

# MECHANISMS OF CHANGE IN PSYCHOSOCIAL TREATMENTS FOR PAIN-RELATED CHRONIC HEALTH CONDITIONS

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# Overview of Symposium

- ❑ Theory of change in psychosocial treatments is specific (e.g., reduction in maladaptive cognitions in CT, increase in mindfulness/acceptance in MBSR)
- ❑ Studies of efficacy greatly outnumber studies of mechanism of change
- ❑ Some studies have assessed theory-specific mechanisms of change (e.g., catastrophizing and CBT)
- ❑ Studies rarely assess theory-specific mechanisms as a potential agent of change in other active treatments that may have a different theoretical basis (e.g., catastrophizing and physical therapy)
- ❑ Certain methodologies and statistical analyses are necessary to study mechanism of change

# Speakers

- ❑ Beverly Thorn (University of Alabama) will discuss conceptual/theoretical basis for studying mechanism of change
  - ❑ she will give an example of examining pre-post correlations between theorized mechanism of change and outcome in a recent RCT
- ❑ John Burns (Rush University Medical Center) will discuss methodological considerations for studying mechanism of change
  - ❑ he will give examples of methodological features and statistical analyses needed to better document mechanism effects
- ❑ Mary Davis (Arizona State University) will explore the utility of diary reports in elaborating common and unique mechanisms of change in randomized clinical trials.

- ▣ Beverly E. Thorn, Ph.D., ABPP
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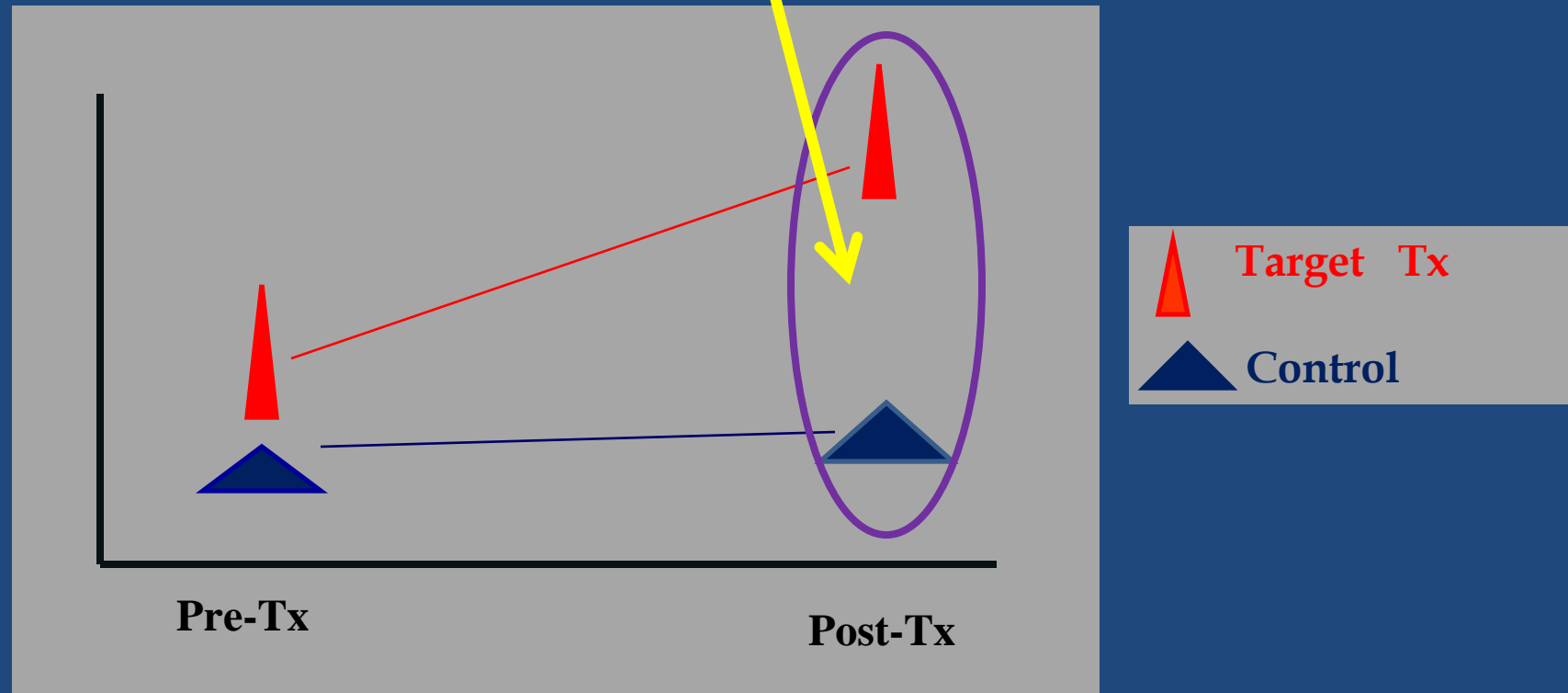
# Conceptual/Theoretical Basis for Studying Mechanisms

*“Does it work?”*

is not the same as

*“How does it work?”*

Much of our “knowledge” of mechanism is based on a questionable assumption: “Since we targeted X as part of our specific technique, and since it changed with our treatment, X must have caused the post-Tx differences between the 2 conditions.”



