REVIEW ARTICLE

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Life adverse experiences in relation with obesity and binge eating disorder: A systematic review

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Background and aims: Several studies report a positive association between adverse life experiences and adult obesity. Despite the high comorbidity between binge eating disorder (BED) and obesity, few authors have studied the link between trauma and BED. In this review the association between exposure to adverse life experiences and a risk for the development of obesity and BED in adulthood is explored. *Methods:* Based on a scientific literature review in Medline, PubMed and PsycInfo databases, the results of 70 studies ($N = 306,583$ participants) were evaluated including 53 studies on relationship between adverse life experiences and obesity, 7 studies on post-traumatic stress disorder (PTSD) symptoms in relation to obesity, and 10 studies on the association between adverse life experiences and BED. In addition, mediating factors between the association of adverse life experiences, obesity and BED were examined. *Results:* The majority of studies (87%) report that adverse life experiences are a risk factor for developing obesity and BED. More precisely a positive association between traumatic experiences and obesity and PTSD and obesity were found, respectively, in 85% and 86% of studies. Finally, the great majority of studies (90%) between trauma and the development of BED in adulthood strongly support this association. Meanwhile, different factors mediating between the trauma and obesity link were identified. *Discussion and conclusions:* Although research data show a strong association between life adverse experiences and the development of obesity and BED, more research is needed to explain this association.

Keywords: trauma, obesity, binge eating disorder

INTRODUCTION

Obesity is one of the major health problems in the United States (Ogden, Yanovsky, Carroll, & Flegal, 2007), being recognized as the leading second cause of death in North America (Stein & Colditz, 2004). The etiology of adult obesity is complex and still unclear, including genetic (Comuzzie & Allison, 1998), behavioral and family factors (Dietz, 1986). In the last decades, empirical and neurological evidence has suggested that adverse childhood experiences occurring in early life, are strongly linked with multiple psychological issues in adulthood (Grilo, Sanislow, Fehon, Martino, & McGlashan, 1999) such as eating disorders (Treuer, Koperdak, Rozsa, & Furedi, 2005; Vanderlinden, Vandereycken, van Dyck, & Vertommen, 1993; Wonderlich et al., 2001), as well as with adult negative physical health outcomes (Anda et al., 2006), including type 2 diabetes (Thomas, Hyppönen, & Power, 2008), metabolic syndrome and heart disease (Lehman, Taylor, Kiefe, & Seeman, 2005; van Reedt Dortland, Giltay, van Veen, Zitman, & Penninx, 2012; Violanti et al., 2006). In this article we review all research data concerning the association between exposure to life adverse experiences and the risk to develop obesity and central adiposity in adulthood, with a particular focus on the mediators of this relationship, such as disordered eating, eating in response to stress, and mood and anxiety disorders. Since the relationship between obesity and binge eating disorders (BED) has been amply established (Bulik & Reichborn-Kjennerud, 2003; Hudson, Hiripi, Pope, & Kessler, 2007), and the fact that approximately 30% of subjects participating in weight control programs have comorbidity with BED syndrome (de Zwaan, 2001), studies investigating the relationship between life adverse experiences and BED were also included.

Definition of adverse experiences

Life adverse experiences are defined as all kinds of traumatic experiences occurring in childhood, adolescence and adulthood, which include emotional abuse, physical abuse, sexual abuse, sexual harassment, rape, bullying by peers, witnessing domestic violence, and serious accidents that threatened the lives of subjects. Exposure to violence in the neighborhood, and exposure to violence in television were excluded to this review.

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METHODS

Inclusion in this review was restricted to studies in people with obesity and/or a diagnosis of BED following American Psychiatric Association criteria (American Psychiatric Association, 1994, 2013). A literature search of articles was conducted in Medline, PubMed and PsycInfo until May 2015. The following keywords were included: overweight, obesity, morbid obesity, waist circumference, waist–hip ratio, abdominal fat, emotional eating, binge eating, and binge eating disorder and childhood abuse, sexual abuse, sexual harassment, physical abuse, physical neglect, physical abuse, bullying by peers, household violence, family violence, post-traumatic stress disorder. We included all the studies in which adverse life experiences were measured by self-reported instruments, clinician interviews or reports of social workers and in which at least one measure of BMI or WC was reported. Studies were divided into 4 groups: (1) studies on traumatic experiences in obese patients; (2) studies on traumatic experiences in obese patients with BED; (3) studies on post-traumatic stress symptoms in adult obese patients; and (4) studies on potential mediators and causal factors explaining the association between life adverse experiences and obesity/BED.

All the studies in this review used the definition of BED syndrome as stated in the DSM-IV and DSM-5 (American Psychiatric Association, 1994, 2000, 2013). BED is characterized by frequent and persistent episodes of binge eating, associated with a strong sense of lack of control over the eating and marked distress in the absence of regular compensatory behaviors. In the fifth edition of the DSM (American Psychiatric Association, 2014), BED is formally recognized as a separate eating disorder. Obesity, overweight and weight gain was evaluated by means of Body Mass Index (BMI) and Waist Circumference (WC).

RESULTS

Study selection

In this review a total of 70 studies were included: 53 studies on the relationship between exposure to trauma and obesity (15 longitudinal studies, 33 cross-sectional studies and 4 case-control studies), 7 studies concerning post-traumatic stress disorder symptoms in relation to obesity and BED (2 longitudinal studies and 5 cross-sectional studies) and 10 research papers regarding the association between trauma and BED (6 cross-sectional studies and 4 case-control studies).

Participants

In total 306,583 subjects (about 203,450 females and 103,133 males) including 50,461 obese individuals and 2,627 participants with BED were included in the analyses.

Adverse life experiences and obesity

Overall, there has been an important interest in this topic. A substantial amount of literature on the topic has been dedicated to investigate the association between interpersonal childhood trauma and subsequent obesity in adulthood. In a previous review Gustafson and Sarwer (2004) show that exposure to childhood sexual abuse is one of the highest risk factors for the development of adult obesity. Another recent review (Midei & Matthews, 2011) highlights that childhood trauma, involving exposure to interpersonal violence may be associated with weight gain, obesity and central adiposity in adulthood.

Cross-sectional studies

The vast majority of studies (Grilo et al., 2005; Grilo, White, Masheb, Rothschild, & Burke-Martindale, 2006; Salwen, Hymowitz, Vivian, & O'Leary, 2014; Wildes, Kalarchian, Marcus, Levine, & Courcoulas, 2008) show that abuse is associated with obesity in later life (see Table 1). Several studies (Grilo et al., 2005, 2006; Salwen et al., 2014; Wildes et al., 2008) report significantly higher levels of different types of abuse (emotional, physical and sexual abuse, and emotional and physical neglect) in two-thirds of a sample of morbid obese patients who underwent gastric bypass surgery compared to a normative sample of adult women, while the rates of maltreatment were roughly two to three times higher in the obese sample. These rates are comparable to those found in psychiatric populations of eating disorders and chronic depression patients (e.g., Carter, Bewell, Blackmore, & Woodside, 2006; Harkness, Bagby, & Kennedy, 2012). Another study (Maddi, Khoshaba, Persico, Bleecker, & VanArsdall, 1997) in a representative sample of morbid obese subjects revealed that a combination of childhood sexual, emotional and physical abuse in the family of origin predicted BMI in adulthood. Clark et al. (2007) reported in a study carried out on a sample of 152 morbid obese patients who underwent gastric bypass a prevalence rate of childhood sexual abuse, adult sexual abuse and childhood physical abuse respectively of 27%, 9% and 19%. Moreover, Sansone, Schumacher, Wiederman, and Routsong-Weichers (2008) found among 121 individuals seeking surgical treatment for obesity finding that 62.7% of respondents indicated having experienced at least one form of childhood abuse. In another study carried out on a sample of bariatric surgery patients Mahony (2010) found prevalence rates of sexual abuse of 15.5%, lower than those reported by other studies that used bariatric surgery patients (Grilo et al., 2005, 2006; Wildes et al., 2008). In two other studies performed on representative large samples (Alvarez, Pavao, Baumrind, & Kimerling, 2007; Rhode et al., 2008), the authors highlighted that both physical and sexual abuse in childhood were positively associated with obesity in adulthood. At the same time Marcus, Bromberg, Wei, Brown, and Kravitz (2007) found that midlife women with an early life history of physical and sexual abuse were more likely to show higher BMI and higher frequency of binge eating than non-abused women. Van Reedt Dortland et al. (2012) found that sexual abuse, physical abuse and emotional abuse independently predicted WC in adulthood. Another series of studies (Aaron & Hughes, 2007; Brewer-Smyth, 2014; Chartier, Walker, & Naimark, 2008; McIntyre et al., 2012; Pinhas-Hamiel, Modan-Moses, Herman-Raz, & Reichman, 2008; Smith et al., 2010)

