Psychotherapy for emerging borderline personality disorder

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The real authors of this presentation

- Prof Carla Sharp, U. Houston
- Prof Patrick Luyten, U. Leuven
- Prof Mario Speranza, U. Versailles
- Prof Michael Kaess, U. Heidelberg
- Dr Christel Hessels, GGz Centraal, NL
- Prof Martin Bohus, IoMH, Menheim

Two recent reviews:

- “Borderline personality disorder in adolescence: An expert research review with implications for clinical practice”, European Child and Adolescent Psychiatry, in press

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What we know about the treatment of emerging BPD

- DBT
- MBT
- ERT
- HYPE
- Pharmacotherapy
Pharmacotherapy

Very limited evidence

- A cautious stance towards medication must be adopted
- Medication should be restricted to the treatment of comorbid conditions

Two observational studies

- 8-week trial of 3mg flupenthixol showed benefits
  - Kutcher, 1995
- Reported benefits for methylphenidate on both BPD and ADHD in adolescents with comorbidity
  - Golubchik, 2008

However, given numerous risks and side-effects, it is strongly recommended to avoid medication with this population leaving us with psychosocial treatments
Are evidence based psychotherapies better than TAU?
We need to understand both disorder and treatment mechanisms to enhance treatment effectiveness.
There are several effective intervention for adults

Only few studies in interventions specific to adolescence

Missed opportunity!

Key period for intervention

Flexibility and malleability of personality traits: *synaptogenesis*

Prevention

Early treatment

Adolescent BPD *responds* to intervention

Kaess et al., 2014; Chanen & McCutcheon, 2013
RCTs of Treatments for Adolescent Suicide Attempters

- **Nine** randomized controlled trials (RCTs)
- **Group therapy** including both cognitive-behavioral and psychodynamic techniques (Wood, Trainor, Rothwell, Moore & Harrington, 2001)
  - *failed to be replicated* in two subsequent follow-up trials (Green et al., 2011; Hazell et al., 2009)
- **Multi-systemic therapy** (Huey et al., 2004) reduce hospitalisation
- **Mentalization-based treatment** (Rossouw & Fonagy, 2012);
- **Integrated CBT** for co-morbid alcohol abuse disorders and suicidal thoughts or behaviors (Esposito-Smythers, Spirito, Kahler, Hunt, & Monti, 2011).
- **Dialectic Behavior Therapy** (Mehun et al., 2014)
- Trials that did **not** yield significant **decreases in suicide attempts**
  - a green card **offering rapid**, no questions asked **hospital admission** if requested (Cotgrove, Zirinsky, Black & Weston, 1995)
  - brief home-based **problem solving intervention** (Harrington et al., 1998)
  - a skills-based approach targeting **problem-solving and affect management** (Donaldson, Spirito, & Esposito-Smythers, 2005)
  - a youth-nominated **support team** (plus a second trial using a slightly modified version of the approach; King et al., 2006, 2009).
Less intensive interventions

Emotion regulation training (ERT)

- Manualised group training
- Developed as add-on to TAU
- Utilises the structure of Systems Training for Emotional Predictability and Problem Solving (STEPPS)
- Complemented with DBT elements and CBT (van Gemer et al., 2009; Bartels, Crotty & Blum, 1997)
- Studies have not shown superiority over TAU (Schuppert et al., 2012)

Borderline Personality Disorder Severity Index (BPDSI-IV) mean values (total score) at baseline and after intervention
Dialectical Behavioural Therapy (DBT)

**General**

- Cognitive-behavioural therapy using **change** and **acceptance techniques** within a **dialectical framework**
- Originally developed **for chronic suicidal adults** with BPD (Miller et al., 1997)

**Adapted for adolescent populations**

- **Developmentally** appropriate themes
- Involves **families** and parents
- **Reduced length**
- **Reduced** number of **skills** taught
- Addition of an adolescent-specific skills module (Miller et al., 1997)

**Evidence**

- Meta-analysis found that DBT for BPD adolescents is **superior** than **TAU**:
  - Reductions in **hospitalizations**
  - **Attrition**
  - Behavioural **incidents** (McPherson et al., 2013)
- A recent **Norwegian RCT** combined DBT (brief, 19 weeks) with other interventions
  - Medium to **large ESs** compared to TAU for **suicidal ideation**, depression and BPD symptoms (Mehlum et al., 2014) maintained at **1 year**
Mentalization-Based Treatment (MBT)

General

- Based on psychodynamic psychotherapy and attachment theory
- Aims at the recovering of MZ to help patients regulate thoughts and feelings
- Aims at achieving functional interpersonal relationships (Bateman & Fonagy, 2010)

Adapted for adolescent populations

- MBT-A consists of weekly individual sessions for 12 months
- Combined with monthly MBT-F (families) sessions (Roussouw & Fonagy, 2012)

Evidence

- RCT on 73 BPD adolescents vs. TAU
  - MBT more effective in decreasing self-harm and depression
  - Positive changes were mediated by increase in ability to mentalize and decrease in attachment avoidance (Roussouw & Fonagy, 2012)
- Naturalistic pilot study showed the feasibility and effectiveness of inpatient MBT-A (N= 11 females)
  - Significant decrease in symptoms
  - Improvements in personality function and quality of life at 1 year of treatment (d= .58-1.46) (Laurensessen et al., 2014)
• Random allocation of young people presenting with self harm to either MBT or TAU
• N=80
• Assessments done every 3 months and at 12 months
• Assessment methods:
  – Risk taking and self harm: RTSHI (Vrouva, 2010)
  – Mood: MFQ (Angold, 1995)
  – BPD traits: BPFSC (Crick, 2005) and CH-BPD (Zanarini, 2007)
  – Dissociation: ADES (Armstrong, 1997)
  – Mentalization: HIF (Sandell, 2008)
  – Attachment: ECR (Brennan, 1998) and IPPA (Armsden, 1987)
## Demographics of sample

