Module:
Psychological Foundations of Mental Health

Week 5
Cognitive therapy: experimental and clinical evidence

Topic in Action 1
Testing the cognitive model – Part 1 of 2

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Lecture transcript

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Cognitive therapy for depression has been with us for over 50 years, and is firmly established as a primary treatment within many mental health services worldwide as an alternative or adjunct to medication. Much of that growth comes from evidence of its effectiveness and efficacy, some of which we reviewed in the last topic. This suggests that CBT and cognitive therapy for depression can be effective in reducing symptoms, either alone or in combination with other treatments.

However, does this tell us anything about why and how it works? In particular, when it is effective, is it really because it targets the key cognitive processes that Beck identifies within his cognitive model? Conversely, when it doesn't work for some people, why not?

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It would be reasonable to expect after the amount of research that has been done over the past 50 years, that we would have a pretty good answer to these questions. However, things are not always as we expect. Although advocates of cognitive therapy and CBT would no doubt disagree, here's a conclusion from a review published in 2007 by Alan Kazdin, a highly respected clinician and researcher from Yale University. As you see, he feels that we can still say little about why cognitive therapy works, when it does.

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Some people may argue that if something works, do we really need to understand how and why? However, for any therapy-- whether psychological or otherwise-- understanding the mechanisms of action is critical. The reasons for this come in the interrelationship between theory and application within translational clinical research.

Our theories and the evidence derived from testing them are fundamental because they allow us to identify one or more critical targets for treatment, a target that is assumed by the model to have a causal role in either the onset or maintenance of the clinical problem. A rational therapy is then developed that aims to intervene directly on that target, one that we predict will be an effective treatment if our theory is correct. The outcome can then be measured and evaluated through clinical trials as we discussed in the last topic.

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Any treatment's effectiveness will depend, in part, on how well it addresses the critical targets proposed by the theory and its supporting evidence. The more accurately the theory can define the critical target, the more precisely the therapy addresses them, and the better the outcomes are likely to be.

However, just as no treatment is perfect, nor, too, is any one theory. Because of this, both need to be continuously tested, revised, and often, eventually replaced by a better one. This allows us to further improve the clinical applications, even if they build on those that went before.

So outcomes from clinical trials themselves provide one important source of evidence in this cycle. They can help us with the final treatments, so they address the hypothesised targets more effectively. For example, we may be able to cut out elements of a complex treatment that are not contributing to the overall treatment effect, allowing us to deliver similar outcomes more quickly and economically.

Alternatively, a specific therapy component may be adjusted so that it better addresses the target. Equally, therapy outcomes provide evidence that relates back directly to the underlying theory and models on which the treatment was based. When the outcomes don't match the predictions of the theory, or better predicted by an alternative one, the theory itself may need to be revised or replaced, rather than tweaking the therapy to make the data fit. And so the process continues with, we hope, evermore accurate theories and evermore effective treatments.

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Let's take 3 of these elements and rearrange them a bit-- the therapy, the target, and the outcome. The target, in this context, can be considered an intervening variable. We used this term in week one when considering the role of an unobservable, but inferred, factor that is transformed by an input-- in this case, the therapeutic intervention-- and is instrumental in bringing about the observed outcome-- the therapeutic change.

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In the case of the cognitive model and cognitive therapy for depression, we can summarise the critical intervening variable as dysfunctional thinking. This lies at the heart of the model and is the explicit target of the various therapeutic techniques of CBT that we looked at last week. When we examine outcomes from clinical trials, what we observe-- at least, on average-- is positive therapeutic change-- a decrease, not just in dysfunctional thinking, but in overall reduction of depression.

Although seemingly plausible and broadly consistent with existing evidence and cognitive psychology, we have to remember that the cognitive model is just that-- a theoretical model, not a statement of fact. It is a hypothesis based on the combination of clinical observation and research. Like any model, it needs to be tested before we know whether or not it is accurate.

What we have not looked at so far is the evidence that cognitive therapy works through changing the dysfunctional thinking, and that this, in turn, is responsible for the clinical benefits that we observe. As Kazdin observed in 2007, there is still a big question mark over the accuracy of this model and mechanism of action of cognitive therapy.

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This brings us to the main focus of the present topic-- how we test and identify potential causal and mediational variables within the cognitive model that may help us understand how and why CBT works so we can continue to improve it. Let's look briefly at 3 key concepts-- cause, mediation, and mechanism. It's important that you understand and use these terms accurately. We can use a physical example to illustrate them.

What happens when we press a switch to turn on a light? There is an obvious association between

the action and the outcome, as shown by the two-headed arrow. But can we say more about how it happens?

However, rather than just an association, we can reasonably consider that pressing the switch is a causal event. The action leads to and is directly responsible for the observed change. The light would not come on without it. We can now represent this as a one-directional arrow.