Table 1. Cross-sectional and case-control studies on the relationship between adverse childhood experiences and development of obesity in adulthood

First author, year	Country/ Study design	Samples	Mean BMI (WC)	Obesity measures	Measures	ACE (*n* or %)	Variables predicted OB	Results
Aaron, 2007	USA CS	*N* = 416 ♀ OB = 90 Age = 37.8	27.8	SR BMI OB = BMI ≥ 30 M-OB = BMI ≥ 35	CSA-Structured interview	CSA = 31%	OB/M-OB = CSA	BMI + in CSA
Alvarez, 2007	USA CS	*N* = 11116 OB = 2509 Age 18–65		SR BMI OB = BMI ≥ 30	TSS	ACE in OB = 29.9%	OB = Tot ACE, CSA & CPA	
Anda, 2006	USA CS	*N* = 17337 (9367 ♀) Age = 57		SR BMI M-OB = BMI ≥ 35	Four questions from CTS ACEs Standardized Questionnaire	CEA = 10.6% CPA = 28.3% CSA = 20.7% DV = 12.7% ≥4 ACE = 12.5%	M-OB = ≥4 ACE	
Bellis, 2014	UK CS	*N* = 1466 (882 ♀) OB = 268 M-OB = 40 Age = 18–70		SR BMI OB = BMI ≥ 30 M-OB = BMI ≥ 40	ACE questions	1 ACE = 287 2–3 ACE = 234 ≥4 ACE = 185	OB = ≥4 ACE	
Brewer-Smyth, 2014	UK CS	*N* = 81 ♀ OB = 35		LM BMI OB = BMI ≥ 30	Muenzenmaier's scale		OB = CSA	
Burke, 2011	USA CS	*N* = 701 (381 ♀) Age = 0–20.9		LM BMI OB = BMI ≥ 85th %	Trauma screen on a chart review Social service report	≥1 ACE = 67.2% ≥4 ACE = 12.0%	OW/OB = ≥4 ACE	≥4 ACE = 38 OB
Buser, 2004	USA CC	*N* = 42 ♀ M-OB CSA = 21 Age CSA = 39.0 Age Non CSA = 36.1	CSA = 56.9 Non CSA = 51.1	LM BMI	Interview	CSA = 21 Non CSA = 21		BMI: CSA = Non CSA
Chartier, 2008	Canada CS	*N* = 8116 (4074 ♀) OB = 829 Age 15–64		SR BMI OB = BMI ≥ 30	Mental Health Supplement of the Ontario Health Survey	CPA in OB = 15.0% CSA in OB = 18.2%	OB = CSA	
Clark, 2007	USA CS	*N* = 152 (111 ♀) Age = 51.3		SR BMI in 73 M-OB LM BMI in 79 M-OB OB = BMI ≥ 30	Semi-structured interview	CSA = 27% ASA = 9% CPA = 19%		
D'Argenio, 2009	Italy CC	*N* = 200 (130 ♀) OB = 65 (42 ♀) OB/PS = 85 (64 ♀) Age OB = 40.4 Age OB/PS = 39.1	OB = 41.3 OB/PS = 38.3	LM BMI OB = BMI ≥ 30	ETLE	ACE in OB = 38.5% ACE in OB/PS = 43.5%	OB = Tot ACE	No ACE differences

(Continued)

Table 1. (Continued)

First author, year	Country/ Study design	Samples	Mean BMI (WC)	Obesity measures	Measures	ACE (*n* or %)	Variables predicted OB	Results
Davis, 2014	USA CS	*N* = 210 (110 ♀) Age = 45.8	30.4	LM BMI OB = BMI ≥ 30 OB = WHR > 2	ELSI AAI	CEA = 23% CPA = 41% CSA = 30% DV = 29%	BMI = ACE WHR = ACE	
Felitti, 1993	USA CS	*N* = 200 (158 ♀) OB = 100 (79 ♀) NW = 100 (79 ♀) Age = 41	>65 pounds MIW	LM of weight Weight = pounds	Clinical interview Nurses information	CSA in OB = 25% CSA in NW = 7% CEA/CN in OB = 29% CEA/CN in OB = 14%		CSA + in OB CEA/CN + in OB
Fuller-Thomson, 2012	Canada CS	*N* = 12590 (6887 ♀) OB = 2787 (1254 ♀) Age = 18–80	N/R	SR BMI OB = BMI ≥ 30	CCHS	♂ CPA = 4.9% ♀ CPA = 9.7%	OB = CPA in ♀	OB + in ♀ CPA
Goedecke, 2013	South Africa CS	*N* = 44 ♀ OB = 24	Black OB = 38.6 White OB = 37.8	LM BMI OB = BMI > 30 NW = BMI < 25	CTQ	Tot CTQ in black OB = 33.2 Tot CTQ in white OB = 45.9 Tot CTQ in black NW = 38.8 Tot CTQ in black NW = 43.1		CTQ: OB = OW
Grilo, 2005	USA CS	*N* = 340 M-OB (282 ♀) BED = 76 M-OB = 203 Age = 51.1	43.1	LM BMI	CTQ	CEA in M-OB = 46.2% CPA in M-OB = 28.8% CSA in M-OB = 31.8% CEN in M-OB = 48.8% CPN in M-OB = 32.1%		CN + in M-OB/BED
Grilo, 2006	USA CS	*N* = 137 M-OB (122 ♀) Age = 42.3	51.8	LM BMI	CTQ	CEA in M-OB = 46.0% CPA in M-OB = 29.2% CSA in M-OB = 32.1% CEN in M-OB = 49.6% CPN in M-OB = 27.7%		No BMI differences
Gunstad, 2006	USA CS	*N* = 696 (339 ♀) OB = 73 (37 ♀) Age = 36.59	OB = 34.8	LM BMI OB = BMI ≥ 30	CAT	CEA in ♂ OB = 17% B in ♂ OB = 30%	BMI = B BMI = CEA ♂ OB = ACE	B + in ♂ OB B + in ♂ OW CEA + in ♂ OB
Hodge, 2014	USA CS	*N* = 459 (336 ♀) Median age = 42	31	OB = BMI ≥ 30 M-OB = BMI ≥ 40	Questions about ACE		BMI = CEA	
Hollingsworth, 2012	Australia CS	*N* = 239 ♀ OB = 63	26.24	LM BMI OB = BMI ≥ 30	CTQ	CEA in OB = 41.3% CPA in OB = 41.9% CSA in OB = 31.9% CEN in OB = 21.4% CPN in OB = 26.3%	OB = CEA, CPA	CEA + in OB CPA + in OB

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Table 1. (Continued)

