

The Importance of Mechanisms in
Developing *Better* Psychosocial Treatments
for Chronic Pain

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Psychosocial Therapy Approaches

- Behavior Therapy (BT)
- Cognitive Therapy (CT)
- Cognitive Behavioral Therapy (CBT)
- Biofeedback

All evaluated – to varying degrees – for efficacy, usually in comparison to wait-list or TAU control conditions

Results have been fairly uniform

- they all “work”
- all appear to offer alternatives or adjuncts to purely biomedical interventions.
- Effect sizes no more than modest

New Psychosocial Therapy Approaches

- Mindfulness-Based Stress Reduction (MBSR)
- Acceptance and Commitment Therapy (ACT)

Also evaluated – to varying degrees – for efficacy, usually in comparison to wait-list or TAU control conditions

Results have been fairly uniform

- they also “work”
- they also appear to offer alternatives or adjuncts to purely biomedical interventions.
- Effect sizes also no more than modest

No lack of imagination in developing new theories, models and psychosocial chronic pain interventions

But new is not necessarily better.

Showing that new Tx is superior to established Tx is an important hallmark of progress.

Is there evidence for superiority? progress?

No!!!

Comparison studies (i.e., new Tx's compared to established Tx's) -- a small minority of studies -- have generally found that different Tx's have similar effects on primary outcomes.

That is, alas, they all reach the finish line in a virtual tie.



Similarity of effects on *primary outcomes* emerges when 2 or more active treatments are compared...

Turner (1982). Relaxation = CBT

Newton-John et al. (1995). CBT = biofeedback

Redondo et al. (2004). CBT = physical exercise program

Smeets et al. (2006). CBT + physical exercise = CBT = physical exercise

Wetherell et al. (2011). ACT = CBT

Wong et al. (2011). MBSR = multidisciplinary program

in the context of additive studies where components are added and compared to single components...

Turner & Jensen (1993). CT + Relaxation = CT = Relaxation

Jensen et al. (2001). CBT + physical exercise = CBT = physical exercise

Smeets et al. (2006). CBT + physical exercise = CBT = physical exercise

Sharpe & Schreiber (2012). CT = BT = CBT

When a psychosocial Tx, enhanced with an additional component, is compared to the treatment alone...

Glombiewski et al. (2009). CBT + Biofeedback = CBT

Kerns et al. (2013). CBT + Motivational Interviewing = CBT

And even when Tx's are compared to active control conditions (eg, pain education) ...

Ersek et al. (2008). Self-management training = pain education

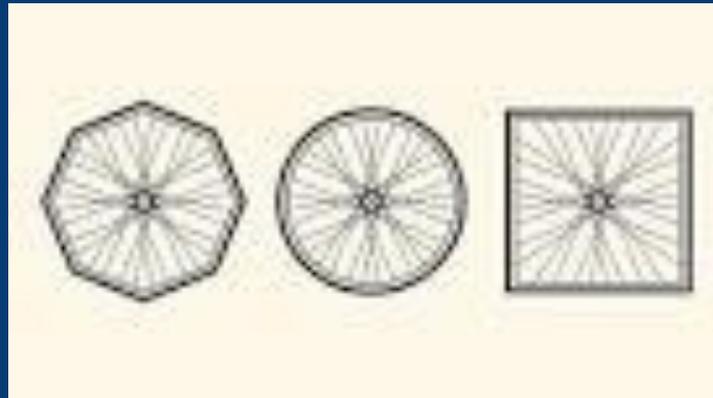
Thorn et al. (2011). CBT = pain education

Schmidt et al. (2011). MBSR \geq supportive group therapy

Disturbing Conclusions

- the drive to enhance outcomes via new and better psychosocial Tx's has languished since advent and dissemination of CBT.
- In terms of improved outcomes – and we have room for improvement -- we have not made any additional headway.
- “Anything” psychosocial has some salutary effect
- Normal RCT research has failed thus far to produce superior treatments

Rather than spending time and effort reinventing adequate but nonsuperior wheels with normal RCT science – target Tx vs inert control...



Perhaps we should stop exclusively asking “Does it work?”

