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Epidemiology of eating disorders part III: Social epidemiology and case definitions revisited

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Abstract

The previous papers in this series (Rosenvinge & Pettersen, 2014a,b) outlined a historical panorama and presented knowledge about the distribution of eating disorders in various populations as well as about putative risk factors. This final paper focuses on social epidemiology, notably the current status of treatment dissemination and prevention research. We also discuss some comorbidity findings and transdiagnostic issues as an alternative to the DSM based definition of an epidemiological case, and how such an alternative definition may introduce new perspectives of prevention.

Key words: eating disorders, epidemiology, review

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The present article is the final one of three about the epidemiology of eating disorders. The first one (Rosenvinge & Pettersen, 2014a) outlined basic epidemiological concepts, approaches and issues. In addition, it outlined a historical panorama of the epidemiology of eating disorders up to the early 1990s depicting, the evolvement of eating disorders from being *one*, curious and rare disorder (i.e., anorexia nervosa) to a *set of* disorders distributed in the population to an extent which has caught public concern and scientific effort for the past 30 or 40 years. This evolvement has resulted in case definitions through the DSM and ICD manuals, and being worldwide disseminated they haves had a major impact on case definition- and identification methods in epidemiological eating disorder research.

The second paper in this series (Rosenvinge & Pettersen, 2014b) outlined recent knowledge about descriptive epidemiology; how many who actually have a DSM eating disorder or significant eating problems at a given point of time (i.e., the point prevalence), or in the past (i.e., the lifetime prevalence), as well as about the number of new cases appearing per year (i.e., the incidence). In addition, we focused on issues within analytic epidemiology, notably what is known about risk factors for developing eating disorders or for staying ill for a longer period of time.

Case *identification* methods require a case *definition* which is useful for analytic and social epidemiological purposes, and not just for describing the prevalence or incidence. Traditionally, the DSM taxonomy has provided a gold standard. This may be questioned in view of mental disorders most likely to come along with eating disorders, most likely to affect their course, and where the comorbidity may point to transdiagnostic factors possibly relevant to epidemiological research. The first aim of this paper is to discuss the relevance and implications of such perspectives in constructing an epidemiological case definition.

Social epidemiology becomes relevant when we have some answers from descriptive epidemiology to the “how many-questions”, and from analytical epidemiology to the “who, and “why”-questions. One part of social epidemiology concerns prevention, and which may take the approach of health promotion at a universal (general population) level, i.e., by enhancing environmental supportive factors as well as individual empowerment and resiliency. The *disease prevention* approach, however targets specific syndromes, e.g., eating disorders. Also this approach operates at a universal level, yet a selective (risk group) or indicative (individual-at risk) level is preferred, depending on the reliability and specificity of

risk factors and the nature of the epidemiological case definition. Compared with the rather dismal first generation of prevention studies up to mid-1990s, the situation gradually improved due to more knowledge about risk factors for eating disorders (Rosenvinge & Pettersen, 2014b) to aid in the setting up of selective prevention programmes. However, risk factor research is highly related to a DSM-taxonomy to define the "unit of epidemiological interest" with respect to eating disorders. This paper's second aim is to outline recent findings about the efficacy of prevention and to discuss perspectives of prevention in view of case definition approaches beyond the DSM phenotypes.

Social epidemiology also concerns the dissemination and implementation of evidence-based treatments. Literature reviews (e.g., Brownley et al., 2007; Bulik et al., 2007; Hay, 2013, Shapiro et al., 2007) show that such treatments comprise dialectical (Bankoff, Karpel, Forbes & Pantalone, 2012) and cognitive (CBT) approaches (Fairburn & Harrison, 2003, Fairburn, 2008), family therapy (Couturier, Kimber & Satzmani, 2013; von Sydow, Behler, Schweitzer & Retzlaff, 2010), and to some extent psychodynamic therapy (Lunn & Poulsen, 2012; Poulsen et al., 2014; Zipfel et al., 2014). Increasingly more consumers and health authorities demand access to such treatments (McHugh & Barlow, 2010), yet the access is limited in general (Proctor et al., 2009) as well as for eating disorders (Lynch et al., 2010). Limited access may be due to inadequate training in residency programmes (Mahr et al., 2014; Ghadirian & Leichner, 1990, Rosenvinge & Pettersen, 2006; Williams & Leichner, 2006), thus increasing medical provider's self-perception of poor intervention skills (Linville, Brown & O'Neill, 2012) or due to a suboptimal *delivery* of the evidence based treatments (Mussell et al., 2000; Shafran et al., 2009; Waller, Stringer & Meyer, 2012). Optimal delivery is highly related to the use of treatment manuals (Loeb et al., 2005), yet only 40-51% of clinicians actually use such a manual regularly (Wallace & von Ranson, 2011; Waller et al., 2013). There are mixed results with respect to what characterises non-manual users (Wallace & von Ranson, 2011; Turner et al., 2014). However, training clinicians to use manuals seems to be a viable strategy (Waller et al., 2013). Discussing this and other dissemination and implementation strategies to improve access to optimal treatment for a broad range of eating disorder sufferers in the community is the final focus of this paper.

Epidemiological case definitions revisited

Building on historical heritages (Rosenvinge & Pettersen, 2014a) the DSM represents the default model for epidemiological case definitions. So far, the DSM-5 classification does not seem to produce very different incidence or prevalence figures. Rather, the distribution of cases within the spectrum of eating disorder diagnoses serves to reduce the prevalence of non-specific cases (Rosenvinge & Pettersen, 2014b). Ever since the 1980 version of the DSM the validity of the eating disorder criteria has been heavily discussed in the clinical literature (in a number of studies which cannot be cited here). Also, the overall classification has been challenged from large sample studies using latent profile analysis, latent class analysis and taxometric analyses (Eddy et al., 2009; Wonderlich et al., 2007a, b), and from clinically developed transdiagnostic approaches (Fairburn, Cooper & Shafran, 2003). Other authors (e.g., Batstra & Frances, 2012; Frances, 2013) have attacked the DSM-5 as a whole for reinforcing the medicalisation trend of modern societies by "potentially creating false epidemics of misidentified pseudopatients" (Frances & Widiger, 2012, p. 122). This critique also includes the new status of binge eating disorder (BED) as an official diagnosis.

