qué tengo dolor lumbar crónico?
Curiosamente, una de las afirmaciones más rotundas a nivel científico con respecto al dolor es que es MULTIFACTORIAL y BIOPSICOSOCIAL. Multifactorial se refiere a que no hay un solo factor que justifique el dolor lumbar crónico, sino que son muchos los factores que causan y mantienen este dolor.

El aspecto biopsicosocial del dolor se refiere a que estos factores que generan o mantienen nuestro dolor son:

- Neurobiológicos. Cómo nuestro cerebro y nuestro sistema nervioso responden ante diferentes situaciones, liberando pequeñas sustancias como las hormonas y los neurotransmisores que influyen en el comportamiento de nuestro organismo. Esto provoca que ante una misma situación a unos les duela la cabeza o tengan dolor y otros, en cambio, no sientan nada de esto.
- Cognitivos. Cómo nuestro cerebro percibe e interpreta lo que sucede a nuestro alrededor. Cada uno de nuestros cerebros puede interpretar la misma situación de diferente manera. Por ejemplo, lo que para unos puede ser un vaso casi lleno, para otros es un vaso casi vacío.
- Sociales o ambientales. Cómo lo que nos rodea, trabajo, familia, sociedad etc, provocan estrés o felicidad lo que influye directamente sobre nosotros como personas.

Le voy a poner un ejemplo:
Les presento a Manoli y a Jordi. Los dos tienen la misma edad y trabajan en una oficina. A los dos les diagnosticaron hace unos años una hernia discal acompañada de problemas degenerativos en las vértebras. Pero solo Manoli tiene dolor lumbar crónico. En cambio Jordi, con el mismo diagnóstico hace una vida normal. Entonces ¿qué es lo que pasó para que solo uno de ellos desarrollara dolor crónico?
Hace unos años, a Manoli le empezó a doler la espalda más de lo habitual y empezó a preocuparse. Le hicieron una resonancia magnética y le diagnosticaron una hernia discal y un principio de degeneración articular en las vértebras. A Manoli, eso de la hernia discal y la degeneración vertebral le pareció algo muy grave. De hecho, ella relaciona su dolor lumbar con un sobreesfuerzo que hizo tras haber levantado unas cajas en su casa, aunque no está seguro del todo. Desde entonces, evita levantar cosas o hacer esfuerzos que impliquen a su zona lumbar ya que tiene miedo de que sus “problemas” en la zona lumbar empeoren y le reproduzcan el dolor. Ya que para ella el dolor significa que la hernia discal está empeorando y de seguir así acabará teniendo que operarse o peor aún, ¡en silla de ruedas! Así que también ha dejado de hacer deporte y cosas que le gustan por la misma razón. Esta falta de actividad también ha repercutido en su musculatura y en sus articulaciones, ¡ya no se siente la misma de antes!

Solamente un 7% de las personas mayores con dolor lumbar tienen limitaciones funcionales.

A Jordi, le pasó algo parecido. Le empezó a doler la zona lumbar y también le hicieron unas pruebas donde le diagnosticaron lo mismo que a Manoli. Pero a J, un profesional sanitario le explicó que la hernia discal y la degeneración articular son un proceso normal en el ser humano. De hecho, la mayor parte de nosotros hemos tenido alguna vez en la vida un episodio de dolor lumbar y muchos de nosotros tenemos hernias discales. Por lo que Jordi, tras unos días de reposo relativo, empezó de nuevo a realizar actividad física supervisado por su fisioterapeuta. De manera que, poco a poco, se dio cuenta de que aunque tenía algunos “problemillas en su zona lumbar” podía seguir haciendo las cosas que le gustaban, progresivamente y bajo la supervisión de su fisioterapeuta.

EL DOLOR ES SIEMPRE REAL

De esta manera, podemos ver que la interpretación que Manoli hace de su dolor lumbar le genera una actitud de excesiva protección. Así que su cerebro, para proteger su zona lumbar, responde generando dolor aunque NO HAGA FALTA, ya que después de unas semanas en las que los tejidos han regenerado ya no hay una alarma. En cambio el cerebro de Jordi interpreta que lo que ocurre en su zona lumbar es normal, por lo que no necesita "sobreprotegerla" y con el paso del tiempo ya no le genera dolor con la actividad.

Volvamos a la evidencia científica. Estudios recientes nos dicen que todas estas alteraciones que podemos observar en las pruebas por imagen, como las de Manoli y Jordi, forman parte del proceso natural de envejecimiento y recuerde que el proceso de envejecimiento empieza sobre los ¡20 años! Además, estas alteraciones aumentan a medida que envejecemos. Pero lo que es más importante, la evidencia científica nos dice que no hay ninguna relación entre estas alteraciones en la zona lumbar y el desarrollo del dolor crónico. Pero esto Manoli no lo sabía ya que nadie se lo había dicho.