First author, year	Country/ Study design	Samples	Mean BMI (WC)	Obesity measures	Measures	ACE (*n* or %)	Variables predicted OB	Results
Jia, 2004	USA CS	*N* = 237 ♀ OB = 53 Age = 38.9	OB = 40.1	LM BMI OB = BMI ≥ 30	CPA-Structured interview CSA-Structured interview	CPA in OB = 34 CSA in OB = 28	OB = CPA OW = CPA	CSA + in OB/OW CPA + in OB/OW
Kestilä, 2009	Finland CS	*N* = 1369 (618 ♀) M-OB = 20 (12 ♀) OB = 101 (40 ♀) Age 18–29		SR BMI M-OB = BMI ≥ 35 OB = BMI ≥ 30	Health Examination Survey Structured interviews Self-administered questionnaires	♀ B = 28% ♂ B = 21%	♀ OB = B	
Knutson, 2010	USA CS	*N* = 571 (282 ♀) OB = 93 OW = 85 Age = 6.3		LM BMI OB ≥ 95th %	56 items about care neglect HEQ		BMI = CN	
Larsen, 2005	the Netherlands CS	*N* = 157 M-OB (144 ♀) Age = 40	CSA = 45.1 Non CSA = 45.7	LM BMI M-OB = BMI ≥ 40	Questions about CSA	CSA = 23%		BMI: CSA = Non CSA
Maddi, 1997	USA CS	*N* = 1027 M-OB (855 ♀) Age = 37.31	46.13	LM BMI M-OB = BMI ≥ 35	Interview	CPA in OB = 11.6% CEA in OB = 10.0% CSA in OB = 12.1% I in OB = 2.3%	BMI = CEA, CPA, CSA	
Mahony, 2010	USA CS	*N* = 573 OB (419 ♀) Age = 40.14			PsyBari	CSA = 89 CPA = 133		
Marcus, 2007	USA CS	*N* = 589 ♀ OB = 213 OW = 176 Age = 42–55	N/R	LM-BMI & WC OB = BMI ≥ 30 OB = WC ≥ 88	12-item questionnaire from National Comorbidity's Study modification of the revised Diagnostic Interview Survey PTSD section	CPA/CSA = 15.6%	BMI = CPA/CSA	
McIntyre, 2012	Canada CS	*N* = 373 (230 ♀) MS = 83 (50 ♀) Age = 42.9	N/R	LM BMI & WC OB in ♀ = WC > 102 OB in ♂ = WC > 88 OB = BMI > 30	Klein Trauma & Abuse-Neglect self-report scale	CPA = 21.9% CSA = 24.9% DF = 39.0% Any ACE = 46.7%		OB + in CSA
Oppong, 2006	USA CS	*N* = 258 M-OB (208 ♀) Age = 40	Non CSA = 52 CSA = 51	LM BMI OB = BMI ≥ 30 M-OB = BMI ≥ 35	Questionnaire on ACE	♀ CSA = 29.6% ♂ CSA = 12.2%		BMI: CSA = Non CSA
Pinhas-Hamiel, 2008	Israel CS	*N* = 145 ♀ OW OB = 42 Age = 10.4	29.9	LM BMI OW = BMI > 95th % M-OB = BMI ≥ 35	Social worker and psychologist interview	CSA in OW = 3.5% CSA in OB = 9.5%		OB + in CSA Δ BMI + in CSA
Rhode, 2008	USA CS	*N* = 4641 ♀ OB = 3251 Age = 52		SR BMI OB = BMI ≥ 30	Four questions from CTQ	CSA = 904 CPA = 709	OB = CSA/CPA	

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Table 1. (Continued)

First author, year	Country/ Study design	Samples	Mean BMI (WC)	Obesity measures	Measures	ACE (*n* or %)	Variables predicted OB	Results
Salwen, 2014	USA CS	*N* = 187 (67 ♀) OB = 29 M-OB = 158 Age = 43.58	45.90	LM BMI OB = BMI ≥ 30 M-OB = BMI ≥ 40	CTQ-short form	Severe CEA = 10.2% CPA = 8% CSA = 4.8% CEN = 8% CPN = 9.6% AIA = 30.5% Any ACE = 61.0%		
Sansone, 2008	USA CS	*N* = 121 OB (104 ♀) Age = 44.6	47.2	LM BMI OB = BMI ≥ 30	Questions about ACE	CEA = 43% CSA = 19% CPA = 17.4% CPN = 9.1% DV = 39%		
Schneiderman, 2012	USA CC	*N* = 454 (231 ♀) ACE = 303 (151 ♀) CG = 151 (60 ♀) Age = 10.93	N/R	LM BMI OB ≥ 95th % OW = 85–95th %	Child Welfare Case Records	CEA = 156 (83 ♀) CPA = 156 (67 ♀) CSA = 64 (41 ♀) CN = 232 (111 ♀)		BMI: ACE Group = CG CG > ACE Group in OB/OW
Smith, 2010	USA CS	*N* = 867 ♀ OB = 310 Age = 47.6	N/R	LM BMI M-OB = BMI ≥ 40 S-OB = BMI ≥ 35 OB = BMI ≥ 30	Three questions about SA		OB = Any SA	
Taylor, 2006	USA CS	*N* = 455 ♀ Age = 20.8	23.7	LM BMI OW = BMI 25–32	CTQ Questions of negative comments about shape and weight	NC = 114 OW NC = 40–50% 152 OW > 90th % on CEA, CEN & CPN	MBS = NC, CEN	
van Reedt Dortland, 2012	the Netherlands CS	*N* = 2755 (1829 ♀) Age = 41.9	WC = 87.0	LM WC	Childhood Trauma Interview used in the Netherlands Mental Survey and Incidence Study	CEA = 10.8% CPA = 2.8% CSA = 1.3% CEN = 20.4%	WC = CPA, CSA & CEA	
Whitaker, 2007	USA CS	2412 children Age = 3	N/R	LM BMI OB = BMI > 95th %	CTSPSC	CEA = 94% CPA = 84% CN = 11%	OB = CN	
Wildes, 2008	USA CS	*N* = 230 M-OB (191 ♀) Age = 44.8	51.4	LM BMI M-OB = BMI ≥ 40 OB = BMI ≥ 30	CTQ	Tot ACE = 151 CEA = 109 CSA = 71 CEN = 71		CEA + in ♀ OB CSA + in ♀ OB

Index: AAI = Adult Attachment Interview; CAT = Child Abuse and Trauma Scale; CCHS = Canadian Community Health Survey; CTQ = Childhood Trauma Questionnaire; CTSPSC = Parent–Child Conflict Tactics Scales; CTS = Conflict Tactic Scale; ELSI = Evaluation of Lifetime Stressors Interview; ETLE = Early Traumatic Life Events; HEQ = The Home Environment Questionnaire; TSS = Traumatic Stress Schedule; AIA = Adult Interpersonal Abuse; ACE = Adverse Childhood Experiences; CSA = Childhood Sexual Abuse; CPA = Childhood Physical Abuse; CEN = Childhood Emotional Neglect; CPN = Childhood Physical Neglect; CEA = Childhood Emotional Abuse; ASA = Adult Sexual Abuse; DV = Domestic violence against mother; D/S = Divorce/ Separation of parents; I = Incarcerated household members; B = Bullied/rejected; DF = Death of family members; NC = Negative comments in family; S-OB = Severe obese; M-OB = Morbid obese; OB = Obese; OB/PS = Obese with psychiatric disorders; NW = Normal weight; BMI = Body Mass Index (kg/m²); WC = Waist Circumference (cm); WHR = Waist–Hip Ratio; MBS = Maximum Body Size (kg).

confirm previous findings showing a strong association between childhood sexual abuse with obesity, suggesting that this variable may be an important risk factor for becoming obese in adulthood.