And instead ask *“How does it work?”*

And thus discover what critical things make the typical wheel work

- *And then devote all our energy to maximizing these*

This would require that we shift our focus to the study of

Treatment Mechanisms

Conceptualizing Mechanisms in Psychosocial CP Treatments

“Mechanism” here refers to thoughts, emotions, behaviors targeted for change thru therapeutic procedures which in turn impact pain and function.

For example, CT alters maladaptive pain cognitions thru cognitive restructuring in order to reduce pain and increase function.

CT → ↓ Pain catastrophizing → ↓ pain

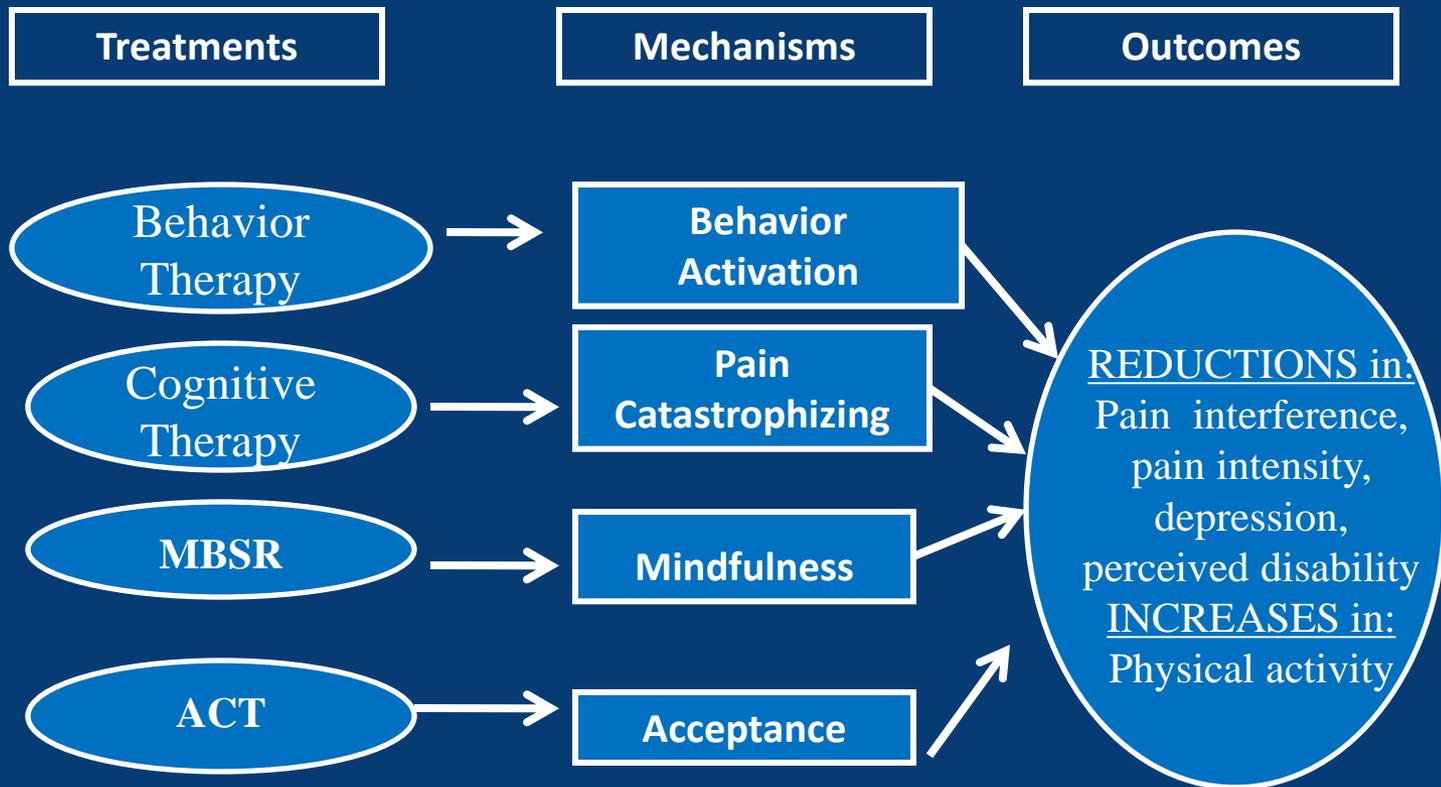
Conceptualizing Mechanisms in Psychosocial CP Treatments

The “default” option informing opinion about how Tx’s work may be termed the **Specific Mechanism Model**

All psychosocial chronic pain Tx’s may get to the same place but do so *via their own theoretically-specified and distinct therapeutic mechanisms*

Something like this....

