

# Breastfeeding and Obesity

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**Neuroprotective Developmental Care lactation fellowship 2025: Endocrinology Session 2**

# Conflicts of Interest

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I have no actual or potential declarations to make in relation to  
this programme.

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**>50%** of Australian mothers entering pregnancy are affected by overweight or obesity.

# Overview

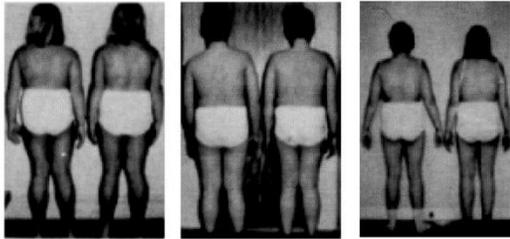
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1. Obesity pathophysiology: a review
2. Obesity stigma
3. Impacts of obesity on breastfeeding
4. Optimising breastfeeding for women with obesity
5. Weight (and weight loss) during breastfeeding

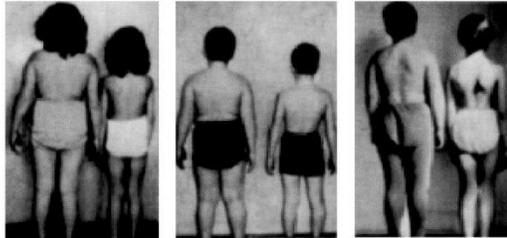
Medical school teaching	How I think now
Obesity is a result of poor individual lifestyle choices	Obesity <ul style="list-style-type: none"> <li>- Has a significant genetic component</li> <li>- Has environmental influences which are systemic and universal</li> </ul>
Obesity is as simple as excess adiposity	Obesity is a complex, dynamic endocrine condition
Obesity is a risk factor	Obesity is a chronic disease, and warrants the same careful attention as other chronic diseases
Weight loss is “mind over matter”	Weight is vigorously defended by brain and hormonal mechanisms
Weight loss is achievable via diet and lifestyle modification if sufficient discipline/ engagement	Only a very small minority (5%) of individuals with severe obesity will achieve sustained weight loss with diet and exercise
We should aim to shift all individuals into a normal BMI range	Weight loss targets should be individualised. For clinically meaningful improvements in metabolic parameters, 5-10% weight loss is a good target