However, pressing a switch does not describe, yet, how pressing the switch leads to the light coming on. What critical variable and mechanism brings this about?

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With our knowledge of electric circuits, we can see that the critical process is the closing of a break in the circuit connecting a power source to the light bulb. In this example, we can say that the closing of the break is a mediating variable. A mediator can be defined as the variable that explains the observed relationship between the event and the outcome.

A mediator may fully explain the relationship-- as illustrated here-- or be a partial mediator, explaining some of the relationship, but not all. Partial mediation is more typical when considering mediation in therapy.

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However, we still have not described the actual mechanism. In this example, the mechanism is not the closing of the break in the circuit, but the electrical current, the flow of electricity caused by the movement of electrons in the copper wire from a negative to a positive charge, and the effect of that current flow in the bulb causing it to emit light energy.

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The cognitive model proposes that the techniques of CBT cause a change in dysfunctional thinking, and that this change causes the overall clinical improvement. The model predicts, first, that there will be an association between CBT and a reduction in depression. This is supported by the evidence from clinical trials.

The cognitive models suggest further that this observed relationship is explained by a change in dysfunctional thinking that is acting as a mediator of clinical recovery. In other words, there is no direct association between therapy and outcome, only an indirect one mediated by change in thinking.

A possible counterargument is that change in dysfunctional thinking is simply a marker of recovery, along with reduction of other symptoms. In other words, that dysfunctional thinking simply reduces as the person gets less depressed.

So mediation cannot be assumed. It has to be demonstrated empirically. Demonstrating mediation is central to any test of the cognitive model. However, demonstrating mediation is not the same as proving causation.

For example, a change in dysfunctional thinking may itself be associated with some other change that is occurring during therapy that is the true mediator. Unless we measure it, we will not be able to assess its importance. So while change in dysfunctional thinking may seem central, it may be a so-called proxy variable, or indicator, of the true variable.

This brings us to the issue of mechanism. Is change in dysfunctional thinking the actual mechanism that is critically responsible for the clinical improvement? Identifying a strong mediator can be helpful in pointing us in the direction of a possible mechanism of change. But proving causation is much more challenging, as we will see.

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How do we set about assessing whether a variable mediates the relationship between therapy and outcome, and whether it is a potential mechanism responsible for the changes that we observe? The first important point to note is that no single piece of evidence will be sufficient to conclusively demonstrate that a variable is a mediator or causal mechanism. We need convergent evidence from a range of different sources.

The review by Alan Kazdin set out a range of criteria that we can apply along with their associated research methodologies. First, while association evidence is never sufficient, those associations that are observed should be strong and reliably demonstrated across the majority of studies. For example, between therapy and change in a proposed mediator or mechanism, and between such change and the desired therapeutic effect.

Second, we would want evidence of specificity of the mediator or mechanism. While there may be more than one process of change, the better we can isolate a specific one predicted by the theory, the more confidence we can have that it plays a central role. Third, we would expect any important mechanism to be relevant across a range of settings and applications. Where a variable is an apparent mediator or mechanism only in one particular setting, it is less likely to be central to the process of therapy more generally.

Clinical trials can be considered as a controlled experiment, but typically don't allow us to isolate and assess the role of a specific factor that may be serving as a mediator or mechanism of change. This is where experimental laboratory experiments come in. In experiments, we have far greater control over manipulating specific variables and measuring their effect on the target outcome.

Laboratory experiments do not aim for clinically meaningful change. Instead, they look for evidence that a specific manipulation changes a variable. This can serve as a marker of clinical improvement, even in the short term.

To infer a causal relationship, we also have to be confident that the temporal order of events is correct. If a variable is a mediator or mechanism of change, it must show change before, or at least simultaneously, with the clinical change. Whether this happens quickly or slowly is not important. If the proposed mediator changes after the clinical improvement, we may simply be observing reverse causation.

For example, people's dysfunctional thinking changes because they are less depressed, not the other way around. If a variable is an important mediator and/or mechanism of change, we might also expect to observe a systematic association between the degree of change and the mediator, and the degree of change in outcome, a so-called gradient. Thus, a small change in dysfunctional thinking would be associated with a small reduction in depression, and a large change in thinking with a greater change in depression.

The final criterion that Kazdin proposes is that of the plausibility or coherence. This refers to the extent to which a proposed mediator or mechanism is consistent with the broader base of scientific evidence. In our situation, this is knowledge of cognitive factors and their interactions with mood and behaviour that may increase vulnerability to mental health problems, maintain them over time, and predict recovery or relapse. Such coherence may also be increased by convergent evidence from other sources, such as animal studies or neuroimaging studies.

In the next section, we will look at examples of such evidence for the role of changing dysfunctional attitudes as a central mediator of clinical improvement following CBT, and as a potential mechanism of change. A word of warning, though, before we begin-- conclusive answers remain stubbornly elusive.

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