# What causes eating disorders?

<table>
<thead>
<tr>
<th>ENVIRONMENT</th>
<th>GENETICS</th>
<th>BIOLOGY I</th>
<th>BIOLOGY II</th>
<th>BIOLOGY III</th>
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</thead>
</table>
| Cessive value based thinness | Female gender most potent risk factor (Jacobi et al, 2004)  
• Biology vs environment | Brain vulnerable to poor nutrition:  
• 20% of calorie intake | Changes in central appetite control: **homeostatic system.**  
*Brain stem, hypothalamus ➔ hunger, satiety* | Alterations in chemical transmitters e.g. 5-HT, opioids and DA |
| Stigmatism based on od/shape/weight | Twin/family studies:  
• Complex genetic diseases  
• Heritability 50-83% | Most EDs emerge during adolescence  
• Period of brain reorganisation | Changes in central appetite control: **drive system**  
*Mesolimbic cortex, striatum ➔ reward, motivation, learning* | Abnormalities in illness-related/ non-illness related info processing:  
*Food, body and exercise stimuli* |
| General adversity:  
• Stress  
• Birth related complications  
• Neglect  
• Physical abuse | GWAS/ linkage studies have identified loci for AN and BN | Starvation shrinks the brain  
• Neurobiologic al changes  
• Behavioural/ psychosocial changes | Changes in central appetite control: **Self-regulation system**  
*Prefrontal cortex ➔ top-down control, contextualises appetite within goals* | Abnormalities in illness-related/ non-illness related info processing:  
*Problems with decision making and reward processing* |
| Easy access to highly palatable food | Around 1/3 of genetic risk for ED and depression/anxiety/ addiction is shared | Some resolve with weight gain | Other central appetite control systems may also be altered  
• Abnormalities in illness-related/ non-illness related info processing:  
*Problems with flexible thinking and central coherence* | |
| General adversity:  
• Stress  
• Birth related complications  
• Neglect  
• Physical abuse | **CREATES NEUROBIOLOGICAL VULNERABILITIES** | **Abnormalities in all 3 have a role in the risk/maintenance of EDs** | Abnormalities in illness-related/ non-illness related info processing:  
*Problems with social cognition and emotional regulation* | |

## ED: A model of rewards vs inhibition

The key neural pathways that are involved in human consummatory behaviours include the corticostriatal limbic circuits and the dorsal cognitive circuits which modulate reward and self-control. Therefore, any extremes of consumption may be related to an altered balance between reward and inhibition, this includes a shared altered reward appraisal which leads to emotional dysregulation. Also inhibition varies with diagnoses depending on whether the cause of the ED is due to cognitive control of dysregulation of processing. The current hypotheses for EDs are:

- **AN:** dysfunctional/diminishes reward sensitivity, exaggerated cognitive control
- **BN and BED:** deficient cognitive control, erratic/ excessive responding to reward stimuli; more research needed