<table>
<thead>
<tr>
<th>Characteristics at Baseline</th>
<th>TAU</th>
<th>MBT</th>
<th>Test Statistic</th>
<th>(p=)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female, n/N (%)</td>
<td>35/40(87.5%)</td>
<td>33/40(82.5%)</td>
<td>(\chi^2(1)&lt;1)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Age, y, mean (SD)</td>
<td>14.8 (1.2)</td>
<td>15.4 (1.3)</td>
<td>(t(78)=2.01)</td>
<td>0.041</td>
</tr>
<tr>
<td>Chronicity of Self harming</td>
<td></td>
<td></td>
<td>(\chi^2(1)&lt;1)</td>
<td>n.s.</td>
</tr>
<tr>
<td>less than 3 months</td>
<td>16/40(40%)</td>
<td>16/40(40%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-5 months ago</td>
<td>4/40(10%)</td>
<td>7/40(17.5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-11 months ago</td>
<td>6/40(15%)</td>
<td>2/40(5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2 years ago</td>
<td>11/40(27.5%)</td>
<td>12/40(30%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>over 2 years ago</td>
<td>3/40(7.5%)</td>
<td>3/40(7.5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression (MFQ≥8), n/N (%)</td>
<td>38/40(95%)</td>
<td>39/40(98%)</td>
<td>(\chi^2(1)&lt;1)</td>
<td>n.s.</td>
</tr>
<tr>
<td>BPD (CI-BPD ≥5)</td>
<td>28/40(70%)</td>
<td>30/40(75%)</td>
<td>(\chi^2(1)&lt;1)</td>
<td>n.s.</td>
</tr>
</tbody>
</table>
Overall number of appointments

Group difference: $\beta=2.95$, 95% CI: -4.28, 10.17, $t(78)=0.81$, $p<0.419$, $d=0.18$
Self harm scores on the RSHI

Group differential rate of change: $\beta=-0.049$, 95% CI: -0.09, -0.02, $t(159)=-2.49$, $p<0.013$, $d=0.39$
Depression scores on the MFQ

Group differential rate of change: $\beta=-0.046$, 95% CI: -0.09, -0.01, $t(159)=-2.25$, $p<0.024$, $d=0.36$
Borderline personality features scores

Group differential rate of change: $\beta=-0.361$, 95% CI: -0.7, -0.03, $p<0.034$, $d=0.34$
Mentalizing scores on the HIFQ for treatment groups

Group differential rate of change: $\beta=1.49$, 95% CI: 0, 2.98, $t(159)=1.99$, $p<0.049$, $d=0.32$
Attachment avoidance scores from Experiences in Close Relationships Questionnaire for groups

Group differential rate of change: $\beta=-0.696$, 95% CI: -1.48, 0.08, $t(159)=-1.75$, $p<0.081$, $d=0.28$
FIGURE 2  Mediation of effect of mentalization-based treatment for self-harm in adolescents (MBT-A) on self-harm scores at the end of treatment. Note: Path coefficients (SE) are shown with the association of MBT-A on self-harm. The coefficient for the path controlling for specific indirect effect of Experience of Close Relationships Inventory (ECR) avoidance and How I Feel Questionnaire (HIF) change is shown in italics. *p < .05, **p < .01, ***p < .001.
NELFT Self-harm treatment study

Observed and Predicted Means for Moods & Feelings Questionnaire: Quadratic Model

Mean Moods and Feelings Q Score

Baseline | 3-months | 6-months | 9-months | 12-months | 18-months FU

Predicted TAU | Predicted Treatment | Observed TAU | Observed Treatment | 95% CI observed | 95% CI predicted

Adjusted for Age: Random Slope

Group differential rate of change: Beta=-0.838, 95% CI: -1.45, -0.23, t(437)=-2.69, p<0.0035, d=0.26
NELFT Self-harm treatment study

Observed and Predicted Means for selfharmr

Mean Selfharm Score

Baseline  3-months  6-months  9-months  12-months  18-months FU

Predicted TAU
Predicted Treatment
Observed TAU
Observed Treatment
95% CI observed
95% CI predicted

Adjusted for Age: Random Slope
Less intensive interventions

Helping Young People Early (HYPE)

- **Team based**, integrated intervention that includes (Chansen et al., 2009)
  - Assertive “psychologically informed” case management
  - **Active engagement** of families
  - **General psychiatric care** (assessment and treatment of comorbidities)
  - Community **outreach**
  - **Crisis team** and brief inpatient care
  - Access to a psychosocial recovery programme
  - Individual and group **supervision** of staff

- All elements organised within a psychotherapeutic framework of **Cognitive Analytic Therapy** (CAT), which obtains faster results than TAU (but not better) (Chanen et al., 2008)

- Focus on **problematic relationships** and their dysfunctional patterns

Evidence for all specialised interventions is still scarce

But it is possible to conclude that specialised early intervention for BPD is more effective than TAU
The effect CBT for depression across time 1977-2014
A meta-analysis by Johnsen & Friborg, 2015

K= 70 published studies
Within-group (pre-post) k=53
Between-groups with waiting list, k= 17
Average quality of studies (RCT-PQRS)= 28.4 (7.5)

N= 2,426
Average n(sd)= 34.6 (34.1)
Males= 30.9%
Patients with comorbidity= 43%

Average CBT sessions= 14.6 (5.12)
Mean baseline BDI= 26.1 (4.1)
Males= 30.9%
Patients with comorbidity= 43%

Patients in remission at post-treatment

57% of patients had remissions

Change in BDI scores at post-treatment

Average weighted effect size for BDI
$g = 1.58$ (1.43 – 1.74)

Change in HDRS scores at post-treatment

Average weighted effect size for HDRS
$g = 1.69$ (1.48 – 1.89)

WHY?

Study quality
Not related to reduction in treatment effects
$\beta = -0.0085$, $p = .45$
$Q_{df=1} = 0.002$, $p = .89$

Client related:
Age
Gender
Comorbidity
Medication
Severity
Associated problems

Therapist related:
Competency

Treatment related:
Number of sessions
Beck’s manual
Adherence check
Analysis method
Study quality

57% of patients had remissions

Number of patients in remission

Analysis method

ITT vs completers

Number of sessions

Use of Beck’s manual

Therapist’s adherence

Adherence check

Study quality

Effect size

Year
Is emerging BPD a valid and useful construct for clinicians?
Four key questions
Is emerging BPD a valid and useful construct?