Specific Mechanisms Model

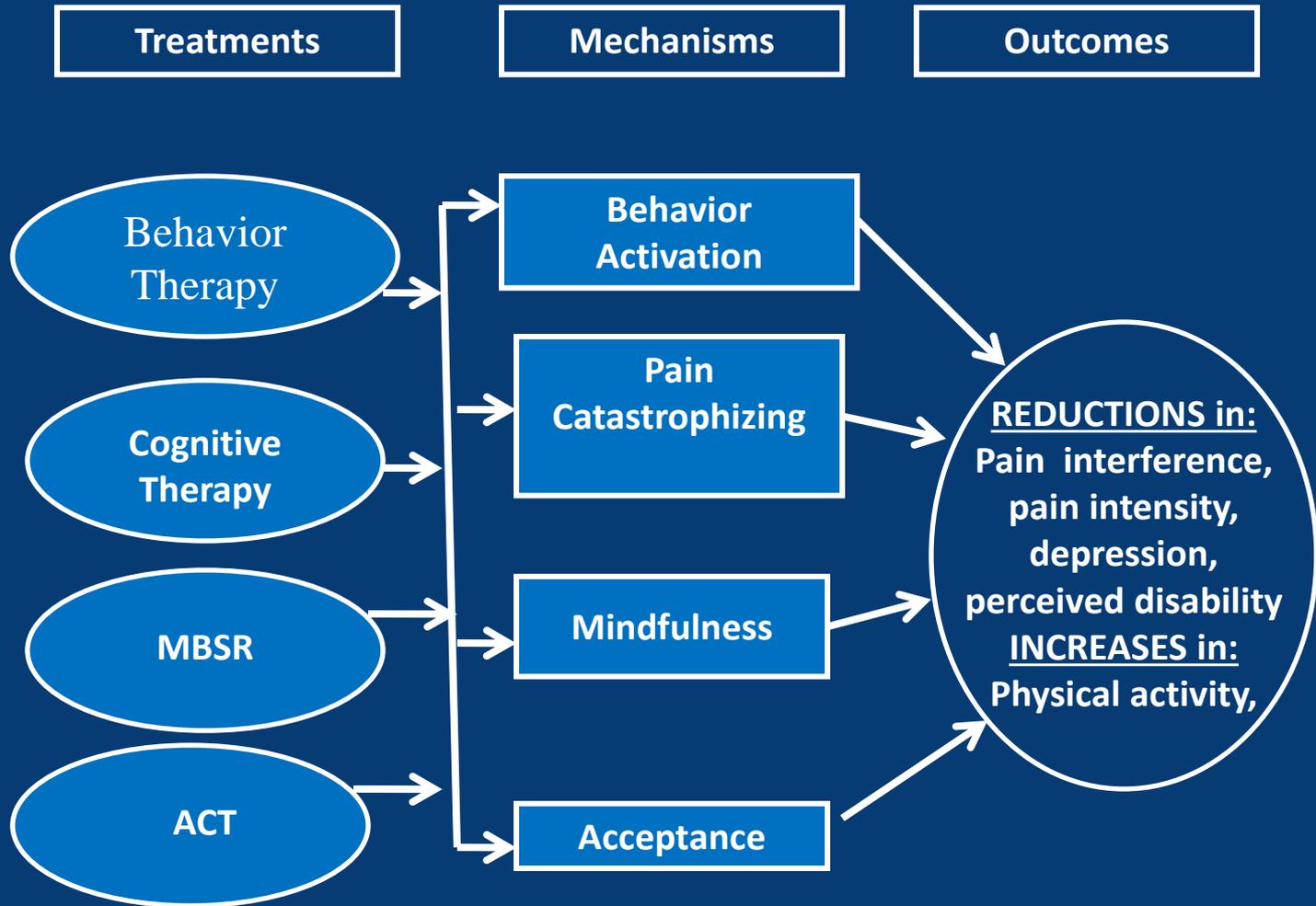


Given the similarity of effects across different Tx's,
alternatively and more parsimoniously is the **Shared
Mechanism Model**

