LITERATURE REVIEWS

Summary and Perspective of Recent Literature
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Werneke M, Edmond S, Deutscher D, Ward J, Grigsby D, Young M, McGill T, McClenahan B, Weinberg J, Davidow A. (2016). Effect of Adding McKenzie Syndrome, Centralization, Directional Preference, and Psychosocial Classification Variables to a Risk-Adjusted Model Predicting Functional Status Outcomes for Patients with Lumbar Impairments*. JOSPT*; 46:726-741.

STUDY'S PUBLISHED CONCLUSION

The small added prognostic capabilities identified when combining McKenzie or pain-pattern classifications with the SCL-BPPM classification did not significantly improve prediction of functional status outcomes in this study.

At first glance, it would appear that MDT classification along with psychosocial classification has no importance when attempting to determine prognosis in patients. However, it is important to first understand the study's design.

WHAT IS THIS STUDY TRYING TO DO?

This study is attempting to determine what independent factors best explain or predict a patient's functional outcome at discharge from physical therapy services. Multiple models were developed in a series examining the predictive power of patient characteristics, therapist characteristics and the effect of adding MDT, Pain Pattern, and Psychosocial classification categories, as well as, a combination of the aforementioned classification paradigms. All eight models (Table 5) were compared in a head-to-head manner. These statistical comparisons allowed the determination of which model had the greatest 'predictive power' (i.e. R^2_{value}) for predicting a patient's functional outcome following treatment.

The 'predictive power' of a model is represented as an R^2_{value}. The greater the R^2_{value}, the stronger the predictive ability of a model for the given dependent variable. The dependent variable we are concerned about is the functional status of the patient at discharge. The functional status of the patient is assessed by Focus On Therapeutic Outcome's (FOTO) lumbar measure. This measure is psychometrically reliable, valid and responsive and has been described in detail elsewhere [1-5]. FOTO uses a 0-100 functional scale to express a patient's overall level of function (0 = 'essentially bed ridden' vs 100 = 'participating in collegiate sports').

In TABLE 5, you will see two R^2_{values} per model; One that is calculated initially with our available data for the study, and a second that is generated by PRESS (Prediction Error Sum of Squares). PRESS is used to avoid 'overfitting'. Overfitting is a problem that can occur in complex statistics when you have many variables to assess. As stated earlier, the purpose of the proposed models is to PREDICT functional changes for future patients. The model, however, is using a data set that has already been collected and, in the worst case scenario, the model generated would essentially 'memorize' the data points used and thereby have 100% prediction for the available data but have no utility with future data. PRESS is used to cross validate the initial findings of each model. To do this, PRESS uses the model's prediction equations on a completely separate collection of patient data and shows how similar the two findings are. To demonstrate validity, you want the model's predictions and PRESS' predictions to be fairly close, if not ideally identical. The findings demonstrate that the margin for error is small.

Only significant independent variables are included in the model to calculate the overall R^2 value. An independent variable's explained variance is represented as a beta coefficient. Beta coefficients are a way of representing to what extent a variable, such as age, has an ability to influence for better or worse a dependent variable relative to all variables measured. The beta coefficients reported in Table 5 indicate the amount of explained variance that each significant independent variable contributes to the predictive power of the model compared to a reference standard.

EXAMPLES:

Model 2 (FOTO and MDT Classifications) demonstrated an additional 2.8% in predictive ability compared to Model 1 (FOTO). Reducible Derangement is the reference standard for MDT classifications. Compared to a Reducible Derangement, Chronic Pain State in this model predicts that the patient will achieve 14.3 fewer points over the course of care. Mechanically Inconclusive is predicted to achieve 5.1 fewer points of functional gains by discharge compared to an individual classified as Reducible Derangement.

Model 7 included the addition of MDT classification and SCL-BPPM to FOTO's original model and resulted in an additional 3.6% predictive power. Again, Reducible Derangement is the reference standard for MDT classifications. Compared to a Reducible Derangement, Chronic Pain State in this model now predicts 13.4 fewer points of function at discharge. This highlights that the strength of each beta coefficient is dependent on all the variables calculated in the equation.