- Is **reliable** and clinically **meaningful** diagnosis possible?
- Do we have a plausible understanding of the **disease mechanisms**?
- Are evidence-based **treatments** available?
- Can they be **disseminated** and implemented in different settings?
Leadership Skills in CAMHS: International Perspective
Summer School 17-21 August, London

- Unique overview of best CAMHS practice
- Cutting edge evidence, policy, outcomes, payments and user participation
- International perspective across different health and welfare systems
- Leadership in service planning, delivery and evaluation
From bench to consulting room

Science of mechanisms

Science of intervention development

Science of implementation
From bench to consulting room

Science of mechanisms

Science of intervention development

Science of implementation
Can BPD be diagnosed in adolescence?
Can BPD be diagnosed in adolescence?

- Almost $\frac{2}{3}$rd (63%) of British psychiatrists considered the diagnosis invalid when surveyed in 2009 (Griffiths, 2011)
- Concerns about stigma (BPD $\rightarrow$ IPD ?)
  - Intense persistent distress
- Difficult to distinguish BPD from ‘normal’ adolescent turmoil
- Incomplete personality development in this age group
Research articles on adolescent BPD 1990-2013

(Number of Publications)

(From Sharp and Tackett, 2014)
BPD in adolescence: prevalence studies

The disorder identifies a group of adolescents with high comorbidity and poor outcome. It predicts current psychopathology, psychosocial dysfunction, and negative longitudinal outcomes.

Similar prevalences to those found in adults:

- Community dwellers: 3%
- Outpatients: 11%
- Inpatients: 50%

Bernstein, 1993; Chanen et al., 2004; 2007; 2008; Crawford et al., 2001; Grilo et al., 1996; Kaess et al., 2014; Landelijk Kenniscentrum Kinder- en Jeugdpsychiatrie, 2011; Zanarini et al., 2003
Diagnosing BPD in adolescence

Main barrier: STIGMA

BPD is highly **stigmatised** among **professionals**

Associated to patient’s “**self-stigma**”

**Delays** beneficial interventions and education

Increases likelihood of inappropriate diagnosis and **iatrogenic** harm

**Duration** of psychiatric disorders is associated to **worse prognosis**

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Laurensen et al., 2013; Kaess, et al., 2014; Aviram et al., 2006; Rusch et al., 2006
Early detection of adolescent BPD: Instruments

Interviews
- Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II)
- ICD-10 International Personality Disorder Examination (IPDE)
- Childhood Interview for DSM-IV Borderline Personality Disorder (CI-BPD)
  - Multiples sources recommended
  - Most interviews still lack developmentally sensitive criteria

Self-reports
- BPD items of the SCID-II Pers. Questionnaire (SCID-II-PQ) (AUC: .84)
- Borderline Pers. Questionnaire (BPQ) (Specificity: .90; Sensitivity: .68)
- McLean Screening Instrument for BPD (MSI-BPD) (Spec: .66; Sens:.69)
- Borderline Personality Features Scale for Children (BPFS-C) and parents (BPFS-P) (Specificity: .84; Sensitivity: .85)
  - BPD should never be diagnosed only through questionnaires
BPD in adolescence as a reliable diagnosis

Personality disorder categories can be applied to children
- Maladaptive traits are pervasive and persistent (≥1 year)
- Considered unlikely to be limited to a developmental stage

Several National Health Guidelines include the diagnosis
- Britain (NICE, 2009)
- Australia (NHMRC, 2013)
- Germany (Bohus et al., 2008)
- The Netherlands (Landelijke Stuurgroep Multidisciplinaire Richtlijnontwikkeling in de GGZ, 2008; Landelijk Kenniscentrum Kinder- en Jeugdpsychiatrie, 2011)

There has been a five-fold increase in empirical studies for BPD in adolescents in the last 10 years (Sharp & Tackett, 2014)

Reliability and validity of BPD diagnosis in adolescence is comparable to that in adulthood (Chanen et al., 2008; Kaess et al., 2014; Miller et al., 2008; Westen et al., 2014)
Best nominated symptom identifiers for BPD in adolescence and early childhood markers of vulnerability

**Core diagnostic features**

- **Identity disturbance** (girls++)
- **Inappropriate anger**
- **Paranoid ideation** (boys++)
- **Chronic feelings of emptiness** (Self-harm, dissociation)
- **hostility**

**Childhood disorder markers**

- Attention deficit/hyperactivity disorder
- Oppositional defiant disorder

**Childhood behaviour markers**

- Controlling and coercive behavior towards attachment figures
- Poorly defined sense of self
- Hostile, distrustful view of the world
- Affective instability
- Relational aggression
- Intense outbursts of anger

Fossati, 2014
BPD in adolescents and adults

**Adolescents**

More likely to present with: ‘acute’ (executive) symptoms of BPD:
- Recurrent self-harm & suicidal behaviour
- Other impulsive & self-damaging behaviours
- Inappropriate anger

**Common to both**

- High rejection sensitivity
- Difficulties with trust & cooperation
- Shame proneness
- Negative self- and body perception
- Intermittent hostility

**Adults**

More likely to present with: enduring characteristic symptoms:
- Unstable relationships
- Identity disturbances

**Heterotypic continuity:** a developmental process of continuing and consistent impairment with changing manifestations
Conceptualizing BPD from a **dimensional**, rather than a **categorical**, approach is particularly pertinent in adolescents, as a dimensional approach may better account for the **developmental variability** and **heterogeneity** observed during this age period.

**Section 3:** Dimensional model of personality pathology

- Impairments in self
- Difficulties in relatedness

A sensitive and precise diagnosis could be achieved by **combining** both approaches.

**Dimensional – Categorical**
DSM-5: BPD in adolescence

DSM-5 maintains the historical caution to attribute personality problems to an adolescent only in "relatively unusual circumstances" (APA, 2013; p. 647)

Criteria A
Judgment of severity of problems in
- identity
- self-direction
- empathy
- intimacy

Criteria B
4 or more of
- emotional lability
- anxiousness
- separation insecurity
- depressivity
- impulsivity
- risk taking
- hostility

ICD 11 has legitimised the diagnosis
Relation of BPD to NSSI and suicidal behavior disorder (DSM-5 section 3)

Non-suicidal self injury (NSSI) disorder

BPD diagnosis possible if NSSI is repetitive

Greater likelihood of BPD diagnosis if adolescents report both NSSI and suicide attempts

Suicidal behaviour disorder

BPD diagnosis possible

Descriptive diagnoses of pure behaviours or symptoms may detract from important underlying psychopathological factors (e.g. dimensional features of personality pathology) and prevent specific interventions
Stability and course of BPD: A summary