A transdiagnostic understanding has been advocated for several disorders (e.g., Nolen-Hoeksema & Watkins, 2011; Watson, 2005). As for eating disorders restrictive eating, overeating, compensatory behaviours, body checking and the overevaluation of the importance of controlling food intake, weight and body shape are posited (Fairburn, 2008; Fairburn et al., 2003; Pallister & Waller, 2008; Waller, 2008) as common factors across the diagnostic categories. This understanding departs from the DSM in reducing rather than increasing the number of diagnostic categories. However, both approaches to case definitions rely on observed (behavioral) as well as latent criteria (i.e., weight phobia and unjust evaluations of self and one's body), yet the number of latent criteria is increased within a transdiagnostic approach by the introduction of maintenance factors, i.e., dysfunctional perfectionism, mood intolerance, interpersonal difficulties and a profound low self-esteem. Such factors are also core clinical features of the DSM cluster B and C personality disorders associated with high anxiety and arousal, disorders which do occur in eating disorders.

The transdiagnostic approach has received scientific support (e.g., Allan & Goss, 2014; Hinrichsen, Waller & Emanuelli, 2004; Hoiles, Egan & Kane, 2012; Dakanalis et al.,

2014; Lampard, Byrne, McLean & Fursland, 2011; Tasca et al., 2011; Wade et al., 2006), yet tempered by *gender* (i.e., less support using male samples), *diagnosis-specific pathways* (i.e., more interpersonal problems related to unspecific eating disorders and more dysfunctional perfectionism related to anorexia and bulimia nervosa) and *risk factors* (Hilbert et al., 2014). Further validations will need research data generated from for instance, genetics and cognitive neuroscience. Following this track of science the NIMH[1] initiated in 2008 research within neuroscience, psychology and genetics to develop a future classification system based on latent variables or "constructs" (Cuthbert & Insel, 2014; Insel, 2014; Clark, Watson & Reynolds, 1995; Hyman, 2010; Insel et al., 2010; van Prag, 2000, Kendell & Jablensky, 2003) to understand the panorama of illnesses in the population beyond the accidential heritage of the ICD and DSM. Some studies relevant for eating disorders have appeared (e.g., Caglar-Nazali et al., 2014), and more are expected to appear in the future.

Comorbidity-based epidemiological case definitions

A purpose of epidemiology is to identify individuals in the population in need of special prevention or treatment efforts. Comorbid disorders may be relevant to include as they may impair quality of life (Mendlowicz & Stein, 2000; Padierna, Quintana, Arostegui, Goncalez & Horcajo, 2000) and increase the risk for a protracted course of eating disorders (Rosenvinge & Pettersen, 2014b). However, to be relevant for epidemiological case definition it is also important to include comorbid disorders with a certain probability of occurrence. These issues are discussed below.

Anxiety disorders

Many previous studies are flawed with methodological shortcomings (Swinbourne & Touyz, 2007), notably inadequate comorbidity diagnostics, uncontrolled designs and low statistical power. Well-powered studies however, indicate a prevalence range of any anxiety disorder from 42-64% (Godart et al., 2003b; Kaye et al., 2004), where the range is mostly accounted for by OCD (i.e., 41%) and social phobia (i.e., 20%), the former comparable to the 31% versus 8% (normal controls) reported by Råstam, Gillberg and Gillberg (1995). The odds ratio (OR) for a comorbid anxiety disorder is also high in bulimia nervosa (OR 5.0; CI[2]$_{95}$ 3.1-8.1) and BED (OR 5.00 CI$_{95}$ 3.0-8.1), but lower for anorexia nervosa (OR 0.9; CI$_{95}$ 0.3-2.6) (Swanson et al.,

[1] NIMH = National Institute of Mental Health (in the USA)
[2] CI = the confidence interval

2011). Lower level of comorbidity (i.e. 11% for any anxiety disorders and 7% for social phobia) has been related to younger age (Bühren et al. 2014), but not to a shorter duration of neither anorexia (i.e., 55%) nor BED (i.e., 50%) (Jordan et al., 2008). A community study (Gadalla & Piran, 2008) comprising over 20 000 individuals with eating problems and aged 15-24 years reported an elevated current risk (OR 3.44; CI_{95} 2.52-5.91) and lifetime risk (OR 3.95; CI_{95} 2.87-5.41) for any anxiety disorder. For epidemiological (but not clinical) purposes such studies are important as one may evade inflated findings usually found in clinical samples. All prevalence findings clearly exceed the community prevalence figures which range from 1-4% for generalised anxiety disorder, social anxiety disorder (2-7%), panic disorder (2-3%) OCD (2-3%) and phobic disorders (7-9%) (APA, 2013).

Mood disorders

Both literature reviews (e.g. Godart et al., 2007) and single studies (Jordan et al., 2008; Lucka, 2006) show that major depression is highly comorbid with anorexia nervosa, i.e., 10-88% for the restrictive and 50-72% for the bulimic subtype. Also, a 58% proportion has been reported for moderate to severe depression (Fennig & Hadas, 2010). Moreover, the recent study by Bühren et al. (2014) which indicates a lower overall proportion of comorbidity still reports a higher level of comorbid mood disorder (35%) than anxiety disorders (11%), notably accounted for by the proportion of major depressive disorder (32%).

The probability of mood disorders to appear seems lower in anorexia nervosa, i.e., OR 0.7; CI_{95} 0.3-2.0 (Swanson et al., 2011) and only slightly increased in subclinical disorders (i.e., OR 1.7; CI_{95} 0.5-5.8, and 1.8; CI_{95} 0.4-9.0 for a major depressive disorder and dysthymia, respectively). In community studies (e.g., Swanson et al., 2011) the comorbidity is likely for any mood disorder in bulimia (OR 5.7; CI_{95} 3.2-9.9) and BED (OR 4.76 CI_{95} 2.7-7.7). Among subclinical bulimia there is a high risk for major depressive disorder (OR 10.2; CI_{95} 4.5-23.1) and dysthymia (OR 6.3; CI_{95} 2.3-17.1). For subclinical BED the risk is only slightly lower (i.e. OR 3.5; CI_{95} 1.8-6.6 for major depressive disorder and OR 3.2; CI_{95} 1.4-7.2 for dysthymia) (Touchette et al., 2011). Other community studies (e.g., Gadalla & Piran, 2008) among adolescents with eating problems shows a high point- and lifetime prevalence of major depressive disorder (i.e., 5.98; CI_{95} 3.96-8.83 and 4.38; CI_{95} 3.21-6.18, respectively). Here, however, the evidence is mixed as other studies (e.g. Leon et al., 1999; Perez, Joiner, & Lewinsohn, 2004; Zaider, Johnson & Cockell, 2000) report that dysthymia (and not a major

depressive disorder) predict future eating problems across genders, notably bulimic symptoms (Santos et al., 2007).