# Genetics of BMI

## Body Mass in Twins



**Monozygotic Twins (Intrapair Correlation = 0.66)**



**Dizygotic Twins (Intrapair Correlation = 0.26)**

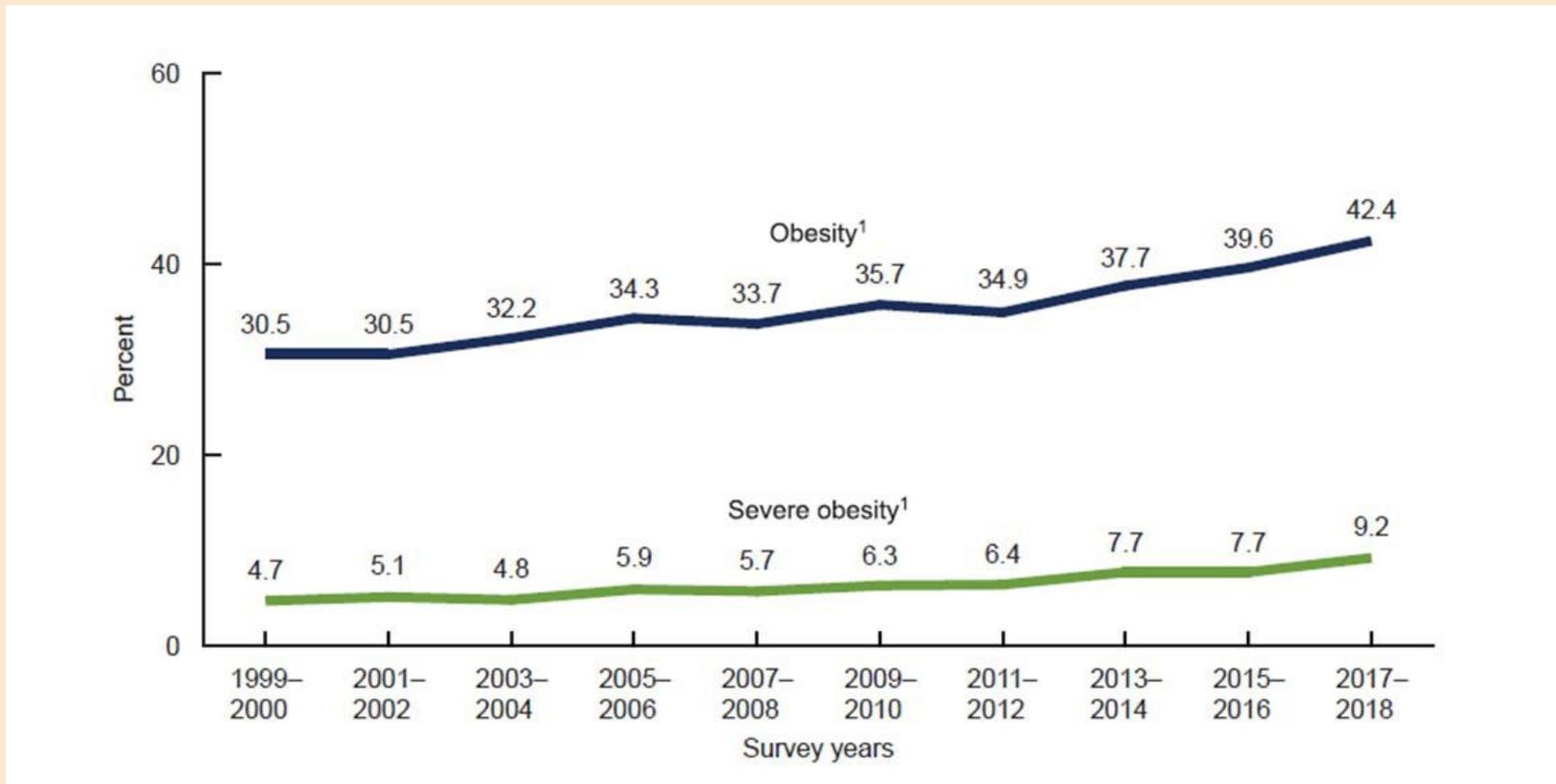
## Current understanding

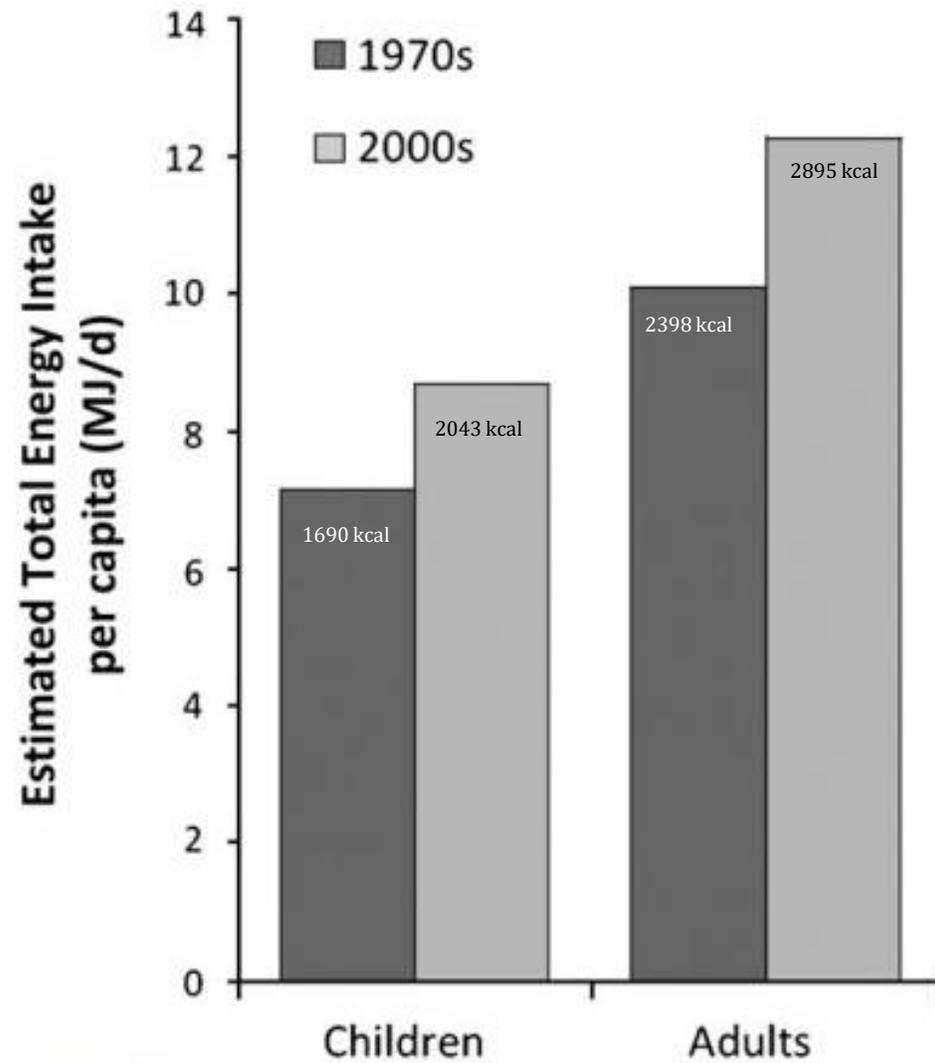
- Genetic contribution to adult BMI **40-70%**
- 1 biological parent with obesity = **3-4 x risk** vs those who do not
- 2 parents with obesity = **10 x risk**

In most cases, obesity is **polygenic** - hundreds of “associated” genetic loci

Probably further modified by epigenetics

# Obesity is a **disease of the modern environment**





**FIGURE 2.** Changes in estimated daily energy intake per capita for US children and adults for the periods 1971–1976 (1970s) to 1999–2002 (2000s).

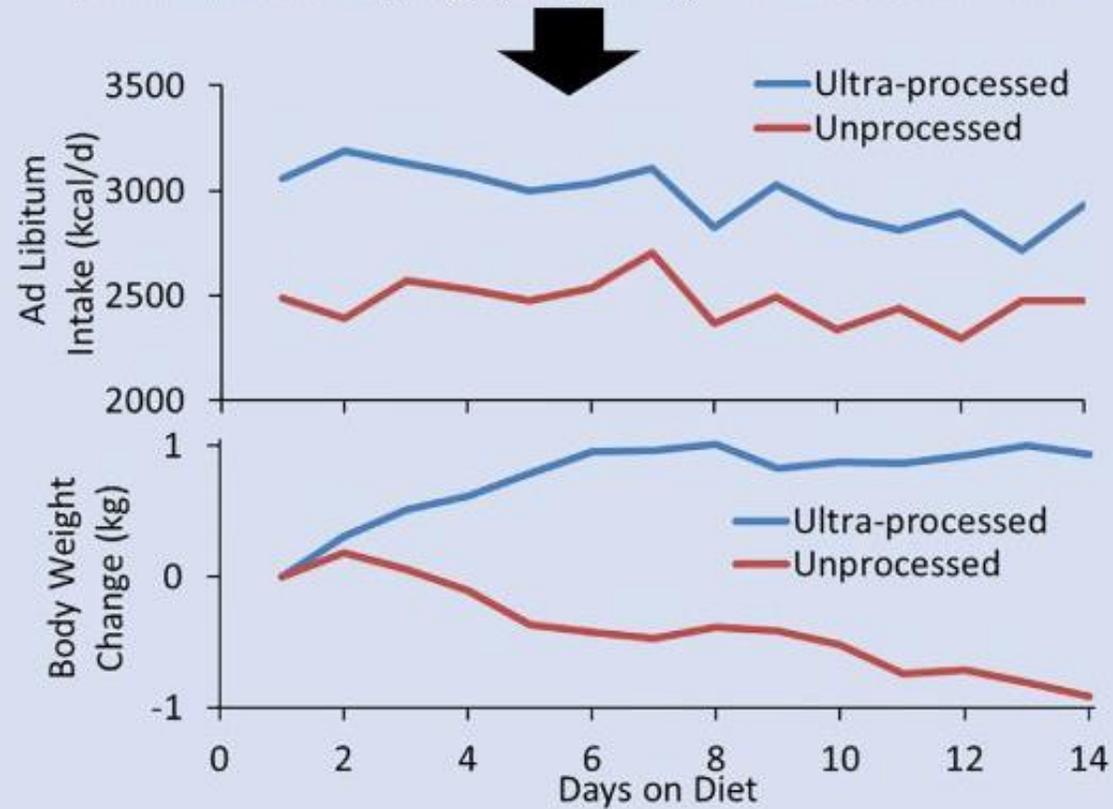
## Ultra-processed Diet



## Unprocessed Diet

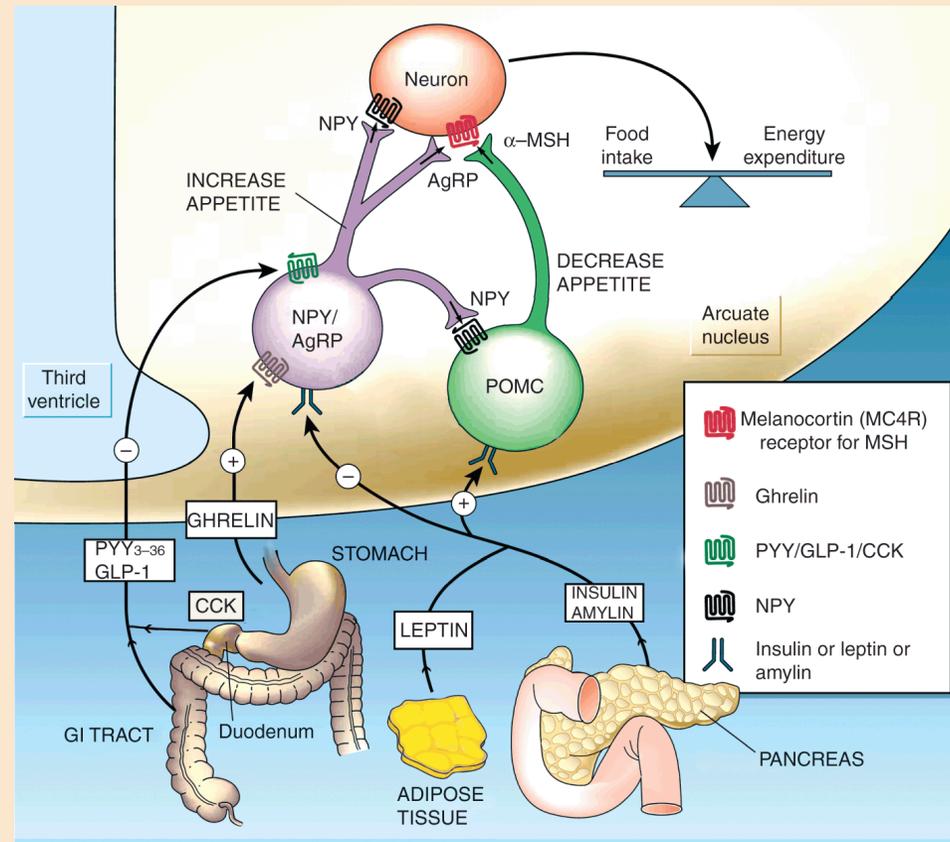


Diets were presented in random order and matched for provided calories, sugar, fat, fiber, and macronutrients