Un dato interesante a conocer es que entre el 50 y el 80% de la población mundial ha sufrido alguna vez en su vida un episodio de dolor lumbar, pero no todos ellos desarrollan dolor crónico aunque tengan diagnósticos parecidos, como el caso de Jordi y Manoli. Además, seguro que la mayoría de nosotros pensaríamos que a medida que nos hacemos más viejos y aumentan estas alteraciones en nuestra zona lumbar, deberíamos sentir más dolor y tener más limitaciones funcionales. Pues resulta que los estudios científicos nos dicen que solamente un 7% de las personas mayores con dolor lumbar tienen limitaciones funcionales. ¡Sólo un 7%! ¿Verdad que hubiéramos pensado que eran más? Pues ya ven que no.

Otro de los datos interesantes que nos aporta la ciencia es que EL DOLOR ESTÁ EN EL CEREBRO. Pero no, no se preocupe, esto no quiere decir que se está inventando su dolor o que su dolor lumbar no sea real. Todo lo contrario, EL DOLOR ES SIEMPRE REAL pero es el cerebro humano el que toma la decisión de si sentir dolor o no. Y esta decisión nuestro cerebro la toma teniendo en cuenta factores como nuestra experiencia previa, los aspectos sociales y ambientales que nos rodean y, sobre todo, cómo interpreta nuestro cerebro lo que está sucediendo. Como han visto en el ejemplo anterior, el cerebro de Manoli interpretaba que lo que tenía en la zona lumbar era algo "peligroso" y que por lo tanto debería ser protegido por lo que su cerebro generaba dolor y así Manoli no hacía actividades que involucraran su zona lumbar. En cambio, el cerebro de Jordi percibía lo que sucedía en su zona lumbar como algo normal y como consecuencia no generaba dolor y eso le permitía a Jordi hacer una vida normal.

¡EL DOLOR SIEMPRE ESTÁ EN EL CEREBRO Y DEPENDE DE CÓMO NUESTRO CEREBRO INTERPRETE LO QUE NOS SUCEDE!

Entiendo que todo lo que ha visto y le hemos dicho en este video, puede llegar a romper de alguna manera con lo que tenía entendido acerca de su dolor. No pretendemos que ahora se vaya a nadar o a correr un maratón, pero sí que nos gustaría que se replanteara sus creencias y actitudes hacia su dolor lumbar. Recuerde que los estudios científicos nos han demostrado que tener una hernia discal, un proceso degenerativo en las vértebras o cualquier alteración a nivel muscular o del nervio, es algo normal en el ser humano, que forma parte de nuestro proceso de envejecimiento y lo que es más importante, no tiene por qué producir dolor crónico ya que hay mucha gente con el mismo diagnóstico y siguen teniendo vidas activas y normales. Recuerde: ¡EL DOLOR SIEMPRE ESTÁ EN EL CEREBRO Y DEPENDE DE CÓMO NUESTRO CEREBRO INTERPRETE LO QUE NOS SUCEDE!

Appendix 5: Written material video 4

Para entender estos procesos es importante recordar que el dolor siempre está en el cerebro y que, por lo tanto, este dolor es siempre real.

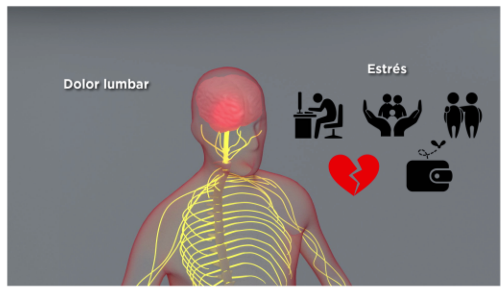
Seguramente, más de una vez habrá relacionado al estrés con su dolor y habrá visto cómo durante situaciones estresantes ha podido tener más dolor. O, por ejemplo, alguna vez habrá anticipado su dolor lumbar antes de realizar un movimiento o actividad; incluso habrá identificado algún movimiento, postura o actividad que apenas realiza por miedo a desencadenar su dolor lumbar. Pero, ¿existe una relación o una conexión entre su dolor lumbar y el estrés y/o el miedo?

Hay dos tipos de estrés que pueden influenciar sobre su dolor. Por un lado, el dolor lumbar en sí mismo puede ser un factor que nos provoque estrés. Por otro lado, tenemos los factores ambientales o psicosociales relacionados con nuestro trabajo, o la falta de éste, la familia, los amigos, las emociones, el dinero. Todos estos factores también nos pueden generar estrés. De manera que el estrés, independientemente de la razón que lo provoque, puede hacer que se inicie nuestro dolor lumbar o que nos duela más de lo normal.

