Module:
Psychological Foundations of Mental Health

Week 5
Cognitive therapy: experimental and clinical evidence

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

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Lecture transcript

Slide 3
In this section, we are going to explore the role of dysfunctional attitudes and beliefs that an individual holds, and their potential role in depression. If you remember back to week five, having a certain schema will shape both the individual's beliefs about the future, themself, and the world, and the nature of their thought content when they are depressed.

However, the model does not just consider the schema as a feature depression, per se, in the same way as low mood. Rather, the schema is seen as an enduring feature of the individual, built up over the course of their life through a range of factors. This can shape their general way of thinking about themselves as a person, about other people, the world around them, and their future.

Slide 4
Dysfunctional attitudes that characterise a schema are thought to represent a diathesis, or vulnerability factor. Holding certain attitudes in the presence of life's stessors may increase an individual's risk of becoming depressed, or for relapsing, following recovery from a previous episode.

Other vulnerability factors may include early adverse life experiences, thinking style, genetic factors, and brain biology. Of course, it is quite possible that these various factors or just different reflections of the same vulnerability factor.

Beck's model is an example of a more general diathesis stress model of psychological disorder that is widely applied, not just to depression. Such models propose that it is the presence of stress, combined with one or more vulnerability factors, that increases the risk of depression and other psychological disorders. The vulnerability factor can serve to magnify the impact of the stressor.

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Before we start to look at the evidence about the role of dysfunctional attitudes, we need to be able to measure them reliably. Measures such as the Beck Depression Inventory do contain individual items that address some of the core negative attitudes-- such as future pessimism, and

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negative self-reference-- and tend to better reflect a current depressive mood than an underlying trait.

However, such items do not capture the full range of dysfunctional attitudes that lie at the heart of the cognitive model. In addition, using individual items from a scale such as the Beck Depression Inventory is not reliable. For these reasons, a number of separate scales have been developed to assess dysfunctional attitudes.

One of the first was the Dysfunctional Attitude Scale, or DAS, published by Weismann and Beck in 1978. Originally comprising 100 items, it was developed into two 40-item versions-- the DAS-A and DAS-B.

Beck's model suggests a variety of dysfunctional attitudes may occur in depression, and in depression-prone individuals, but does not assume that all attitudes are affected equally, even within an individual.

A 24-item version of the DAS-A was proposed by McPower and colleagues in 1994, here at the IOPPN. This version seemed to reliably measure three subtypes of dysfunctional attitudes. First, those relating to personal achievement, such as these two examples shown here. If I fail at work then I'm a failure as a person. Or if a person asks for help it is a sign of weakness.

Second, there were items that tap attitudes relating to dependency on others, such as these two. My happiness depends more on other people than it does on me. Or if others dislike you, you cannot be happy.

Finally, there are items relating to self-control, such as these last two examples. I should always have complete control over my feelings. Or a person should do well at everything he or she undertakes.

A more recent analysis of the DAS-A by Ester de Graaf and colleagues was based on a large population sample of 8,000 Dutch individuals. They tested how well previously proposed subscales structures fitted the actual data.

In the end, they suggested yet another version, this one of 17 items that seemed to identify two reliable subscales, one relating to perfectionism and performance achievement, and the second to dependency. This recent 17-item version has not been widely used to date.

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With a reliable measure dysfunction assumptions, we would expect to find that their level is higher in people with depression than people who are not depressed, and that severity of depression is associated with the level of dysfunctional assumptions held. Data from the large de Graaf study confirms both predictions.

First, in the graph, we see the mean scores of the two subscales and the total score in the non-depressed group and the 8% of the population studied that had major depressive disorder. All comparisons were highly statistically significant. Although, we have to remember that, with large samples such as these, even relatively small effects can be significant without being very strong or important.

However, overall, we see that the depressed patients endorsed dysfunctional attitudes over 50% more strongly than the non-depressed sample, with the biggest difference observed for the attitude relating to perfectionism and performance.