Addictive disorders

A literature review shows a median of 23% (range 3-49%) comorbidity between bulimia and substance abuse and a higher proportion of substance abuse in bulimia nervosa (i.e., 41%) than in anorexia nervosa (i.e., 10%) (Holderness, Brooks-Gunn, & Warren, 1994). Similarly, a meta-analysis of newer clinical studies (Gadalla & Piran, 2007) showed significant relationships with moderate to strong effect sizes (ES) for the comorbidity between alcohol abuse and bulimia (ES 0.53) as well as EDNOS (ES 0.72), but not with respect of anorexia nervosa (ES 0.06). In community samples, however, the ES has been reported higher (i.e., 0.24), although still lower than for bulimia (i.e. 0.56) and EDNOS (i.e., 0.36). A large general population study (Root et al., 2010) reiterated this pattern across all eating disorder diagnostic groups. Compared with normal controls the probability of suffering from alcohol abuse was high for bulimia (OR 4.29; CI_{95} 3.13-5.87), and again, statistically significant lower for anorexia nervosa (OR 1.90; CI_{95} 1.35-2.66). A meta-analysis restricted to drug abuse (Calero-Elvira et al., 2009) reiterated the pattern of more abuse in bulimia nervosa both compared to controls and compared to restrictive anorexia nervosa. Thus, across studies the pattern of more substance abuse in eating disorders and highest among those with bulimia nervosa has been rather consistent.

Personality disorders (PDs)

Personality disorders (PDs) and eating disorders is perhaps the comorbidity most comprehensively researched, and summarized in several meta-analyses (i.e., Bornstein, 2001; Cassin & von Ransom, 2005, Friborg et al., 2014; Martinussen et al., 2014; Rosenvinge et al., 2000) covering the literature between 1987 and 2010.

Overall, proportions of PDs among patients with anorexia (i.e., 19-50%) and bulimia nervosa (i.e., 25-59%), BED (i.e., 29%) and EDNOS (i.e., 37%) are significantly higher compared with healthy controls (i.e., 10%). Across all eating disorder diagnoses proportions of PDs ascend from the cluster A to the cluster C disorders. Thus, the proportions of *cluster A* diagnoses range between 11-12%; CI_{95} 0-53, and 7-22% (anorexia nervosa), 13-27% (CI_{95} 7-22, and 0-56%) for bulimia (Rosenvinge et al., 2000; Martinussen et al., 2014), 13% (CI_{95} 2-

51%) for BED, and EDNOS (i.e. 8% CI_{95} 2-25%). The *cluster B* proportions range from 15-23% (CI_{95} 16-32) for anorexia nervosa, from 33-44% (CI_{95} 26-40, and CI_{95} 12-66) for bulimia, 20% (CI_{95} 5-56) for BED, and 22% (CI_{95} 10-43%) for EDNOS. As for *cluster C* PDs the anorexia and bulimia nervosa proportions hover around 43-45% (CI_{95} 33-54, and CI_{95} 10-77) (Martinussen et al., 2014; Rosenvinge et al, 2000), for BED, 33% (CI_{95} 21-48%), and 36% (CI_{95} 26-49) for EDNOS (Friborg et al., 2014).

Taken together, the findings indicate some important transdiagnostic similarities, notably related to the *avoidant PD,* which has been found to run through all the eating disorders, i.e., 25% for anorexia nervosa, 20% for bulimia nervosa and EDNOS, and 12% for BED. Although catching a smaller range of severity, social phobia also relates to the avoidant PD, and where similar proportions have been found. Some specific relations have also been detected related to the *dependent PD* (18%), but predominantly the *borderline PD,* where higher proportions have been found for bulimia nervosa (25%) as well as for BED and EDNOS (i.e., 11%). For anorexia nervosa, however, the proportion of *OCD PD* is twice as high (i.e., 22%) compared with bulimia (i.e., 12%) and BED (i.e., 10%).

Cross-sample findings and succession of onset studies

Eating disorders have found to occur among 11-42% of OCD-patients (Becker, DeViva, & Zayfert, 2004; Bulik, Sullivan & Joyce, 1995; Micali et al., 2011) and among 20% of patients with social phobia (Brewerton et al., 1993). Such figures may be deflated as commonly used tools to assess anxiety disorders fail to detect up to 80% of cases (Becker et al., 2004). Also, among bipolar patients the prevalence ranges from 14-16% for BED and anorexia nervosa, yet lower for bulimia (5%) (McElroy et al., 2011; Hudson et al., 2007; Fornaro et al., 2011). Still, there is an increased risk (OR 4.5; CI_{95} 1.1-17.6) for also having a bipolar disorder given a diagnosis of bulimia nervosa (Lunde et al., 2009), and rising (OR 9.1; CI_{95} 1.1-73.6) when including affective temperaments. Such temperaments may indicate a latent factor in terms of dysregulation, notably of weight, eating and a cyclicity of behavioural activation (Fairburn & Harrison, 2003; McElroy et al., 2002). A dysregulation perspective is also indicated by the high prevalence of cross-sample prevalence of bulimia nervosa (8-41%) and anorexia nervosa (2-10%) among patients with substance abuse (Holderness et al., 1994).

Also, a 17% prevalence of eating disorders has been found among patients with PDs, notably a risk for bulimia in the borderline PD (OR 3.57; CI_{95} 1.55-8.23) and EDNOS (OR 3.26; CI_{95} 1.61-8.99) and for anorexia nervosa in the OCD PD (OR 3.57; CI_{95} 4.48-18.50) (Reas, Rø, Karterud, Hummelen & Pedersen, 2013). Moreover, patients with an avoidant PD also had a higher risk of having bulimia (OR 2.64; CI_{95} 1.15-6.06) and EDNOS (OR 2.37; CI_{95} 1.17-4.79), however, not anorexia nervosa (OR 0.15; CI_{95} 0.12-1.27). The latter finding stands in contrast to the high proportions of the avoidant PD among patients with anorexia nervosa outlined in the above section. At least three other similar studies have appeared in the literature. In the study by Zanarini et al. (2010) patients with a borderline PD were about two times more likely to report any eating disorder (relative risk ratio (RRR) = 1.95, and where the RRR-values actually were lower for bulimia (i.e., 1.71) compared with anorexia nervosa (i.e., 2.33) and EDNOS (i.e., 2.26). Furthermore, among female patients admitted for a PD, higher proportions than in community samples have been reported for anorexia nervosa (8%), bulimia (12%) and EDNOS (22%), yet with no statistically significant differences across PDs for neither of the eating disorder diagnostic groups (Skodol et al., 1993). Grilo et al. (1993) on the other hand, argue against a meaningful eating disorder-PD comorbidity when base rates of eating disorders are controlled for.

