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RESEARCH ARTICLE

PEDIATRIC OBESITY, ADDICTION AND FAMILY DYNAMICS: CONCEPT OF CO-OBESITY

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ABSTRACT

Background: The obesity epidemic has been a crucial health concern over the past few decades. Multiple contributing factors have been identified at various levels: genetic, biological, environmental, social, economic etc. In many ways obesity presents some similarities with substance dependence and abuse. The term "co-dependency" originates from the realm of addiction. Co-dependency mechanisms can also appear between parents and children and enable obesity. **Methods**: Between January 2020 and May 2020, a literature search based on electronic bibliographic databases as well as other sources of information (grey literature) was conducted in order to investigate the most recent data on obesity, addiction and co-dependencies. Some clinical examples of these concepts applied to everyday life were chosen to illustrate how they are all linked together, especially in terms of familial co-obesity (between children and parents). **Results:** Many studies link obesity and addiction, even though, the Diagnostic and Statistical Manual of Mental Disorders (DSM) does not list obesity as a psychiatric disorder. Both obesity and addiction share a common neural basis and use the same reward pathways which has been described and studied through many works. Dopamine certainly plays an important part in that system. Pediatric obesity is particularly worrying but might offer an opportunity for intervention, and once tackled, reduce the severity of adult obesity. Working with children, means working with families, and investigating intra-familial contributing dynamics. The concept of co-obesity emerged from the addiction model applied to pediatric obesity. Clinical examples illustrate an integrative biopsychosocial model of parent-child co-dependencies in obesity. **Conclusion:** Co-obesity often emerges from great altruism, tolerance towards inappropriate and maladaptive behaviors and emotions that are difficult to manage. A new and promising model of intervention is developed, based on addiction techniques (withdrawal/abstinence) associated to behavioral strategies (distraction, alternative behaviors, distress tolerance, trigger avoidance and stress lessening).

INTRODUCTION

Obesity is a medical condition characterized by the excessive accumulation and storage of body fat; to the point it has a negative impact on health. In 2013, obesity was classified as a disease by the American Medical Association. It has also been described as an epidemic (Taylor, Curtis and Davis, 2010). To measure obesity in adults, the body mass index (BMI) is used as it correlates with the amount of body fat a person has (COobesity, What is Obesity). The BMI is calculated by dividing an individual's weight (in kilograms) by their height (in meters) squared. People are considered overweight with a BMI between 25-30 kg/m^2 and obese once their BMI is over 30kg/m^2. Growth charts should be used to measure obesity in children as they are not fully grown (Obesity Facts for Kids 2017). Various causes of obesity were identified; the main ones being a combination of (Obesity Healthism 2018) excessive food intake, lack of physical exercise, genetic susceptibility, medical illnesses (hormonal problems), medications and psychiatric diseases. But in 2006, Keith, Redden, Katzmarzyk *et al*., 2006) also reported 10 other putative contributors to the secular increase of obesity: "Insufficient sleep, endocrine disruptors (environmental pollutants interfering with metabolism of lipids), decreased variability in ambient temperature, decreased rates of smoking, increased use of specific medications (like anti-psychotics or anti-depressants) as their indications are getting wider,

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proportional increases in ethnic and age groups that tends to have a higher weight, pregnancy at a later age (which might also be a factor in children obesity), epigenetic risk factors passed on generationally, natural selection for higher BMI and assortative mating resulting in an increased concentration of risk factors for obesity". New studies have shown that "healthy obesity" does not exist. Reinberg (2014) said that "*even without high blood pressure, diabetes or other metabolic issues, overweight and obese people have higher rates of death, heart attacks and stroke after 10 years compared with their thinner counterparts.*"