- **Categorical stability** of BPD is **modest** in both adolescents and adults
- **Dimensional stability** is **moderate**
- BPD symptoms usually **appear in adolescence, peak in early adulthood**, then **decline**
- Some individuals **do not** experience age-related **decline** of symptoms
- While **impulsive** symptoms **reduce** over time, **affective symptoms** are more likely to **persist**
- Need to **distinguish** acute mental **states** from **traits** that indicate a more general pattern of maladaptive & dysfunctional behaviours
- **Remission from** categorical **diagnosis** does **not** imply remitted patients are **healthy**
Comorbidity

High psychiatric comorbidity and low psychosocial functioning

- Significant percentage of BPD adolescents meet criteria for externalising problems relative to other inpatients
  - ADHD
  - Oppositional disorder
  - Conduct disorder

- Substance-related disorders

- Internalising disorders
  - Mood disorders
  - OCD
  - PTSD
  - Separation anxiety
  - Social phobia

- Up to 60% of BPD adolescents have complex comorbidity
  - Confluence of internalising and externalising disorders
    - e.g. having any mood or anxiety disorders plus a disorder of impulsivity

Disruptive behaviour disorders and depressive symptoms in childhood predict adolescent BPD diagnosis

Ha et al., 2014; Eaton, 2011

Stepp, 2012
Comorbidity

High psychiatric comorbidity and low psychosocial functioning

Comorbidity in adolescent inpatients

<table>
<thead>
<tr>
<th>Disorder</th>
<th>BPD</th>
<th>Non-BPD psychiatric controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mood disorders</td>
<td>70.60%</td>
<td>39.20%</td>
</tr>
<tr>
<td>Anxiety disorders</td>
<td>67.30%</td>
<td>45.50%</td>
</tr>
<tr>
<td>Externalising disorders</td>
<td>60.20%</td>
<td>34.40%</td>
</tr>
</tbody>
</table>

Ha, Balderas, Zanarini, Oldham & Sharp, 2014
Complex comorbidity of BPD in adolescence

Externalizing problems: ADHD, oppositional & conduct disorders

Internalizing problems: Mood & anxiety disorders

Increased likelihood of BPD diagnosis

High levels of both internalizing and externalizing problems may indicate possible BPD in adolescents and warrant specific diagnostic assessment.
What we know about the mechanisms of BPD in adolescence
What we know about the mechanisms of BPD in adolescence

- Genetics
- Neuroimaging
- Neurobiology
- Environmental factors
- Psychological mechanisms
Are core impairments in BPD intrinsically related?

- 3 recent large family twin studies suggest a common pathway model with one highly heritable general BPD factor
  - Distel et al, 2010; Gunderson et al, 2011; Reichborn-Kjennerud et al, 2013

- Factor analytic studies in adolescents suggest that BPD in adolescence is best represented by a single hierarchical superordinate factor
  - Sharp et al, 2012; Michonski et al, 2013
Mechanisms of BPD in adolescents

Recent twin studies suggest a common pathway to BPD with one highly heritable general BPD factor. BPD in adolescence is best represented by a single hierarchical superordinate factor.

Genetics

- Heritability of 40-50% in adults
- No specific gene has been associated to BPD
- Adolescents (9-15 y.o.) who carry the s-allele of the 5-HTTLPR have higher levels of BPD
- History of maltreatment predicted BPD features at age 12 for those young people with family history of psychopathology

Environment

- Key factors associated to BPD: abuse and neglect, problematic family environment, and low SES
- Maltreatment increases likelihood of BPD (adj OR: 7.7)
- Low SES is a totally independent predictor of BPD
  - Countries with larger income inequality have greater prevalence of BPD and associated problems
- Attachment problems are strong predictors
  - Maternal withdrawal at 18 months predicts BPD in late adolescence
  - Early adversity, disorganised attachment and parental hostility predict BPD features in middle childhood, adolescence and adulthood
- Peer to peer abuse (bullying, e-bullying, peer rejection, teen dating violence, chronic exclusion)

Fonagy, Speranza, Luyten, Kaess, Hessels, Bohus, submitted
GxE interaction predisposing to BPD: A vicious cycle

Gene polymorphisms (5-HTTLPR, DAT-1)

Genetic/constitutional vulnerability

Heritability 42-60%

Environmental adversity

Social exclusion, early maternal separation

Abuse, neglect, maladaptive parenting, peer bullying
Antecedents and co-morbidities of BPD related characteristics in 12 year old children (Belsky et al., 2012): Age 5 ToM

Figure 1. Psychiatric Antecedents and Comorbidities of Borderline Personality Related Characteristics in 12 Year Old Children

<table>
<thead>
<tr>
<th>Child Characteristics</th>
<th>Correlations (Pearson’s r) Between Child Characteristics and Borderline Personality Related Characteristics:</th>
<th>Means and 95% Confidence Intervals (a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive Functioning (5 yrs)</td>
<td>r</td>
<td>95% CI</td>
</tr>
<tr>
<td>IQ</td>
<td>-0.11***</td>
<td>(-0.16, -0.06)</td>
</tr>
<tr>
<td>Executive Function</td>
<td>-0.05*</td>
<td>(-1.11, -1.00)</td>
</tr>
<tr>
<td>Theory of Mind</td>
<td>-0.11***</td>
<td>(-0.16, -0.07)</td>
</tr>
<tr>
<td>Behavioral and Affective Probs (5 yrs)</td>
<td>Interviewer Rating of Temperament</td>
<td>Lack of Control</td>
</tr>
<tr>
<td>Lack of Control</td>
<td>0.10***</td>
<td>(0.04, 0.15)</td>
</tr>
<tr>
<td>Approach</td>
<td>0.01</td>
<td>(-0.04, 0.06)</td>
</tr>
<tr>
<td>Inhibition</td>
<td>-0.01</td>
<td>(-0.07, 0.04)</td>
</tr>
<tr>
<td>Mother &amp; Teacher Rating of Impulsivity, Behavioral &amp; Emotional Problems</td>
<td>Impulsivity</td>
<td>(Mother Rating)</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>0.34***</td>
<td>(0.29, 0.38)</td>
</tr>
<tr>
<td>(Teacher Rating)</td>
<td>0.22***</td>
<td>(0.16, 0.28)</td>
</tr>
<tr>
<td>Externalizing Problems</td>
<td>(Mother Rating)</td>
<td>r</td>
</tr>
<tr>
<td>Externalizing Problems</td>
<td>0.44***</td>
<td>(0.38, 0.49)</td>
</tr>
<tr>
<td>(Teacher Rating)</td>
<td>0.24***</td>
<td>(0.17, 0.30)</td>
</tr>
</tbody>
</table>
Interaction between family history of psychiatric illness and history of maltreatment on BPD symptoms