Es importante tener en cuenta que muchas veces no somos conscientes de que estamos sufriendo estrés pero aun así, el estrés sigue actuando dentro de nuestro organismo.

Es importante tener en cuenta que muchas veces no somos conscientes de que estamos sufriendo estrés pero aun así, el estrés sigue actuando dentro de nuestro organismo. Ahora, veamos qué sucede dentro de nuestro cuerpo cuando ese dolor lumbar o esas situaciones ambientales nos producen un estrés que se mantiene en el tiempo.

Cuando sufrimos estrés se activan unas áreas concretas en nuestro cuerpo, entre ellas la amígdala y el eje Hipotalámico-Pituitario-Adrenal. La amígdala, al activarse, envía una señal al hipotálamo que segrega una hormona llamada CRH. Esta hormona activa la glándula pituitaria que a su vez libera otra hormona llamada ACTH. Finalmente, la hormona ACTH activa las glándulas suprarrenales y estas liberan cortisol. El cortisol tiene funciones importantísimas en nuestro organismo, nos ayuda a despertar por la mañana, nos aporta energía si estamos en una situación comprometida y tenemos que salir de ella y, además, es un potente antiinflamatorio que mantiene a raya las sustancias proinflamatorias que se liberan también en situaciones de estrés. El problema viene cuando sufrimos de estrés crónico ya que entonces nuestro organismo agota el cortisol y se produce una situación de desequilibrio en el que las sustancias proinflamatorias superan a las antiinflamatorias, pudiendo producir dolor o aumentando el dolor que ya teníamos sin necesidad de que se produzca una lesión en nuestra zona lumbar.



La evidencia científica nos dice que los procesos inflamatorios producidos por el estrés están implicados en diferentes dolencias como la osteoporosis, la fibromialgia, el dolor lumbar y la ciática. De hecho, cuando se liberan sustancias proinflamatorias en las raíces nerviosas de la zona lumbar se puede producir el típico dolor ciático sin necesidad de que el nervio esté lesionado.
Veamos un ejemplo práctico de lo que acabamos de explicar.

Manuel tiene 50 años y desde hace 5 tiene dolor lumbar recurrente. Cada vez que tiene un episodio de dolor lumbar su cerebro, concretamente el córtex prefrontal, le da un significado al dolor. Manuel piensa que de seguir así puede acabar en silla de ruedas y que deberá pasar por quirófano. Además, todo esto le hace pensar que va a tener que pedir la baja en el trabajo y dejar de hacer el deporte o las actividades que le gustan. Evidentemente, estos pensamientos le producen miedo o preocupación acerca de las consecuencias futuras de su dolor lumbar. Además, el miedo provoca estrés y como hemos visto anteriormente, el estrés hace que se active la amígdala dando como consecuencia la activación del eje Hipotalámico-Pituitario-Adrenal y la liberación de cortisol. Si esta situación se mantiene en el tiempo, nuestro cuerpo se queda sin cortisol y como consecuencia tendremos más dolor ya que habrá más sustancias inflamatorias en el organismo. Además, este miedo/preocupación por nuestro dolor y sus consecuencias nos produce ansiedad y esto se traduce en un aumento de la percepción del dolor y una disminución de los movimientos y actividades que involucran a nuestra zona lumbar.

La evidencia científica nos dice que los procesos inflamatorios producidos por el estrés están implicados en diferentes dolencias como la osteoporosis, la fibromialgia, el dolor lumbar y la ciática.

Es importante comprender que a veces la causa de nuestro dolor lumbar no está solo en la propia espalda sino que tiene que ver con situaciones como el estrés, el miedo, la ansiedad o la preocupación.

Estos factores producen cambios a nivel neurofisiológico que se traducen en la aparición de dolor o en el aumento de la percepción de dolor.

Nos gustaría que pensara en las cosas que ha dejado de hacer por culpa de su dolor lumbar y analizara hasta que punto el estrés, ya sea producido por su dolor lumbar o producido por diferentes factores relacionados con su vida, puede tener que ver con su situación actual.

Disminuir el estrés, las preocupaciones y eliminar los pensamientos catastróficos e incorrectos acerca de nuestro dolor lumbar, nos ayudará a mejorar nuestra situación con el resultado de ser más activos y tener una mejor calidad de vida.

Appendix 6: Written material video 5

En este texto queremos explicarle cómo la actividad física influye positivamente y ayuda en el tratamiento del dolor crónico. También hablaremos de los aspectos negativos derivados de mantener una vida sedentaria.