Some previous studies demonstrated a relationship between food insecurity and obesity: food insecurity is when an individual does not know when their next meal will be and where it will come from. The U.S. department of Agriculture identified 2 types of food insecurities (USDA) : "*Low food security; reports of reduced quality, variety or desirability diet. Little or no indication of reduced food intake*"." *Very low food security; reports of multiple indications of disrupted eating patterns and reduced food intake*" (USDA; Davidson and Morrell, 2018). Unfortunately, the cheaper foods which are the only ones some people (low-income families on welfare) can access, are also the unhealthy ones. These foods are loaded in fat, carbs and sugars and do not contain the right nutrients needed by growing children. Children fed on these, do not get the right balance that will motivate them to go outdoors and physically exercise. This causes obesity: Children just want to stay indoors, watch TV, go on their computers and play video games because they do not have the energy to go and play outside to be healthy (CO obesity; Davidson and Morrell, 2018). In 2017, Harvard T. H. Chan explained how families influence children's dietary choices and risk obesity in many ways. The term "co-dependency" originates from the realm of addiction psychology. It is essentially used to explain how addicted people are enabled to maintain their addiction due to other people. In this context, an obese child can be enabled by a parent who facilitates, voluntarily or not, their/that addiction(Gomes, Barros and Pereira , 2017).

MATERIALS AND METHODS

Between January 2020 and May 2020, a literature search based on electronic bibliographic databases as well as other sources of information (grey literature) was conducted in order to investigate the most recent data on obesity, addiction and co-dependencies. Some clinical examples of these concepts applied to everyday life were chosen to illustrate how they are all linked together, especially in terms of familial co-obesity (between children and parents).

RESULTS, DISCUSSION AND CONCLUSION

Obesity and Addiction: There are some similarities between obesity/overeating and addiction/substance abuse. The concept of addiction is complex; traditionally it was applied to excessive ingestion of substances leading to physical dependence, characterized by tolerance and withdrawal (Holden, 2001). It was solely used regarding the abuse of drugs that activated the brain's mesolimbic reward pathways. But the term has now a broader meaning as it is also applied to designate some addictive behaviors rather than the sole use of psychoactive substances. This was established as research showed that the mesolimbic reward system was also activated by pleasurable activities (Kelley, Schiltz and Landry, 2005). Altogether, the addiction process includes a compulsive pattern undeterred by some negative health and social repercussions. Obesity has a very plural etiology, but chronic overeating is certainly a crucial part of it. When this food overconsumption becomes compulsive, excessive and out of control, it can be classified as "food addiction" which has caused intense clinical and scientific controversy (Davis, Curtis, Levitan, Carter, Kaplan and Kennedy, 2011). Multiple works have shown there was a relationship between substance abuse and obesity. Both disorders disrupt the systems in the brain that balance immediate reward and need with survival requirements. When people use drugs, they temporarily feel good or experience pleasure; the same occurs when people eat food: they go through intense though short-lasting feeling of pleasure (Addiction Campuses Editorial Team, 2018). Exactly like drugs, food induces tolerance over time; which means that in order to reach and maintain a certain effect (toxicity for drugs/ satiety food wise), you need to increase your intake. These compulsive behaviors (using drugs or overeating) cycle between withdrawal and relapse; which suggest a similar brain functioning. Dieting for example can cause dysphoria and distress, while certain triggers (worries and stress or significant life events) can lead to relapse. Actually, a high incidence of relapse is noted with both drugs and food compulsive use (Volkowand O'Brien, 2007).

So far, a connection between obesity and substance abuse has been identified at multiple levels. First regarding biology and genetics(how much/where body fat can be stored and how the body burns calories and converts food into energy), brain functioning (reward pathways), environmental/developmental risk factors (peer pressure, trauma, stress, parenting) health repercussions, intense urges and cravings, social circumstances, poor quality of life but also social stigma and discrimination(as both problems are dismissed as "poor self-control disorders" and often considered as "self-inflicted life choices") (Yeomans and Gray, 1997; Addiction Campuses, 2018). Studies utilizing the Yale Food Addiction Scale (YFAS) (Gearhardt, Corbin and Brownell, 2009); support the notion of food addiction in adults (Davis, Curtis, Levitan, Carter, Kaplan *et al*., 2011; Mogul, Irby and Skelton, 2014).