MDT clinicians consider classification essential to guiding treatment and setting long-term expectations (prognosis) for our patients. We found that classification categories were significant and generated large beta coefficients within all classification models examined (except for fear avoidance model) yet when comparing models in a head-to-head manner as we did in this study, the conclusion appears to contradict the data reported in Table 5.

IS THE CONCLUSION CORRECT?

We observed that the addition of classification variables added an extra 4% in R^2_{value} after controlling for patient and therapist characteristics (i.e. 44% vs 40%), but R^2_{values} were not statistically different between models. At first glance, if the reader only read the abstract, they are left with the impression that classification was not only statistically insignificant, but clinically unimportant.

Although the differences in R^2_{value} between models were not statistically different, that does not mean that classification was not important!!! The devil is in the details. Understanding the statistical complexity of the study design, knowledge of previous prediction models developed and published in the physical therapy literature, and careful interpretation of the data presented in Table 5 offers a different perspective.

<u>IMPORTANT DATA FINDINGS from TABLE 5 (below)</u>

- Model 7 (addition of MDT and SCL-BPPM) improved the original Model by 3.6%.
    - MDT Classification beta coefficients were generally larger values (i.e. -5.0, -13.4) than SCL-BPPM beta coefficient values of -3.3 and -3.2. Therefore, MDT is a greater prognostic variable then SCL-BPPM.
- Model 8 (addition of Pain Pattern and SCL-BPPM) improved the original Model by 3.9%.
    - Pain Pattern Classification beta coefficients were generally larger values (i.e. -8.1, -3.2) than SCL- BPPM beta coefficient values of -3.3 and -4.0. Therefore, Pain Pattern Classification is a greater prognostic variable then SCL-BPPM.
- Chronic Pain Syndrome (MDT Classification) had the greatest beta coefficient of all at -13.4 (Model 8).
- FABQ had NO statistical benefit in predicting outcomes.

Due to the original study's design of comparing models in a head-to-head fashion, it is correct that statistically a 3 - 4 % prediction (achieved by MDT / Pain Pattern / Psychosocial) is insignificant compared to a 40% prediction (achieved by FOTO's original baseline model).

We recommend for future studies to examine what variables added sequentially in a single model have the best predictive capabilities. If we look at this data from the perspective of what variables explain the largest amount of variance, things appear different. The DISCUSSION section of the article highlights these important facts and expands upon the clinical importance of interpreting classification beta coefficients.