Más o menos todos sabemos que llevar una vida sedentaria no es bueno pero, ¿hasta qué punto no es bueno? Tanto la Organización Mundial de la Salud como los Centros para el Control y la Prevención de Enfermedades nos avisan de que llevar una vida con una actividad física insuficiente es un importante factor de riesgo a nivel mundial tanto para desarrollar enfermedades no transmisibles, como por ejemplo el cáncer y la diabetes; como para provocar la muerte. Además, tener una comportamiento sedentario, aparte de no reducir los síntomas de nuestro dolor crónico, aumenta la sensación de dolor, aumenta las posibilidades de generar discapacidad, nos hace perder fuerza, reduce nuestro rango de movimiento y nos limita físicamente ya que nuestras capacidades han disminuido. Por otro lado, realizar actividad física produce una serie de efectos beneficiosos, no solo sobre nuestra salud en general, sino también sobre nuestro dolor crónico:

Realizar actividad física produce una serie de efectos beneficiosos, no solo sobre nuestra salud en general, sino también sobre nuestro dolor. crónico

- Disminuye la posibilidad de generar discapacidad
- Disminuye la severidad y la intensidad del dolor
- Disminuye la duración de la baja laboral
- Disminuye el riesgo de enfermar
- Mejora los aspectos cognitivos
- Mejora nuestro humor
- Mejora el sueño
- Influye positivamente en nuestra calidad de vida
- Disminuye el riesgo de desarrollar hipertensión
- Disminuye el riesgo de desarrollar enfermedades cardiovasculares
- Disminuye el riesgo de desarrollar diabetes tipo 2
- Disminuye el riesgo de desarrollar osteoporosis
- Disminuye el riesgo de desarrollar obesidad.
- Mejora nuestra independencia

Como habrá apreciado, la lista de beneficios es larga e importante como para incluir la actividad física en nuestra vida diaria. De hecho, la evidencia científica es cada vez mayor a favor de la utilización de la actividad física cómo tratamiento de las enfermedades crónicas gracias a los beneficios anteriormente mencionados.

Aunque queda claro que la actividad física es beneficiosa en el tratamiento del dolor crónico, son muy pocos los profesionales sanitarios que la prescriben y, por lo tanto, son muy pocos los pacientes con dolor crónico lumbar que la incluyen en sus rutinas diarias. Algunos de los motivos que llevan a no utilizar la actividad física como tratamiento del dolor crónico derivan de la falta de información o están basados en creencias erróneas, como por ejemplo creer que el reposo es mejor que permanecer activos. Incluso algunos pacientes argumentan que no tienen tiempo para realizar actividad física o que les produce miedo, no vaya a ser que su dolor empeore. Además, no saben qué actividad física realizar o quién es el profesional sanitario que debería pautar y supervisar su actividad física. De manera que, antes de seguir adelante, es necesario clarificar todos estos aspectos.

La evidencia científica es cada vez mayor a favor de la utilización de la actividad física cómo tratamiento de las enfermedades crónicas

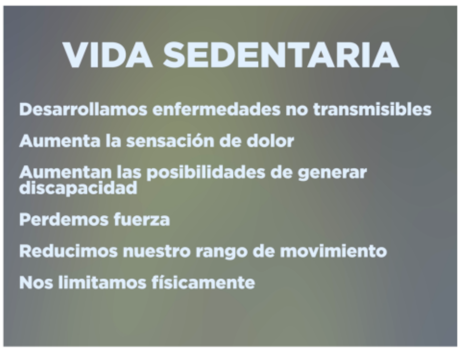
Primero, es importante destacar que la actividad física puede ser tanto de baja como de alta intensidad. Por lo tanto, no solamente ir a correr o nadar se considera actividad física sino que ir a caminar también lo es. Muchas de las personas que sufren de dolor crónico llevan una vida sedentaria por culpa de su dolor. Seguramente son personas que anteriormente practicaban algún tipo de actividad física pero por culpa de su dolor la han dejado de practicar. Es muy normal cuando se sufre dolor crónico tener miedo a realizar actividad física pero, ¿ese miedo tiene fundamento real o simplemente es por falta de información?

Por un lado, la evidencia científica nos dice que realizar actividad física es generalmente seguro y que los posibles y escasos efectos secundarios son temporales y se pueden evitar con una buena información al paciente y realizando actividad física de manera progresiva. Es más, la evidencia científica nos dice que la actividad física es efectiva en la prevención del dolor lumbar y que, especialmente en aquellos pacientes que llevan una vida sedentaria y quieren empezar a ser más activos, una actividad física de baja intensidad también produce beneficios. Así que ya ve que realizar actividad física aunque sufra de dolor lumbar crónico es beneficioso y seguro.

Realizar actividad física aunque sufra de dolor lumbar crónico es beneficioso y seguro.