Obesity and the DSM: The correspondence/parallel between obesity and addiction (substance abuse and dependence) reached such an extent, that some researchers questioned the need for 'food addiction' or 'excessive overeating' (leading to obesity) to be listed as a psychiatric disorder in the Diagnostic and Statistical Manual of Mental Disorder (DSM). The DSM 5 (American Psychiatric Association, 2013), which was released in May 2013 by the American Psychiatric Association, lists the Binge eating Disorder (BED) as an actual eating disorder for the very first time. The previous DSM-IV(American Psychiatric Association, 2000) (released in 1994) was only listing BED in Appendix B and it had to be diagnosed with the non-specific "EDNOS" (Eating Disorder Not Otherwise Specified). This BED criteria includes loss of control over eating and consumption of large quantities of food over a short period of time. Nethertheless, BED remains distinct from obesity (as most obese individuals do not engage in recurrent binge eating) (American Psychiatric Association, 2013). There is currently no diagnostic category for chronic overeating or food dependence(Barry, Clarke and Petry, 2009), knowing that food differs from drugs as it is impossible to abstain from it as food is necessary for life (Devlin, 2007).

Neural Basis and Reward System: Recent researches suggests that the global obesity epidemic isn't triggered by a lack of motivation to lose weight, but that "some foods, or some substances added to them, can trigger an addiction process by activating in the brain the same reward system generated by drugs, the mesolimbic system via dopamine" (Campana, Brasiel, de Aguiar, and Dutra, 2019). Dopamine is a neurotransmitter that signals reward in our brain. The levels of dopamine increase when an individual uses drugs or eats palatable food. This surge of dopamine also impacts other cerebral areas relating to self-control, which explains how the ability to control strong urges (for drugs or food) is compromised when suffering from compulsive, excessive and chronic overconsumption(Addiction Campuses, 2018).The metabolism of the brain is mainly controlled by "the arcuate nucleus and a 'cognitive' brain allowing interactions with the environment that offers food, including its search and storage" (Campana, Brasiel, de Aguiar, and Dutra, 2019).

Surrounded by the prospect of food, our brain is sensitive to signals announcing palatable food around us. Seeing, smelling or even just being close to some delicious food will excite our brain, predicting we will eat soon; increasing dopamine levels in anticipation ofthe reward. But once we have eaten (feeling full and satisfied), eating does not appeal to us anymore and is no longer rewarding. Our brains are designed and hardwired to respond to rewards. Without reward, survival is compromised. Indeed, both our behavior and actions are motivated by reward. For example, having sex is a pleasurable experience while producing offspring which is crucial for the species to survive. Similarly, eating keeps humans alive by maintaining health and energy, while enjoying the taste of food is the reward (Addiction Campuses, 2018). For Volkowand O'Brien (2007)and Taylor, Curtis and Davis(2010), the "mesolimbic reward pathway evolved to reinforce the motivation to approach and engage in naturally rewarding behaviors like eating, promoting survival in times offamine".

But over the last century, our food environment has changed; industries have used new food technologies in order "to create or modify certain foods by artificially enhancing their rewarding properties(our innate preferences) in an attempt to increase sales in highly competitive market" (Kessler, 2009); offering to our Western societies some highly palatable, ready to eatand well-advertised foods (high in calories, and containing large amounts ofsugar, salt and fat). Unfortunately, many of these abundant and easily accessible foods won't create the above-mentioned feeling of satiety, fullness or satisfaction. Highly rewarding foods will only produce short-lasting and intense feelings of gratification, leading the most vulnerable (biologically or environmentally) to "misuse" food in a similar way addicts misuse drugs(Taylor, Curtis and Davis, 2010). Food intake being controlled by a set of cognitive and emotional factors, Campana and hiscolleagues (2019)report that morbidly obese people present "with a reduction in dopamine D2 receptors and can develop a resistance to leptin, conducting to compulsive eating". Overeating has been shown to increase "*the release of endogenous opiates, augmenting the desire for food by determining the weights gain and obesity*" (Campana, Brasiel, de Aguiar and Dutra, 2019).

