

# Biological & Psychological Risk Factors for Eating Disorders in Type 1 Diabetes

## BACKGROUND

Women with type 1 diabetes (T1D) are 2.4 times more likely to develop an eating disorder than their non-diabetic peers.<sup>1</sup> Although there are few prevalence studies that include males, it's presumed that their risk is also elevated. In addition, multiple studies over the last 20 years report that 30-35% of women with T1D restrict or omit insulin in order to lose weight.<sup>2-3</sup>

Eating disorders are widely considered to be biopsychosocial disorders. It is also recognized that having T1D is an additional risk factor associated with the development of an eating disorder.<sup>4-8</sup> The majority of investigations conducted to determine the reasons for this have been done with questionnaires and interviews.<sup>9</sup> From those, certain factors involved in having diabetes have been identified as reasons for the higher risk:

- weight gain at diagnosis and/or with intensive diabetes management<sup>10-12</sup>
- emphasis on food intake, meal planning, food rules, and dietary restraint<sup>11,13-14</sup>
- intense focus on numbers (BG, A1c, carbs, dosages, etc.) and being judged as a number<sup>11,15</sup>
- constant attention on losing or maintaining weight<sup>16-18</sup>
- attitude toward insulin and/or toward diabetes<sup>19</sup>
- feelings related to diabetes, including feeling out of control<sup>16</sup>

These factors generally fall into the social and emotional categories. We know that T1D ends production of BOTH insulin and amylin, impacts other parts of the metabolic system, and can result in higher eating disorder psychopathology.<sup>3,20</sup> While some research has been done on the correlation between T1D biology/psychology and eating disorders, little has been done on causation.

## OBJECTIVE

To identify the biological disruptions and psychological pressures unique to a person with T1D that create the higher risk for developing an eating disorder.

## DESIGN & METHODS

Reviewed literature, articles and research studies on biological systems impacted by T1D, and on the psychological factors involved in eating disorders. Gathered experiential evidence from Diabulimia Helpline clients and diabulimia online support group members through discussions, posts and responses to questions. Connections were made between T1D biology/psychology and eating disorders, then cause and effect processes were posited.

### Emotional dysregulation

Anxiety Depression  
Binge/Guilt Burn out  
Fear Negative Cognition  
Dissatisfaction



## RESULTS – PSYCHOLOGICAL FACTORS

It is suggested that a number of character traits associated with eating disorders<sup>36</sup> can be exacerbated with a type 1 diabetes diagnosis.

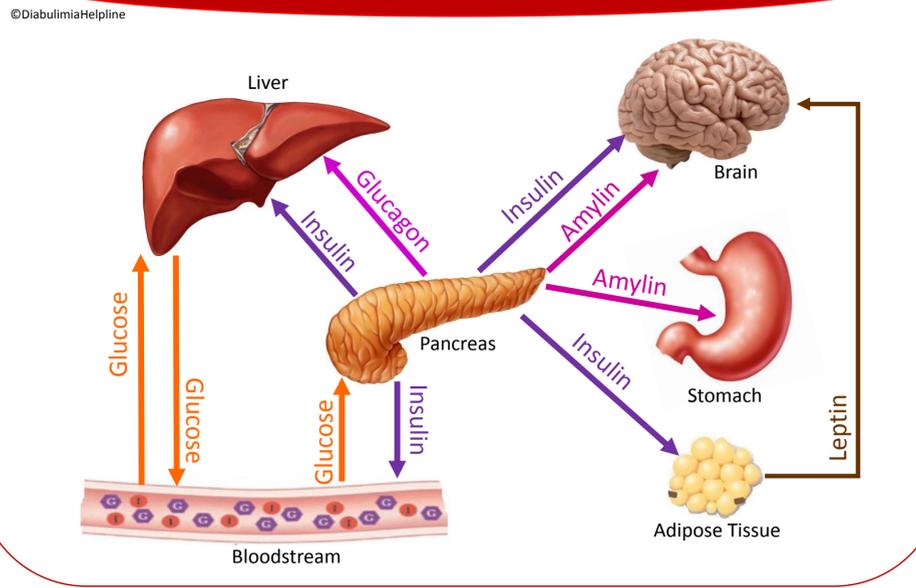
- Anxiety - heightened with worries about high BG, low BG, new/unknown foods, carrying supplies, traveling, etc.<sup>3</sup>
- Perfectionism - exacerbated as one focuses on “perfect” glycemic goals or “perfect” metabolic control.
- Obsessionality - dealing with an illness that requires 24/7 attention can amplify tendencies toward obsession or compulsion.
- Rigidity - the many rules, behaviors and calculations required for diabetes management may feed into or stress a rigid personality.
- Avoiding Thoughts - a person already prone to avoiding mental events is more likely to experience diabetes burnout; a precursor for diabulimia.
- Avoiding Emotions - attitude about chronic illness is a known risk for diabulimia, especially in a person prone to avoiding emotions.<sup>14</sup>
- High Harm Avoidance – these individuals will avoid conversations that invoke confrontation or negative feedback;
  - They may say everything is fine or avoid doctor appointments if they feel they will be judged or criticized for their diabetes management.<sup>37-38</sup>
- Control - there is an emphasis on the need for tight BG control to prevent complications, yet to “control” a disease is extremely difficult.<sup>10</sup>
- Predictability – extremely challenging when one can make the same diabetes management decisions with widely different results.
- Low Self-directedness – a lack of unity with oneself is impaired even more with a diabetes diagnosis.<sup>37,39</sup>
- Low Cooperativeness - diabetes first separates a person from their peers, then insulin omission separates them from the diabetes community.<sup>38</sup>