TABLE 5

MODEL	1	2	3	4	5	6	7	8
Dependent variable: FS at discharge (n = 723)								
R^2[1]	0.398 (0.345, 0,451)	0.426 (0.374, 0.478)	0.428 (0.376, 0.480)	0.408 (0.356, 0.461)	0.398 (0.345, 0.452)	0.399 (0.346, 0.452)	0.434 (0.382, 0.485)	0.437 (0.385, 0.489)
Increase in R^2 compared to basic model	...	2.8%	3.0%	1.0%	0.0%	0.1%	3.6%	3.9%
Sample validation predicted R^2[2]	0.349	0.368	0.375	0.355	0.347	0.348	0.372	0.380
Variables[3]								
Intercept	68.5 (<.001)	71.5 (<.001)	71.9 (<.001)	72.6 (<.001)	67.7 (<.001)	66.8 (<.001)	74.9 (<.001)	75.6 (<.001)
Intake FS	0.5 (<.001)	0.5 (<.001)	0.5 (<.001)	0.5 (<.001)	0.5 (<.001)	0.5 (<.001)	0.4 (<.001)	0.4 (<.001)
Age groups								
18 to <45 y (reference)	...	...	...	...	...	...	...	...
45 to <65 y	−1.9 (.096)	−1.6 (.153)	−1.7 (.144)	−2.1 (.071)	−1.9 (.106)	−1.8 (.115)	−1.8 (.122)	−1.8 (.115)
65 to <75 y	−2.1 (.339)	−2.4 (.266)	−2.4 (.262)	−2.6 (.230)	−2.1 (.341)	−2.1 (.341)	−2.7 (.207)	−2.8 (.188)
≥75 y	−0.9 (.740)	−0.8 (.777)	−1.9 (.494)	−1.5 (.572)	−0.9 (.742)	−0.9 (.731)	−1.1 (.677)	−2.3 (.398)
Acuity								
0-21 d (reference)	...	...	...	...	...	...	...	...
22-90 d	−5.8 (<.001)	−5.1 (.001)	−5.0 (.001)	−5.9 (<.001)	−5.8 (<.001)	−5.8 (<.001)	−5.2 (.001)	−5.1 (.001)
>90 d	−9.6 (<.001)	−8.6 (<.001)	−8.8 (<.001)	−9.5 (<.001)	−9.6 (<.001)	−9.6 (<.001)	−8.6 (<.001)	−8.8 (<.001)
Surgical history								
No related surgery (reference)	...	...	...	...	...	...	...	...
≥1 related surgeries	−6.2 (<.001)	−6.3 (<.001)	−5.3 (<.001)	−6.2 (<.001)	−6.2 (<.001)	−6.1 (<.001)	−6.3 (<.001)	−5.4 (<.001)
Payer								
HMO/PPO (reference)	...	...	...	...	...	...	...	...
Medicare	−1.0 (.599)	0.1 (.973)	−0.0 (.998)	−0.7 (.732)	−1.0 (.599)	−1.0 (.602)	−0.5 (.881)	−0.1 (.976)
Medicaid	−6.3 (.137)	−4.7 (.262)	−5.5 (.183)	−6.3 (.132)	−6.2 (.139)	−6.3 (.136)	−5.0 (.231)	−5.7 (.168)
Workers' compensation	−2.7 (.224)	−1.1 (.616)	−1.4 (.527)	−1.9 (.387)	−2.8 (.208)	−2.8 (.203)	0.5 (.805)	−0.7 (.756)
Litigation, no fault, auto insurance, indemnity	0.3 (.927)	0.6 (.871)	−0.1 (.987)	−0.4 (.916)	−0.5 (.899)	−0.3 (.934)	0.1 (.960)	0.3 (.887)
Patient, other, no charge	3.7 (.147)	−2.7 (.284)	3.5 (.162)	−3.4 (.183)	−3.7 (.146)	−3.6 (.051)	−2.6 (.305)	−3.2 (.197)
Number of comorbidities								
None (reference)	...	...	...	...	...	...	...	...
1-2	−2.8 (.244)	−2.3 (.329)	−2.7 (.244)	−2.6 (.281)	−2.8 (.246)	−2.9 (.229)	−2.2 (.354)	−2.5 (.277)
3	−4.0 (.061)	−3.9 (.065)	−4.2 (.048)	−3.8 (.073)	−4.0 (.063)	−4.1 (.056)	−3.7 (.078)	−4.0 (.059)
≥4	−8.1 (<.001)	−7.9 (<.001)	−8.1 (<.001)	−7.0 (.001)	−8.1 (<.001)	−8.2 (<.001)	−7.1 (.001)	−7.1 (.001)
Therapist								
Therapist 1 (reference)	...	...	...	...	...	...	...	...
Therapist 2	−3.7 (.324)	−2.2 (.568)	−4.3 (.243)	−3.6 (.328)	−3.6 (.329)	−3.9 (.296)	−2.4 (.521)	−4.3 (.239)
Therapist 3	−4.5 (.120)	−3.8 (.176)	−3.5 (.214)	−4.9 (.084)	−4.5 (.119)	−4.4 (.123)	−4.1 (.153)	−3.9 (.164)
Therapist 4	−9.5 (.001)	−10.8 (<.001)	−10.7 (<.001)	−9.7 (.001)	−9.5 (.001)	−9.6 (.001)	−10.9 (<.001)	−10.8 (<.001)
Therapist 5	−4.3 (.007)	−5.3 (.001)	−6.0 (<.001)	−4.6 (<.001)	−4.3 (.007)	−4.4 (.006)	−5.4 (.001)	−6.1 (<.001)
Therapist 6	−2.0 (.797)	−2.1 (.780)	0.3 (.968)	−2.9 (.698)	−2.0 (.797)	−2.0 (.789)	−3.0 (.688)	−0.7 (.925)