Intra-Familial Dynamics/Factors and Pediatric Obesity: Obesity is unfortunately one of the most common nutritional disorders in childhood, with a lot ofheavy consequences short, medium and long-term, including the increased risk for adult obesity and numerous associated diseases (Nguyen, Larson., Johnson and Goran, 1996). According to the WHO(2015) the worldwide prevalence of combined overweight and obesity in children rose by more than 47% over the past 30 years. In 2013, 23,2% of children were overweight or obese in developed countries and 13,2% in developing countries (Ng., Fleming, Robinson, Thomson, Graetz, *et al*., 2014; Pretlow and Corbee, 2016). Therefore, it is essential to identify the factors contributing to the development ofobesity in children, in order to possibly find some ways to treat this critical health concern. The causes of childhood overweight and obesity are multiple and inter-related (Brødsgaard, A., Wagner, L. and Poulsen, I., 2014).Having previously mentioned multiple biological, genetic, social and environmental contributors, this section will particularly focus on how factors from within the family participate to the spread ofobesity.

Already in 1991, Klesgesand his team identified that young children tend to choose food that contains high levels of fat, sodium and sugar, as well as food that they are familiar with. He also showed the role of parental influences on children's food intake. In 1996, the works of Nguyen(Nguyen, Larson., Johnson and Goran, 1996) showed "that dietary intake patterns within the family can contribute to developing obesity in families with children who have obese parents".Lamerzand his team (Lamerz, Kuepper-Nybelen, Wehle, Bruning, Trost-Brinkhues *et al*., 2005) established that there was a strong connection between parental years of education and childhood obesity; the less educated the parents were, the lower their economic status was and the higher the risk for the child to become obese. In 2013, Chari (Chari, Warsh, Ketterer, Hossain and Sharif, 2014; Ventura and Birch, 2008)came to a similar conclusion when studying how health literacy (HL) was connected to obesity. Her research concludes that "obesity in school-aged children is associated with parental factors (including obesity and parental HL), while obesity in adolescents is strongly associated with the adolescent's HL".
The parenting style has also been investigated as possibly having an influence on children developing obesity (Golan, Kaufman, and Shahar, 2006; Ventura and Birch, 2008).The term parenting style designates differences among parental attitudes and styles of interacting with children (Darling and Steinberg, 1993). Classically, there are four specific types of parenting style based on demonstration of demandingness and responsiveness (Ventura and Birch, 2008; Darling and Steinberg, 1993): authoritative, authoritarian, indulgent and neglectful.

If parenting was proven to influence child eating and weight, child eating and weight were also found to influence parenting; making it a bidirectional model(Golan, Kaufman, and Shahar, 2006; Birch 2006).Families with overweight children were shown to use more "*permissive or indulgent feeding styles, maladaptive control strategies and less supportive strategies than families with non-overweight children*" (Ventura and Birch, 2008).Indeed, indulgent parenting is linked to lower levels of self-regulatory skills, as these parents are more responsive than demanding. Golan's research (2006) presents improvement in parental authoritative style as associated to increased weight loss in children; as long as parents are assertive, providing their offspring with clear, consistent and firm directions, but remain not intrusive nor restrictive. Child feeding practices also play a part in the etiology of pediatric obesity (Birch, 2006).

Young children depend on their parents or care-givers to be provided with food that will promote healthy growth and development. Child feeding practices can determine which food the child will eat and in which quantity (portion size), how often food is served and in which social context eating occurs. They are part of tradition and culture and therefore not always adjusted to the conjuncture. For example, our child feeding practices have evolved from the time when food scarcity was the main nutritional threat to children's growth. Today the situation is quite different as the risk is rather to be exposed to too much food. But the feeding practices might not have adapted to our present eating environment and can still include "offering food frequently, and as a first response to child distress, giving palatable, preferred foods, when available and coercing or even forcing children to eat" (Birch, 2006).