Anxiety disorders also tend to develop before the onset of eating disorders. Thus, lifetime prevalence of anxiety disorders amounts to 83% (anorexia nervosa) and 71% (bulimia nervosa) notably social anxiety as the most prevalent condition (i.e., 49-55% and 59-68%, respectively) (Godart et al., 2000; 2003b). These findings are on par with other studies and reviews (e.g., Bulik, Wade & Kendler, 2000; Swinbourne et al., 2012). Some premorbid anxiety disorders seem to confer a specific risk i.e. OCD in anorexia nervosa (OR 11.8) and social phobia in bulimia (OR 15.5) but the risk for premorbid overanxious disorder seems increased for both anorexia (OR 13.4) and bulimia nervosa (OR 4.9) (Bulik et al., 2007) and associated with more severe eating pathology and extreme personality traits (Raney et al., 2008). A premorbid OCD has also been reported in other studies (e.g. Micali et al., 2011) and that a general anxiety disorder as well as trait anxiety predicts the onset of anorexia nervosa (Jacobi et al., 2004; Yackobvich-Gavan et al., 2009). A lover (8%) lifetime prevalence among younger teenage eating disorder patients (Bühren et al., 2014) may indicate an age effect in

need of further investigation, but overall, overall, this evidence suggests that anxiety disorders may be included in the construction of an epidemiological case definition.

Also, mood disorder symptoms may precede, and confer a higher probability of developing eating disorders (Godart et al., 2007; Rosenvinge & Pettersen, 2014b). Notably, at least one year before the onset of an eating disorder, about 25-71% has experienced a clinically significant depressive disorder. However, a premorbid mood disorder seems less common (i.e. 6%) among younger people. Mood disorders may improve after weight restoration (Meehan, Loweb & Attia, 2006), but the premorbid occurrence as well as the persistence throughout the long-term outcome (Rosenvinge & Pettersen, 2014b) indicate that mood disorders are not only a complication to eating disorders.

Towards possible latent factors relevant for an epidemiological case definition

Possible shared aetiological mechanisms

Comorbidity studies and family studies indicate that eating disorder patients' family history of both anxiety and mood disorders (Gershon et al., 1984; Hudson et al., 1983a; Logue et al., 1989; Rivinus et al., 1984; Strober, Freeman, Lampert & Diamond, 2007; Strober et al., 1990). Twin studies of adult populations support a substantial shared genetic etiology between depression and generalized anxiety disorder (Jardine et al., 1984; Kendler, 1996; Kendler et al., 1992; Roy et al., 1995), between eating disorders and major depression (Wade et al., 2000; Walters et al., 1992), and between eating disorders and anxiety disorders (Kendler et al., 1995). Furthermore, Silberg & Bulik (2005) demonstrated that comorbidity between eating disorders, anxiety disorders and depressive symptoms is accounted for by shared genetic factors, and with the personality trait "harm avoidance" as a possible pathway. Harm avoidance has been shown to be elevated in individuals with anxiety (Kennedy, Schwab, & Hyde, 2001), depression (Grucza, Przybeck, Spitznagel, & Cloninger, 2003; Kennedy et al., 2001; Marijnissen, Tuinier, Sijben, & Verhoeven, 2002) and eating disorders (Bulik, Sullivan, Carter, & Joyce, 1995; Bulik, Sullivan, Weltzin, & Kaye, 1995; Klump et al., 2000). Moreover, activation of a common biological pathway that influences anxiety, depression, and eating (e.g., the serotonergic system) may also be plausible as such activity has been consistently shown to be abnormal in women with anorexia and bulimia nervosa both during the acute phase of the illness and after recovery (Kaye, 1997a,b; O'Dwyer, Lucey, & Russell, 1996; Wolfe, Metzger, & Jimerson, 1997). Moreover, serotonin influences

both feeding behavior (Blundell, 1992; Leibowitz & Shor-Posner, 1986) and mood and anxiety (Charney, Woods, Goodman, & Heninger, 1987; Kahn, van Praag, Wetzler, Asnis, & Barr, 1988).

Personality traits along a perfectionism-impulsivity dimension

Dysfunctional perfectionism is characterised by maintenance of personal standards that exceed one's abilities along with avoidance and a profound fear of failure (Frost, 1991; Frost & DiBartolo, 2002). Such perfectionism is frequently reported in anorexia nervosa, and may be extended to domains of life beyond the drive to fulfil unrealistic standards related to the control of food, shape, and weight (Fairburn et al., 2003) to a severity compatible with the avoidant and the OCD PDs (Frost, 1991; Frost & DiBartolo, 2002). Recent reviews (Bardone-Cone et al., 2007; Egan, Wade & Shafran, 2011) present evidence for perfectionism as a risk and maintaining factor for eating disorders, mood disorders, and anxiety disorders.

Impulsivity and afffective regulation represent the "companion" to perfectionism. Here, food and eating serves the purpose of regulating emotions like anger, excitement, anxiety, or sadness (Favaro et al., 2005; Lampard, Tasca, Balfour & Bissada, 2012). These dysfunctional regulatory strategies may be extended to, for instance, self-injury, uncritical sexual behaviours or substance abuse. For instance, both substance abuse and overeating serve affect regulation purposes (Root et al., 2010) by temporarily reduce negative affects. Also, a more sophisticated regulation mechanism should be considered with reference to "alexithymia" (i.e., the poor ability to identify and describe one's emotions (Spence & Courbasson, 2012) and the findings Thorberg et al., 2011; Lyvers et al., 2012) linking substance intake to the temporary access to one's feelings, a regulating mechanism associated with insecure and anxious parent-child basic attachment patterns. Impulsivity and regulation problems are highly present among patients with bulimia, and are core elements in the borderline PD. The spectrum of dysregulated emotions shared between PDs and eating disorders is also reflected in the DSM cluster model of PDs. Thus, eating disorder patients with high levels of impulsivity may fall into the cluster B, whereas the overcontrolled/constricted subtype fall into the cluster C PDs (Claes, Vandereycken, & Vertommen, 2005; Gazzillo et al., 2013; Skodol et al., 1993). The impulsivity-perfectionism dimension is thus found both in eating disorders (Lilenfeld et al., 2006; Fairburn et al., 2003; Fairburn et al., 2008; Gazzillo et al., 2013) and in PDs (Gazzillo et al., 2013; Skodol et al.,

1993). The majority of eating disorder patients move between anorexia- and bulimia nervosa (Eddy et al., 2008; Milos et al., 2005; Castellini et al., 2011). The character trait "low self-directedness" has been found to strongly predict such crossovers due to alternations between restraints and disinhibition (Tozzi et al., 2005), and such alternations has been particularly related to the borderline PD (Jylhä et al., 2013). Hence, this dimensional trait may run through the eating disorder diagnoses as a common underlying factor, predicting the negligible differences in the proportions of PD comorbidity between anorexia nervosa and bulimia nervosa that have been reported in meta-analyses. A review of the literature (Fassino et al., 2004) also show that the traits "harm avoidance" and "low self-directedness" tend to run through all the eating disorder categories as well as through the mood and anxiety disorders. Moreover, individuals with a combination of perfectionism and impulsivity may suffer from a very high eating disorder symptom load (Boone, Claes & Luyten, 2014) and it has been suggested (Luyten & Blatt, 2011) that obsessions and overcontrol may act as an overcompensation strategy to control impulses. Other traits like "neuroticism" is associated with the development of anxiety and depression (Clark, Watson & Mineka, 1994) as well as eating disorders (Ghaderi & Scott, 2000).