Ahora vamos a ver cómo la actividad física ayuda a nuestro organismo a combatir el dolor crónico y mejorar su salud.

Cuando realizamos actividad física aunque sea de baja intensidad, como caminar a un ritmo rápido, se producen una serie de cambios dentro de nuestro organismo que afectan a nuestra salud en general y a nuestro dolor lumbar en particular. Cuando realizamos actividad física, aunque sea de baja intensidad:



•Aumentan el número de pulsaciones
•Aumenta la presión sanguínea
•Se producen contracciones musculares

Estos mecanismos a su vez producen tres efectos muy importantes para nuestro organismo:

•Efecto analgésico
•Disminución de la actividad en las vías que activan el dolor
•Efecto antiinflamatorio

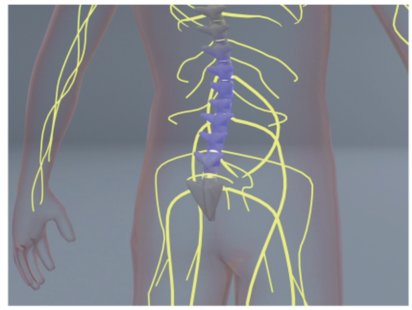
Ahora veremos cómo cada uno de estos efectos ayuda a nuestro organismo en el tratamiento de nuestro dolor lumbar crónico.

El efecto analgésico se produce cuando nuestro organismo, al realizar actividad física, libera sustancias como las B-endorfinas y los factores de crecimiento. Las B-endorfinas son liberadas por la pituitaria y el hipotálamo. De manera que cuando las libera la pituitaria producen un efecto periférico en nuestro organismo y cuando las libera el hipotálamo producen un efecto a nivel central. De este modo, cuando el organismo libera las B-endorfinas, éstas producen una disminución del dolor, lo cual no está nada mal, ¿verdad? En cambio, los factores de crecimiento ayudan al organismo a reparar los tejidos que puedan estar dañados lo que a su vez, también produce un efecto analgésico. Cuando realizamos actividad física disminuye la actividad en las vías que activan el dolor ya que nuestro organismo libera diferentes sustancias como los endocannabinoides, las catecolaminas y el óxido nítrico. Estos elementos hacen que nuestro sistema nervioso libere menos productos excitadores tanto a nivel periférico como a nivel de la médula espinal, de manera que disminuye la posibilidad de que nuestro cerebro genere dolor.

Finalmente, pero no por ello menos importante, la actividad física produce un efecto antiinflamatorio muy importante. Este efecto antiinflamatorio se produce por diferentes mecanismos. Por un lado, la actividad física hace que nuestro organismo libere una sustancia llamada IL-6 que a su vez, libera en el organismo productos con un efecto antiinflamatorio (IL-10, IL-1RA). Además, el realizar actividad física hace que disminuya la masa de grasa visceral en el organismo lo que es muy importante ya que disminuye la liberación de unas sustancias inflamatorias llamadas adipocinas. Por último, la actividad física produce un efecto de estrés agudo que es beneficioso para nuestro cuerpo ya que se liberan los corticoestereoides que son unas sustancias con un efecto antiinflamatorio muy relevante. Así que realizar actividad física nos ayuda a luchar contra nuestro dolor lumbar crónico y nos ayuda a tener una vida más saludable y con una mejor calidad.

Cuando realizamos actividad física disminuye la actividad en las vías que activan el dolor

Es importante que tenga en cuenta que la intención de este texto no es de que ahora mismo se vaya a correr, ya que seguramente sería contraproducente debido a que su organismo no está aún preparado para ello. Nuestra intención es que tenga la información suficiente para que empiece a realizar actividad física de muy baja intensidad al principio. Que vuelva a recuperar esa vida activa que ha dejado de lado por culpa de su dolor lumbar. Puede empezar simplemente por incorporar el caminar a su rutina diaria y, poco a poco, incrementar la velocidad del paso. Hacer esto de manera frecuente producirá los efectos que hemos comentado anteriormente. Por último, si lo que quiere es aumentar la actividad física que realiza, ya que esto le ayudará aún más en el futuro, le aconsejamos lo siguiente:



- Primero visitar a su médico de familia para que compruebe que su salud general es la adecuada.
- En segundo lugar, debe ponerse en manos de un fisioterapeuta, ya que es el profesional sanitario adecuado a la hora de prescribir y supervisar la actividad física en las personas con problemas de salud. Es lo que nosotros llamamos Ejercicio Terapéutico.

Para finalizar, y volviendo a la evidencia científica, los efectos del ejercicio terapéutico son más beneficiosos cuando se adapta a la persona, se progresa lentamente y se realiza bajo supervisión del profesional sanitario adecuado.
Así que ¡ya lo sabe! ¡Caminar también es sano!