Another predominant risk factor for childhood obesity that has been identified is parental obesity (Klesges, Stein, Eck, Isbell, and Klesges, 1991; Svensson, Jacobsson, Fredriksson, Danielsson, Sobko *et al*., 2011). This can be explained by acombination of genetic, epigenetic, social and environmental factors. The influence of parental relative weight primarily affects the severity of childhood obesity (which is strengthened as the child grows into adolescence)rather than the timing/the age of onset (Svensson, Jacobsson, Fredriksson, Danielsson, Sobko *et al*., 2011; Villamorand Jansen, 2016). In 2012, Berge(2013)demonstrated that family functioning (which includes communication, closeness, problem solving and behavioral regulation) can be a protective factor for teenagers' weight and weight-related health behaviors across all races/ethnicities. Haines and colleagues (Haines, Rifas-Shiman, Horton, Kleinman, Bauer *et al*. (2016)showed similar results in 2016. As obesogenic factors include low physical activity and poor diet, toddlers are a very vulnerable group for obesity. Hager and her team (Hager, Calamaro, Bentley, Hurley, Wang, *et al*., 2016) showed how insufficient sleep and poor sleep behaviors (late bedtime, multiple night awakenings, sharing beds, lack of sleep routine) put toddlers and children at risk for obesity, especially in low-income families.

A Novel Theory in Pediatric Obesity: Addiction, Co-Obesity and Clinical Examples

In 2009, Merlo (Merlo, Klingman, Malasanos, and Silverstein, 2009)and her team were pioneers in investigating food addiction symptoms in children. They showed that levels of pediatric obesity were related to food attitudes and weight management and that some children experience a drive to eat that can be compared to that of an addiction. The YFAS for Children (YFAS-C) has subsequently been validated in children (Gearhardt, Corbin and Brownell, 2009). In 2017, Burrows(Burrows, Skinner, Joyner, Palmieri, Vaughan *et al*., 2017) studied the association between food addiction in children with obesity, parental food addiction and parental feeding practices. The results demonstrated that children with raised YFAS-C scores may be at greater risk for eating-related issues. Indeed "*in children, food addiction was significantly associated with higher BMI z-scores. Children with higher food addiction symptoms had parents with higher food addiction scores and exercising feeding practice with higher levels of restriction and pressure to eat but no monitoring*" (Burrows, Skinner, Joyner, Palmieri, Vaughan *et al*., 2017). A novel explanation for pediatric obesity emerged from the observation that parents give 'treats' to their children and extra food at meals to gain love and affection from them (Page and Brewster, 2009). This could also apply to pets, which just like children, depend on their pet-parents to eat and also develop some frustrating obesity. In 2015, German speculated that "obesity in pets may be an intriguing model for childhood obesity" (German,2015; Pretlow andCorbee,2016). Both in children's and in pets' obesity, it has been noticed that treating interventions produce modest weight loss at short term, and that the weight is often regained in the long term; the individuals having been able to successfully lose weight and maintain it being a minority. To explain this, the addiction model was applied to pediatric obesity and led to identification of the concept of 'co-dependence' or 'co-dependency'. A parent can become psychologically addicted (dependent) to this treat induced affection, and by doing so enable eating addiction in the child, as a means of positive interactions with the parent (Katz, Murimi, Pretlow, and Sears, (2012).The term 'co-obesity' emerged from the fact that both the parent and the child become psychologically dependent on the treats and the extra food provided to the child, reinforcing and enabling each other (voluntarily or not). This co-dependency is often initially based on positive reinforcement; this is quite well understood and used by the food industry to advertise their products: " give them food, get the love".

Clinical example 1: Sam is five and enjoys shopping in supermarkets with his mother. He often picks some sweets and chocolate bars he saw advertised on TV and ask his Mum to buy them for him. He even tells her that "he will be so happy if she gets him the treat, he will give her a big kiss and will behave as a good boy". Mum often buys the requested treat: it costs little money, avoids a fight with the child, keeps him quiet and makes Mum happy to have such a loving and well behaved little boy. It can of course transition in negative reinforcement (avoiding emotional pain), when the extra food and treat are given by the parent to avoid hostility or rejection from the child.

Clinical example 2: The situation becomes difficult when Mum refuses to buy the treat requested by Sam. She would like to resist sometimes, knowing that these food high in fat, in sugar and sodium are not good for him, but she knows that if he does not get him the desired treat, she will have to deal with an angry, crying and shouting Sam. She can't stand her son to be unhappy and even less angry at her, therefore she does no dare not to buy the treat Sam wants. In that case both parent and child become hostage to the treats/food (Pretlow and Corbee, 2016). Actual addictive tolerance may develop; as the child might request more and more treats/ food and highly pleasurable/palatable food(Kessler,2009) or the parent independently might want to give more treats/ food to the child (fearing that the child does not get enough for example). Grand-parents and close relatives (aunt, uncle, godmother…) can also be part of this co-dependence enabling overeating and obesity (Schalkwijk, Bot, de Vries, Westerman, Nijpels *et al*., (2015).