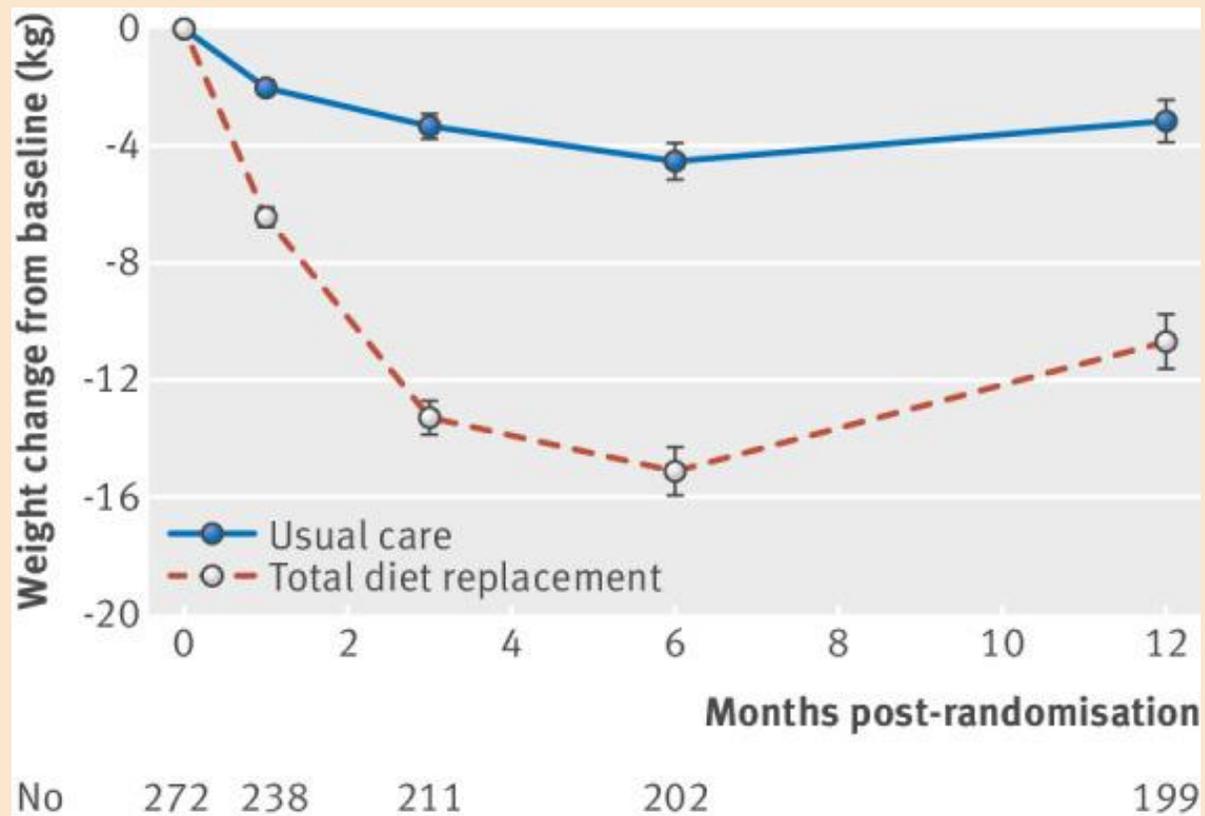


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# Obesity is a brain disease