Analysis of Extreme Borderline Group Membership*

<table>
<thead>
<tr>
<th>Extreme Borderline Group Prevalence</th>
<th>Positive Family History</th>
<th>No Family History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maltreatment</td>
<td>42%</td>
<td>8%</td>
</tr>
<tr>
<td>No Maltreatment</td>
<td>7%</td>
<td>3%</td>
</tr>
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</table>

N=2,119

<table>
<thead>
<tr>
<th>Extreme Group</th>
<th>Comparison Children</th>
<th>RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>++</td>
<td>20</td>
<td>28</td>
</tr>
<tr>
<td>+-</td>
<td>48</td>
<td>562</td>
</tr>
<tr>
<td>-+</td>
<td>3</td>
<td>42</td>
</tr>
<tr>
<td>--</td>
<td>44</td>
<td>1,372</td>
</tr>
</tbody>
</table>

Departure from Additivity = 9.73  95% CI (1.90 , 15.73)

Interaction between family history of mental illness and maternal negativity

**Panel A. Analysis of Dimensional Borderline Personality Related Characteristics Scale Score**

<table>
<thead>
<tr>
<th>Model</th>
<th>Maternal Negative Expressed Emotion</th>
<th>Family History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test of Diathesis-Stress Interaction, Between Families</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.</td>
<td>2.05 (0.000)</td>
<td>1.41 (0.000)</td>
</tr>
<tr>
<td>II.</td>
<td>1.71 (0.000)</td>
<td>0.03 (0.474)</td>
</tr>
<tr>
<td>Test of Diathesis-Stress Interaction, Within Families</td>
<td></td>
<td></td>
</tr>
<tr>
<td>III.</td>
<td>1.45 (0.000)</td>
<td>--</td>
</tr>
<tr>
<td>IV.</td>
<td>1.18 (0.000)</td>
<td>--</td>
</tr>
</tbody>
</table>

II. Between Families Interaction Beta=0.92 p<0.001
IV. Within Families Interaction Beta=0.92 p<0.001

Mechanisms of BPD in adolescents

**Neurologic**

- Reduced volumes of **amygdala, hippocampus, OFC and ACC** in adults
  - Key areas for *emotion regulation* and social *information* processing
  - Average *decrease* in size of 11% for the **hippocampus** and 13% for the **amygdala**
  - These *results* are *contradictory* in *adolescence*, but **ACC** and **OFC** volume reductions are

- Hypothalamic-pituitary-adrenal (HPA) axis (maladaptive stress response in the development of the disorder in the presence of trauma history)

- Dysfunctions in hypothalamic-pituitary-adrenal (HPA) axis (maladaptive stress response in the development of the disorder in the presence of trauma history)

- BPD adolescents present decreased *fractional anisotropy* in the fornix and inferior longitudinal fasciculus
  - Reflect reduced fibre density, *axonal diameter*, and *myelination* in white matter
  - Not found in adults, suggest a transient impairment of a developing BPD

- Amygdala hyper responsive, as in adults, associated to repeated NSSI

- Dysfunctions in hypothalamic-pituitary-adrenal (HPA) axis (maladaptive stress response in the development of the disorder in the presence of trauma history)
GxE interaction at the neurological level?

- Reduced volumes of left ACC and right OFC
- Atypical hippocampal asymmetry
- Unclear if brain abnormalities in adolescents with BPD reflect trauma or general vulnerability for psychopathology
- Decreased fractional anisotropy in fornix, inferior longitudinal fasciculus
- Reduced volumes of amygdala and hippocampus in some studies
The challenges of adolescence

Prefrontal cortex and STC undergoing major structural reorganization

Amygdala hyperactivity (Monk et al, 2003)
The challenges of adolescence
The challenges of adolescence

Developing social network of friendships and romantic relationships
Since 1986 activity of YP with their families decreased, in favour of activities with peers.

Low socioeconomic status is an independent risk factor for adolescent BPD.

New educational challenges and competition.

Bullying, peer rejection experiences.

Adolescent with BPD are more vulnerable to media influence.

The evolutionary advantage of being able to adapt to hostile environments in infancy (e.g. maltreated children become more sensitive to threats) could generate chronic epistemic hypervigilance.

Mechanisms of BPD in adolescents

Psychological

- BPD patients present heightened affective instability compared to controls
  - Not exclusive of BPD: also found in PTSD and Binge Eating

- Social emotions are central for BPD
  - Shame, disgust, fear of social rejection
  - May give rise to marked dissociative symptoms
  - Dissociative symptoms, in turn, are related to hypoalgesia

- Rejection sensitivity, provocation of aggressive behaviour, inability to become involved in trustful and cooperative behaviour

- Impairments in mentalizing
  - Mentalizing brain areas undergo massive synaptogenesis during adolescence
  - Characterised by hypermentalizing: excessive or overinterpretative

Fonagy, Speranza, Luyten, Kaess, Hessels, Bohus, submitted
Diathesis-stress approaches

Linehan and cols (1993; 2009)
- Trait vulnerability
  - Sensitivity-reactivity
  - Impulsivity
- Aberrant socialisation mechanisms in the family
  - Acquisition of poor emotion regulation skills

Fonagy and cols (2000; 2009)
- Heritability
  - Innate ToM
  - Sensitive temperament
- Early attachment experiences
  - Development of social cognition:
    - MENTALIZATION (hypermentalizing)

BPD
Inside Out: A Major eMotion Picture from PIXAR
Social cognition in BPD

Facial emotion recognition

- **Hypersensitivity** to subtle facial cues of negative emotions
- Increased arousal that impairs recognition of overt emotions
- **Faster eye movements to** the eyes of **negative faces**
  - Enhanced *amygdala* activation
  - Reduced by administration of *oxytocin*

Trust appraisal and rejection sensitivity

- **Neutral** faces are **less trustworthy**
  - NOT reduced by administration of *oxytocin*