Despite the large amount of studies, demonstrating a strong association between traumatic experiences and the development of obesity, some studies show some conflicting data. For instance, Jia, Li, Leserman, Hu, and Drossman (2004) showed that in obese and overweight patients who reported a history of physical or sexual abuse only the rates of physical abuse significantly increased the odds of being overweight. Some other studies (Goedecke, Forbes, & Stein, 2013; Larsen & Geenen, 2005; Oppong, Nickels, & Sax, 2006), showed that sexually abused patients did not differ significantly in BMI compared with patients reporting no sexual abuse.

In a cross-sectional study performed on a sample of pre-pregnancy women Hollingsworth, Callaway, Duhig, Matheson, and Scott (2012) found that the rates of childhood emotional and physical abuse were higher in obese women compared with normal weight women. Furthermore, these authors revealed that pre-pregnancy obesity was associated with a self-reported history of emotional or physical abuse. On the other hand, Fuller-Thomson, Sinclair, and Brennenstuhl (2012) in a cross-sectional study carried out in a large and representative sample of adult subjects found that among women with childhood physical abuse compared to non-abused women, the odds of obesity were 35% higher, while childhood physical abuse was not associated with adult obesity among men. Two other studies clearly showed that being bullied or rejected during childhood predicted BMI and obesity in adulthood (Gunstad et al., 2006; Kestilä, Rahkonen, Martelin, Lahti-Koski, & Koskinen, 2009). Three cross-sectional studies (Anda et al., 2006; Bellis, Lowey, Leckenby, Hughes, & Harrison, 2014; Burke, Hellman, Scott, Weems, & Carrion, 2011) showed that children who were exposed to different types of adverse life experiences have a higher risk to develop overweight, obesity and morbid obesity in adulthood compared with subjects without traumatic experiences in childhood. Consistent with these studies Davis et al. (2014) found that different types of adverse life experiences predicted central obesity measured respectively with waist–hip ratio and BMI.

In contrast with the great interest in physical, and sexual abuse, only few studies focused on the impact of childhood neglect documenting a positive association between parental neglect in childhood and increased risk of obesity both in childhood (Knutson, Taber, Murray, Valles, & Koeppl, 2010; Taylor et al., 2006) and adulthood (Whitaker, Phillips, Orzol, & Burdette, 2007). One cross-sectional study reported that only verbal abuse but not sexual and physical abuse were predictive of adult obesity (Hodge, Stemmler, & Nandy, 2014).

Case-control studies

Only a few case-control studies were carried out (see Table 1). One case-control study (Felitti, 1993) showed that obese patients report a higher incidence of different childhood traumatizing life events compared with and age and sex matched control sample. Another case-control study (D'Argenio et al., 2009), comparing obese subjects to both obese subjects with a current psychiatric diagnosis, and to non-obese and non-psychiatric subjects, showed that also marital conflicts and separation of one or both parents, may be a risk factor for developing obesity during adulthood. Again some studies show some contrasting findings and do not find a higher risk to develop obesity in obese patients with a history of trauma compared to a non-trauma sample (Buser, Dymek-Valentine, Hilburger, & Alverdy, 2004; Schneiderman, Mennen, Negriff, & Trickett, 2012).

Longitudinal studies

Several longitudinal studies report a positive association between abuse and later obesity (see Table 2). Two studies (Midei, Matthews, & Bromberger, 2010; Power, Pinto, & Li, 2015) showed that childhood physical and sexual abuse and childhood neglect were positively associated with obesity, BMI and WC increase in adulthood. Another study (Thomas et al., 2008) revealed that verbal abuse and physical abuse was related with increases of both BMI and WC in adulthood, and that physical punishment, neglect, witnessing violence and living in a conflictual family, predicted overall the growth of WC in adulthood. At the same time another longitudinal study (Bentley & Widom, 2009) found that only physical abuse predicted the growth of BMI. Boynton-Jarrett, Rosenberg, Palmer, Boggs, and Wise (2012) found that in comparison with non-abused women, those reporting severe physical and/or sexual abuse had the highest ratios of obesity and waist circumference, and that both severe sexual and physical abuse were positively associated with body weight. Two other longitudinal studies (Greenfield & Marks, 2009; Williamson, Thompson, Anda, Dietz, & Fellitti, 2002) corroborate the finding that in adults reporting childhood physical, sexual and psychological violence from parents, greater odds of obesity was observed.

Noll, Zeller, Trickett, and Putnam (2007) showed that sexually abused women were 2.85 times more likely to be obese during young adulthood compared to non-abused women. Some longitudinal studies (Johnson, Cohen, Kasen, & Brook, 2002; Lissau & Sørensen, 1994; Shin & Miller, 2011; Vámosi, Heitmann, Thinggaard, & Kyvik, 2011) clearly show that the prevalence of obesity and greater rates of increase of BMI were significantly higher among subjects who had experienced neglect in the prior years compared to those growing up in a supportive and harmonious family environment. Only one study (Fuemmeler, Diedert, McClemon, & Beckham, 2009) showed that only in men, but not in women, obesity and overweight were positively associated with early life history of sexual abuse. Finally, Sweeting, Wright, and Minnis (2005), in line with the findings of Gunstad et al. (2006) and Kestilä et al. (2009) found that victimization at school was positively related to obesity at age 11 but that this association disappeared at age 15. Only one longitudinal study is in conflict with previous findings (Bennett, Sullivan, Thompson, & Lewis, 2010) since the chronicity of neglect was associated with lower BMI at 7 and 9 years but was unrelated to BMI at an earlier age.

Table 2. Longitudinal studies on the relationship between adverse childhood experiences and development of obesity in adulthood

First author, year	Country	Samples (follow-up)	Mean BMI/WC	Obesity measures	Measures	Prevalence of ACE (*N* or %)	Variables predicted OB	Results
Bennett, 2010	USA	*N* = 185 (103 ♀) Age = 6.1	N/R	LM BMI BMI ≥ 30 BMI ≥ 95th %	Mother interview CTSPSC Child Protective Service allegation	CN = 91 CPA = 12		BMI – in CN
Bentley, 2009	USA	*N* = 807 (426 ♀) OB in ACE = 294 CG = 303 Age (T2) = 41	CPA = 31.9 CSA = 30.7 CN = 29.7	LM BMI SR BMI (*n* = 180) BMI ≥ 30	Official records	CPA = 68 CSA = 54 CN = 335	OB = CPA	
Boynton-Jarrett, 2012	USA	N = 33298 ♀ OB = 14418 Age = 49 (21–69)	No ACE = 30 CPA/CSA = 31 CPA + CSA = 32	SR BMI and WC OB = BMI ≥ 30 OB = WC ≥ 35 inch	CTS PAAS	CPA/CSA = 3513 CPA + CSA = 804	Δ BMI = CPA/CSA Δ BMI = CPA + CSA Δ WC = CPA/CSA	
Fuemmeler, 2009	USA	*N* = 15197 OB = 1778 (905 ♀) Age = 22.0	N/R	SR BMI BMI ≥ 30 BMI ≥ 35	Survey	CSA in ♀ OB = 4.4% CPA in ♀ OB = 16.2% CN in ♀ OB = 5.3% CSA in ♂ OB = 6.9% CPA in ♂ OB = 13.8% CN in ♂ OB = 6.3%		CSA + in ♂ OB CSA + in ♂ OW
Greenfield, 2009	USA	*N* = 1650 (891 ♀) OB = 478 Age = 56.6	N/R	SR BMI BMI ≥ 30	Items from modified version of CTS	Rarely CPA or CEA = 18% Frequently CPA or CEA = 6% Frequently CPA & rarely CEA = 12% Frequently CPA + CEA = 22%	OB = frequently CPA + CEA	
Johnson, 2002	USA	*N* = 782 (385 ♀) OB = 93 Age = 22	N/R	SR BMI	Official records Parental Interview Neglect Scale	CN in OB = 21%	OB = CN	OB + in CN
Lissau, 1994	Denmark	*N* = 756 OB = 38 Age = 19–20	N/R	LM BMI (T1) SR BMI (T2) BMI > 95th % BMI > 30	Teachers and parents questionnaire	CN in OB = 18%	OB = CN	