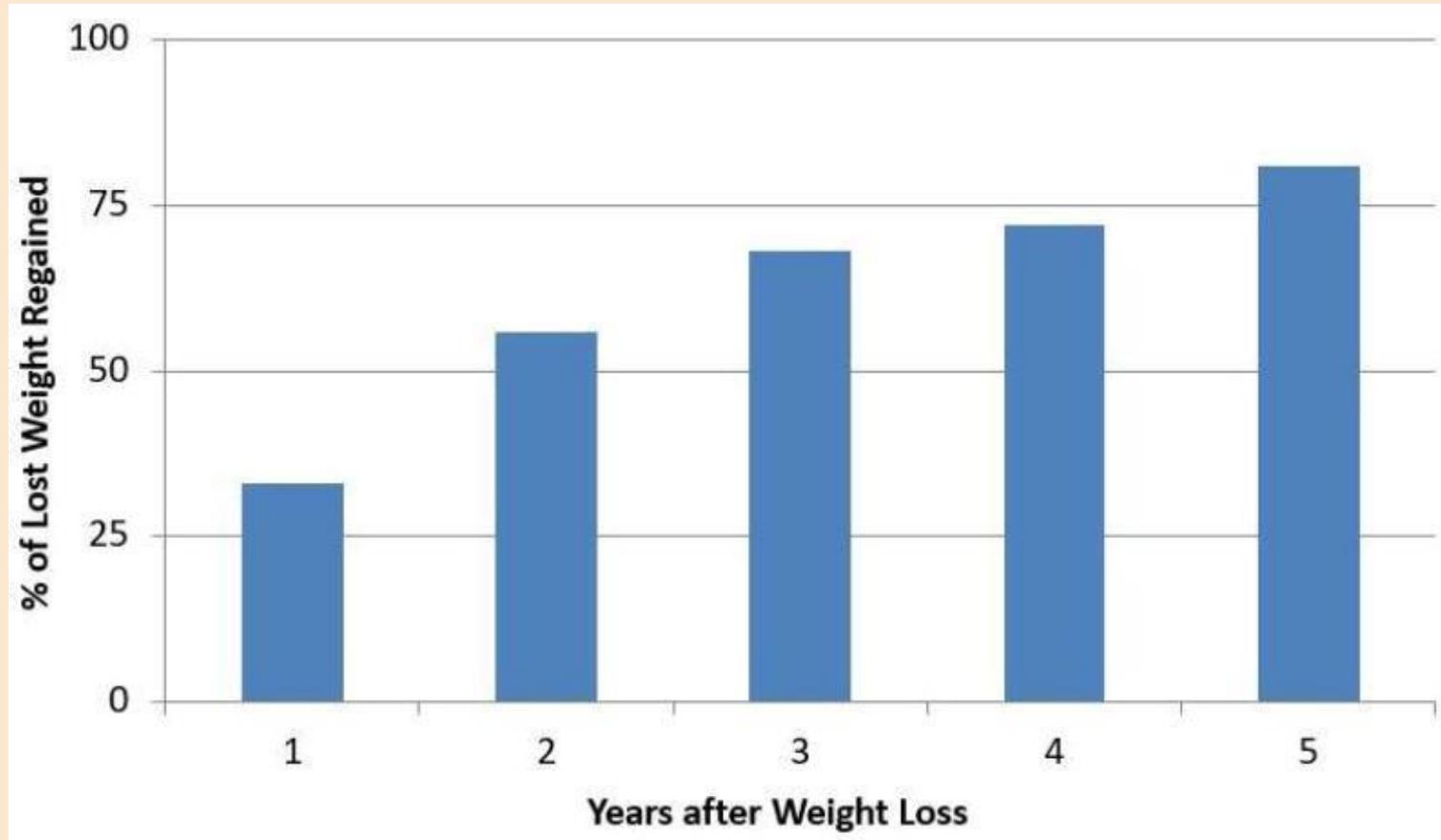


## Diets DO work – but only in the short term

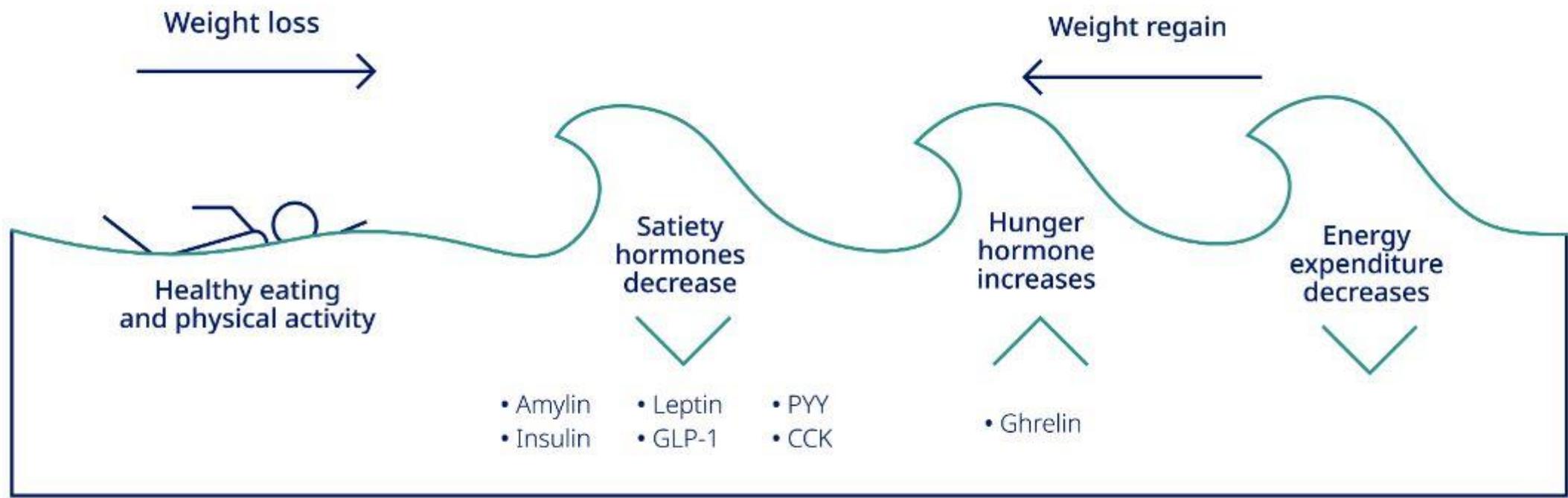


“Compared with regular weight loss support from a practice nurse, a programme of weekly behavioural support and total diet replacement providing 810 kcal/day seems to be tolerable, and leads to substantially greater weight loss and greater improvements in the risk of cardiometabolic disease”.

# Diets DO work – but only in the short term



Hall and Kahan, Med Clin North Am, 2018 Jan; 102(1): 183–197.



# Weight stigma

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Weight stigma is a **pervasive stereotypical and discriminatory perception that being larger-bodied is inherently pathological**

and

that **individuals are solely responsible—and thus subject to judgement and blame—for their own weight.**

It is associated with the view that a **particular body size is more valuable or worthy than another.**

# Weight stigma

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Increasingly acknowledged as a **chronic social stress** that may be a key contributor to the adverse health outcomes of obesity.

↑ BP, oxidative stress,  
cortisol, HbA1c

↑ mortality up to 60%  
(even after BMI accounted  
for)

↑ risk for further weight  
gain and obesity  
longitudinally

Gendered – more than  
twice as frequently  
experienced by women  
than men, and at lower  
BMIs

Particularly common in  
healthcare settings

Particularly common in  
preconception, pregnancy  
and postpartum – a major  
issue now that 29% ♀  
reproductive age have  
obesity

# THE OBESITY BIAS

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Critical Conversations About: Patient-First Language

**DON'T:** Use language that implies you see a patient's medical condition before their personhood or as a moral or personal failing.

“  
An overweight  
patient  
”

“  
An obese  
patient  
”



**DO:** Talk to the patient as neutrally about their weight as you would speak about any other condition.

“  
A person who  
would like to  
lose weight  
”

“  
A person  
undergoing  
treatment for  
obesity  
”

“obese” is an identity

whereas “obesity” is a  
disease

# Obesity and breastfeeding: the **benefits**

Most recent/ comprehensive SR to date (2023, commissioned by WHO):

- n=169 studies included (cohort, case control and cross-sectional)
- BF **protective against overweight and obesity for offspring, OR 0.73 (0.71, 0.76)**
- Persisted with subgroup analysis of **ONLY very high quality** studies (n=19): OR **0.85 (0.77, 0.93)**



# Obesity and breastfeeding: the epidemiology

## Maternal obesity is associated with earlier weaning.

- Initiation rates generally only slightly lower among women with obesity, but BMI  $\geq 30$  associated with **significantly higher odds of stopping exclusive (or any) BF in early post-partum.**



Prepregnancy BMI (kg/m <sup>2</sup> )	Initiated BF, %	Exc BF to 4mo PP, %
<18.5	99.0*	57.8*
18.5–24.9	99.6	62.9
25.0–29.9	98.8	54.4
30.0–34.9	98.7	45.4
$\geq 35.0$	97.4	37.8

Norwegian Mother and Child Cohort Study, n=49 309 mother/ child pairs

- This observation typically persists after adjustment for other important socio-demographic factors.

# Obesity and breastfeeding: the epidemiology

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## Breastfeeding difficulties in lactating mothers with obesity manifest early.



- Infants born to mothers with obesity have **higher odds of requiring medically-indicated supplementation during hospital stay**, even after correction for conditions such as diabetes/ C-section delivery/ IOL/ PET <sup>1</sup>
- Odds of **maternally-requested formula use** higher in mothers with obesity <sup>1</sup>
- **Delayed onset of lactogenesis (DoL)**, defined as >72h, in >50% of women with obesity in some large US cohort studies <sup>2,3</sup>

<sup>1</sup> Colling et al, Breastfeed Med 2019; 14(4): 236-242.

<sup>2</sup> Chapman and Perez-Escamilla, J Am Diet Assoc 1999; 99(4): 455-456.

<sup>3</sup> Nommsen-Rivers et al, Am J Clin Nutr 2010; 92(3):574-584.

# Obesity and breastfeeding: the **mechanisms**

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## Physiological

- impaired PRL dynamics
- insulin resistance
- inflammation

## Clinical

- higher rates of obstetric and delivery complications (e.g. C-sections, NICU)
- practical difficulties – fit and hold etc

## Psychosocial

- negative self-perceived body image
  - inequitable support
- allostatic load of weight stigma

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# Obesity and breastfeeding: the **mechanisms**