Clinical example 3: Laura is 8y old. Her Mum, Frances is a nurse and after 8y of staying at home, recently decided to go back to work. This means that Laura has to stay at her Granny's after school and do her homework there till Frances finishes her shift and come to collect her at Granny's. Laura loves staying at Granny's because she always has plenty treats in the press: crisps, biscuits, chocolate, even cake sometimes. Every evening, while waiting for Mum to collect her and take her home for super, Laura is provided with crisps and snacks,

which she enjoys on a daily basis. Laura's weight has become an issue as she put on quite a few pounds over the last 6 months. Despite her weight increase, Laura refuses to abstain from the snacks at Granny's. She is even waiting for them. Frances knows her daughter is provided with unhealthy food and is aware of the negative impact it has on Laura, but won't step in as she would fear to lose the care arrangement with Granny for Laura. At a stage, the parent might notice that the child has become overweight and might attempt to cut back on the treats and the extra food. But parental withdrawals symptoms (resulting from the loss of love/affection from the child if treats are reduced) may prevent them to do so. Likewise, the child does not want to go through withdrawal and may reject the parent or display angry behavior because of the loss of 'food fixes'. Aversion to anger or 'cold shoulder' from the child, motivates and perpetuates then the parental enabling, as the fear of rejection may surpass the desire for love. Clinical example: Livie is 7y old. Her parents separated last year. Livie used to stay with her Mum during the week in the family home and was very reluctant to visit Dad in his new flat over the weekend. Mum has always been promoting healthy food, and Dad was doing same before the separation. To please Livie, Dad buys her take-away, pizza and junk food when she visits him. Livie enjoys the treat and is even furious if for some reason, Dad cancels access as she is longing for these foods that are forbidden by Mum. Dad knows it is very unhealthy to feed his daughter that way, but can't forgo her visits and the affection she gives him when she visits. Pretlow and his colleague (Pretlow and Corbee, 2016)identify a combination of circumstances in the Western world as possibly contributing to the pediatric obesity epidemic and leading to eating addiction: (a) cheap, highly accessible, highly pleasurable/palatable food, (b) increased distress in children like anxiety/depression/boredom and (c) comfort eating (easing pain and distress and reinforcing the behavior in the brain so it is maintained)(Di Segni, Patrono, Patella, Puglisi-Allegra, and Ventura, (2014).

Conclusion

New ways of thinking: A New Treatment for Pediatric Obesity Based on the Addiction Model and Co-Obesity. Family-based interventions for pediatric obesity should take account of the addiction model, as traditional approaches (including lifestyle modification of diet and exercise) have failed to give significant long-term results. Pretlow (Pretlow, Stock, Allison, and Roeger, 2015) proposed a model of intervention in which classic addiction medicine techniques of withdrawal/ abstinence are combined with behavioral addiction treatment methods. This intervention start with a procedure of staged food withdrawal for both children and parents. Food causing cravings and difficult to resist to, when available (problem foods), are removed in the first place. Children withdraw from problem foods, one by one, by total abstinence from the food for at least 10 consecutive days. Parents also have to go through withdrawal by not having the problem foods in the home and not supplying them outside the home. The snacks (non-specific food eaten between the meals) will be withdrawn in second place before reducing the size of the portions served at meals. Behavioral addiction treatment methods involve alternative behaviors (like going for a walk), reducing the stress levels, distraction techniques, distress tolerance and trigger avoidance (Pretlow and Corbee, 2016).

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Glossary of Abbreviations (3rd page)

BMI: Body Mass Index.
DSM: Diagnostic and Statistical Manual of Mental Disorders.
USDA: United States Depratment of Agriculture.
EDNOS: Eating Disorder Not Otherwise Specified.
BED: Binge-Eating Disorder.
WHO: World Health Organization.
HL: Health Literacy.
YFAS: Yale Food Addiction Scale.
TV: Television.

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