Therapist 7	-3.0 (.045)	-3.9 (.010)	-3.9 (.011)	-3.3 (.030)	-3.0 (.045)	-3.0 (.044)	-4.1 (.007)	-4.1 (.008)
Therapist 8	2.4 (.221)	0.2 (.921)	0.7 (.742)	1.3 (.491)	2.3 (.228)	2.2 (.265)	-0.6 (.773)	-0.3 (.879)
Therapist 9	6.6 (.069)	4.5 (.214)	3.6 (.323)	5.2 (.155)	6.6 (.070)	6.4 (.078)	3.3 (.363)	2.2 (.539)
Therapist 10	5.2 (.026)	2.9 (.211)	4.0 (.093)	4.1 (.079)	5.1 (.029)	5.0 (.031)	2.1 (.373)	3.0 (.206)
Therapist 11	17.4 (<.001)	15.0 (<.001)	14.5 (<.001)	16.1 (<.001)	17.4 (<.001)	17.4 (<.001)	14.1 (<.001)	13.4 (<.001)
Therapist 12	-1.5 (.421)	-2.8 (.137)	-2.4 (.216)	-1.9 (.305)	-1.6 (.402)	-1.6 (.401)	-2.9 (.117)	-2.7 (.156)
McKenzie classification								
Reducible derangement (reference)	...	...	...	...	...	...	...	...
Irreducible derangement		-5.0 (.009)					-5.0 (.008)	
Dysfunction		-0.7 (.834)					-1.2 (.727)	
Chronic pain syndrome		-14.3 (<.001)					-13.4 (<.001)	
Surgery		-0.8 (.756)					-1.1 (.660)	
Mechanically inconclusive		-5.1 (.008)					-5.0 (.010)	
Spinal stenosis		-2.5 (.514)					-3.1 (.415)	
Spondylolisthesis, hip, sacroiliac joint dysfunction, other		-7.0 (.009)					-7.2 (.008)	
Patient response classification								
DP/CEN (reference)	...	...	...	...	...	...	...	...
DP/non-CEN			-3.2 (.694)					-3.2 (.010)
DP/NC			1.6 (.810)					-1.3 (.510)
No DP/non-CEN			-8.1 (<.001)					-8.1 (<.001)
No DP/NC			-1.6 (.793)					-1.9 (.384)
SCL BPPM								
Low risk (reference)	...	...	...	...	...	...	...	...
Medium risk				-3.3 (.005)			-3.3 (.006)	-3.3 (.005)
High risk				-4.6 (.003)			-3.2 (.041)	-4.0 (.009)
FABQ-W								
Low fear of work (reference)	...	...	...	...	...	...	...	...
High fear of work					-0.4 (.726)			
FABQ-PA								
Low fear of physical activity (reference)	...	...	...	...	...	...	...	...
High fear of physical activity						-0.1 (.350)		

(permission granted from JOSPT to use this table)

PLACING THE RESULTS IN PERSPECTIVE

Predictive models for patient functional change are seeking the GOLD STANDARD of 50%. The gold standard would be able to explain 50% of the variation in patient outcomes from start to finish of an episode of care. However, this gold standard does not yet exist.

The highest predictive capabilities to date in published literature is FOTO at 35 - 40%. If you remember, combining MDT / Pain Pattern with psychosocial (SCL-BPPM) resulted in a 3 - 4 % prediction of outcomes. When you add MDT / Pain Pattern / SCL-BPPM to FOTO, you have a predictive capability of nearly 44%. That is a TREMENDOUS FEAT!

Considering that the variables used to account for FOTO's numbers have as little as 1% prediction, a variable that demonstrates 3% is on that scale BIG.

Reality = MDT Classification (~3%) is a BIG / STRONG variable in predicting outcomes.

Reality = Pain Pattern Classification (~3%) is a BIG / STRONG variable in predicting outcomes.