Cognitive empathy

- **Impaired ToM** – impaired perspective taking
- Enhanced performance in **RME**
  - It does not require explicit meta-representation of the other’s mind
- **Lower activation** of theory of **mind brain circuit**
  - Even during enhanced performance at RME

Affective empathy

- Automatic (**unconscious**) imitation of **negative expressions**
  - Enhanced right-mid insular activity (self-origin of emotions)
  - Reduced anterior insula (other-origin of emotions)
Correlation Between Movie for the Assessment of Social Cognition (MASC) and Borderline Personality Features Scale for Children

Hypermentalizing leads to emotion disregulation which leads to borderline personality features (Sharp et al., 2011, J.Am. Acad. Child. Adol. Psychiat., 60, 563-573.)

\[ 0.42^{***} (0.19^{*}) \]

\[ \text{Hypermentalizing (MASC)} \rightarrow 0.27^{*} \rightarrow \text{Emotion Regulation (DERS)} \rightarrow 0.75^{**} (0.69^{**}) \rightarrow \text{BPD (BPFSC)} \]

*p < .05, **p < .01, ***p < .001

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>R²</th>
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<tr>
<td>DERS</td>
<td>.375</td>
<td>.036</td>
<td>.686**</td>
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</table>
Attachment and adolescent BPD (Sharp et al. submitted)

- **Attachment representations** of adolescents with emergent PD
  - **Rejection** and abandonment
  - **Incoherent** and disorganized representations of close relationships

- **Moderate continuity** from childhood to adulthood
  - **Long lasting** effects on developing relationships (increasingly important in transition to adulthood)

- **Increased demand** on capacities for attachment may overwhelm some youths as they negotiate **new intimate relationships** ➔ peaking of PD symptoms goes beyond parental influence
The effect of attachment-related stress on the capacity to mentalize (Nolte, Hudac, Mayes, Fonagy & Pelphrey, 2013)

Subjects to make **two types of judgments** in three conditions:
- **Attachment** stress story related to their personal history,
- **General** stressful memory (e.g. exam) and
- **No stress**.

**Stimuli & Design**

Examples of single trial stimuli, RMET (top), control task (bottom).

*Frontiers in Human Neuroscience, 7, Article 816*
Regions that showed differential activation between mental state and age judgments in the baseline RMET-R that were modulated by stress induction type. (Attachment related stress versus general stress) Nolte et al. (2013)

Attachment Stress Induction resulted in reduced mentalization-related activation in the left posterior superior temporal sulcus (STS), left inferior frontal gyrus and left temporoparietal junction (TPJ).
The Menninger Study of Adolescent Personality Disorder

- N = 259 (mean age 15.42, SD = 1.43)
- 63.1% females
- 31% (n = 80) met criteria for BPD

**Measures**

- **Child Attachment Interview** (Target et al., 2007) – Coherence scale
- **Movie Assessment of Social Cognition** (Dziobek et al., 2006)
- **Difficulties in Emotion Regulation Scale** (Gratz & Roemer, 2004)
- **Borderline Personality Disorder Features Scale** (Crick et al., 2005)
Multi-mediational model

Attachment Security

Emotion Dysregulation

Hypermentalizing

Borderline Features

-0.315*
-1.742
.617**
.352***
-.156
Conclusions

• **Attachment stress** specifically derails mentalizing judgments (Nolte et al., 2013)

• **Attachment schemas** predict mentalizing in adolescence (see e.g. Dykas & Cassidy, 2011; Sharp, Fonagy, & Allen, 2012).

• Potentiating affect **attachment insecurity** in **derailing** the development of **optimal mentalizing** capacity is proposed.

• **MZ and ER compete** in a mediational model.
**CONTEXTUAL RISK AND VULNERABILITY FACTORS**
- Sexual and physical abuse
- Maladaptive parenting (maternal inconsistency; over-involvement)
- Peer victimization experiences
- Attachment disorganization

**GENOTYPE** (Distal)
- Heritability 42-60%
- Polygenetic
- Gene–environment interactions
- Polymorphisms in serotonin transporter gene (5-HTTLPR)
- Polymorphisms in dopamine transporter gene (DAT1)

**NEUROBIOLOGICAL ENDOPHENOTYPE** (Proximal)
- Frontolimbic networks in adults
- No fMRI in youth
- Reduced OFC volumes
- Decreased ACC volume
- Attenuated cortisol responses
- Oxytocin abnormality

**INTERMEDIATE ENDOPHENOTYPE** (Proximal)
- Negative social-cognitive bias
- Social perspective coordination
- Hypermentalizing
- Impulsivity
- Emotion dysregulation

**BPD PHENOTYPE**
- Anger
- Affective instability
- Emptiness
- Identity diffusion
- Paranoia/ Dissociation
- Abandonment fears
- Self-harm/suicide
- Impulsivity
- Interpersonal

**CONTEXTUAL PROTECTIVE FACTORS**
- Secure attachment; relatedness

Etiological factors in the development of adolescent BPD.
Resilience and BPD: A developmental view
Life-course structure to psychopathology

Need for longitudinal research designs

• **Extant research** on structure of psychopathology focuses on individuals who report **symptoms within** a specified **period**
  – Biggest puzzle is why people change clinical presentations over time (adolescent conduct problem adult depression)

• **Mixing single-episode**, one-off cases **with recurrent** and chronic cases which differ in:
  • **extent** of their **comorbid** conditions
  • the **severity** of their conditions
  • **etiology** of their conditions.