What about the association between attitudes and depression severity? The table on the right shows the Pearson correlation coefficients between the subscales and the total score in the

measure of depression severity.

Both subscales and the total score were significantly associated with the severity of depressive symptoms. To judge their magnitude, the square of the correlation, as shown, is an indication of the amount of variability explained.

We see that the measures of dysfunctional assumptions can account for approximately a quarter to one-third of the variability in depression, and vice versa. This is a substantial amount, although it suggests that the majority of variability remains unexplained, some of which may be accounted for by other potentially more important mediators that were not assessed.

Slide 7
So dysfunctional attitudes are elevated in depression, as measured by scales such as the DAS. Other research has shown similar effects for the frequency of actual dysfunctional thinking-- the negative automatic thoughts themselves.

Scales such as the automatic thoughts questionnaire, developed by Holland and Kendall in 1980, have also been widely used to measure this aspect of thought content. The cognitive model would predict that CBT, which seeks to target both the dysfunctional cognitive processes-- the attitudes-- and the thought content itself, will lead to reduction in both aspects of negative thinking as the patient recovers.

Further, such findings should be general ones, seen across the majority of studies. The best way to assess these predictions is, as always, ideally, not through results of individual trials, but through meta-analysis.

The forest plot, here, shows results in the systematic review by [INAUDIBLE] and colleagues from 23 RCTs published between 1987 and 2012. These all reported mean change in dysfunctional thinking, either attitudes as measured by the DAS, or content as measured by the ATQ.

As predicted, there is a consistent mean positive reduction in dysfunctional thinking in the CBT group, compared to the control intervention, in every study. The one exception, shown here, was for one component of an early study by Scogin in older adults for an intervention based on coping skills, rather than cognitive therapy, more specifically. Not all of the studies showed significant change, typically because of small sample size. But the overall effect size for the pool data was moderate and significant at 0.51. Similar effects were observed for the studies that used the DAS and those that used the ATQ.

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However, just because there is a reduction in dysfunctional thinking, is this associated with clinical recovery? This question was addressed in the study using the technique meta-regression.

This is another meta-analytic technique. However, rather than analysing differences in mean change in the outcome, it assesses the relationship between that outcome and another variable reported in the studies.

In this figure, we see the mean change in dysfunctional thinking in the CBT condition for each of the trials on the horizontal x-axis, expressed as a standardised effect size, and the corresponding change in depression score on the vertical y-axis. This shows us that the greater the mean change in dysfunctional thinking, the greater the mean reduction in depression symptoms. This offers further support for an association between the degree of change in the presumed intervening variable, and clinical recovery.

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We have evidence that depression is reliably associated with dysfunctional thinking, though CBT

can reduce such thinking, and that the degree of reduction is associated with the degree of positive therapy outcome. However, this still does not help us answer questions about causality, mediation, or mechanism. Rather than the hypothetical model shown here, the evidence that we have examined could be equally explained by the model below-- that people's thinking changes because they are less depressed, not the other way around.

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One way to address this is to measure dysfunctional thinking before and after a person develops a negative mood. In effect, can thinking-change follow the mood change.

This was addressed in a classic and still influential study by Jeanne Miranda and Jacqueline Persons in 1988. They did not rely on the spontaneous occurrence of depression, but induced a temporary mood change using standard laboratory induction techniques, such as those you learned about in week 3.

The dysfunctional thinking was measured using the DAS, before and after either a negative depressive mood induction, or a positive euphoric one. The inductions were successful in producing reliable, if transient, change in mood in the expected directions, as shown in the graph, here.

A high mood school on the scale we used is more negative. We see that mood improved following the positive induction, and worsened during the negative one.

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Levels of dysfunctional thinking was low in both groups before the induction, and changed in opposite directions follow an induction with a significant statistical interaction. There was evidence of a significant decrease on the DAS in the positive group, and a small but non-significant increase in the negative group. This offers some partial support for dysfunctional thinking being mood-related, or mood-congruent.

