measure) are indeed true, this has negative implications for societies. It makes it less likely that any particular intervention, such as language training, could compensate for the cognitive and behavioral problems. An unfortunate implication of poverty.

However, it is possible that the global neural effect of low SES is the result of a combination of a multitude of environmental effects, and that each of these can be identified and targeted. Future research might thus highlight the role of specific environmental factors in affecting cognitive development, which could help inform policy decisions.

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DOI:10.1002/wps.20934

The evolving epidemiology and differential etiopathogenesis of eating disorders: implications for prevention and treatment

Profound changes in the classification of eating disorders have occurred over the past decades. The expanded diagnostic spectrum of feeding and eating disorders now ranges from conditions characterized by food restriction (anorexia nervosa and avoidant/restrictive food intake disorder, ARFID) through to those typified by food craving and overeating (bulimia nervosa and binge eating disorder).

Since the advent of the DSM-5 in 2013, amenorrhea is no longer required to diagnose anorexia nervosa, and binge eating disorder is a fully recognized diagnostic entity. Most previous differences between the ICD and DSM have now been eliminated: the ICD-11 is broadly similar to the DSM-5, the only important difference being that subjective binges are accepted for an ICD-11 diagnosis of binge eating disorder.

About 1.4% of women and 0.2% of men experience anorexia nervosa during their lifetime; 1.9% of women and 0.6% of men are affected by bulimia nervosa, while 2.8% of women and 1.0% of men develop binge eating disorder. So, binge eating disorder is the most prevalent eating disorder[1].

To judge time trends in the occurrence of new cases, only longitudinal incidence studies on large population-representative samples can provide clarity. Incidence studies count new cases of eating disorders in dynamic populations, meaning that individuals can enter or leave the underlying population by, for example, immigrating to a country or dying. Therefore, each individual in the population is followed up for a different time period. These individual follow-up durations are summed to the total follow-up time expressed in person-years. New cases per person-year are measured by incidence rates.

Although diagnostic specifiers have evolved over time, the incidence of anorexia nervosa and bulimia nervosa presenting to primary care, in countries (such as the UK and the Netherlands) where this is an entry point for secondary care, has been relatively stable over the last six decades[2]. On the other hand, admissions for inpatient treatment for anorexia nervosa have rapidly increased in several European countries, despite most guidelines recommending this as a tertiary form of management. The explanation for this discrepancy in service use is uncertain. One possibility is that a reduced mortality rate has allowed those with a severe form of illness to survive for longer. Another possibility is that environmental protective factors may have decreased whilst perpetuating factors have increased.

There are many contrasts in the clinical features and underlying etiopathogenesis between anorexia nervosa and binge eating disorder. Anorexia nervosa has an earlier onset in the peripubertal period. In binge eating disorder, the female:male ratio is lower, the risk in ethnic minorities is higher, and a developmental and/or family history of higher weight is commonly present. As binge eating disorder is such a recent diagnosis, incidence studies with sufficient follow-up time have not yet been performed[2].

There are no genome-wide association studies on bulimia nervosa or binge eating disorder, but emerging work suggests that the genetic risk profile differs from that of anorexia nervosa. For example, a study using the UK Biobank cohort found that adults who engage in binge eating carry a polygenic liability to higher body mass index (BMI) and attention-deficit/hyperactivity disorder (ADHD)[3]. This contrasts to the negative genetic association with BMI and variables related to the metabolic syndrome in anorexia nervosa[4].

Over the past 70 years, the food environment has changed rapidly. Food technology has increased access to cheap, highly palatable foods (combining salt, sweet and fatty elements), refined for rapid absorption. This has contributed to changes in eating behaviour, such as the reduction in social eating and increase in fast food consumption. These changes in the food environment are likely to have contributed to an increased prevalence of binge eating.

Another key social determinant is weight stigma (social rejection, teasing, bullying and devaluation because of a bigger body), particularly if the body shaming induced is internalized. Weight stigma may be compounded by other forms of trauma, alienation and discrimination that may occur in marginalized groups.

