

Hope-based Psychoeducation Seminars

Objectives

1. To enhance understanding of the diagnosis and terminology (i.e. “negative symptoms”);
2. To enable a more active role and confidence in communication with health professionals (active monitoring/discussion of symptoms, side effects and options);
3. To enable a more active role and confidence in using health services;
4. To enhance understanding/monitoring/control of symptoms (psychotic, negative, depressive);
5. To reduce fear of the disorder (fear of being irreversibly “defective”, fear of degeneration, of lifetime chronicity, of inheritability);
6. To reduce self-stigma (maladaptive beliefs about the future);
7. To reduce passivity towards the disorder;
8. To reduce insecurity/avoidance of life-important goals/decisions such as getting engaged/married, having children, etc.;
9. To enhance understanding and confidence in pharmacotherapy;
10. To detach stigma from medication intake;
11. To shift focus from psychotic symptoms to depressive and anxiety symptomatology;
12. To reduce fear and a sense of guilt for being stressed or depressed;
13. To encourage very small lifestyle changes.

Session 1.

- A. Explanation of diagnostic symptoms and explanation of the concept of positive and negative symptoms, giving examples with the use of clinical terms. Obj.: 1, 2, 3, 4
- B. Giving examples of positive symptoms and management tips. Obj.: 4
- C. Discussing the three myths of psychosis: myth of violence, lifetime chronicity and the degenerative nature of the disorder. Obj.: 5, 6

D. Bringing in research evidence from systematic reviews for the outcome heterogeneity of schizophrenia, demonstrating with examples from studies the high chances for recovery and that recovery can be achieved in the face of several relapses, (e.g. AlAqeel & Mergolese, 2012; Harding, 1988; Zipursky, Reilly, & Murray, 2012). Obj.: 5, 6

Session 2.

A. Explaining the stress-vulnerability model. Demonstrate the difference between theoretical hypothesis and research evidence and explain that vulnerability is not necessarily genetic. Explain that genes associated with schizophrenia are also associated with other psychopathological disorders (e.g. Züchner, Roberts, Speer, & Beckham, 2007) and that the genetic linkages in schizophrenia currently seem not consistent across studies (e.g. DeLisi et al., 2002). Obj.: 5, 6, 7

B. Challenging the common belief that schizophrenia is highly heritable, bringing in evidence from research on parents' of patients with schizophrenia, siblings and twins and discuss how heritability is not necessarily genetic (e.g. Al-Jeshi, Epstein, & Zipursky, 2006; Hans, Auerbach, Styr, & Marcus, 2004; Tienari et al., 1987, Tienari et al., 2000; Tienari et al., 2003). Obj.: 5, 6, 7, 8

C. Explaining the difference between causality and correlation in studies about brain morphological architecture. Demonstrate research evidence for the morphological differences correlated with schizophrenia but also with anxiety-related disorders (e.g. Bowirrat et al., 2010; Czeh & Lucassen, 2007; DeQuardo et al., 1994) as well as with general population lifestyle habits (such as smoking cigarettes, sedentary lifestyle, consumption of alcohol), (e.g. Zipursky et al., 2012). Obj.: 5, 6, 7, 8

D. Bringing in evidence for the inconsistent results across studies for the neuro-developmental hypothesis and across studies about brain morphological architecture and discuss methodological problems (e.g. Brown, 1976; Morgan, 1997; Opler et al., 2013). Obj.: 4, 5, 7, 8

E. Explaining biochemical mechanisms. Demonstrate research findings for the effects of altered neurotransmission (serotonin, GABA, dopamine and norepinephrine) and explain how antipsychotic medication work. Specify with examples the relations of pharmacotherapy with psychotic symptoms' alleviation and the relations of pharmacotherapy with normalisation of stress, sleep and appetite, as well as the effects of sudden disruption (e.g. Bleich, Brown, Kahn, & van Praag, 1988; Gaur et al., 2008; Guillin, & Laruelle, 2005). Obj.: 2, 3, 9, 10

Session 3.

A. Bringing in research evidence for the comorbidity of depression and anxiety disorders with schizophrenia (e.g. Buckley, Miller, Lehrer, & Castle, 2009) and discuss their importance in schizophrenia relapse and in the disorder's course. Obj.: 11, 12

B. Explaining the vicious cycle between immune-inflammation, oxidative stress and depression and explain the role of exercise, diet and sleep on biological mechanisms, on anxiety and depression (e.g. Lopresti, Hood, & Drummonda, 2013). Obj.: 9, 11, 12, 13

C. Bringing in evidence from research that underlines the role of very small lifestyle changes (i.e. decrease in saturated fat consumption, small increase in everyday activities/small changes in amount of physical exercise, small increase in consumption of vegetables, wholegrain fibre and consumption of raw olive oil) on biological mechanisms (i.e. immuno-inflammation paths, hypothalamic-pituitary-adrenal axis, oxidative stress) (e.g. Lopresti et al., 2013). Obj.: 7, 9, 11, 12, 13

Session 4.

A. Recap of previous sessions with information that help reduce the sense of being physically different from other people and information for the benefit of using medication. Obj.: 10

B. Revising examples of positive symptoms and management tips. Obj.: 4

C. Explaining the term “early signs of relapse” with specific examples (increasing stress, increasing social isolation, increasing sleep/diet disturbance, increasing hygiene neglect, etc.). Bring in research for the role of early signs of relapse on the onset of acute episodes (e.g. Eisner et al., 2013). Obj.: 1, 2, 3, 10, 12

D. Give management tips (i.e. relaxation tips, anchor symptoms/asking for help, contact with the psychiatrist). Obj.: 2, 3, 10, 12

E. Explaining the importance of the role of maintenance of a daily routine/the role of keeping active, selecting a number of activities to be involved in daily. Obj.: 7, 12, 13