This study also addressed a separate question-- what is the impact of depression history on such mood congruence effects? The groups were separately analysed according to whether or not they had a prior history of depression.

Having been depressed previously did not affect the level of dysfunctional attitudes before the mood induction. However, those with a history of depression showed a markedly greater increase in such negative thinking after mood induction, compared to those with no history.

These results suggest that a tendency toward dysfunctional thinking may either remain as a sort of psychological scar following a previous episode of depression, or may reflect a trait-like vulnerability factor, as suggested earlier. While such thinking is not evident most of the time, when not depressed, a change in mood can trigger dysfunctional thinking, or perhaps, make the depressive schema more accessible. In other words, dysfunctional thinking may be a trait, but one that is mood-dependent to become visible.

This suggests that reductions in dysfunctional thinking following successful treatment of depression may be a consequence of improved mood, and not the other way around. The dysfunctional thinking style remains latent, and as a vulnerability factor for later relapse.

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Simple pre/post-treatment assessments of change in depression, and other variables, will never be able to conclusively resolve potential mediation and causality questions. As noted by Kazdin earlier, we need to assess the timeline of change in the two measures to see if one changes before the other, or whether they changed simultaneously.

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What does a therapy response look like over the course of CBT? One possible pattern is that the process of clinical improvements is smooth and continuous over the course of treatment, as shown by this hypothetical example. This would suggest that whatever is mediating the change is happening continuously and steadily, offering few clues about possible causal mechanisms, because there is no clear before or after.

In practise, therapeutic response is rarely smooth and continuous. A frequent observation is that some patients show a sudden gain between one session and the next. Such gains tend to be maintained.

Indeed, showing rapid gains is suggested as an important predictor of long-term outcome. Clearly, something has happened between session 4 and 5 in the example shown here, with a clear drop in depression score from 17 to 7.

If we can identify something that happened in therapy, before or during session 4, we may have a clue about a possible mediator, or even a cause of the improvement observed the following week. If that same thing is reliably shown across other patients, who also show sudden gains, the evidence is even stronger.

Let's look at one of the first studies to look at predictors of sudden gains during CBT for depression.

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We see, here, some real average data from a group of 29 patients with initial severe depression, who showed sudden gains in the course of two clinical trials of CBT.

Depression severity was measured at each session. A sudden gain was defined using a series of criteria to make sure that it was large in absolute terms, and relative to the level of depression for the first three sessions prior to the change, and that it was sustained afterwards.

In these 29 patients, the sudden gain was at least 7 points on the BDI, with an average of 11.2 points-- a very substantial improvement. For half of the patients, the gain occurred in the first half of therapy, between sessions 4 and 10, with session 5 being the most common for the gain to occur.

On the figures shown here, the session marked n is the one just before the change happened-- the pre-gain session, and the session n plus, the one where the sudden change was observed-- the gain session. Rather than use self-report data, the authors analysed audio recordings of the actual therapy sessions. These were done blind with the person doing the rating knowing whether the session happened before or after the sudden change.

The authors were specifically interested in the time course of any indications of cognitive change. Various indicators were used, and formed the basis of the patient cognitive change scale, used by the researchers rating the tapes.

Also rated was a measure of therapeutic alliance, the extent to which the client and patient worked closely together-- another potential mediator of therapy gain. Having established a timeline, the authors then investigated what had changed at session n, immediately before the sudden gain.

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Figure A shows the mean scores for cognitive change in the 29 patients for the pre-gain session, n minus 1, and the gain session, n plus, and the session following that, n plus 2. Between sessions n minus 1 and n plus, there was a marked mean increase in indicators of cognitive change, as measured by the PCCS, and is indicated by the arrow which stayed high for the n plus 2 session.

Graph B shows the corresponding mean levels of depression before and after the sudden gain.