All psychosocial chronic pain Tx's get to the same place via
*core critical mechanisms that are shared across
ostensibly different approaches.*

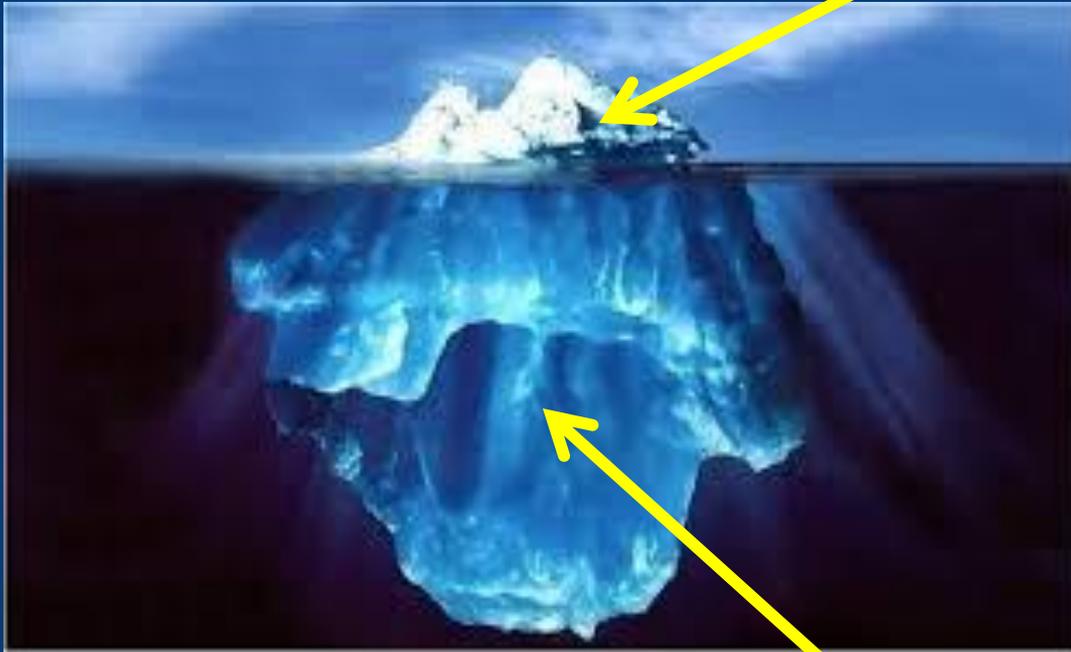
Something like this...

Shared Mechanisms Model



If so, theoretical distinctions and elegance may be just the tip of the therapeutic iceberg...

**Theoretical distinctions
(eg cognitive theory,
Mindfulness),**



**The true core mechanisms
shared by all Tx's**

But uncovering mechanisms is hard work...



“Does it work” is much easier to address

- Normal RCT methods comparing target Tx to control group
- Pre-Tx, Post-Tx, follow-ups on outcomes

“How does it work” requires us

- to operationalize & measure mechanisms
 - cognitive contents
 - cognitive processes
 - behaviors
- to have designs and statistics to be able to distinguish mechanism effects
 - Frequent assessments
 - Cross-lagged panel designs
 - Compare multiple Tx's
 - Assess multiple putative mechanisms

So what do we *actually* know about mechanisms in psychosocial Tx's of chronic pain?