• Some individuals more **prone to persistent psychopathology**.
Bi-factor model with the item-loadings

Community-based sample aged 11-14 years (N=23,477)

### Logistic regression predicting future caseness

<table>
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<tr>
<th>Predictor</th>
<th>B</th>
<th>Wald Chi-square</th>
<th>Odds-ratio</th>
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<td>76.4</td>
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<td><strong>Bi-factor model</strong></td>
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<tr>
<td>Externalising</td>
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<tr>
<td>P-Factor</td>
<td>2.33***</td>
<td>479.01</td>
<td>10.30</td>
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</table>

N=10,270
BPD as the ‘g/P-factor’ of personality pathology (Sharp et al 2015)

- Evaluated a **bifactor model** of PD pathology in which a **general (g) factor** and several **specific (s) factors** of personality pathology account for the covariance among PD criteria

- **966 inpatients** were interviewed for 6 DSM–IV PDs using **SCID-II**

- Confirmatory analysis **replicated DSM-IV PDs**, with high factor correlations
P factor in PDs: the DSM factor structure

Sharp et al., 2015 *Journal of abnormal psychology*

N=966 inpatients

**BPD**
- Avoids abandonment
- Interpersonal Instability
- Identity disturbance
- Self-harming impulsivity
- Suicidality
- Affective instability
- Emptiness
- Intense anger
- Transient dissociation

**AVPD**
- Avoids social work

**OCPD**
- Orderly
- Perfectionistic

**SZTPD**
- Ideas of reference
- Odd beliefs

**NPD**
- Grandiose
- Preoccupied with fantasies

**ASPD**
- Failure to conform
- Deceitfulness
- Impulsivity
- Irritable, aggressive
- Disregard for safety
- Irresponsible
- Lacks empathy
- Envious
- Arrogant
- Lacks remorse

**UNACCEPTABLE MODEL FIT**

Comparative Fit Index (CFI) <95
Tucker-Lewis Index (TLI) <95

Sharp et al., 2015 *Journal of abnormal psychology*
### P factor in PDs: the DSM factor structure

<table>
<thead>
<tr>
<th></th>
<th>BPD</th>
<th>AVPD</th>
<th>OCPD</th>
<th>SZTPD</th>
<th>NPD</th>
<th>ASPD</th>
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<td>-</td>
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<td>.31</td>
<td>.04</td>
<td>.16</td>
<td>.56</td>
<td>-</td>
</tr>
</tbody>
</table>

In spite of internal coherence at a criterion level, DSM personality disorders, within individuals, are not neatly separable. They are not discrete phenomena.

Sharp et al., 2015 *Journal of abnormal psychology*
P factor in PDs: does EFA replicate the DSM factor structure?

Excellent model fit:

\[ \chi^2(897) = 1110.58, \ p < .001 \]

RMSEA = .02 [0.01, 0.02], \ p = 1

CFI = .97

TLI = .97

N=966 inpatients

Sharp et al., 2015 Journal of abnormal psychology
Excellent model fit:
\[ \chi^2_{(897)} = 1030.09, \ p < .001 \]
RMSEA = .02 [.01, .02], \( p = 1 \)
CFI = .98
TLI = .97

Only factor loadings >|30| are shown

Sharp et al., 2015 *Journal of abnormal psychology*
The ‘P’ Factor (Caspi et al., 2013)

- Ungendered chronic Psychotic conditions
- Partially gendered Personality disorder
- Gendered ‘Neurotic’ conditions

Impairment

Externalizing

Internalizing

Female

Male

Gendered Style

Persistence
Understanding the ‘P’ or ‘g’ factor as an absence of expected resilience
From disease- to health-oriented research: A paradigm shift
Formerly: Investigating the mechanisms that lead to stress-related illness
Now: Investigating the mechanisms that protect against illness
Basic assumption of resilience research: Resilience is not simply due to an absence of disease processes but reflects the work of active adaptation mechanisms with a biological basis.

(Kalisch et al)
Active refers to any resource demanding process and may apply to cognitive as well as behavioral processes (Kalisch et al., in press)
Resilience has been conceptualised variously as a...
The ability of a system to resist dynamically a perturbation or adverse condition that challenges the integrity of its normal operation and to preserve function as a result in reference to some initial design or normative functional standards.
Bringing order to the conceptual chaos

Factors
- Social support
- Social status
- Personality
- Life history
- Coping style
- Genetic background
- Brain function

May overlap conceptually and/or interact statistically

Mediating mechanisms
- Psychological
- Biological

Outcome

RESILIENCE
What is it that patients with BPD lack?

- Individuals with intense persistent distress (high ‘P’ scorers) are by definition not resilient:
- They are oversensitive to possibly difficult social interactions (they cannot interpret the reasons for other’s actions reliably)
- Cannot set aside (put out of their mind) potentially upsetting memories of experiences leaving them vulnerable to emotional storms
How appraisal shapes our experience

Not

Enough

Except our experience is social: not with physical objects but with people
The type, quality and extent of emotional reactions (including stress reactions) are not determined by simple fixed stimulus-response relationships…

The process underlying resilience is driven by top-down cognition
Appraisal (higher order cognition) theory

Stimulus

Mental representation

Higher order cognition

Emotional response

...but by context-dependent evaluation of motivational relevance
Brains can preserve core aspects of the functional architecture of information processing that sustains higher order cognition in spite of substantial structural damage (Rudrauf, 2014, Advances in Neuroscience). Full AD diagnosed postmortem in 25%-67% of elderly with no prior cognitive impairment (Dubois et al., 2012).

“Higher-order cognition” unites in a functionally integrated subjective frame:
- executive functions
- attention,
- self-awareness
Positive appraisal style theory of resilience (PASTOR)

Factors | Mechanism | Outcome
---|---|---
F₁ | 1. Positive appraisal style | Resilience
F₂ | 2. Positive reappraisal |
F₃ | 3. Interference inhibition |
F₄ |
F₅ |
F₆ |
F₇ |
F₈ |
‘P’ Factor

Resilience
‘P’ Factor

Resilience

Normal/neurotic
‘P’ Factor

Resilience

BPD
Can we draw these constructs into a unifying conceptualisation?
‘P’ Factor

Resilience

Can we draw these constructs into a unifying conceptualisation?
The current bio-psycho-social MZ model of BPD as an absence of resistance to social stress

- The ‘P’ factor of general vulnerability to psychopathology is actually an indication of the absence of resilience (psychological equivalent of immune system response, Higgitt & Fonagy, 1992)
  - The nature of the stressor (abuse, bullying, neglect, maltreatment or everyday social stress) is not relevant
  - Most toxic stressors attack the mechanisms of resilience
- While patients with ‘neurotic’ problems (regardless of severity) have high resilience (unlikely to be effected by subsequent stressors) those with BPD have low resilience and are likely to succumb to psychosocial stress
The current bio-psycho-social MZ model of BPD as an absence of resistance to social stress