Depression measured in the sessions was reporting on mood in the preceding week. Hence, the data is shown in the space between sessions on the graph.

What we see is that depression levels were high in the week leading up to the sudden gain session, and fell only subsequently in the following week, remaining low afterwards. We see further that therapeutic alliance also changed, but not until after the change in depression.

So we see, here, evidence that rapid cognitive change with CBT seemed to precede the marked symptomatic improvement, and that, in turn, preceded other aspects of therapy that may contribute to overall outcome.

This looks good for the cognitive model, and the role of cognition. However, nothing is as simple as it seems, as sudden gains are also commonly seen with other forms of psychotherapy, and even in patients being treated by medication.

So while sudden gain is a good predictor of outcome, it does not appear to be specific to CBT. Each treatment may be mediated by a different variables, or all of them may work on the same process, regardless of the treatment model.

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The evidence that we have been looking at is generally supportive of the idea that cognitive factors are important in depression, that they change with CBT, and that the order of change is consistent with them being a possible mediator, or even mechanism, of change. Given the central focus on cognition, it is reasonable to expect that cognitive therapy and CBT will be more effective at changing cognitions than other therapies, whether alternative psychotherapies or antidepressant medication.

Let's look at whether this expectation stands up to scrutiny by rem visiting the systematic review meta-analysis by Ioana Cristea and colleagues in 2015. If you recall, overall, CBT was more effective in changing dysfunctional thinking compared to all controlled conditions, with an effect size of 0.51.

However, what do we see if we look at specific comparisons between CBT and other active psychotherapies? Of the 26 studies included, 14 of them compared CBT with another therapy condition that did not have the same focus on cognition.

Was CBT more effective in this specific analysis? The answer was no. Across all 14 studies, there was no advantage for CBT, although a small difference was found in the five studies that used the DAS only to measure dysfunctional thinking.

A moderate advantage was also found on follow up data, although this was based on only five studies, and so may not be a reliable result. These days offer rather weak support for there being something specific about the mode of action in CBT in targeting cognition so deliberately.

What about the effect of medication compared to CBT? Even if there was a nonspecific effect of psychotherapy, would we expect medication to have as big an impact on cognitive change? While the prediction might be no, the evidence suggests that there is no difference, at least in the four studies reviewed.

It seems that CBT, and other psychotherapies, and even antidepressant medication, may all change aspects of negative thinking. Even if changing cognition is important for symptom change, it is far from clear that CBT is the best or only way to achieve it.

Slide 16
While all therapies may produce reductions in dysfunctional thinking, synchronised with overall clinical improvement, is CBT more effective in reducing the subsequent reactivity of such thinking

in response to the presence of negative mood? What happens to dysfunctional thinking in people who have recovered from depression following CBT, compared to those who were treated with medication?

This study by Segal and colleagues, in 1999, repeated the design of the Miranda and Person study, this time comparing the two treatment groups pre and post a negative mood induction using the Dysfunctional Attitude Scale. The DAS score of the two groups was the same before the mood induction.

To assess any differential effect of mood induction in the two groups, the study used regression procedures. The study does not report the means of the actual data. Instead, the figure, here, reflects the results of the regression model, indicating the change in DAS scores before and after the mood induction, related to the relative magnitude in negative mood increase following induction.

It shows that the medication-treated group showed a positive relationship, with a larger increase in dysfunctional thinking, with larger increases in negative mood. In the CBT-treated group, the opposite result was seen.

Interpreting this, we can say that dysfunctional thinking seems less likely to be reactivated by high levels of negative mood, if previously treated with CBT, compared to medication. A further interesting aspect of this study was a recontact with the participants, some years later, to see which of them had experienced a recurrence of their depression.

Combining the two treatment groups, they found that the level of dysfunctional thinking before the mood induction was not associated with the likelihood of recurrence. Instead, it was their level of dysfunctional thinking after the mood induction. The more reactive their thinking to low mood, the more likely they were to have experienced a subsequent episode of depression.

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