Appendix 7: Visual Analogue Scale

Escala EVA del dolor.

Por favor, comenzando por la izquierda de la siguiente barra, desplace la barra hacia la derecha indicando la intensidad de su DOLOR LUMBAR en las últimas 24 horas:

Sin dolor I________________________________I Máximo dolor imaginable

Appendix 8: Fear-Avoidance Beliefs Questionnaire

CUESTIONARIO FAB

Aquí están algunas cosas que otros pacientes nos han dicho sobre su dolor. Por favor, para cada afirmación haga un círculo en un número del 0 al 6 para indicar hasta qué punto las actividades físicas tales como inclinarse, levantar peso, caminar o conducir afectan o afectarían a *su* dolor de espalda.

	En total desacuerdo		Ni de acuerdo ni en desacuerdo			Completamente de acuerdo	
1. Mi dolor fue causado por la actividad física	0	1	2	3	4	5	6
2. La actividad física hace que mi dolor empeore	0	1	2	3	4	5	6
3. La actividad física podría dañar mi espalda	0	1	2	3	4	5	6
4. No debería hacer las actividades físicas que empeoran mi dolor, ni las que podrían empeorarlo	0	1	2	3	4	5	6
5. No puedo realizar las actividades físicas que empeoran mi dolor, ni las que podrían empeorarlo.	0	1	2	3	4	5	6

Las siguientes afirmaciones se refieren a cómo su trabajo normal afecta o afectaría a su dolor de espalda.

	En total desacuerdo		Ni de acuerdo ni en desacuerdo			Completamente de acuerdo	
6. Mi dolor se debe a mi trabajo, o a un accidente en el trabajo	0	1	2	3	4	5	6
7. Mi trabajo agravó mi dolor	0	1	2	3	4	5	6
8. Estoy recibiendo o tramitando algún tipo de compensación por mi dolor de espalda, como una baja laboral, una pensión o una indemnización de cualquier tipo	0	1	2	3	4	5	6
9. Mi trabajo es demasiado pesado para mí	0	1	2	3	4	5	6
10. Mi trabajo empeora mi dolor, o podría empeorarlo	0	1	2	3	4	5	6
11. Mi trabajo puede dañar mi espalda	0	1	2	3	4	5	6
12. Con mi dolor actual, no debería hacer mi trabajo normal	0	1	2	3	4	5	6
13. Con mi dolor actual, no puedo hacer mi trabajo normal	0	1	2	3	4	5	6
14. No podré hacer mi trabajo normal hasta que mi dolor haya sido tratado	0	1	2	3	4	5	6
15. No creo que pueda regresar a mi trabajo habitual en los próximos 3 meses	0	1	2	3	4	5	6
16. No creo que sea capaz de volver nunca a mi trabajo habitual.	0	1	2	3	4	5	6

Appendix 9: Tampa Scale for Kinesiophobia

CUESTIONARIO TSK-11SV

Tampa Scale for Kinesiophobia (Spanish adaptation. Gómez-Pérez, López-Martínez y Ruiz-Párraga, 2011)

INSTRUCCIONES: a continuación se enumeran una serie de afirmaciones. Lo que Ud. ha de hacer es indicar hasta qué punto eso ocurre en su caso según la siguiente escala:

1	2	3	4
Totalmente en desacuerdo			**Totalmente de acuerdo**

1. Tengo miedo de lesionarme si hago ejercicio físico.	1	2	3	4
2. Si me dejara vencer por el dolor, el dolor aumentaría.	1	2	3	4
3. Mi cuerpo me está diciendo que tengo algo serio.	1	2	3	4
4. Tener dolor siempre quiere decir que en el cuerpo hay una lesión.	1	2	3	4
5. Tengo miedo a lesionarme sin querer.	1	2	3	4
6. Lo más seguro para evitar que aumente el dolor es tener cuidado y no hacer movimientos innecesarios.	1	2	3	4
7. No me dolería tanto si no tuviese algo serio en mi cuerpo.	1	2	3	4
8. El dolor me dice cuándo debo parar la actividad para no lesionarme.	1	2	3	4
9. No es seguro para una persona con mi enfermedad hacer actividades físicas.	1	2	3	4
10. No puedo hacer todo lo que la gente normal hace porque me podría lesionar con facilidad.	1	2	3	4
11. Nadie debería hacer actividades físicas cuando tiene dolor.	1	2	3	4

Appendix 10: Roland–Morris Questionnaire

FUNDACION KOVACS

ESCALA DE ROLAND-MORRIS

Cuando le duele la espalda, puede que le sea difícil hacer algunas de las cosas que habitualmente hace. Esta lista contiene algunas de las frases que la gente usa para explicar cómo se encuentra cuando le duele la espalda (o los riñones). Cuando las lea, puede que encuentre algunas que describan su estado de *hoy*. Cuando lea la lista, piense en cómo se encuentra usted *hoy*. Cuando lea usted una frase que describa como se siente hoy, póngale una señal. Si la frase no describe su estado de hoy, pase a la siguiente frase. Recuerde, tan solo señale la frase si está seguro de que describe cómo se encuentra usted hoy.