(Continued)

Table 2. (Continued)

First author, year	Country	Samples (follow-up)	Mean BMI/WC	Obesity measures	Measures	Prevalence of ACE (*N* or %)	Variables predicted OB	Results
Midei, 2010	USA	*N* = 311 ♀ OB = 109 Age = 45.7	28.6 (87.6 cm)	LM BMI & WC BMI ≥ 30	CTQ	Tot ACE = 36.0% CEA = 19.6% CPA = 16.7% CSA = 14.1% CEN = 6.4% CPN = 14.8%		Δ BMI + in ACE Δ BMI + in CSA Δ BMI + in CPA Δ WC + in ACE Δ WC + in CSA
Noll, 2007	USA	*N* = 173 ♀ OB in CSA = 35 OB in CG = 25 Age = 6–27	N/R	LM BMI BMI ≥ 30	Substantiated abuse through child Protective Services	CSA = 84	OB = CSA	Δ BMI + in CSA OB + in CSA
Power, 2015	UK	*N* = 17638 OB = 2101 (952 ♀) Age = 45	♂ = 28.1 ♀ = 26.8	LM BMI SR BMI OB = BMI > 95th % PATH OB = BMI ≥ 30	Interview of parents	CPA in ♀ = 6.14% CEA in ♀ = 11.7% CSA in ♀ = 2.71% CN in ♀ = 18.5% CPA in ♀ = 5.95% CEA in ♀ = 8.29% CSA in ♀ = 0.48% CN in ♀ = 21.8%	♂ OB = CPA ♀ OB = CPA, CSA Δ BMI + in ♂ = CPA CN Δ BMI + in ♀ = CPA, CSA, CN	
Shin, 2011	USA	*N* = 8471 Age = 28.3	29.0	SR BMI	CASI Interview	CPA = 11% CSA = 1% CN = 22%	Δ BMI = CN	
Sweeting, 2005	Scotland	*N* = 2127 (1043 ♀) OB = 139 (77 ♀) Age = 15	♀ = 24.2 ♂ = 25.6	LM BMI BMI > 95th %	2 items on B	B in ♀ at 11 years = 21.8% B in ♂ at 11 years = 29.8% B in ♀ at 15 years = 12.4% B in ♂ at 15 years = 9.9%	OB at 11 years = B Δ BMI = B	
Thomas, 2008	UK	*N* = 9310 (4658 ♀) OB = 2227 (1104 ♀) Age = 45	♀ = 27.5 ♂ = 26.5	LM BMI & WC BMI ≥ 30 WC in ♂ ≥ 102 cm WC in ♀ ≥ 88 cm	PBI ACEs Study ACE-reports of social workers Parents and teachers interview	CEA = 1420 CPA = 558 CSA = 147 CN = 1425 PP = 705 DV = 557 CF = 366	Δ BMI = CEA, CPA Δ WC = CN, PP, DV, CF	

(Continued)

Table 2. (Continued)

First author, year	Country	Samples (follow-up)	Mean BMI/WC	Obesity measures	Measures	Prevalence of ACE (*N* or %)	Variables predicted OB	Results
Vámosi, 2011	Denmark	*N* = 236 (159 ♀) ♀ Age = 41.4 ♂ Age = 40.4	♀ = 29.5 ♂ = 28.8	SR BMI OB = BMI ≥ 30	CECA.Q	Maternal CN = 50 Paternal CN = 105 Maternal antipathy = 37 Paternal antipathy = 41	OB at age 40 = Maternal CN OB at age 40 = Maternal antipathy	
Williamson, 2002	USA	*N* = 13177 (6720 ♀) OB = 3294 M-OB = 1014 Age = 55.7	27.4	LM BMI BMI ≥ 30 BMI ≥ 40	ACEs study survey	Any ACE = 1318 CEA = 6238 CPA = 2710 CSA = 2860	OB/M-OB = Any ACE, 4 ACE M-OB = CEA, CPA	

Index: CECA.Q = The Childhood Experience of Care and Abuse Questionnaire; CTQ = Childhood Trauma Questionnaire; CTS = Conflict Tactic Scale; CTSPSC = Parent–Child Conflict Tactics Scales; PAAS = Pregnancy Abuse Assessment Scale; PATH = Personality and Total Health Through Life Project; PBI = Parental Bonding Inventory; ACE = Adverse Childhood Experiences; CSA = Childhood Sexual Abuse; CPA = Childhood Physical Abuse; CN = Childhood Neglect; CEN = Childhood Emotional Neglect; CPN = Childhood Physical Neglect; CEA = Childhood Emotional Abuse; PP = Physical Punishment; DV = Domestic violence against mother; B = Bullyed/rejected; M-OB = Morbid obese; OB = Obese; OB/PSY = Obese with psychiatric disorders; BMI = Body Mass Index (kg/m^2); SR BMI = Self-reported BMI; LM BMI = Laboratory measure of BMI; WC = Waist Circumference (cm).

Table 3. Studies on the relationship between PTSD and development of OB/BED in adulthood

First author, year	Country/ Study design	Samples	Mean BMI	Obesity measures	Measures	PTSD/ACE (*N* or %)	Variables predicted OB	Results
Dedert, 2010	USA CS	*N* = 148 ♀ OB = 43 OW = 50 Age = 39.10	30.9	LM BMI OB = BMI > 30 OW = BMI > 25	TLEQ DTS	PTSD = 49% CSA = 45% CPA = 46%	BMI = CSA, CPA BMI = PTSD WC = PTSD	OB + in CSA OB + in CPA
Duncan, 2015	USA L	*N* = 3699 ♀ OB = 497 Age = 15–24		SR BMI OB = BMI ≥ 30	Standard traumatic event checklist adapted from the NCS	PTSD in OB = 7.4% CSA in OB = 25.3% CPA in OB = 31.2% CN in OB = 6.0%	OB = CSA OB = CSA+CPA +CN OW = CSA, CPA	CSA + in OB
Grilo, 2012	USA CS	*N* = 105 ♀ BED Age = 42.7	38.4	LM BMI OB = BMI ≥ 30	SCID-I/P EDE for BED	PTSD = 25		No BMI differences
Kubzansky, 2014	USA	*N* = 50504 ♀ OB = 5250 OW = 9019 Age = 34.5	23.6–24.1	LM BMI OB = BMI ≥ 30 OW = BMI ≥ 25	Modified BTQ 7-item screening scale for DSM-IV PTSD	ACE + PTSD = 51.1% Only ACE = 30.3%	OB/OW = ACE + PTSD	ACE + PTSD > Only ACE in OB/OW ACE + PTSD > Only ACE in Δ BMI
Pagoto, 2012	USA CS	*N* = 20013 (10527 ♀) BED = 440 OB = 4863 Age = 44.2		SR BMI Class I OB = BMI ≥ 30 Class II OB = BMI ≥ 35 Class III OB = BMI ≥ 40	CIDI for PTSD CIDI for BED	1 year PTSD = 3.4% Past PTSD = 3.3%	OB = PTSD in ♀ & ♂	OB + in 1 year PTSD
Roenholt, 2012	Denmark CS	*N* = 2981 (1425 ♀) OB = 197 OW = 650 Age = 28	23.8	SR BMI OB = BMI ≥ 30	4 PTSD questions	1/2 symptoms of PTSD = 21% 3/4 symptoms of PTSD = 6% CEA = 9% CSA = 2% CN+CPA+CEA = 2%	OB = CN + CPA + CEA	PTSD + in OB ACE + in OB & OW
Violanti, 2006	USA CS	*N* = 101 (40 ♀)	♀ WC = 80.6 ♂ WC = 97.2	LM WC WC > 88 in ♀ WC > 102 in ♂	IES	Severe PTSD = 6 Moderate PTSD = 23 Mild PTSD = 19 Sub-clinical PTSD = 53		WC + in Severe PTSD WC > 88 in 9 ♀ WC > 102 in 23 ♂