The literature is filled with studies demonstrating the importance of psychosocial variables. FABQ was demonstrated to contribute nothing to the prediction of functional outcomes for patients. The SCL-BPPM was shown to be a significant single variable at 1%. Compared to the single variable of MDT Classification or Pain Pattern Classification, psychosocial variables predictive ability is not nearly as important. Once again, this study supports previous findings that eliciting or failing to elicit Centralization / classifying or failing to classify as Derangement is a stronger predictor of patient outcomes then psychosocial variables.

Secondary findings observed trends in outcomes related to McKenzie level of postgraduate education / training and the treating therapist. Dip.MDT achieved significantly greater functional scale outcomes then those with Cert.MDT. However, the treating therapist was also a greater predictor of functional change then the level of MDT training. Essentially, clinician characteristics that drive them to pursue advanced training may have an increased desire to excel professionally and develop stronger therapeutic alliances with patients.

TAKE AWAY MESSAGE

This and other powerful literature supporting MDT published in peer-reviewed journals is the end result of the hard work and dedication of our MDT research group dedicated to collecting data on a daily basis in the clinic to scientifically expand upon the MDT literature and to report on the merits of what we observe during every day practice.

We, as clinicians, are learning every day a bit more about what is best treatment and why some treatments are more beneficial than others. If you want to be a force in molding where the profession is going, collect data then join your colleagues on FOTO. It will be a humbling experience and one that will challenge you to be the best clinician you can be.

Please feel free to contact me, Brian McClenahan, bmcclen@gmail.com, with any questions or let me know if you are interested in joining our MDT research group. Become active in research driven by clinical practice. Walk the walk. Don't just talk the talk!

LET THE SYSTEM BE YOUR GUIDE.

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Summary and Perspective of Recent Literature
Adrian Wozny, PT, Dip. MDT and Richard Rosedale, PT, Dip. MDT

Cook JL, Rio E, Purdam CR, Docking SI. (2016). Revisiting the continuum model of tendon pathology: what is its merit in clinical practice and research? *British Journal of Sports Medicine; 50:1187-1191*

In 2009, Cook and Purdam presented a model of load-induced tendinopathy with an emphasis on encouraging clinicians not to treat all tendon problems in the same way. The authors stated that both overloading and unloading can produce the same degenerative changes and there are various hypotheses that try to explain the process of tendon pathology. They asked the question of whether the various pathologies that have been described could be seen to be on one continuum. The authors presented a new model of tendon pathology which proposed three stages:

1. Reactive tendinopathy
2. Tendon disrepair
3. Degenerative Tendinopathy

It was suggested, based on the available evidence at the time, that these changes form a continuum of tendon pathologies (Figure 1) and that these changes are reversible as long as the tendon is not in the degenerative tendinopathy stage, even though they still acknowledge the possibility of healing at this stage. The authors note that if degeneration is extensive or loads sufficiently high, rupture can occur.