The source of this intolerance is widespread, ranging from family, peers, friends, the online community, the medical profession and policy makers. For example, public health interventions targeting obesity may harm through weight stigmatization and increasing body dissatisfaction[5]. Indeed, data from three ongoing birth cohorts in the UK suggest that weight control behaviours have increased in adolescents of both genders (almost 50% report dieting), which may produce a steep increase in eating disorders within the next decade[6].

The implications drawn from the epidemiology of disordered eating and the emerging genetic associations suggest that complex interactions between the environment and somatic and psychological factors are causally involved in the development of eating disorders. A wide range of variables can moderate the expression of these vulnerabilities. A broader approach to the prevention of both eating disorders and obesity is needed, with a central focus on reducing weight stigma and increasing healthy forms of eating and exercise behaviours rather than promoting unhealthy patterns of food restriction. Eating disorders affect individuals of all body weights, shapes and sizes, and it is of concern that heavier patients may not be considered "ill enough" either by themselves or by the gatekeepers of financially constrained eating disorder services, thus missing the opportunity for early intervention.

At the other end of the care pathway, new approaches are being developed for people with eating disorders who have failed to respond to standard treatment. Advances in the management of binge eating disorder include treatments targeting psychological processes believed to precede and perpetuate the disorder, such as reward sensitivity, inhibitory control, ADHD tendencies and interoceptive awareness. One example is represented by strategies that focus on increasing inhibitory effortful control[7].

In severe anorexia nervosa, there are intriguing case reports describing the use of metreleptin, a recombinant human leptin analogue often used to treat excess appetite in people with lipodystrophies. The seemingly counterintuitive rationale for this is based on experimental work derived from activity-based animal models of anorexia nervosa[8]. Metreleptin led to an immediate reduction in depression, and a later resolution in eating disorder behaviours[9]. A similar profile of change has been seen following neuromodulatory techniques.

Thinking forward, advances in our understanding of the evolving epidemiology and differential etiopathogenetic factors associated with eating disorders can improve prevention and treatment, and hopefully reduce the incidence of these conditions.

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DOI:10.1002/wps.20935

Mental health of children and parents after very preterm birth

Having a baby at less than 32 weeks gestation is a highly stressful and potentially traumatizing experience for parents. For almost all parents, there is heightened anxiety about their baby's health and well-being. In some cases, the birth itself may be traumatic, and women may require an intensive care admission and/or longer stay in hospital. Parents may be separated from their baby for extended periods of time unless there is provision for them to stay alongside their baby in the neonatal intensive care unit.

As well as the immediate risks to their baby's health, parents are faced with uncertainty about their baby's longer-term health and development. There is increasing recognition that children born very preterm (<32 weeks gestation) are vulnerable to mental health difficulties in childhood and adolescence[1]. In the preschool period, they are more likely to experience internalizing and dysregulation difficulties compared with term-born peers[2]. There is also evidence of an increased risk for symptoms and diagnoses of attention-deficit/hyperactivity disorder (ADHD) in the preschool period for children born extremely preterm (<28 weeks gestation)[1]. By school age, compared with term-born children, children born very preterm have three times higher odds of meeting criteria for any psychiatric disorder[3].

The pattern of mental health difficulties and psychiatric diagnoses in children born very preterm appears to be clustered around the key areas of attention, social and emotional difficulties. This is reflected in increased rates of autism spectrum disorder (ASD) and ADHD diagnoses by school age for those born very or extremely preterm[1,3]. This pattern continues into early adolescence, with the most prevalent diagnostic categories for those born preterm being ADHD, ASD and anxiety disorders[4]. Over time, the risk for psychiatric disorder associated with preterm birth appears to decrease, although some studies report ongoing differences in mental health outcomes[5], with implications for quality of life and functioning.

Mothers and fathers of infants born very preterm experience elevated levels of depressive, anxiety and post-traumatic stress symptoms compared with parents of term-born babies. One study found that approximately 40% of mothers and fathers experienced depressive symptoms and almost 50% reported anxiety symptoms