1.- ☐ Me quedo en casa la mayor parte del tiempo por mi dolor de espalda.

2.- ☐ Cambio de postura con frecuencia para intentar aliviar la espalda.

3.- ☐ Debido a mi espalda, camino más lentamente de lo normal.

4.- ☐ Debido a mi espalda, no puedo hacer ninguna de las faenas que habitualmente hago en casa.

5.- ☐ Por mi espalda, uso el pasamanos para subir escaleras.

6.- ☐ A causa de mi espalda, debo acostarme más a menudo para descansar.

7.- ☐ Debido a mi espalda, necesito agarrarme a algo para levantarme de los sillones o sofás.

8.- ☐ Por culpa de mi espalda, pido a los demás que me hagan las cosas.

9.- ☐ Me visto más lentamente de lo normal a causa de mi espalda.

10.- ☐ A causa de mi espalda, sólo me quedo de pie durante cortos períodos de tiempo.

11.- ☐ A causa de mi espalda, procuro evitar inclinarme o arrodillarme.

12.- ☐ Me cuesta levantarme de una silla por culpa de mi espalda.

13.- ☐ Me duele la espalda casi siempre.

14.- ☐ Me cuesta darme la vuelta en la cama por culpa de mi espalda.

15.- ☐ Debido a mi dolor de espalda, no tengo mucho apetito.

16.- ☐ Me cuesta ponerme los calcetines - o medias - por mi dolor de espalda.

17.- ☐ Debido a mi dolor de espalda, tan solo ando distancias cortas.

18.- ☐ Duermo peor debido a mi espalda.

19.- ☐ Por mi dolor de espalda, deben ayudarme a vestirme.

20.- ☐ Estoy casi todo el día sentado a causa de mi espalda.

21.- ☐ Evito hacer trabajos pesados en casa, por culpa de mi espalda.

22.- ☐ Por mi dolor de espalda, estoy más irritable y de peor humor de lo normal.

23.- ☐ A causa de mi espalda, subo las escaleras más lentamente de lo normal.

24.- ☐ Me quedo casi constantemente en la cama por mi espalda.

Appendix 11: Ethical Committee approval

IDIAP Jordi Gol

STATEMENT FROM THE CLINICAL INVESTIGATION ETHICS

Rosa Morros Pedrós, President of the Clinical Ethics Committee of the IDIAP Jordi Gol and Gurina.

To Certify:

That this Committee in its convocation on Decembre 17th 2014 evaluated the research project (P14/138) titled: ***Influence of a biopsychosocial educational internet-based intervention, on pain, dysfunction, and pain cognitions, in chronic low back pain patients in primary care: A mixed methods approach (Chronic low back pain)***, presented by Jorge Soler Gonzalez.

In consideration of the ethical principles and methodology of the investigation, definitive approval has been granted for the abovementioned project.

Rosa Morros

Signed in Barcelona December 2014.

Appendix 12: Publication of the study protocol

Valenzuela-Pascual *et al. BMC Medical Informatics and Decision Making* (2015) 15:97
DOI 10.1186/s12911-015-0220-0

BMC Medical Informatics and Decision Making

STUDY PROTOCOL Open Access

The influence of a biopsychosocial educational internet-based intervention on pain, dysfunction, quality of life, and pain cognition in chronic low back pain patients in primary care: a mixed methods approach

Fran Valenzuela-Pascual[1,2,3*], Fidel Molina[2,4], Francisco Corbi[5], Joan Blanco-Blanco[1,2,3], Rosa M. Gil[6] and Jorge Soler-Gonzalez[2,7,8]

Abstract

Background: Low back pain is the highest reported musculoskeletal problem worldwide. Up to 90 % of patients with low back pain have no clear explanation for the source and origin of their pain. These individuals commonly receive a diagnosis of non-specific low back pain.

Patient education is a way to provide information and advice aimed at changing patients' cognition and knowledge about their chronic state through the reduction of fear of anticipatory outcomes and the resumption of normal activities. Information technology and the expedited communication processes associated with this technology can be used to deliver health care information to patients. Hence, this technology and its ability to deliver life-changing information has grown as a powerful and alternative health promotion tool.