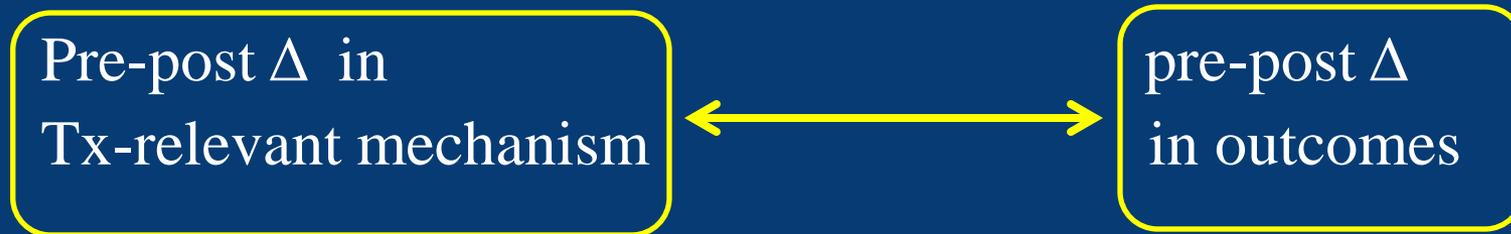
So what do we *actually* know about mechanisms in psychosocial Tx's of chronic pain?



Some recent research has focused on study of therapeutic mechanisms

- Secondary analyses of uncontrolled and controlled clinical trials
- Host studies not designed to comprehensively address mechanism questions
- At present, wide array of findings derived from analyses and designs with varying degrees of rigor.

Covariation



- CT-CBT:
 - Jensen et al. (2001); Spinhoven et al., (2004); Thorn et al. (2007); Turner et al. (2007)
- ACT:
 - Vowles & McCracken (2008; 2010)
- MBSR:
 - Schmidt et al. (2011)

Change in mechanism precedes change in outcome

Early-Tx Δ
in mechanism

>

Early-Tx Δ
in outcome

– Burns et al. (2003).

- early-Tx changes in pain catastrophizing > early-Tx changes in outcomes

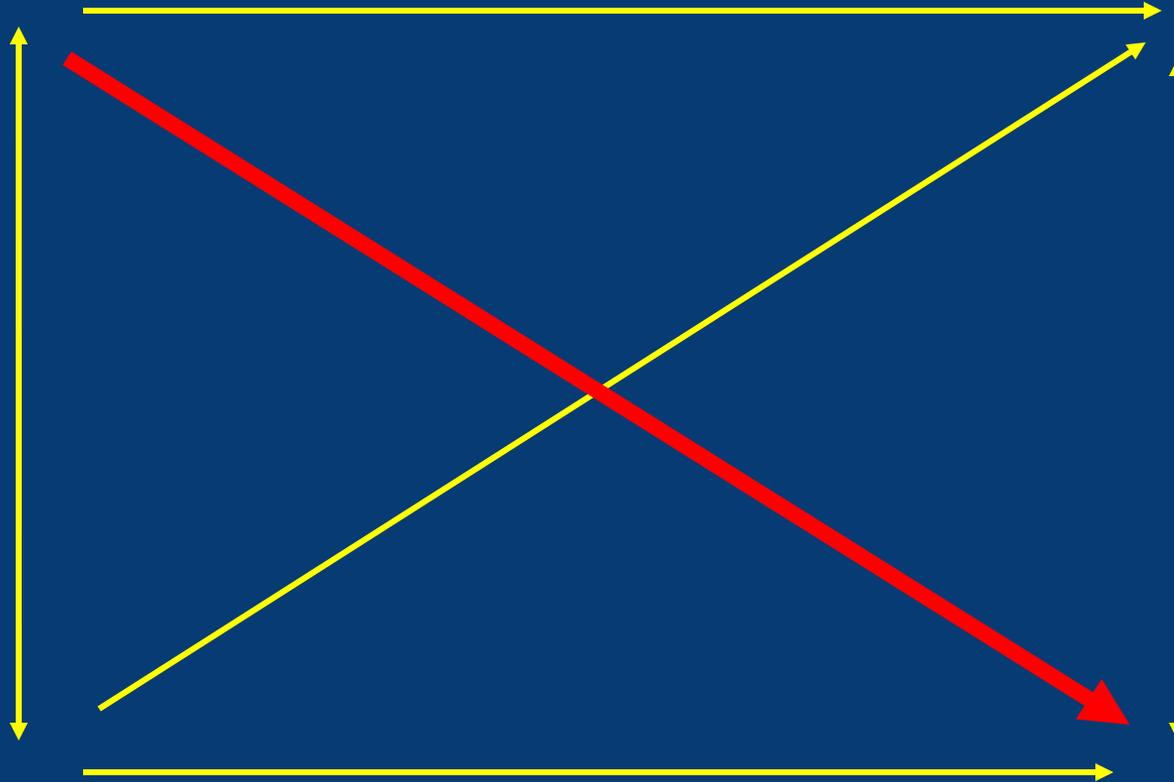
– Burns et al. (in press).