Index: BTQ = Brief Trauma Questionnaire; CIDI = World Health Organization Composite International Diagnostic Interview; DTS = Davidson Trauma Scale; IES = The Impact of Event Scale; NCS = National Comorbidity Scale; SCID = Structured Clinical Interview for DSM Axis I Disorders; TLEQ = Traumatic Life Events Questionnaire; ACE = Adverse Childhood Experiences; CSA = Childhood Sexual Abuse; CPA = Childhood Physical Abuse; CN = Childhood Neglect; CEA = Childhood Emotional Abuse; OB = Obese; OW = Overweight; NW = Normal weight; UW = Underweight; BMI = Body Mass Index (kg/m^2); SR BMI = Self-reported BMI; LM BMI = Laboratory measure of BMI; WC = Waist Circumference (cm); IES = Impact of Event Scale; TLEQ = Traumatic Life Events Questionnaire.

Table 4. Studies on the relationship between adverse childhood experiences and development of BED in adulthood

First author, year	Country/ Study design	Samples	Mean BMI	Obesity measures	Measures	ACE in BED	Variables predicted OB/BED	Results
Allison, 2007	USA CC	*N* = 271 (204 ♀) BED = 176 (139 ♀) NES = 57 (41 ♀) OB = 38 (24 ♀) Age of BED = 44.9	BED = 35.6	LM BMI	CTQ EDE SCID-I/P	Tot ACE = 82% CEA = 54% CPA = 31% CSA = 29% CEN = 69% CPN = 50%		Tot ACE + in BED/ NES CEA + in BED/NES CN + in BED No differences in BMI
Becker, 2011	USA CS	*N* = 137 ♀ BED Age = 43.9	35.7	LM BMI OB = BMI ≥ 30	CTQ SCID-I/P EDE	CEA = 71 CPA = 38 CSA = 42 CN = 156	OB onset = CPA	
Dalle Grave, 1997	Italy CC	*N* = 64 ♀ OB BED = 29 OB = 35 Age = 36.4	*N* = 35.7 BED = 35.0 OB = 36.3	LM BMI OB = BMI > 30	Clinical Standardized Interview for Trauma Semistructured Interview for BED diagnosis	ACE in OB = 26% ACE in OB/BED = 41.3%		ACE + in OB-BED
Gabert, 2013	Canada CS	*N* = 500 M-OB (441 ♀) BED = 148 Age = 43.7	47.9	LM BMI M-OB = BMI ≥ 35	Interview about CSA	CSA in ♀ = 23.6% CSA in ♂ = 8.5%		BED + in CSA CSA = non CSA in BMI
Grilo, 2001	USA CC	BED = 145 (111 ♀) OB/BED = 105 NS = 1125 ♀ Age BED = 43.6	BED = 37.9	LM BMI OB = BMI ≥ 30	CTQ SCID-I EDE-Q CPA = 35.8% QEWP-R	Any ACE = 82.8% CEA = 59.3% CPA = 35.8% CSA = 30.3% CEN = 69% CPN = 48.6%		OB/BED = BED in any ACE BED > NS in Any ACE
Grilo, 2002	USA CC	BED = 116 (90 ♀) NS = 1125 ♀ Age BED = 44.2 Age NS = 42			CTQ SCID-I	Any ACE = 81.9% CEA = 52.6% CPA = 31.9% CSA = 25.9% CEN = 67.2% CPN = 49.6%		BED > NS in Any ACE
Knoph Berg, 2011	Norway CS	*N* = 45644 ♀ BED = 931 Age = 29.9		SR BMI	2 questions about CPA 2 questions about CSA Diagnostic algorithms based on DSM-IV for BED At least 1 binge per week	CPA in BED = 23.6% CSA in BED = 24.0%	BED = CSA, CPA	

(Continued)

Table 4. (Continued)

First author, year	Country/ Study design	Samples	Mean BMI	Obesity measures	Measures	ACE in BED	Variables predicted OB/BED	Results
Mitchell, 2012	USA CS	$N = 5692$ (3310 ♀) BED = 105 (75 ♀) Age ♂ = 43.2 Age ♀ = 44.0			CIDI	Any ACE = 90.3% Any Int-ACE = 63.7% R = 19.5% CSA = 34.8% CPA = 13.71% DV = 30.8% Stalking = 33.8%		Any ACE + in BED Any Int-ACE + in BED CSA + in BED R + in ♂ BED CPA + in BED DV + in BED Stalking + in BED
Pike, 2006	USA CC	BED = 162 ♀ (101 OB) CG = 162 ♀ (32 OB) PC = 107 ♀ (23 OB) Age BED = 30.8 Age CG = 30.0 Age PC = 20.5	BED = 34.6 NC = 25.6 PC = 25.9	LM BMI OB = BMI ≥ 30	RFI SCID-IV Abbreviated version of EDE	CSA = 8.6% CPA = 16.7% NC = 42.9%	BED = > 3 ACE	CSA + in BED CPA + in BED NC + in BED
Striegel-Moore, 2002	USA CC	BED = 162 ♀ CG = 251 ♀ Age BED = 30 Age CG = 30			Oxford assessment	CSA = 56.7% CPA = 65% B = 46.7%		CSA + in BED CPA + in BED B + in BED

Index: EDE = Eating Disorder Examination; CTQ = Childhood Trauma Questionnaire; SCID-I/P = Structured Clinical Interview for DSM-IV-TR Axis I Disorders Patient edition; SCID-I = Structured Clinical Interview for DSM-IV-TR Axis I Disorders; SCID-IV = Structured Clinical Interview for DSM-IV-TR Disorders; ACE = Adverse Childhood Experiences; CSA = Childhood Sexual Abuse; CPA = Childhood Physical Abuse; CN = Childhood Neglect; CEA = Childhood Emotional Abuse; DV = Domestic violence; B = Bullied/rejected; R = rape; NC = Negative comments in family about shape and weight; BED = Binge Eating Disorder; BEB = Binge Eating Behaviors; M-OB = Morbid obese; OB = Obese; BMI = Body Mass Index (kg/m^2); SR BMI = Self-reported BMI; LM BMI = Laboratory measure of BMI; WC = Waist Circumference (cm); QEWP-R = Questionnaire of Eating and Weight Patterns-Revised; CIDI = World Health Organization Composite International Diagnostic Interview (CIDI) for DSM-IV and ICD-10 diagnosis; RFI = Oxford Risk Factor Interview.

Post-traumatic stress disorder and obesity

Some studies evaluated the presence of a post-traumatic stress disorder (PTSD) in adult obese patients and this way indirectly measured the presence of trauma (see Table 3). Violanti et al. (2006) showed that waist circumference in police officers exposed to some form of traumatic experience was related to PTSD symptoms. In line with these findings, other researchers found a significant association between obesity and PTSD symptoms (Duncan et al., 2015; Pagoto et al., 2012), and that both PTSD symptoms and major depressive symptoms had a significant effect on BMI and waist–hip ratio (Dedert et al., 2010). Roenholt, Beck, Karsberg, and Elklit (2012) also found a strong positive association between BMI and the presence of PTSD symptoms. Moreover, Grilo, White, Barnes, and Masheb (2012) found in a sample of obese patients with BED a great number of subjects had comorbid PTSD. Grilo et al. (2012) also demonstrated that BED patients with a diagnosis of PTSD had a greater percentage of binge eating behaviors compared with BED patients without PTSD. In another longitudinal study Kubzansky et al. (2014) demonstrated that women with PTSD symptoms reported significantly more both overweight and obesity compared with women with no PTSD symptoms. Moreover, BMI trajectories over time indicated a faster rate of weight gain among women who experienced trauma or PTSD symptoms relative to women who never experienced trauma or PTSD.