A possible overarching latent variable summarizing the above findings may then be a *dysregulation core construct.* As discussed below, a second, general latent construct may be interpersonally related, and it connects to research indicating shared aetiological mechanisms between anxiety disorders and eating disorders.

Anxiety disorders and eating disorders

Several authors have suggested that the comorbidity between anxiety- and eating disorders can point to important latent factors (e.g., Bulik, 1995; Waller, 2008; Pallister & Waller, 2008; Rosenvinge, 2008). Notably, arguments (Waller, 2008) for collapsing these disorders build on the high proportions of comorbidity, the fact that the PDs most comorbid with eating disorders (e.g., the avoidant, borderline, and obsessive-compulsive PDs) are characterized by high levels of anxiety and arousal, the succession of disorders as well as a possible shared genetic vulnerability as discussed above. Also, shared seems to be certain core cognitive beliefs (Waller, Ohanian, Meyer & Osman, 2000; Hinrichsen et al., 2004) leading to the understanding of eating disorder symptoms as a variant of safety behaviours typical of anxiety disorders. Hence, symptoms like bingeing and purging may be regarded as

actions to regulate feelings of negative activation and negative affects, or to dissociate from the anticipatory anxiety if coming into contact with emotionally loaded thoughts or impulses. Moreover, symptoms of perfectionism and body checking may be viewed as an avoidance strategy serving the same protective purposes.

A possible shared vulnerability has been empirically tested with a special reference to generalised anxiety disorder (GAD), i.e. the prevalence of which that may fit best with the predictions of the shared vulnerability model (Pallister & Waller, 2008; Waller, 2008). Specifically, a cognitive model of GAD (Dugas, Gagnon, Ladouceur & Freeston, 1998) suggests that worry, as the main component of this anxiety disorder consists of four components, i.e., intolerance of uncertainty, positive beliefs about the good reasons for worrying, poor problem orientation, and cognitive avoidance. Intolerance of uncertainty has been associated with deviant eating attitudes (Konstantellou & Reynolds, 2010), and in clinical samples (Konstantellou et al., 2011) with both eating disorders and GAD. In the latter study all comorbid patients scored significantly higher on all components compared with normal controls, and vulnerability factors for GAD were present in eating disorder even in cases of subclinical GAD. This gives support to understanding eating disorders as a part of GAD, but also through social anxiety, OCD (Boelen & Reijntjes, 2009; Holoway, Heimberg & Coles, 2006, Tolin, Abramowitz, Brigidi & Foa, 2003) and mood disturbances (Dugas, Schwartz & Francis, 2004; Yook, Kim, Suh & Lee, 2010; McEvoy & Mahoney, 2011).

Furthermore, shared psychopathology between anxiety disorders and eating disorders may predict certain attachment disturbances, as the common psychological reason for why various forms of safety behaviours are needed (Tasca & Balfour, 2014). It is important to note that developmental theory does not posit that attachment disturbances are inevitably leading to psychopathology; they only raise the risk for such pathology (Sroufe, 2005). Nevertheless, both disorganized (i.e., the simultaneous seeking and avoiding parental care) and insecure attachment states have been related to psychopathology both in meta-analyses (Bakermans-Kranenburg, & van Ijzendoorn, 2009; Caglar-Nazali et al., 2014) with ES up to 1.31, and in longitudinal studies (Sroufe, 2005). In addition, the review by Tasca and Balfour (2014) points to the evidence for a relationship between attachment insecurity and more severe eating disorder symptoms, as this attachment style is particularly associated with dysfunctional affect regulation and interpersonal sensitivities. A special

variant of safety behaviours relevant to eating disorders is the dysfunctional perfectionism, and one recent study (Dakanalis et al., 2014) reports evidence that dysfunctional perfectionism mediates the association between the insecure, anxious, and avoidance attachment patterns and eating disorder symptoms across all the DSM-IV diagnostic categories. Another study (Keating, Tasca & Hill, 2013) also supported the role of attachment anxiety and attachment avoidance in predicting body dissatisfaction. Here, a mediator (i.e., alexithymia) was a proxy for a class of safety behaviours (e.g., dissociation, suppression or overregulation) to avoid anxiety and where an undue focus on weight and shape may be understood as a consequence of social insecurity and a hypersensitivity to perceived societal standards. Also, dysfunctional attachment dimensions relate to negative mood through affect regulation problems (Wei et al., 2005) and to mood and eating disorder symptoms (Tasca et al., 2009). Despite sophisticated statistical methods used, these studies are limited by cross-sectional designs. Importantly then, four of the longitudinal and prospective studies (Allen et al., 2009; Nicholls & Viner, 2009; Tanofsky-Kraff et al., 2011; Zerwas et al., 2014) lend equivocal support to the predictive validity of maternally reported childhood anxiety relative to eating disorder symptoms. One study (Milan & Acker, 2014) also found that an insecure attachment style mediated by an elevated pubertal BMI predicted eating disorder symptoms .

Parental bonding is a concept closely related to attachment, but focuses more on the parental contributions. Reviews of this literature (Ward, Ramsay & Treasure, 2000; Tetley, Moghaddam, Dawson & Rennoldson, 2014) supports attachment findings in the sense that overall, eating disorder patients across diagnostic groups tend to remember their parents as less affectionate and more controlling compared to normal controls. However, this may seem like a general vulnerability as the same pattern apply to other mental disorders as well, as it is related to perfectionism, but also to a poor self-concept and cognitive beliefs like being internally flawed and defective (Ward et al., 2000; Perry et al., 2008).

The above findings may be subsumed under a "*dysfunctional basic relations"* core construct, and along with a dysregulation core construct may contribute to understanding eating disorders and their comorbid disorders beyond a descriptive level. It should be recognised that the full understanding of the possible shared *causal* mechanisms across eating disorders and the comorbid disorders awaits future research. However, for

epidemiological purposes, the evidence may be sufficient to consider such latent constructs as elements in an epidemiological case definitions.

Towards new potentials for epidemiological research?

What then, about future studies using the DSM to define "the epidemiological case"? Consistent with the changes made in the DSM-5, the epidemiological studies done after its release do not substantially change the distribution of eating disorder in the population save for fewer unspecific cases (Rosenvinge & Pettersen, 2014b). Also considering the relative stability over time (Rosenvinge et al., 2014a) one may argue, then, that further descriptive epidemiological studies of this distribution are unnecessary.