**Character traits persist after recovery which means T1D exacerbators persist after recovery. Treatment must address both in order to be sustained.**

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## Pancreatic Hormone Functionality



### Insulin (co-secreted by Beta cells with Amylin)

- In the **bloodstream**, enables glucose to pass from the bloodstream into the cell.
- In the **liver**, triggers the conversion of glucose into glycogen.
- In **adipose** tissue, promotes the production of leptin.
- In the **brain**, suppresses neuropeptide Y and promotes dopamine turnover.

### Glucagon

- In the **liver**, triggers the conversion of glycogen to glucose.

### Amylin (co-secreted by Beta cells with Insulin)

- In the **stomach**, slows gastric emptying reducing blood glucose spikes.
- In the **brain**, promotes satiety.

### Leptin

- In the **brain**, inhibits hunger and promotes satiety.

## RESULTS – BIOLOGICAL FACTORS

### Loss of Natural Hunger Cues

- Meal initiation and meal components become based on external signals.<sup>21-22</sup>

### Stomach Empties Too Quickly

- Without amylin to slow gastric emptying, stomach empties too fast.
- Faster emptying causes malabsorption of amino acids; lack of nutrients interferes with neurohormonal signals to stop eating.<sup>23</sup>

### Reduced or Non-existent Satiety

- Without amylin or sufficient leptin in the brain, there is nothing to signal satiety.<sup>24-25</sup>
- Lack of amylin also disrupts satiety mechanisms controlled by the rate of gastric emptying.<sup>23</sup>

### Uninhibited Hunger / Increased Appetite

- Reduction of leptin in the brain which normally inhibits hunger<sup>26</sup> can lead to uncontrolled food intake.<sup>27-28</sup>
- Without amylin to help match caloric intake to energy expenditure can lead to overeating.<sup>24,29-30</sup>
- Low or no insulin means no suppression of neuropeptide Y and no suppression of appetite.<sup>31</sup>

### Lack of reward or pleasure with food intake

- Low carb meals and Low or no insulin reduce dopamine turnover<sup>32-34</sup> resulting in reduced reward or pleasure in eating.<sup>35</sup>

### Lipohypertrophy (“diabelly”)

- Small lumps of fat accumulates under the skin at injection sites.

**Many biological risks become reinforcers of insulin omission and propel disordered eating into an eating disorder.**

While insulin analogs have made great strides, they still do not perfectly mimic natural insulin, thus they do not perfectly mimic insulin’s interactions with other hormones, peptides and neurochemicals in the body.

## When Biology Becomes Psychology

- Fatty acids from high blood glucose, Lack of amylin, and Lack of leptin stimulate overeating<sup>40</sup> which can create guilt<sup>41</sup> and trigger insulin restriction.
- Low insulin and/or low carb reduces dopamine turnover<sup>34</sup> which:
  - reduces reward/ pleasure<sup>35</sup> which can lead to:
    - food restriction (no pleasure, why bother) or
    - overeating (continuously trying to achieve pleasure).
  - can increase depression and novelty seeking<sup>42</sup> - eating disorder traits.
- Reduction of leptin has a negative impact:
  - on regulatory and emotional control of appetite<sup>43</sup> creating a risk of bingeing which creates risk of insulin omission.
  - on cognition similar to Alzheimers<sup>44</sup> impacting decision making.
- Absence of amylin can lead to excess glucagon production<sup>45</sup> which leads to high BG despite proper insulin, causes frustration and burn out – eating disorder risk.
- Insulin omission provides a false sense of control and predictability.
- Floating high blood sugar numbers “numbs” thoughts and emotions; a positive for anyone trying to escape emotional pain.

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