Adverse life experiences in binge eating disorder (BED)

In contrast with the large number of obesity studies, only 10 studies on the relationship between childhood maltreatment and BED are published (see Table 4). In one of the first case-control studies, Dalle Grave, Oliosi, Todisco, and Vanderlinden (1997) reported significantly higher rates of childhood trauma in BED compared to an OB sample. Pike et al. (2006) investigated the occurrence of stressful life events at least 12 months before the onset of BED ($N = 162$ BED patients). Compared to a normal control sample, BED subjects reported more physical abuse and critical comments about shape and weight, compared to psychiatric subjects. In another series of studies, high prevalence rates of abuse in BED are reported namely between 35% and 82% (Allison, Grilo, Masheb, & Stunkard, 2007; Grilo & Masheb, 2001, 2002) reaching peaks of 90% (Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Mitchell et al. (2012) found that compared to a normal sample, BED patients reported significantly more exposure to any form of interpersonal trauma. Striegel-Moore, Dohmn, Pike, Wilfley, and Fairburn (2002) in a case-control study carried out in 60 black and 102 white women with BED, showed that both white and black women with BED reported significantly higher scores of sexual abuse, physical abuse, and bullying by peers than a healthy comparison group. Rates of sexual abuse were significantly higher in black women with BED than those of a psychiatric group. Allison et al. (2007) studied the relationship between childhood emotional neglect and abuse in 176 women with BED compared to 57 women with night eating syndrome (NES) and to 37 women with OB. In this study the rates of both emotional abuse and emotional neglect were significantly higher in BED women, compared to the other two groups, whereas physical neglect was higher in NES women compared to BED women. In two cross-sectional studies (Grilo & Masheb, 2001, 2002) obese-BED patients reported two to three times higher rates of both emotional and physical neglect and emotional, physical and sexual abuse compared to the rates of a normative sample. However, Grilo and Masheb (2001) showed that none of the 5 forms of maltreatment were associated with age at onset of the first binge eating episode and with the severity of both obesity and binge eating behaviors. One cross-sectional study (Becker & Grilo, 2011), showed that obese women with BED who reported sexual, physical and emotional abuse, had higher BMI and waist circumference compared to non-abused women. Moreover, all of these forms of childhood abuse in the family of origin were positive predictors of later obesity. On the other hand, Knoph Berg et al. (2011) in a large and representative sample of pregnant women found that physical abuse and sexual abuse increased the incidence of BED respectively of 1.68- and 1.57-fold. Finally, in another cross-sectional study in a large sample of morbidly obese patients Gabert et al. (2013) found that 21.8% of patients reported sexual abuse and that the BED diagnosis was significantly higher in this patient sample. All these findings further support the hypothesis that childhood maltreatment may be associated with a greater risk to develop BED symptomatology.

POTENTIAL MEDIATORS AND MECHANISM INVOLVED IN THE RELATIONSHIP BETWEEN ADVERSE LIFE EXPERIENCES AND OBESITY

Since the mechanism by which traumatic experiences are linked with obesity has not yet been clarified, several studies have looked at possible mediating variables which could explain this link. According to some authors, PTSD symptoms could play an important role to understand the mechanism underlying the association between traumatic experiences and development of obesity in adulthood (Dedert et al., 2010; Heppner et al., 2009; Mitchell et al., 2012; Roenholt et al., 2012). Mitchell et al. (2012) also conclude that PTSD symptoms may in part explain the association between trauma and BED. Abuse-related PTSD symptoms are associated with hyper-activation of HPA axis and with subsequent increases in peripheral cortisol, which in turn have been linked to accumulation of fat in adipose tissues with a consequent increase in abdominal obesity (Glaser, 2000; Pasquali, Vicennati, Cacciari, & Pagotto, 2006). In line with these findings, the hyperactivation of HPA axis with an exaggerated cortisol response to stress, has been observed in obese patients (Marin et al., 1992), and were also put in relation with stress-induced eating (Vicennati, Pasqui, Cavazza, Pagotto, & Pasquali, 2009), with night eating syndrome (NES) (Birketvedt et al., 1999), and with waist adiposity in BED patients (Gluck, Geliebter, & Lorence, 2004).

Another mediating factor can be the presence of dissociative symptoms. Although few studies have analyzed the relationship between trauma, dissociation and BED, some

authors have been suggesting that dissociation may play an important mediating role between the presence of early trauma and the development of eating disorders (Beato, Rodriguez, & Belmonte, 2003; McShane & Zirkel, 2008; Oliosi & Dalle Grave, 2003; Treuer et al., 2005; Vanderlinden et al., 1993). In agreement with the theory of escape from self-awareness (Heatherton & Baumaister, 1991), it is hypothesized that when negative emotional states are activated, a shift towards lower levels of cognition and self-awareness is initiated, which involves cognitive processes similar to dissociation. This mechanism tends to remove the inhibitions, thereby facilitating the start of binge eating or overeating, both in clinical (Engelberg, Steiger, Gauvin, & Wonderlich, 2007; Heatherton & Baumaister, 1991; Vanderlinden et al., 1993) and in non-clinical subjects (Lyubomirsky, Casper, & Sousa, 2001). In line with this interpretation, some studies showed a positive association between trauma, dissociative symptoms and BED. For instance, Dalle Grave et al. (1997) highlighted that obese subjects with a diagnosis of BED had higher scores of both early traumatic experiences and dissociation compared to obese non-BED subjects, and that dissociation was significantly associated with traumatic experiences. Recently Rodriguez-Srednicki (2001) found that the presence of childhood sexual abuse was the strongest predictive factor of binge eating episodes, and that dissociation fully mediated the relationship between childhood abuse and the severity of binge eating symptoms. In another study dissociation was the only variable able to predict the frequency of binge eating episodes (La Mela, Maglietta, Castellini, Amoroso, & Lucarelli, 2010). These studies seem to support the hypothesis that dissociation may have a mediating role in the abuse and binge eating link.