- early-Tx changes in action attitude > early-Tx changes in outcomes

Cross-lagged panel design

Early-Tx
Mech Change

Later-Tx
Mech Change

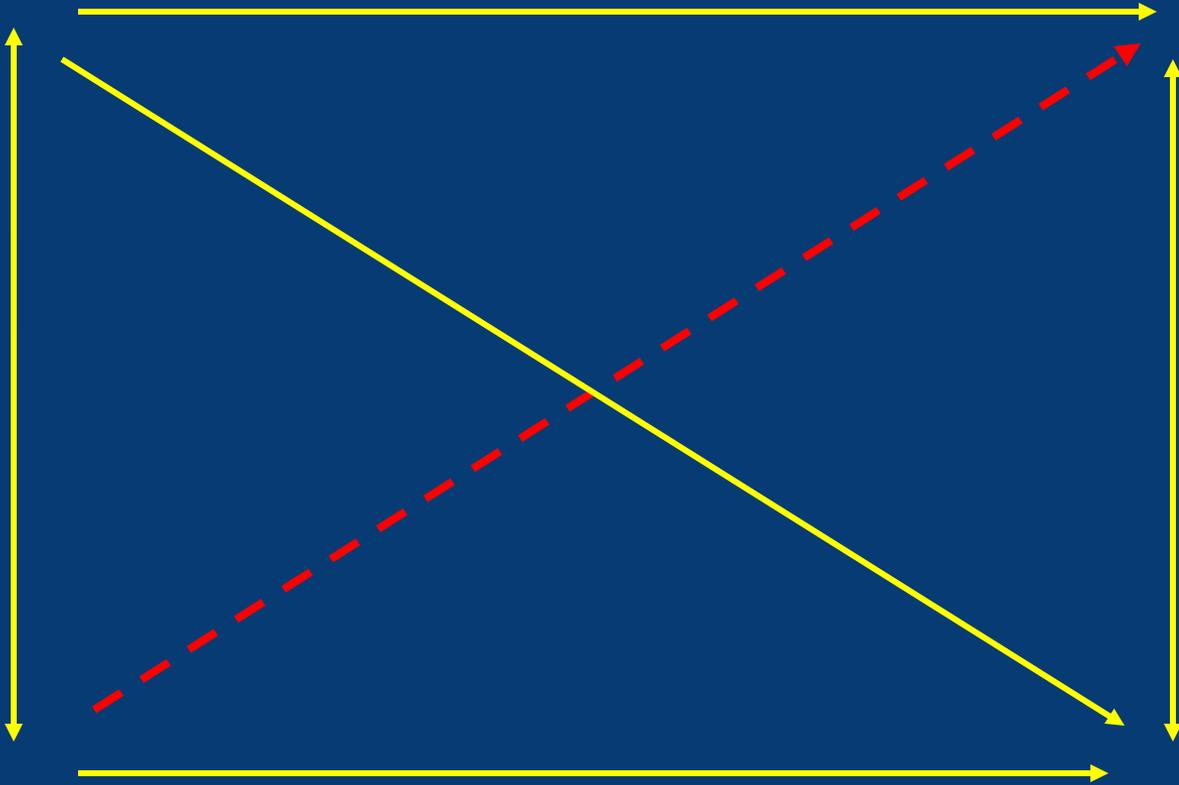


Early-Tx
Outcome Change

Later-Tx
Outcome Change

**Early-Tx
Mech Change**

**Later-Tx
Mech Change**



**Early-Tx
Outcome Change**

**Later-Tx
Outcome Change**

- Burns et al., (2003a,b).
 - early-Tx changes in pain catastrophizing predicted late-Tx outcome gains, but not vice versa
- Bergborn et al. (2012).
 - early-Tx changes in pain catastrophizing were related significantly to pre- to post-treatment changes in disability
 - did not test cross-lagged effects