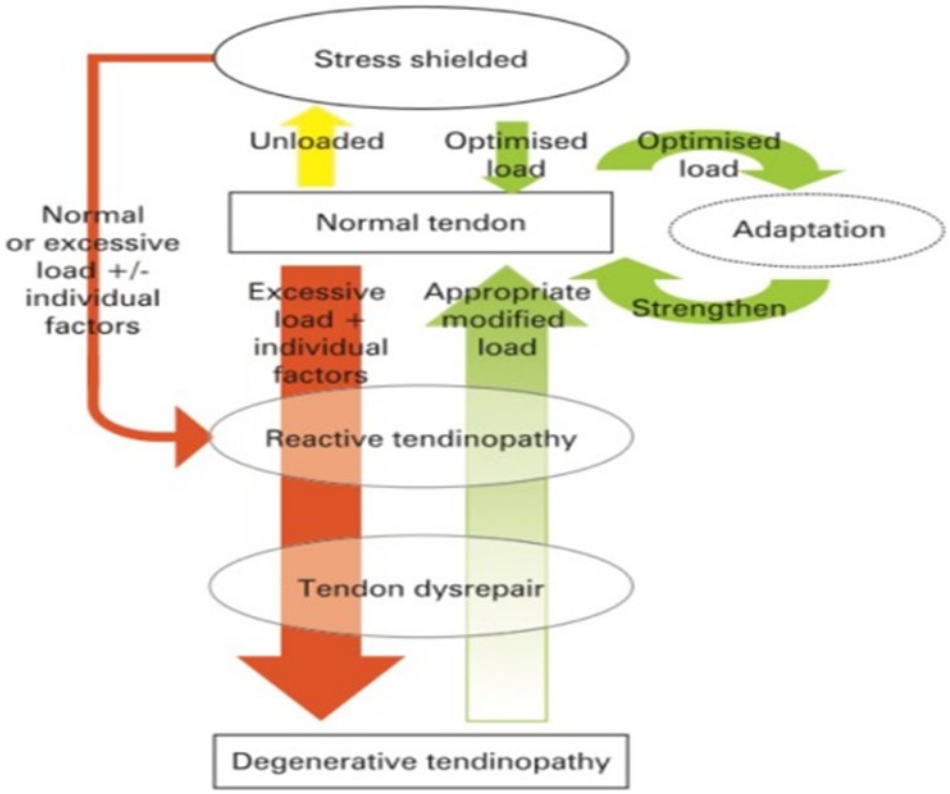


Figure 1 Pathology continuum; this model embraces the transition from normal through to degenerative tendinopathy and highlights the potential for reversibility early in the continuum. Reversibility of pathology is unlikely in the degenerative stage. Figure from Cook JL, Purdam CR. Br J Sports Med 2009; 43: 409-416.

The clinicians are then guided in clinical decision making by dividing the continuum model into two clear groups with implications for management (Figure 1):

1. **Reactive/early tendon disrepair**
   Management entails identifying the ‘abusive load’ and then focusing on load reduction and modification, allowing the tendon to normalize and become less reactive.

2. **Late tendon disrepair/degenerative tendinopathy**
   Progressively loading the tendon positively stimulates cell activity and matrix restructuring and offers pain relief. Eccentric exercise is especially “beneficial for pain, function and return to activity.”

This brings us to the 2016 “revisit” by Jill Cook et al. “Revisiting the Continuum Model of Tendon Pathology: What is its Merit in Clinical Practice and Research?” They reflect on the original model and its relevance to sports medicine and attempt to answer some of the questions that have been raised in the literature since.

First of all, the authors summarise the main categories of tendon pathology models, of which the continuum model is one:

1. **Collagen disruption/tearing hypothesis**
   This model is challenged as a primary event of disruption. Normal tendon cannot tear as a result of day to day loading unless there has already been changes in the collagen matrix.

2. **Inflammation**
   Although changes in the level of inflammatory markers occur in response to cyclic load, there is not the support that inflammation is the primary event or ‘key driver’ of tendon pathology. So, this model is also challenged.

3. **Tendon cell response**
   This model suggests that loading (sensed by the tendon call) is the key factor affecting the collagen fibers and adaptation that occur.

The authors state that “It is unlikely that any one model fully explains all aspects of the pathoaetiology of tendon pathology”. It is a complex process, especially in regards to the relationship between structure, pain and function.