Close to 20% of patients in outpatient clinics have undetected eating problems (Fursland & Watson, 2014) and their need for eating disorder treatment is identified by their comorbid disorder. Such findings argue for including in the epidemiological case those disorders which are most likely to appear along with eating disorder. Hence, epidemiological research should go beyond the descriptive level based on the DSM-5 taxonomy to better capture the proportion of individuals in need of special attention, and to estimate the need for dissemination of prevention and treatment.

We do recognize that the RDoC-initiative from the NIMH in a far future may bring about a complete restart of epidemiological research. In the meantime, but following the same line of reasoning, we suggest elements in a case construction consisting of individuals who 1) display depressive or anxiety symptoms, 2) diet or use other measures (substance abuse and self-injurious behaviours included herein) to control or regulate emotions, and where regulatory mechanisms also serve to regulate interpersonal relations for the purpose of compensating or coping with attachment and bonding, and 3) display traits related to temperament and personality, notably "neuroticism" and "harm avoidance". Moreover, risk factors for eating disorders are relevant, because of, and not despite their lack of specificity relative to eating disorders. Hence, by including operational definitions of latent variables an epidemiological case definition may truly cross the border from a "comorbidity" perspective to a transdiagnostic understanding. Compared to a DSM way of defining a case, such an enhanced definition may allow a screening among younger children. We do recognize

challenges in accomplishing studies based on an enhanced case definition, but to discuss these goes beyond the scope of this paper.

An enhanced case definition as an independent variable and a set of end pints (dependent variables), may in prospective epidemiological designs serve the purpose of testing the predictive value of transdiagnostic, latent factors on a large scale level. The output of such research may help in designing selective prevention programmes, and with a broader aim than only preventing eating disorders. An enhanced case definition as a composite dependent (outcome) variable may revitalise universal disease prevention research, and if converted to positive entities, universal health promotion purposes as well. These issues are discussed in more detail below.

From analytic to social epidemiology: An update and future suggestions

Prevention

Prevention of eating disorders is challenged by the literature (Rosenvinge & Pettersen, 2014b) showing that eating problems tend to level off as individuals approach adulthood, by the fact that those who need an intervention tend not to show up, and the fact that incident cases can be unrelated to the risk status (Rose, 1993). Thus, findings from prevention trials may be flawed with false positive findings. In the literature, over 60 programmes have been identified, most of them with no or just short-lived effects (Ciao, Loth & Neumark-Sztainer, 2014). Hence, the issue has pertinently been raised (Becker et al., 2014) about the feasibility of spending societal money as well as time and energy on disseminating ineffective programmes. On the other hand, though, adolescence is a high risk for the onset of many common mental disorders. A prospective study of adolescents 15-30 years (Patton et al., 2014) confirms a decreasing trend in overall morbidity provided a duration of symptoms less than 6 months. Likewise, a short duration of symptoms also predicts a favourable eating disorder outcome (Rosenvinge & Pettersen, 2014b). Hence, and even when the natural course is considered, there is a need for effective prevention of eating disorders as well as of mental disorders in general.

Several meta-analyses have served their purpose of summing up this field and pointing out new directions. A Cochrane initiated systematic review (Pratt & Woolfenden, 2002) updated in 2004 and covering studies from 1996 found limited support for the effects of both universal and high-risk strategies targeting eating attitudes and behaviours,

knowledge about eating disorders, self-esteem or sociocultural body ideals. Important reasons for this situation were poor methodology, no implementation of evidence-based methods of attitudinal (and behavioural) change and lack of knowledge about risk factors for eating disorders. Meta-analyses (Fingeret, Warren, Cepeda-Benito & Gleaves, 2006; Stice & Shaw, 2004) evaluated universal and selective prevention programmes from 1980-2003. Overall, knowledge about eating disorder reached moderate to high effect sizes at post-test and follow-up, respectively (i.e.,1.01 and 0.68), and the majority of studies significantly reduced at least one risk factor, notably body dissatisfaction, yet the effect sizes were low at post-test (i.e., 0.19 and 0.15, respectively. For other outcome variable like dieting, thin body internalisation and general eating pathology the summarized effect sized were also low (i.e., 0.24 and 0.20 at post-test and follow-up, respectively. Notably, main effects were not assessed, i.e., whether the intervention programmes actually lowered the incidence of eating disorders. As for new research directions moderator analyses (Stice & Shaw, 2004; Stice, Shaw & Marti, 2007) have supported selective rather than universal strategies, and have related a probability of success to limiting the target group to female participants above 15 years of age, to the use of multisession approaches focusing teaching new skills rather than to educate about eating disorders or presenting programmes as aiming to prevent eating disorders in particular.

Evidence-based programmes have implemented knowledge about risk factors, moderators and notably about attitudinal and behaviour change derived from social psychology, e.g., an elaboration-likelihood approach (Petty & Cacioppo, 1986) and the theory of cognitive dissonance (Festinger, 1962). In the latter, a change is initiated by introducing a discomfort-eliciting discrepancy between actions and attitudes which individuals seek to resolve by a change in attitudes. For instance, such discomfort may be created by making body dissatisfied girls argue against factors raising body dissatisfaction. The effect of prevention using cognitive dissonance is heavily documented in meta-analyses and reviews (e.g.,, Stice, Shaw & Marti, 2007; Mitchell, Mazzeo, Rauch & Cooke, 2007; Stice, Shaw, Becker & Rhode, 2008a; Stice, Becker & Yokum, 2013b). In particular, two programmes (Stice et al., 2008b; Stice et al., 2013c) have shown stable long-term effects and a 60% reduction in eating disorder onset. However, given the impact of non-specific factors and demand characteristics (McMillan, Stice & Rhode, 2011) more research is needed to

identify the active intervention components. A review of controlled studies mainly using an elaboration-likelihood approach (Yager et al., 2014) shows moderate effect sizes ranging from 0.23-0.48 for programmes aiming to improve body image. Notably the programme *Life Smart* has been successfully evaluated showing longitudinal (i.e. up to 2.5 years) universal and selective level effects across genders, and for individuals with a comorbid mood disorder (Wilksch, 2010; Wilksch & Wade, 2009; 2014).

Dissemination challenges

The overall low ESs for eating disorder prevention are comparable to those obtained for anxiety (Fisak et al., 2011), depression (Horowitz & Garber, 2006) and substance abuse (Gottfredson & Wilson, 2003), but may still be discouraging. However, in contrast to treatment studies even low ESs may be important from an epidemiological and public health perspective in the sense that the overall risk level for incident cases is lowered. Such a public health impact requires however, observable and stable effects preferably at a universal (population) level.