- Burns et al, (in press).
 - early-Tx changes in action attitudes predicted late-Tx outcome gains, but not vice versa
- Wideman et al. (2009).
 - showed mostly null associations between early-Tx pain catastrophizing and later-treatment changes in outcomes.

Best evidence for specificity of mechanism would come from testing multiple mechanisms...

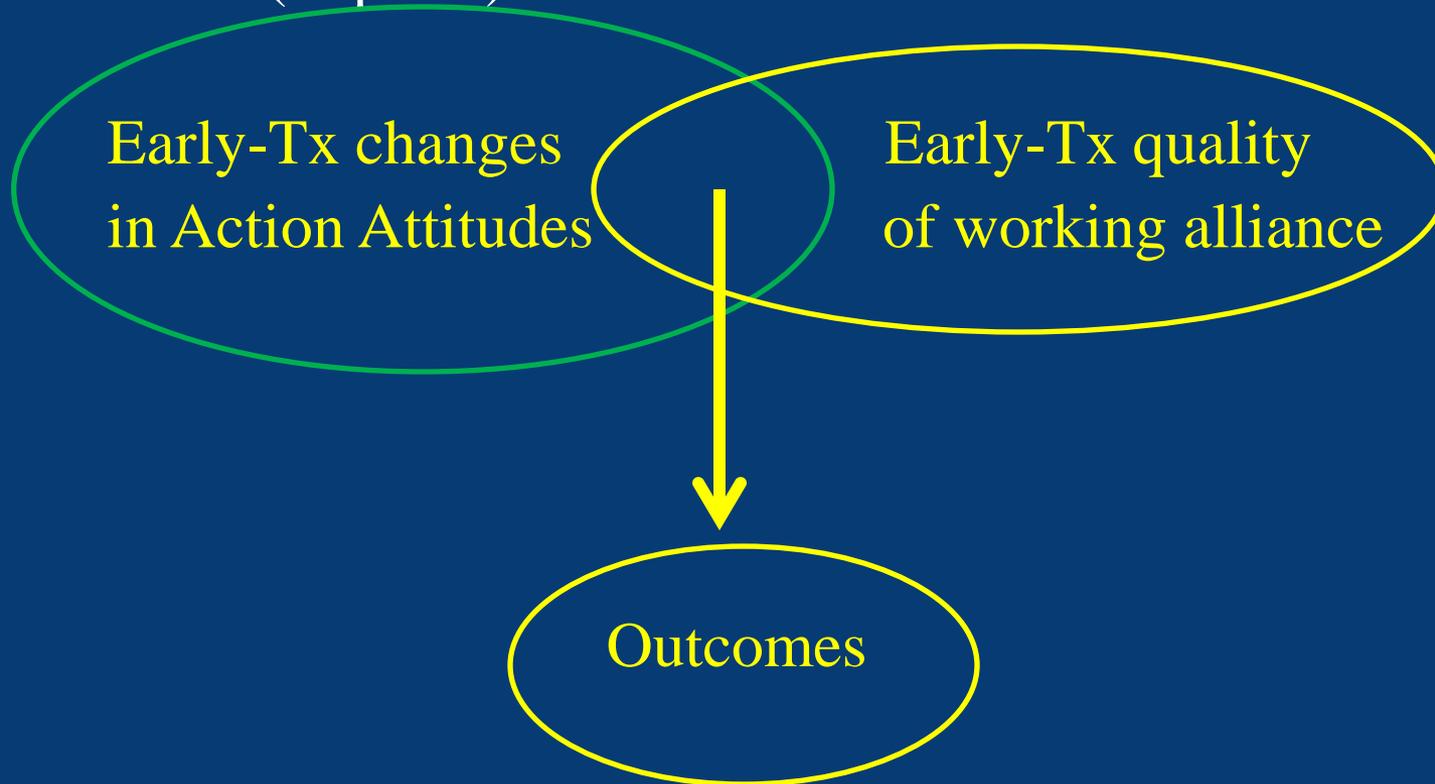
- Vowles et al. (2007): interdisciplinary program w/CBT
 - Signif pre-post changes in Acceptance and pain catastrophizing.
 - Changes were correlated
 - pre-post changes in Acceptance and pain catastrophizing predicted mostly common variance in pre-post outcomes.