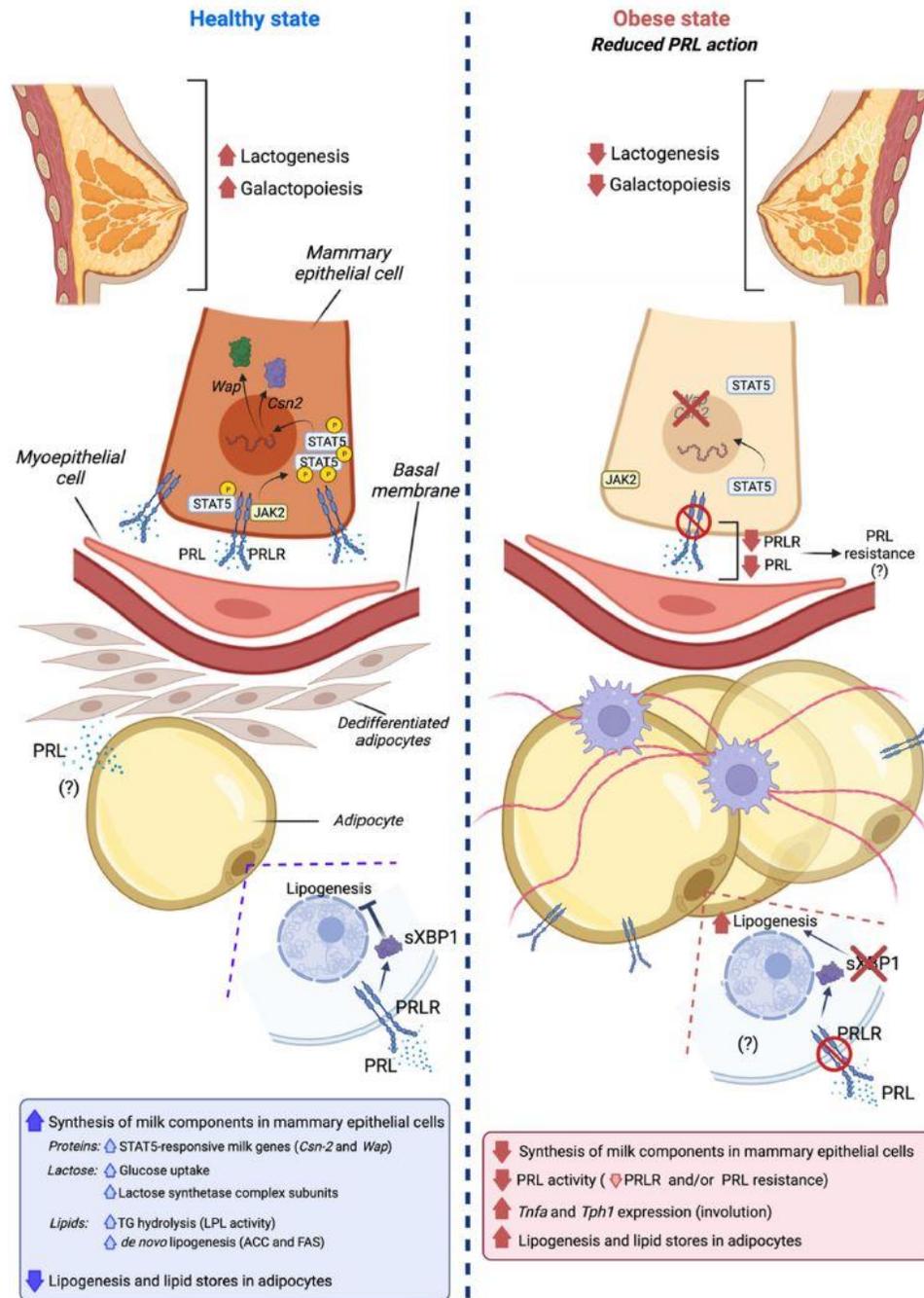
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## **Physiological explanations**

### **1. Abnormal prolactin dynamics**

-rodents that are obese and/ or are fed an obesogenic diet during lactation experience reduced milk yields due, at least in part, to **PRL resistance** (lower levels of PRL receptor expression, and reduced downstream PRL signalling)

## PRL actions in the lactating mammary gland



# Obesity and breastfeeding: the mechanisms

## Physiological explanations

### In humans, ?blunted PRL response to suckling

- New York, early 2000s
- n=23 normal weight, n=17 overweight or obesity
- PRL measured at baseline and +30min of suckling episode at 48h PP
- Women with obesity had significantly lower PRL response to suckling compared with normal-weight women

**TABLE 2.** Prolactin Concentration (ng/mL) Before and After Infant Suckling at 48 Hours and 7 Days Postpartum by Maternal Prepregnant BMI Category

Prolactin Sample	BMI Category*	
	Normal-Weight (n = 23)	Overweight/Obese (n = 17)
48 H		
Baseline	183.7 ± 77.0 (n = 22)	193.2 ± 57.0
30 Min	215.2 ± 62.1	182.9 ± 54.7
Response to suckling	26.0 ± 61.5 (n = 22)	-10.3 ± 28.3†

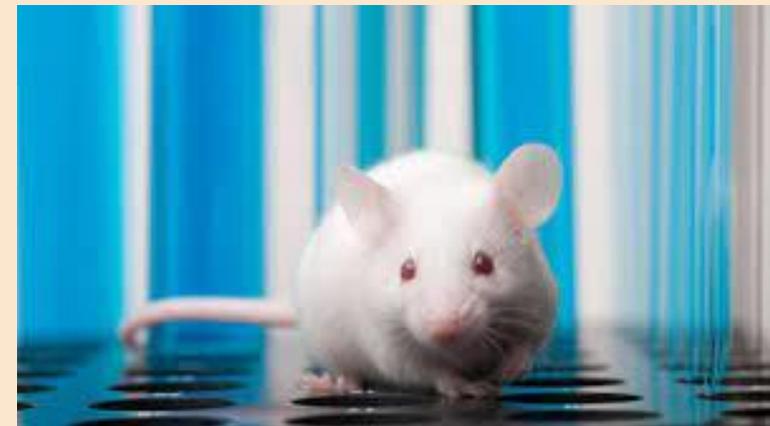
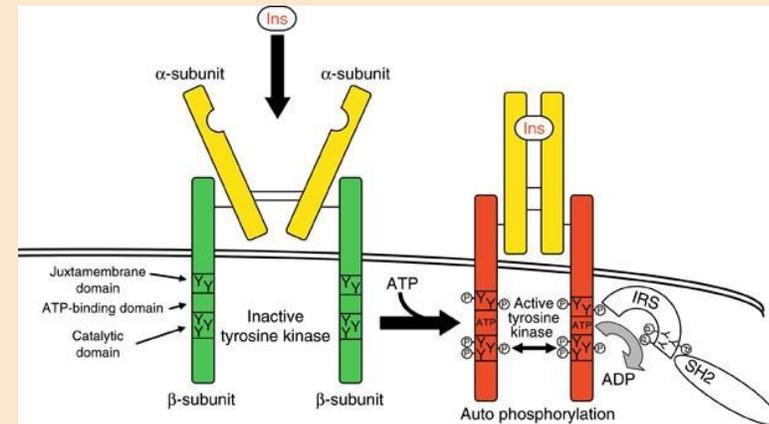
# Obesity and breastfeeding: the mechanisms