The conditions for large-scale dissemination may be present, e.g., that smaller (Becker et al., 2008) yet significant effects may be produced even at low-level dissonance induction (McMillan et al., 2011), without the use of highly qualified personnel (Becker et al., 2006; Stice et al., 2013a), and using a naturalistic (universal) setting with mixed high and low risk subjects (Becker et al., 2008). Preliminary findings (Stice et al., 2013d) indicate the feasibility and effectiveness of a dissonance-approach for large-scale dissemination, and this merit future research.

As over 95% of females have access to the internet and spending on average 1.2 hours daily using it (Odell et al., 2000; Rideout, Roberts & Foehr, 2005). Web-based prevention programmes may thus offer a platform for effective dissemination, interaction as well as enduring and competent delivery. Systematic reviews (Melville & Casey, 2010; Newton & Ciliska, 2006; Yager & O'Dea, 2008) show, however that attrition is a huge problem, and that many programmes fail in providing significant effects. Again, though, dissonance-based programmes to combat body dissatisfaction do seem to perform equally well in large scale contexts (Stice, Rhode, Durant & Shaw, 2012; Stice et al., 2013b), indicating that such programmes should be further developed as a universal strategy. Currently, at least six large-scale online programmes are available (i.e. "Student Bodies",

"Set Your Body Free", the "eBody Project", "Essprit"/"YoungEssprit" and the ProYouth", the latter supported by the EU's Health Programme), and all of these show some positive effects which clearly need to be followed up by more rigorous research (Bauer et al., 2013).

Systematic reviews allow for optimism for the future of internet-based health promotion strategies with respect to promoting physical activity (Hamel, Robbins & Wilbour, 2010) and healthy eating (Hamel & Robbins, 2012) at least on a short-term-basis, provided that programmes are integrated in the school curriculum, are using peers as motivating coaches and include tailored personalised feedback from those responsible for the programme. A recent review (Hart, Cornell, Damiano & Paxton, 2014) however, showed that parents are invariably involved in prevention programmes and that the evidence for the effects of such involvement is mixed. Whether, or in what way parents should be involved in children's programmes remains to be clarified in future studies. However, the importance of including parents is rather likely considering parents as role models and a primary source of values. Another review (Clarke, Kuosmanen & Barry (2014) also report the impact of motivation and support, but also the need for designing future studies according to higher methodological standards.

Perspectives of prevention in view of case definition approaches

Treating variable risk markers as independent (selection) variables obviously leads to more high risk group studies within a disease-prevention paradigm. This direction of research face some challenges as the risk factor research also shows the impact of multiple trajectories and cumulative risk factors, which refutes the idea that a prevention strategy can rely on a one-size-fits-all approach.

Hence, to avoid such challenges a revitalisation of a universal prevention approach is called for using risk factors and latent variables as dependent (outcome) variables. Then, one may avoid screening procedures which in order to obtain acceptable effectiveness can be rather costly (Wright et al, 2014). The social epidemiological benefit of lower incidence figures do include a cost-benefit perspective. Thus, the savings of improved detection may be considerable. For instance, 1 case of bulimia nervosa detected would save USD 33999 in medical costs and 0.7 quality-adjusted life years (Wang, Nicholls & Austin, 2011). Still, for social epidemiological and health promoting purposes small changes in the general population may have a more favourable cost-benefit-ratio in a prospective than a post-hoc

perspective, where a reduction in incident cases may be rather ambitious. Indeed, a review study (Heckman, 2006) shows that while a huge amount of money is invested in preventing adolescent illness, the highest payout per year per dollar invested in human capital programmes lies in the teaching of basic health promotion and social skills to preschool and primary school children. Thus, there may be a huge pay-off by promoting resiliency by reversing the risk factor perspective. Indeed, this health-promoting perspective imply a change of paradigm rather than just a change of programmes (Rosenvinge & Børresen, 1999). Within such a paradigm, prevention may take the form of stimulating children's inherited positive body image and to include some very general psychoeductional efforts. Then, a large scale dissemination project should include informing parents with small children to avoid developing a family climate coloured by poor bonding and fears of rejection, where teasing children for their body, shape or weight is allowed, where undue perfectionism is allowed to grow, where mealtimes develop into an arena of tension or conflict. Moreover, parents should be informed about the value of introducing small, but viable lifestyle changes to fight childhood obesity. A next step in such a programme is to show how these goals may be accomplished.

Epidemiological case definition based on comorbidity and transdiagnostic latent factors may pave the way for new ways of thinking about prevention at a universal level of action.

We devote a final word on prevention to indicative approaches to reach out to long-term, treatment-resistant patients with eating disorders who are easily defocused given the attention to active treatments and the prevention of juvenile eating problems. Few authors have outlined care programs (e.g., Robinson, 2009) or principles of care (e.g., Strober, 2004) for these patients. In our opinion, this is a social epidemiological concern which needs future professional attention.

Treatment dissemination and implementation

All dissemination strategies boil down to making high quality treatment more available to more individuals who need it. Using a DSM case definition epidemiological research indicates that 30-50% (anorexia nervosa), 6% (bulimia) and 20% (BED) are not seen in the clinics (Hoek & van Hoecken, 2003; Keski-Rahkonen et al., 2007; 2009; Reicborn-Kjennerud, Bulik, Sullivan, Tambs & Harris, 2004; Wentz, Gillberg, Gillberg & Råstam,

2001). Also, a systematic review (Hart, Granillo, Jorm & Paxton, 2011) estimated a pooled proportion of only 23% (CI_{95} 16.6-31.4) of community cases seeking treatment for eating disorders. Using an enhanced epidemiological case definition approach we would predict an increased number of undetected cases. Detecting the undetected is a social epidemiological concern, as a proportion of untreated or delayed-treated individuals are exposed to an increased risk for a poor prognosis as a long duration of illness stands out as a rather robust outcome predictor (Rosenvinge & Pettersen, 2014b).

How may such figures be explained? To some extent, they reflect "*patient's delay* in seeking treatment due to shame, poor insight and a natural resistance to change. The failure to temporarily or permanently seek treatment may also relate to the fact that sufferers know that evidence-based treatments are unavailable to them. This turns our focus to *therapist delay* in implementing such treatments in their clinical contexts.