# Why Study Mechanisms?

- ❑ To test treatment-specific theories that underlie the rationale for undertaking a particular intervention
- ❑ To test whether the things we deliberately target to bring about outcomes are actually among the “active ingredients”
- ❑ To facilitate translation/ dissemination of efficacious treatments from controlled RCTs to real world clinical practice, we must identify aspects of tx that are *critical and cannot be diluted* vs. those that are not critical to tx success



# Why Study Mechanisms?

- ❑ To show public health value of psychosocial pain treatments
- ❑ To foster acceptance and dissemination of these approaches
  - we must be able to verify that our treatments produce desirable outcomes
  - we must be able to verify that they do so precisely because of the time-, energy-, and resource-consuming therapeutic procedures that the interventions entail
- ❑ To avoid the conclusion that a treatment “seems to work, but we’re not sure why”

# Causal Assumptions Should be Tested

- Need “evidence rules” and/or “conditions” on which to build the “case” for a mechanism
- Need to specify and define mechanisms
  - The target (theory-specific) mechanism
  - competing theory-specific mechanisms
  - general mechanisms (e.g., working alliance; pt expectations)
- Need to measure indexes of mechanisms
- Need to analyze effects of mechanisms

## To build the case for a mechanism, at least 5 conditions should be examined

- Changes in mechanism and outcomes are correlated
- Substantial change in mechanism precedes substantial change in outcome
  - “cause” (e.g., decreased maladaptive cognitions) must precede “effect” (e.g. decreased pain severity).
- Early change in mechanism predicts later change in outcome (i.e., lagged correlation), but not vice versa
  - “cause” predicts “effect,” but “effect” does not predict “cause”
- Change in mechanism is specific to the treatment approach
  - cognitive restructuring as part of CBT invokes greater decreases in maladaptive cognitions than meditation does as part of MBSR
- Mechanism change has some degree of unique relationship with outcome changes beyond effects of general mechanisms (e.g., working alliance, patient expectations)

Typical RCTs, with focus on pre-post efficacy, do not adequately address the 5 conditions, and give only circumstantial evidence regarding mechanisms

To derive sounder evidence, we must

- Examine time course of mechanism and outcome changes
  - multiple assessments during Tx
- Examine lagged effects between mechanism changes and outcome changes
- Examine the unique and overlapping effects among
  - the target (theory-specific) mechanism
  - competing theory-specific mechanisms
  - general mechanisms (e.g., working alliance; pt expectations)

## Using Cognitive-behavioral Approaches as an Example:

- CBT theory posits that appraisals and interpretations of events affect subsequent emotional and behavioral responses
- Tenet of CBT is that alteration of maladaptive patterns of appraisals and interpretations through cognitive restructuring can lead to a correction of these problematic responses.
- Cognitive change is a therapeutic mechanism specified by CBT (a theory-specific mechanism)

## CBT Mechanism Studies

A number of studies have examined correlations between pre-post Tx changes in maladaptive cognitions/coping and pre-post Tx changes in outcomes.

- E.g., Jensen, Turner & Romano, 2001; Turner, Holtzman, & Mancini, 2007; Spinhoven, Kuile, Kolen-Sijders, Mansfeld, Ouden & Vlaeyen, 2004.
- But these results document JUST correlation.

# The Next Step Up: Combine RCT Approach with Correlational Method

- ❑ If CBT works specifically via reductions in maladaptive cognitions, we would expect cognitive change to be greater in CBT than in other treatments.
- ❑ Smeets, Vlaeyen, Kester, & Knottnerus (2006):
  - ❑ CBT only, Physical Conditioning only, CBT + Physical Conditioning, Wait-list Control
  - ❑ 3 active conditions did not differ on pre-post changes in pain catastrophizing (all were significant)
  - ❑ Pre-post changes in catastrophizing equivalently predicted pre-post changes in most pain-related outcomes.

# Burns, Day, & Thorn, 2012

- ❑ Secondary analysis of RCT with rural patients with chronic pain (Thorn, Day, Burns et al., 2011)
- ❑ Two conditions: CBT (n=32), Pain Education (n=29)
- ❑ Pain Education participants given pain-relevant information about cognitions and behavior, but not given skills training nor homework
  - ❑ Conditions did not differ on pre-post changes in pain catastrophizing (both conditions produced significant reductions)
  - ❑ Pre-post changes in catastrophizing equivalently predicted pre-post changes in Quality of Life, perceived disability, pain intensity, & pain interference for both groups
  - ❑ Pre-post changes in catastrophizing significantly predicted reductions in depression for CBT group only



# Meaning....

- ❑ If CBT mechanism is reducing maladaptive cognitions, changes in cognition should primarily occur and predict pain-related outcomes in CBT (and to a much lesser degree in different txs)
- ❑ Neither Smeets et al. (2006) nor Burns et al. (2012) found this
- ❑ Ss in PT (Smeets) & EDU (Burns) reduced their catastrophizing, but not via a protocol that explicitly targeted cognitive change

# Possible Conclusion

- ❑ Reductions in catastrophizing , by whatever means, may be a potent broad therapeutic mechanism not specific or limited to CBT.
- ❑ Strict adherence to cognitive restructuring via CBT may not be necessary to achieve these therapeutically important cognitive changes
- ❑ (with the possible exception of depression)

# Summary

- ▣ We are in our infancy of examining mechanisms of action of psychosocial treatments
- ▣ Changes are needed in our research design and analyses to examine mechanism
- ▣ I have presented two examples of “lower-tier” examination of mechanism
- ▣ John Burns and Mary Davis will present “higher-tier” methods