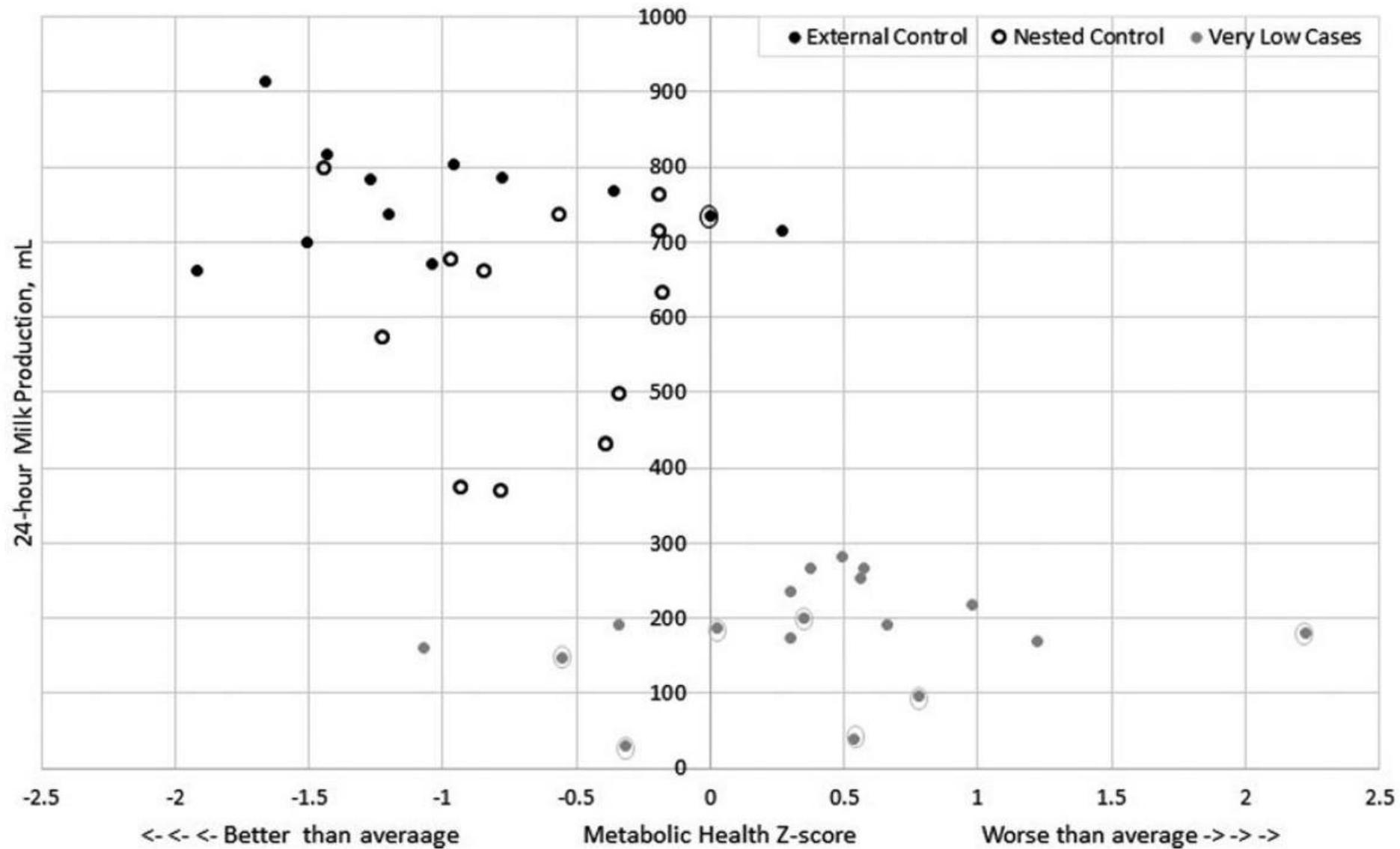
## Physiological explanations

### 2. Insulin resistance

- **Secretory activation** = time of dramatically upregulated gene expression in mammary epithelium.
- Increasingly realising that many of the key pathways are insulin-sensitive (require insulin signalling to be effectively activated)

Insulin probably critical to breast development and milk synthesis!  
Low insulin availability, and/ or insulin resistance (common in obesity), may impair milk production at the level of the lactocyte.





**FIG. 2.** Scatterplot of 24-hour milk production by Metabolic Syndrome Severity  $z$ -score, where 0,  $>0$ , and  $<0$   $z$ -scores signify average, worse than average, and better than average metabolic health profiles, respectively, as compared to all U.S. adults aged 20–65. *Solid black circles*: external control group,  $n=12$ ; *Open black circles*: nested control group,  $n=12$ ; *Solid gray circles*: severely low milk output cases,  $n=18$ . Encircled markers of any color indicate gestational diabetes mellitus diagnosis,  $n=7$  cases and  $n=1$  external control. GDM, gestational diabetes mellitus.

# Obesity and breastfeeding: the mechanisms



- age, parity, post-partum day and frequency of breast emptying similar between groups, but **nearly every measure of metabolic health was significantly worse in the low supply group:**

↑ BMI, ↑ waist circumference, ↑ fasting glucose, ↑ fasting Tg, ↑ SBP, ↓ HDL

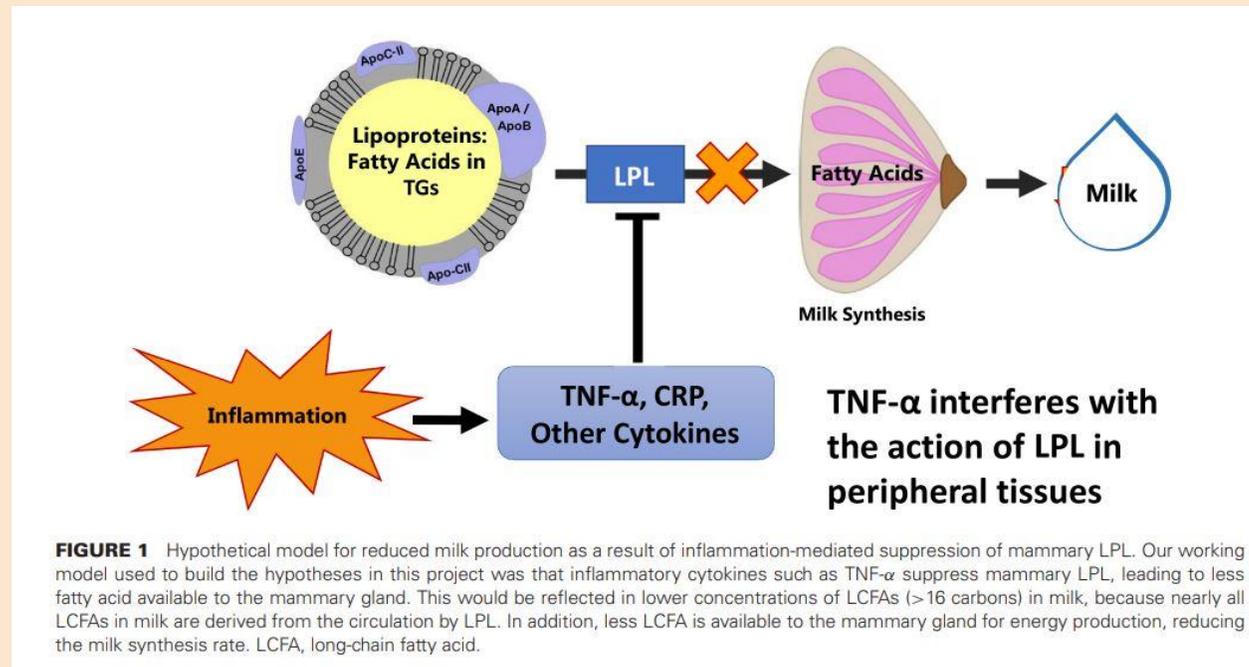
**AND...**

- when milk-derived lactocyte RNA was sequenced from a subset of the cohort, low-supply women (n=5) also had far **fewer RNA copies of the insulin-sensitive genes** in their lactocytes compared with “good supply” controls (n=4) <sup>1</sup> → implicates defective insulin signalling as a contributor to the low supply observed

# Obesity and breastfeeding: the mechanisms

## Physiological explanations

### 3. Inflammation → impaired substrate availability



# Obesity and breastfeeding: the **mechanisms**

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- impaired PRL dynamics
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## Clinical

- higher rates of obstetric and delivery complications (e.g. C-sections, NICU)
- practical difficulties – fit and hold etc

## Psychosocial

- negative self-perceived body image
  - inequitable support
- allostatic load of weight stigma

# Obesity and breastfeeding: the **mechanisms**

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## **Clinical explanations**

- Maternal obesity associated with increased rates of pregnancy and neonatal complications (PET, PTB, Caesarean sections, SCN admission, birth defects, hypoglycaemia and jaundice)
  - delayed early skin-to-skin
  - dyad separation
  - supplemental feeding
  - reduced access to support
- Research suggests that obesity relationship persists even after correction for key obstetric complications, delivery method etc

# Obesity and breastfeeding: the **mechanisms**

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## Clinical explanations

*"Overly large breasts usually betrayed a true poverty of milk, for the heavy fat parts impeded the separation of the milk and its free passage through the narrow conduits to the nipples."*

- Pragmatic difficulties – fit and hold more challenging, difficulty visualising areola, broader areola, weight of breast on infant chest <sup>1</sup>
- Some evidence for higher rates of nipple shield use in mothers with obesity <sup>2</sup>

# Obesity and breastfeeding: the **mechanisms**

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# Obesity and breastfeeding: the **mechanisms**

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## Psychosocial factors

### 1. Negative self-perceived body image

- more common in women with obesity compared with lean counterparts
- body dissatisfaction known to influence BF duration
- women with obesity most commonly cited “decency” as a reason not to initiate BF in one study, and were more likely to feel uncomfortable BF in public at 3mo than reference-weight women <sup>1</sup>



<sup>1</sup> Mok et al, Paediatrics 2008; 121(5): e1319-1324.