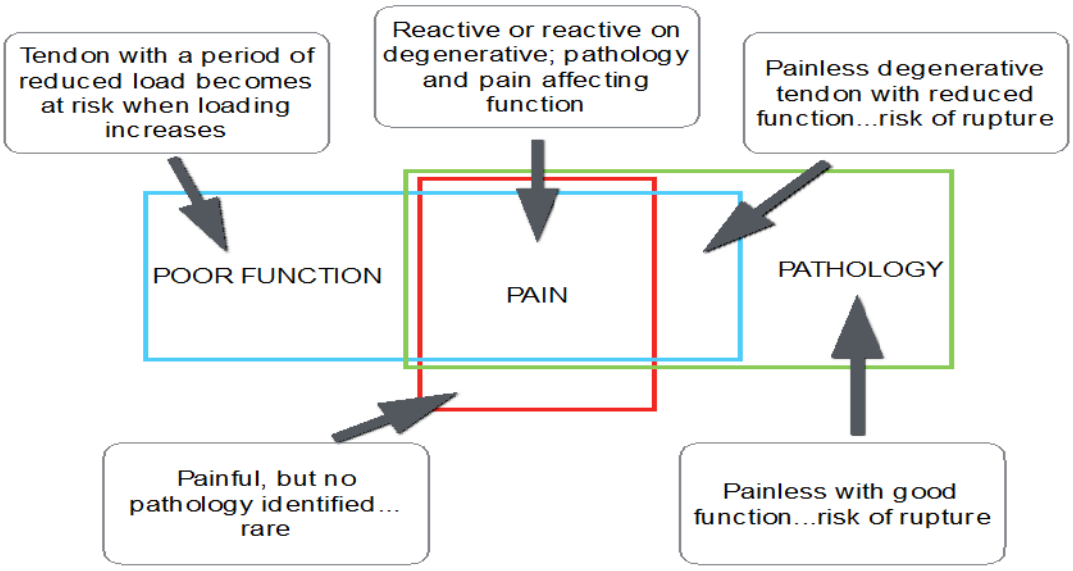
In revisiting the Continuum Model, the authors propose a hybrid of reactive and degenerative pathology, which is ‘reactive-on-degenerative tendinopathy’.

Where pain fits into the continuum is discussed. It falls into two categories in the revised Continuum Model:

1. Reactive tendon with first presentation of tendon pain following acute overload
2. Reactive-on-late disrepair/degenerative tendon pathology

The authors ‘strongly’ suggest that there is a local nociceptive driven pain, hypothesizing that either of the above situations may “increase expression of nociceptive substances and their receptors, stimulating the peripheral nerve and be interpreted as pain”. They acknowledge a potential role for the central nervous system in influencing the pain experience, but suggest that local nociceptive driven pain is critical.

The figure on the following page is adapted from the paper summarises the complex interplay between structure, function and pain.



What is most valuable for clinicians is the analysis by the authors on how to optimise treatment by 'tailoring' it to the stage of tendon pathology. Exercise and 'load management' are at the core of treatment. Authors discuss those interventions in three ways:

1. **Interventions treating pain.**
   Pharmaceutical and modality interventions can reduce pain in the short-term, but without addressing tissue capacity this may result in recurrence. Isometric exercises have a potential to reduce pain and improve strength. Loading programmes are deemed to have broader structural, cortical and functional benefits that may lead to a better outcome.

2. **Interventions addressing function and load capacity.**
   This aspect has had little research to guide the clinician and it is acknowledged that it is difficult to quantify function and for the clinician to get a clear sense of the tendon load capacity. Hopefully, further research will elucidate on these issues.

3. **Interventions targeting structure.**
   This is where the Continuum Model can provide a framework to understand the potential of the tendon to regain normal structure. It is important to understand that in the reactive stage, heavy loading with eccentric exercises may be highly provocative. At this stage, unloading is the key in order to allow the tendon to regain its normal structure. In the degenerative stage, interventions to change structure are not necessarily successful. Treatment should be aimed at building loading capacity and 'optimizing adaptation' in the healthy or in the reactive stage tissue rather than the degenerated portion of the tissue i.e. 'treat the donut, not the hole'. However, for long term tendon health and outcomes, treatment must progress to improve load capacity of the degenerated portion through progressive loading rehab.

In summary, it looks like we are far from having all the answers to the questions surrounding our understanding and management of tendon injury and pathology. It is not likely that any one model will be entirely comprehensive in accounting for all the complex changes that occur, especially in relation to pain and the implications for rehabilitation. For now, the paper's elucidation of these two overlapping phases and the need to manage these phases very differently gives us some guidance to target our interventions. To complete the picture as MDT clinicians, we are always looking towards the patient, their environment and tissue demands, their needs, their expectations and goals in order to tailor our management to achieve the best outcomes we can, putting the patient first.

http://bjsm.bmj.com/content/early/2016/04/28/bjsports-2015-095422.abstract