- ‘P’ and ‘R’ are inversely related because they are identical at the level of mechanisms
  - Low ‘R’ reflects an adaptation consequent on serial communication problems in development combined with genetic vulnerability characterized by epistemic hypervigilance which prevents or undermines a reappraisal process and results in apparent rigidity (imperviousness to social influence)
  - The failure to engage in meaningful reappraisal creates a general vulnerability to psychosocial stress (low ‘R’) which yields to the high prediction of future psychopathology from ‘P’
  - Increasing mentalizing increases epistemic trust which in turn generates resilience through improved capacity for appraising and re-appraising stressful events
Being mentalized in the context of an attachment relationship

EPISTEMIC TRUST

Ability to form and learn from social connections
Ability to reappraise via mentalizing where necessary to repair, preserve, develop and increase these connections throughout life
The nature of psychopathology in PD

- Social adversity (most deeply trauma following neglect) is the destruction of trust in social knowledge of all kinds → rigidity, being hard to reach

- Cannot change because cannot accept new information as relevant (to generalize) to other social contexts

- Personality disorder is not disorder of personality but inaccessibility to cultural communication relevant to self from social context
  - Partner
  - Therapist
  - Teacher
  }
  }
  }
  Epistemic Mistrust
Judgment bias for approachability and trustworthiness of faces.

Nicol et al., 2013 *Plos One*

**P < .001**

Direction of bias

**Approachable as Unapproachable**

**Unapproachable as Approachable**

**Trustworthy as Untrustworthy**

**Untrustworthy as Trustworthy**
Epistemic mistrust not believing what one is told

- It is the consequence of **high levels of epistemic vigilance** (the **over-interpretation** of motives and a possible consequence of **hyper-mentalization**, Sharp et al., 2011)
- The recipient of a communication assumes that the communicator’s **intentions are other than those declared** and therefore not treating the communication deferentially
- Mostly it consists of **misattribution of intention** and seeing the reason’s for someone’s actions as malevolent and to be treated with **epistemic hyper vigilance**
- Most important consequence is that the **regular process of modifying stable beliefs** about the world (oneself in relation to others) remains closed
Implications: The nature of psychopathology

- Epistemic mistrust which can follow perceived experiences of maltreatment or abuse leads to epistemic hunger combined with mistrust
  - Therapists ignore this knowledge at their peril
- Personality disorder is a failure of communication
  - It is not a failure of the individual but a failure of learning relationships (patient is ‘hard to reach’)
  - It is associated with an unbearable sense of isolation in the patient generated by epistemic mistrust
  - Our inability to communicate with patient causes frustration in us and a tendency to blame the victim
  - We feel they are not listening but actually it is that they find it hard to trust the truth of what they hear
Epistemic hypervigilance / High ‘P’ factor/ absence of expected resilience

Epistemic trust / Resilience/ low ‘P’ factor
Building a social network in adolescence
When the capacity to form bonds of trust is shaky and tends to break down...
...we lose our safety net
Reconceptualising BPD: understanding not in terms of disease mechanisms...
...but as an absence of expected resilience or lack of epistemic trust...
...which was once adaptive
Can’t show differences too easily
If therapies worked the way indicated some should work better than others
Can we do any better than agreeing with the Do Do Bird?

“Everybody has won, and all must have prizes.”
What happens when you ask a room of psychotherapists whose approach is the most effective?

What can be done to end this unseemly behaviour?
The DoDo bird sounds like a pigeon

If we can’t do better than say everything works than my career as a treatment developer is over and I might as well turn into a DoDo bird!
Oh dear! Better come up with an answer quick!
The paradigmatic common factor is...

“Can we pull a rabbit out of a hat here?”
All together now…

Mentalize!
Cognitive Behaviourism: The value of understanding the relationship between my thoughts and feelings and my behaviour.

Systems Theory: The value of understanding the relationship between the thoughts and feelings of family members and their behaviours, and the impact of these on each other.

Psychodynamic: The value of understanding the nature of resistance to therapy, and the dynamics here-and-now in the therapeutic relationship.

BIOLOGICAL, SOCIAL and ECOLOGICAL: The value of understanding the impact of context upon mental states: development, deprivation, opportunity, hunger, fear...
How do you think your audience might be feeling right now?

Bored

Sleepy

Is it time for coffee yet?

Fonagy should write a new talk
Therapists listening to an account of mentalizing as the effective component of all therapies
Time for a change?

What?? You didn’t like the mentalizing rabbit???
Do EBPs outperform TAU?

Common factors in successful treatment of BPD

1. extensive effort to maintain engagement in treatment (validation in conjunction with emphasis on the need to address behaviors that interfere with therapy)

2. a valid (evidence-based) model of pathology that is explained and feels relevant to the patient

3. an active therapist stance—that is, an explicit intent to validate and demonstrate empathy and generate a strong attachment relationship

4. the reinforcement of epistemic trust (Sperber et al., 2010)—that is, facilitating a belief in the possibility that something can be learned in therapy
Do EBPs outperform TAU?

Common factors in successful treatment of BPD

5. focus on **emotion processing** and the connection between **action and feeling** (e.g., suicidal ideation is associated with abandonment feelings)

6. inquiry into patients’ **mental states** (behavioral analysis, clarification, confrontation)

7. **a structure** that provides increased **activity, proactivity, and self-agency** (that is, the therapist avoids the expert stance and rather “sits side by side” with the adolescent in a partnership)

8. the structure is **manualized** and **adherence** to the manual is **monitored**
Common factors in successful treatment of BPD

9. method of therapy can be taught as part of a relatively brief training programme

10. both therapist and adolescent must feel a commitment to the approach

11. supervision is essential to identify deviation from the manualized structure and provide support for adherence
Do EBPs outperform TAU?

EFFECTIVE TREATMENTS FOR BPD ARE RICH IN THE FOUR ‘C’S

1. **Coherence**: offering a coherent (understandable) approach to illness and cure that provides the patient with hope

2. **Consistency**: identifying a well-balanced set of interventions based on the theory of disorder & its cure

3. **Continuity**: adherence to model throughout the treatment, without which re-establishment of epistemic trust is inconceivable

4. **Communication**: no communication is possible without the communicator having in mind the perspective of the
Do EBPs outperform TAU?

INGREDIENTS IN COMMON

1. A clear and credible treatment frame: serves as an ostensive cue priming the patient to pay attention
2. Giving the patient the experience of having their mind held in mind and being treated as an agent (being mentalized) \(\rightarrow\) increased epistemic trust
3. Increased epistemic trust \(\rightarrow\) patient is resilient enough to learn from experiences in the social environment beyond therapy, if the environment is sufficiently benign