And from testing multiple Tx's...

- Smeets et al. (2008) compared CBT, exercise treatment, CBT + exercise, wait-list control
 - 3 active Tx's did not differ on pre-post changes in pain catastrophizing
 - changes in pain catastrophizing equivalently predicted pre-post changes in outcomes across 3 conditions

What about non-specific mechanisms? Working alliance ...

- Burns et al (in press). CBT+ MI vs CBT



Summary

- Some evidence for correlation, lagged correlation, and precedence for mechanisms alleged to be specific
- Reductions in pain catastrophizing, increases in mindfulness, attitudes about pain self-management and self-efficacy
 - *ALL may be potent mechanisms*
- But may be brought about both via Tx's that deliberately target a specific mechanism (eg, pain catastrophizing in CBT) and those that do not (physical exercise; PE)
- Non-specific factors also play a role

Summary

- Little evidence to support Specific Mechanism Model
- Our ostensibly different treatments
 - Not only work to about the same extent
 - But appear to work via mechanisms (including non-specific ones) shared across, and not unique to, different Tx approaches.
- Predominant evidence for Shared Mechanism Model

- But little “evidence” overall.

If we acknowledge similarity of outcomes and shared mechanisms, we may seize the opportunity to

- revise “normal” RCT science
- embrace a new paradigm focused on “how does it work”
- answer the question: “if different Tx’s work for many of the same reasons, *then what are these reasons?*”
- find the active mechanisms that make all treatments work
- find the inert and/or redundant factors
- eliminate the latter
- amplify the former

Step 1: Identify Key Mechanisms within Extant Treatment Approaches

Combine RCT with state-of-the-art methods and statistical techniques to uncover mechanisms in Tx's we already have

- Ongoing trial (N=400; Burns, Keefe, Thorn, Jensen et al.)
 - CT, MT, BT and TAU
 - Assume CT, MT, BT will produce similar outcomes
 - With weekly assessments of putative mechanism and outcome factors, examine
 - magnitude of unique and shared effects of mechanism changes on outcome gains across Tx's
 - role of non-specific factors
 - timing of mechanism and outcome changes

Results with methods like these have the potential to show...

- whether alleged mechanisms are indeed actual mechanisms
- *which mechanisms are the primary, secondary and tertiary drivers of treatment effects*
- *which mechanisms should be primary target(s) of any and all treatment procedures and techniques.*

Step 2: Search for New Mechanisms and Techniques to Activate Them

- The conceptualization, design and testing of new and theoretically distinct treatments *must not end*. But, we must avoid normal RCT science
 - uncover new mechanisms that reduce pain and improve function
 - determine which techniques best activate them
 - ***overarching goal to boost efficacy over the Tx's we already have by pursuing NEW mechanisms that exert unique effects***
- New NIMH plan (RFA-MH-15-300)...

Instead of just altering thoughts, feelings and behavior – what they all apparently do – look for new phenomena that affect pain via new pathways

This search requires casting a wide net.

- Increasing function of endogenous opioids
 - Aerobic exercise
- Altering brain region function
- Biopsychosocial mechanisms
 - Work with family and significant others to...
 - Reduce solicitousness, invalidation and criticism
 - Increase reinforcement of well-behaviors

Instead of spending 90% of our time and money spiffing up the 10% of the therapeutic iceberg we can see, let's spend 90% of our time and money understanding and improving the 90% of the iceberg on which we're ALL floating.

We owe it to the field, our patients and to taxpayers to actually increase the effectiveness of our chronic pain treatments.

We need new thinking and methods to actually do so.

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