<sup>2</sup> Kair and Colaizy, Matern Child Health J 2016; 20(3): 593-601.

<sup>3</sup> Nommsen-Rivers et al, Curr Obstet Gyn Reports 2023; 12: 147-157.

# Obesity and breastfeeding: the mechanisms

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## Psychosocial factors



### 2. Implicit and explicit weight bias in healthcare setting, leading to **inequitable access to breastfeeding support**

- large US study (n=19145): 19% of mothers had obesity
- those with obesity less likely to receive BF information, assistance, or a telephone number for help
- also less likely to BF in the first hour post-delivery or be encouraged to BF on demand
- findings remained significant following adjustment for mode of delivery and key sociodemographic factors <sup>2</sup>

### 3. **Chronic stress of experiencing weight bias** = increased allostatic load → inflammation, which may physiologically impair milk production <sup>3</sup>

<sup>1</sup> Mok et al, Paediatrics 2008; 121(5): e1319-1324.

<sup>2</sup> Kair and Colaizy, Matern Child Health J 2016; 20(3): 593-601.

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# Supporting breastfeeding in the context of obesity

## Evidence for structured interventions?

- **Pumping?** No!
- **Structured support programmes?** It depends...
- **Metformin?** Good in theory, but in practise?

*Original Research*

international  
lactation consultant  
association

Journal of Human Lactation  
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**Feasibility and Acceptability of Metformin to Augment Low Milk Supply: A Pilot Randomized Controlled Trial**

Laurie Nommsen-Rivers, PhD, RD, IBCLC<sup>1</sup> ,  
Amy Thompson, MD<sup>1</sup>, Sarah Riddle, MD, IBCLC<sup>1,2</sup>,  
Laura Ward, MD, IBCLC<sup>1,2</sup>, Erin Wagner, MS<sup>1</sup>,  
and Eileen King, PhD<sup>1,2</sup>

1. Nommsen-Rivers et al, Curr Obs Gyn Reports 2023; 12: 147-157.

2. Nommsen-Rivers et al, J Human Lactation 2019; 35(2): 261-271.

# Supporting breastfeeding in the context of obesity

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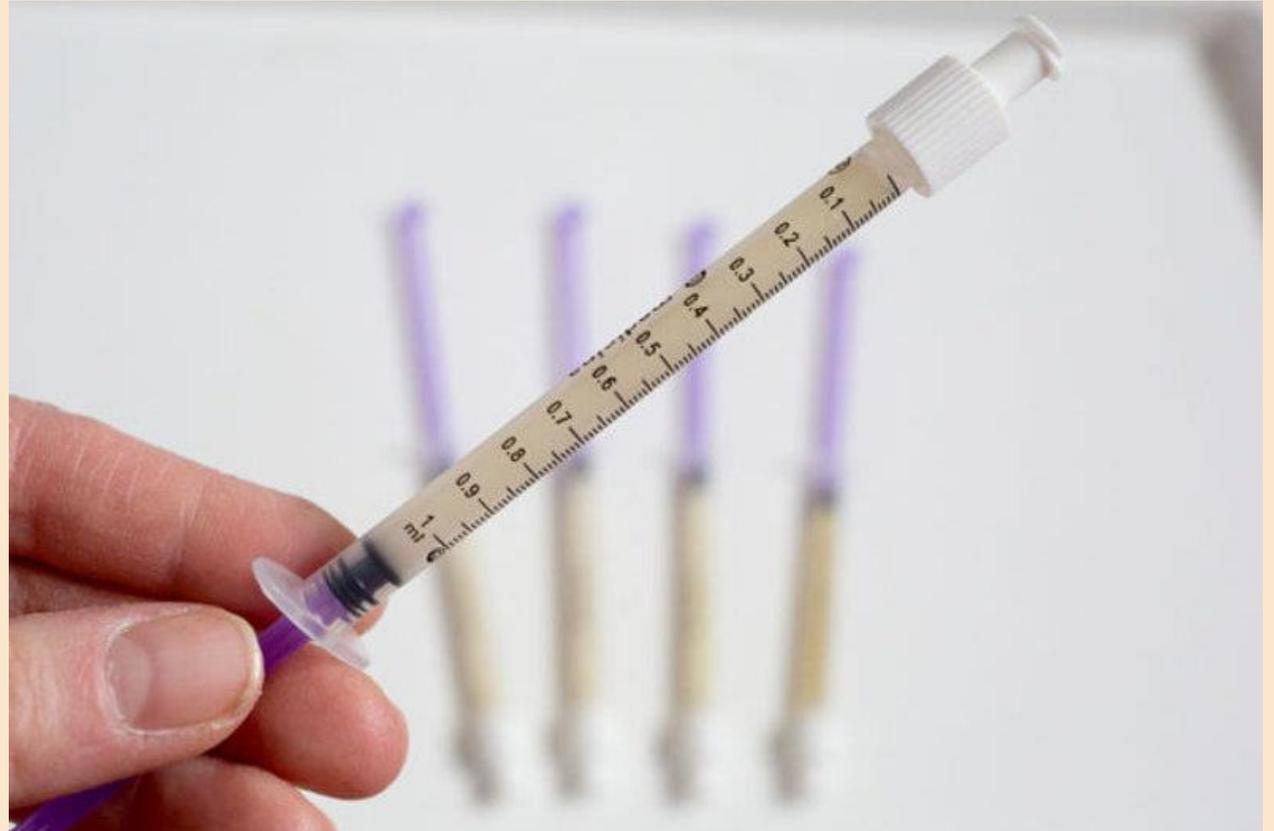
## Practical tips – prenatal

- Respectful care at every size: may emphasise ‘movement’ (over exercise), and ‘low-inflammatory diet patterns’, ‘reduced processed foods’; over calories and portion sizes; person-centred language
- Evaluation of breast size at prenatal visit:
  - widely spaced and conical – IGT?
  - previous breast reduction surgery and approach?
- Identify and share local resources for nursing bras, tops, and baby carriers that accommodate larger bodies.
- *“What have you heard about breastfeeding?”*

# Supporting breastfeeding in the context of obesity

## Practical tips – prenatal

- Validate concerns about milk supply – *“some mothers with obesity make lots of milk, and others make less”*.
- Advise that transition from colostrum to milk may be later than 72 h – options?
- If LGA infant is anticipated, or diabetes, review monitoring for hypoglycaemia.
- Consider antenatal expression – evidence of safety in women with diabetes, but no evidence for meaningful changes to longterm BF success. Highly individual decision!



# Supporting breastfeeding in the context of obesity

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## Practical tips – immediately post-partum

- **Immediate skin-to-skin**, including for Caesarean delivery
- **Positioning support:**
  - lying-back position may help, or football hold – or try breastfeeding lying down
  - may need rolled-up blanket or washcloth to elevate breast enough to visualise attachment (or use mirror!)
  - may need to support or lift breast to keep it from covering baby's nose to allow them to breathe/ swallow, but keep fingers away from areola or baby's mouth
  - properly-fitted maternity bra essential!