Several studies have demonstrated that websites can change and improve chronic patients' knowledge and have a positive impact on patients' attitudes and behaviors. The aim of this project is to identify chronic low back pain patients' beliefs about the origin and meaning of pain to develop a web-based educational tool using different educational formats and gamification techniques.

Methods/design: This study has a mixed-method sequential exploratory design. The participants are chronic low back pain patients between 18–65 years of age who are attending a primary care setting. For the qualitative phase, subjects will be contacted by their family physician and invited to participate in a personal semi-structured interview. The quantitative phase will be a randomized controlled trial. Subjects will be randomly allocated using a simple random sample technique. The intervention group will be provided access to the web site where they will find information related to their chronic low back pain. This information will be provided in different formats. All of this material will be based on the information obtained in the qualitative phase. The control group will follow conventional treatment provided by their family physician.

(Continued on next page)

* Correspondence: fvp1969@infermeria.udl.cat
[1]Universidad de Lleida, Facultad de Enfermería y Fisioterapia, Montserrat Roig 2, Lleida 25198, Spain
[2]Grupo de Estudios Sociedad, Salud, Educación y Cultura, Universidad de Lleida, Pl. de Víctor Siurana 1, Lleida 25003, Spain
Full list of author information is available at the end of the article

Appendix 13: Presentation preliminary findings 16th World Congress on Pain

YOKOHAMA 2016
16TH WORLD CONGRESS ON PAIN®

CERTIFICATE OF PRESENTATION

The following poster was submitted and presented at the
16th World Congress on Pain, held at the Pacifico Convention Center in Yokohama, Japan,
September 26-30, 2016.

PTH172

PATIENTS' BELIEFS ABOUT THE MEANING OF THEIR CHRONIC LOW-BACK PAIN AND ITS RELATIONSHIP WITH EXPLANATIONS PROVIDED BY HEALTH PROFESSIONALS IN PRIMARY CARE: A QUALITATIVE STUDY IN SPAIN

F. Valenzuela Pascual[1], E. Briones Vozmediano[1], F. Molina Luque[1], J. Blanco Blanco[1], F. Rubí Carnacea[1], C. Climent Sanz[1], J. Soler González[1]
[1] University of Lleida, Lleida, Spain

The 16th World Congress on Pain is organized by the International Association for the Study of Pain.

Presented: Thursday, September 29, 2016

Claudia Sommer, M.D.
Chair, Scientific Program Committee

Sponsored by:

IASP®

Appendix 14: Letter of support from the Dean of the Faculty

Universitat de Lleida
Facultat d'Infermeria

D. Joan Blanco Blanco como Decano de la Facultad de Enfermería de la Universidad de Lleida,

HACE CONSTAR:

Su total apoyo al profesor de la Universitat de Lleida, D. Francesc Valenzuela Pascual, en el desarrollo del proyecto que lleva por título: "Influence of a biopsychosocial educational internet-based intervention, on pain, dysfunction, and pain cognitions, in chronic low back pain patients in primary care: A mixed methods approach".

Y para que conste y surta los efectos oportunos firma este documento en Lleida, a 10 de octubre de 2014.

D. Joan Blanco Blanco

Faculty of Nursing | C/ Montserrat Roig, 2 | 25198 Lleida | Phone: 973702443 | deganat@infermeria.udl.cat

Appendix 15: Research stay at the University of Toronto

UNIVERSITY OF TORONTO
SCHOOL OF GRADUATE STUDIES

International Visiting Research Student

Francesc Valenzuela Pascual
Facultat d'Infermeria Universitat de Lleida Avda.
Alcade Rovira Roure, 44
Spain 25198

February 12, 2013

Dear Mr. Valenzuela Pascual,

On behalf of the School of Graduate Studies, I am pleased to offer you full-time admission to the graduate unit described below as an international visiting graduate student to participate in short-term research activities.

Sessions: Winter 2013 and Summer 2013
Student Number: 1000959812
Date of Birth (Immigration purposes): August 15, 1969
Approved Research Period Begins: March 11, 2013
Approved Research Period Ends: June 11, 2013
Status: Full-time
Faculty: Nursing
U of T Supervisor: Dr. Michael McGillion

It is intended that the above information will facilitate the issuing of a Study Permit for Francesc Valenzuela Pascual.

To make the most of your research experience at the University of Toronto, please review the Information for New Students webpage: ***www.sgs.utoronto.ca/current/new_students.asp.*** We look forward to having you join us.

Yours Sincerely,

Heather Kelly
Director, Student Services
School of Graduate Studies
E. & O.E.

Cc: Miranda Cheng, Centre for International Experience

63/65 St. George Street Toronto Canada M5S 2Z9