Other researchers identified some specific psychological variables that function as mediators in the relationship between childhood abuse, obesity and BED, such as depression (Moyer, Di Pietro, Berkowitz, & Stunkard, 1997), trait anger (Midei et al., 2010) and perceived stress (Alvarez et al., 2007). Moyer et al. (1997) even suggest that depression may be the only significant variable explaining the link between childhood abuse and adult obesity. Depression is consistently associated with obesity and central obesity (Katz et al., 2000). A possible way to interpret the link between childhood abuse, depression and obesity are emotional eating. Some studies have shown that childhood abuse and neglect contribute much to psychological etiology of emotional eating (Burns, Fischer, Jackson, & Harding, 2012; Kong & Bernstein, 2009). Moreover, Michopoulos et al. (2015) revealed that depression has an important mediating role in the relationship between childhood trauma and emotional eating, and hence also with weight gain (Hays & Roberts, 2008) and obesity (Cornelis et al., 2014). However, Midei et al. (2010) reported that trait anger but not depression, mediated between childhood abuse and both BMI and waist circumference. Trait anger seems to be related to the increase of visceral adipose tissue (Räikkönen, Matthews, & Kuller, 1999), and is also associated with emotional eating (Appelhans, Whited, Schneider, Oleski, & Pagoto, 2011). Moreover subjects with adverse childhood experiences have a higher risk of developing maladaptive coping strategies, including stress-induced emotional eating (Evers, Stok, & de Ridder, 2010). Alvarez et al. (2007) found that perceived stress may explain the link between child abuse and the development of obesity in adulthood. Activation of the stress response can lead to emotional dysregulation that has been associated with increased appetite, a preference for foods high in sugar and fat (Adam & Epel, 2007; Dallman, 2010; McEwen, 2007; Torres & Nowson, 2007), fat visceral accumulation and obesity in adults (Cohen, Janicki-Deverts, & Miller, 2007; Torres & Nowson, 2007) and adolescents (De Vriendt et al., 2012). In this regards some authors revealed that overweight subjects tend to gain weight when stressed (Dallman, 2010) and that obese individuals increase their food intake after having experienced negative emotions and perceived stress (Barrington, Beresford, McGregor, & White, 2014; Telch & Agras, 1996). Laboratory studies have demonstrated that acute physical or emotional distress were followed by high cortisol reactivity, which induces increased intake of 'comfort' foods (Epel, Lapidus, McEwen, Brownell, 2001; Garg, Wansink, & Inman, 2007; Newman, O'Connor, & Conner, 2007). Stress-related adaptation involves the concept of allostasis, which is the ability to achieve the physiological balance through the change of the internal environment (McEwen, 2007; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Sinha & Jastreboff, 2013). Conditions of repeated or incontrollable chronic stress are followed by higher cortisol response and tend to activate a state of allostatic load resulting in neural and emotional dysregulation, which contribute to maladaptive behaviors such as repeated consumption of high caloric food (McEwen, 2007), lack of control over eating and binge eating (Gluck, Geliebter, & Lorence, 2004; Gluck, Geliebter, Hung, & Yahav, 2004; Groesz et al., 2012). These results suggest that psychophysiological responses to stress may influence subsequent eating behavior and hence may also mediate between the trauma and eating disorder link.

Only two studies have identified the eating disorder symptoms itself as potential mediators between childhood trauma and later obesity. Rhode et al. (2008) found that childhood physical and sexual abuse predicted the outcomes of body dissatisfaction, binge eating and obesity. In addition body dissatisfaction and binge eating predicted obesity. Meanwhile, Greenfield and Marks (2009) found that using food in response to stress partially mediated the relationship between childhood abuse and obesity. Moreover, some studies have questioned the mediating role of BED in the link between the presence of PTSD symptoms and the development of obesity (Pagoto et al., 2012; Rhode et al., 2008). Finally, some authors studied the mediating role of interpersonal factors in the relationship between childhood adverse experiences and obesity paying particular attention to the attachment style. D'Argenio et al. (2009) demonstrated that anxious attachment mediated the association between childhood adverse experiences, and the risk of becoming obese in adulthood. This finding is supported by the data from other studies which highlighted that insecure children had higher odds of obesity than secure children (Anderson & Withaker, 2011). Indeed, according to attachment theory, abused children, may develop difficulties in emotion regulation, which in turn is linked with eating in response to stress in absence of hunger (Macht, 2008;

Yokel, 2012), with emotional eating (Pinaquy, Chabrol, Simon, Louvet, & Barbe, 2003) and with both overeating and binge-purge behaviors (Wonderlich et al., 2007). It is hypothesized that some obese subjects use binge eating aiming to cope with negative affect. Furthermore, some authors highlighted the role played by parents in the monitoring the eating habits of their children as a possible mechanism linking childhood neglect and the development of obesity. These researchers also revealed that neglect experienced in childhood was related to obesity in adulthood (Johnson et al., 2002; Shin & Miller, 2011; Whitaker et al., 2007). These authors hypothesize that parental failure in promoting and monitoring a healthy lifestyle and a low-calorie diet may explain the relationship between childhood neglect and pathological growth of BMI in later life.

SUMMARY, LIMITATIONS AND CRITICAL REMARKS

An important quality of this review is the fact that the data are based on the findings of a very large sample (more the 300,000 participants). In this review 60 studies in obesity and 10 studies in BED were analyzed. The vast majority of these research data (61 studies, 87%), strongly support the hypothesis of an association between adverse life experiences and the development of obesity and BED. In 45 out of 53 studies (i.e., 85%) a statistically significant association was found between at least one type of trauma and obesity. With the exception of one study (Grilo et al., 2012), all other studies (86%) found a positive association between PTSD symptoms and an increase in waist circumference or BMI in adult individuals. These data show that not only childhood trauma but also traumatic events occurring in adulthood, may increase the risk of developing obesity in adulthood. Furthermore out of 10 studies on the relationship between BED and trauma, 9 studies strongly support the association between trauma and the development of BED and in adulthood. This association appears to be particularly strong when the abuse started at an early age and when the abuse was more severe. Only one study did not support this finding (Grilo & Masheb, 2001). Although these studies show some different results concerning the association between the type of abuse and BMI, probably due to differences in methodology, sampling, and research design, the vast majority came to the conclusion that adverse life experiences may play an important risk factor for the development of adult obesity and BED.

Different mediating factors between adverse life experiences, obesity and BED were found in this review namely different psychological factors such as PTSD, depression, trait anger, perceived stress, body dissatisfaction, dissociation, food intake in response to stress, insecure attachment style and neurobiological factors. An important limitation of most obesity studies is the fact that only a few used adequate control samples matched for age, gender and social status. Only in three longitudinal studies focusing on the association between life adverse experiences and obesity, adequate control samples were included (Bentley & Widom, 2009; Boynton-Jarrett et al., 2012; Noll et al., 2007). The same limitation applies to the BED studies: only a few studies incorporated adequate control samples matched for age, gender, social status and BMI. Another limitation is the fact that a wide variety of measurements and questionnaires have been employed to evaluate the different types of trauma. This makes the comparison of the results of the different studies quite difficult. Moreover, in the majority of these studies the report of childhood adversities was retrospective and could thus have led to a certain recall bias (Hardt & Rutter, 2004). In addition, different definitions of childhood adversities have been employed and this may also have created a bias in the results obtained by the different studies. Finally, the assessment of obesity and binge eating was limited in many studies to the validity of the self-reports of weight and height by the respondents. As stated before, in this review BMI was used to evaluate the severity of the obesity, consisting of 3 categories: overweight (BMI $\geq$ 25 kg/m^2), obesity (BMI $\geq$ 30 kg/m^2) and morbid obesity (BMI $\geq$ 35 kg/m^2). However many studies differ in the method used to determine the BMI, given that some used objective laboratory measures, while others used self-reported measures, and still others have used 85th and 95th percentiles of BMI adjusted for age and sex, as cut-point to determine, respectively, overweight and obesity (Sweeting et al., 2005; Whitaker et al., 2007). Both self-reported measures and laboratory measures were included in this review.

Despite these limitations, the results of the vast majority of studies suggest that there is a significant association between both childhood and adult adversities and development of both obesity and BED supporting the hypothesis that trauma increases the risk to develop obesity and binge eating. In this association between adversities and the development of obesity and BED, several mediating factors have been identified such as depression, self-criticism, dissociation, specific interpersonal factors such as the attachment quality, eating disorder symptoms such as binge eating and some neurobiological changes.

Our results have some important clinical implications. Clinicians must be aware of the full range of different traumatic experiences and systematically evaluate these experiences in the standard assessment procedure. Such a careful evaluation may indicate the need for an appropriate psychotherapeutic support. Next, because childhood adversities are associated with adult obesity and a higher risk to develop BED, increased attention must be given to the prevention of these adversities (Bruffaerts & Demyttenaere, 2009; Felitti & Williams, 1998).

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