Also therapists can be resistant to change. Some may resist fitting their clinical practice to evidence based treatments due to a principal, epistemological rejection of the relevance of findings from controlled treatment trials to their clinical practice (Lilienfeld, 2013). Such a position is often defended by the development of attitudes about the importance of therapeutic alliance and the uniqueness of therapist factors, which are at variance with research findings about the importance of general effect variables (e.g., DeRubeis, Brotman & Gibbons, 2005; Stirman, DeRubeis, Crits-Cristoph & Rothman, 2005). Resistance to change practice coincides with overevaluating the impact of "treatment as usual". Clinicians obviously like to believe that their treatments are effective. This belief is reinforced by the confirmation bias (Lilenfeld et al., 2013), resulting in the attribution of patient recovery to therapy factors, and treatment failures to patient characteristics. Implementation may also fail due to inadequate training in evidence-based treatment, a failure that is likely to reinforce prejudices and negative beliefs about such treatments. Hence, the widely held assumption among treatment researchers that evidence based treatments automatically will "diffuse" into ordinary clinical practices is mistaken. Rather, active dissemination and implementation strategies will have to be used.

A traditional way of dissemination is to train students and residents within a situated-learning paradigm (Lave & Wenger, 1991). This procedure is obviously time-consuming and costly, and is not suited for large-scale levels of dissemination. Another way is to launch treatment guidelines. A raised probability of use in ordinary clinical settings is associated with a less complicated clinical syndrome in question, requiring clinical procedures which can be easily explained and tried out in clinical practice without much effort or resources. These prerequisites are not applicable to eating disorders. Not surprisingly then, the evidence (Currin et al., 2007; Rosenvinge & Pettersen, 2006) shows that save for minor impacts on monitoring routines guidelines for eating disorders are not very well known or used in clinical practice. Moreover, a listing and recommendation of evidence based treatments is not useful in the sense that guidelines do not offer an educational platform to train clinicians in how to practice such treatments.

"A training the trainer" approach using interactive web-sites (Fairburn & Wilson, 2013) seems highly promising, and has proven efficient to disseminate effective treatments for panic disorders (Wade, Treat & Stuart, 1998). One variant is a "snowball-recruitment approach" is used, where specialists train a small number of clinicians in a mental health organisation and these trainees then become trainers for the remaining staff members. However, a web based variant access to an "internet-enhanced training" may be scalable to a regional or national level of dissemination. Here an infinite number of clinicians have open access to a website, which provides training and supervision in the proper delivery of the treatment in question. For eating disorders such a site is available (credo-oxford.com). Recent studies outline the rationale and basic principles (Fairburn & Cooper, 2011), and shows that clinicians prefer such a website to illustrate clinical procedures and segments of sessions (Helgadottir & Fairburn, 2014). Several treatments may be relevant here, but CBT seems particularly well suited due to the efficacy across eating disorders as well as for comorbid disorders, the treatment manuals available, the systems to monitor therapist's adherence as well as the reinforcing impact of proximal effects if properly delivered. The transparency of the basic treatment principles has also been launched as an argument for the suitability of large-scale dissemination of CBT in the sense that the basic principles may be learned without years of basic training and education. This argument still lacks detailed empirical evidence given the broad diversity

of educational backgrounds across the mental health workforce (Ellis, Konrad, Thomas & Morrissey, 2009) and the diversity of clinical severity. To get around this problem, videoconferences involving a clinical specialist, and transacting with the general practitioner has been proposed (Comer & Barlow, 2014), at least for the complex cases. A systematic review of 10 randomised control trials (Garcia-Lizana & Munoz-Mayorga, 2010) calls for future optimism for the tendency of videoconference-based therapy to obtain the same results as face-to-face treatments in terms of symptoms, quality of life and patient satisfaction across a broad range of mental health disorders. This optimism is strengthened by positive expectations of usefulness (Dimeff et al., 2009; Monthuy-Blanc, Bouchard, Maiano & Seguin, 2013) as a predictor of intention to use.

Dissemination strategies may also target sufferers directly by the use of the internet in order to reach out to those who are not detected or treated in the health care system. Such strategies include e-therapy and the use of mobile applications (apps). Such reach out is possible given the number of web technology users. For instance, by the end of 2012 global mobile penetration reached 91%, and with 4.3 billion unique mobile phone subscribers. Hence, a large number of individuals worldwide are reachable through apps targeting mental health ("mHealth") which may offer treatment accessibility, real-time monitoring of symptoms and tracking of treatment progress through ecological momentary assessments. Community surveys indicate that about 76% of the population would be willing to use such apps (Proudfoot et al., 2010).

Dissemination through web based platforms does imply some technological, legal and psychological challenges. On the other hand, there is a high consumer satisfaction reported for e-therapy (McClay, Waters, McHale, Schmidt & Williams, 2013; Sanchez-Ortis et al., 2011a). It seems thus that web based disseminations are valued by making a detour around the fear of general practitioners' way of handling self-stigmatised individuals, and for providing help even to those who love in remote geographical areas with limited access to a face-to-face services.

Similar to web-based prevention studies attrition is a matter of concern. Drop-out is only a mouse-click away, resulting in drop-out rates from online intervention ranging from 3-81% across all disorders (Melville, Casey & Kavanagh, 2010), and from 20-40% for

eating disorders (Bell, 2001; Dejong, Broadbent & Schmidt, 2012). A literature review (Brachel et al., 2014) shows similar reasons as in face-to-face treatments for drop-outs, i.e., symptom severity (e.g., low weight and a comorbid PD), and a poor treatment alliance (e.g., a disparity between patient and therapist expectations and a failure to engage the patient in the treatment). Brief and efficient screening and support greatly reduce attrition in e-therapy (Marks & Cavanaugh, 2009). Thus, a controlled trial shows the supportive aspects of e-therapy for individuals with anorexia nervosa (Grover et al., 2011), and the supportive aspects of web-based therapist-guided self-help is highlighted in some studies (Sanchez-Ortiz et al., 2011a, b) which may account for the effect sizes (i.e., 0.42-0.67) reported in systematic reviews (e.g., Breitner, Jacobi & Schmidt, 2014). The majority of apps lack scientific evidence about their efficacy, but a systematic review (Donker et al., 2013) shows a fairly good efficacy at posttest (ES 0.29-2.28) and somewhat weaker at follow-up (ES 0.01-0.48) for depression, stress and substance abuse. A systematic review (Ardoom et al., 2013) indicate that web-based treatments may be effective for binge eating disorder patients without any comorbid disorders, but more research is needed both with respect to apps and web-based treatments. In a wired world then, effective dissemination through unconventional channels and platforms challenges the conventional thinking about therapy and the role of a therapist.

Conclusions

As outlined in this series, many previous challenges have been resolved, allowing for more accurate estimations of prevalence, incidence, risk factors and outcome distributions. However, a case has been made for a definition of the epidemiological case of interest based on comorbidity and transdiagnostic latent variables. This definition may forecast a closer integration between the descriptive, analytical and social epidemiology. Also, progress has been made in the field of prevention, but further advances may be possible using the case definition outlined in this paper. The social epidemiology agenda of making treatment more available to the public may move forward through the implementation of web-based technology targeting both therapists and sufferers.

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