# Supporting breastfeeding in the context of obesity

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## Practical tips – outpatient postpartum followup

In the context of low supply

- May perform endo workup for cases of “true” low supply (TSH, PRL and pit hormones for Sheehan’s syndrome, check for retained placenta)
- Advise triple feeding with caution and for a time-limited period (a few days), followed by review around its sustainability
- **Be open to** partial/ mixed feeding, which can often be a satisfying, long-term relationship

# Supporting breastfeeding in the context of obesity

## Practical tips – outpatient postpartum followup

- **For post-bariatric surgery patients** – adherence to **B12 supplementation** vital!
  - B12 deficiency in up to 64% of pt at 36mo following **gastric bypass**
  - Case reports of exclusively BF infants with profound cortical atrophy and pancytopenia due to maternal B12 deficiency in this context
  - Advise close FU with bariatric dietician throughout preg and lactation





***“It’s time to start losing weight now, doc!”***



- Breastfeeding energy-expensive process, ~500 kcal/ day

...so, does it help weight loss?

- BF women in the **first 3 months** post-partum probably ↑ calories and ↓ activity to meet energy demands of lactation,
- whereas **beyond 3mo**, lactating women more likely to mobilise fat stores.  
MRI and skinfold studies show that *prolonged* lactation is associated with mobilisation of fat from the supra-iliac and mid-thigh regions (compared with non-lactating controls).

- overall SR conclusions – heterogenous pops, **no clear relationship**
- “many other interrelated psychosocial and behavioural variables”

- Post-partum weight loss of **0.5-1.0kg per week** shown not to adversely impact infant growth, and considered safe
- Lactation requires at least 210g of CHO per day – low-carbohydrate/ keto diets can precipitate **lactation ketosis** and should be avoided

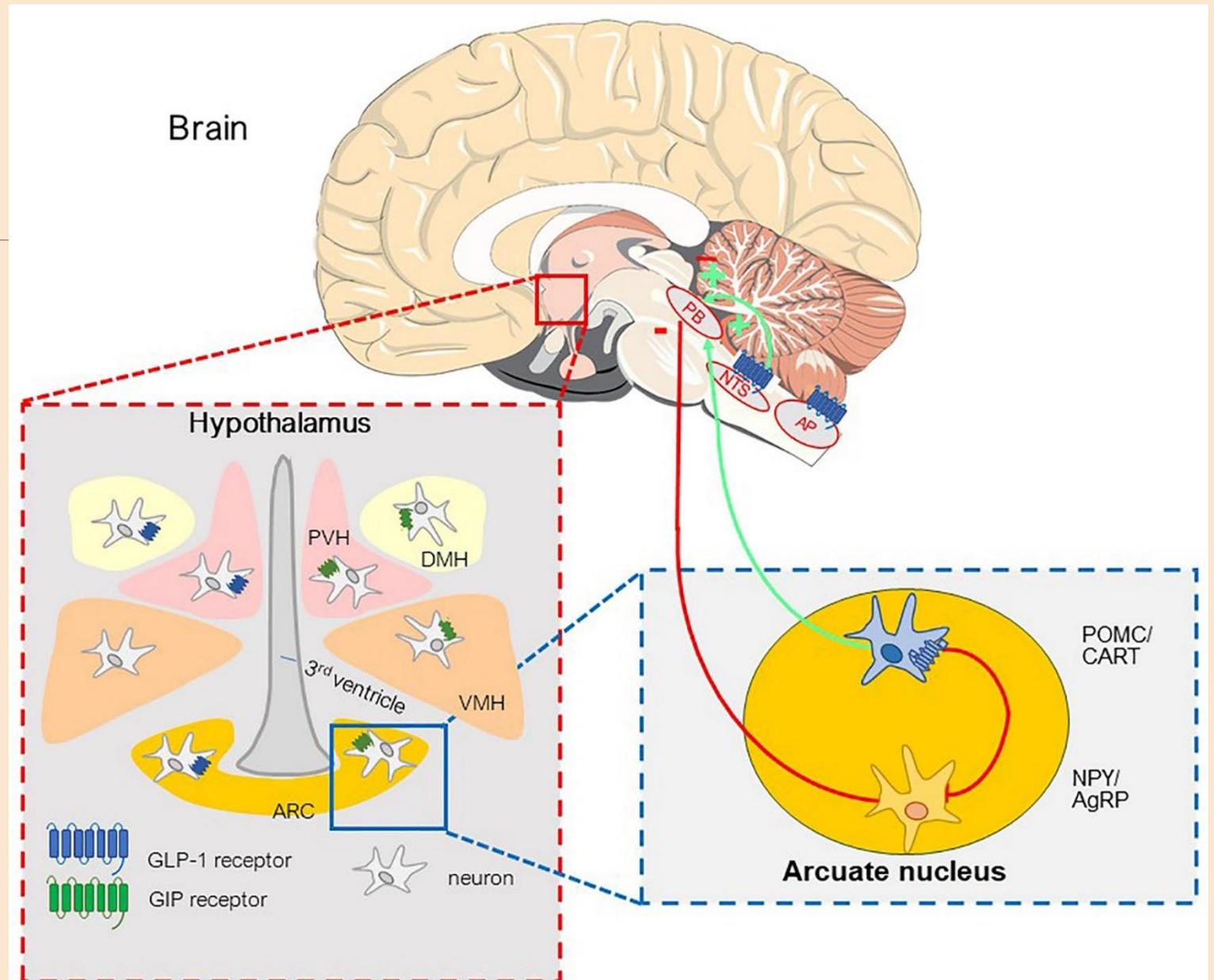
# Lactation ketosis

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- Been described in **veterinary** literature since early 1900s → often occurs in post-partum cows when unable to maintain sufficient energy intake and hepatic GNG to meet substrate demands of lactation.
- Result is a hypoglycaemic, hypoinsulinaemic state → adipose tissue breakdown → ketone formation
- Treatment = iv glucose
  
- Handful of cases in human literature – presents similar to DKA with nausea, vomiting, **increased anion gap acidosis with high blood and urine ketones**
  
- Mainly in lactating women with restricted access to CHO (e.g. keto diets, religious fasting, or severe illness) and/ or with increased lactation demands (e.g. twin lactation)
- Treatment = i.v. fluids and adequate carbohydrate!



# “What about Ozempic, doc?”



***“What about  
Ozempic, doc?”***

---

Drug information calls pertaining to  
**semaglutide use during lactation**  
to the InfantRisk Call Center at Texas Tech University increased  
by **over 500%** from 2021 to 2024.

# “What about Ozempic, doc?”



## Semaglutide

- In pregnant rats → offspring with reduced growth, increased skeletal and visceral malformations
- In monkeys → increased early preg losses and smaller offspring
- **Advice is to discontinue 2-3 months before pregnancy**
  
- **In lactation...**
  - Large molecule and >99% protein bound
  - Amount in milk thus theoretically likely to be very low
  - Even if entered milk, only 0.4-1% orally absorbed and thus theoretically unlikely to adversely affect BF infant

### Use in lactation

In lactating rats, semaglutide was excreted in milk. A risk to a breast-fed child cannot be excluded. Semaglutide should not be used during breast-feeding.

#### Breastfeeding Safety

##### Lactation Risk



L3 - No Data-Probably Compatible

##### Overview

Semaglutide is a human GLP-1 receptor agonist (or GLP-1 analog). Semaglutide reduces blood glucose through a mechanism where it stimulates insulin secretion and lowers glucagon secretion, both in a glucose-dependent manner. Thus, when blood glucose is high, insulin secretion is stimulated, and glucagon secretion is inhibited. The mechanism of blood glucose lowering also involves a minor delay in gastric emptying in the early postprandial phase. This product is large in molecular weight, is not absorbed orally in the human. Due to its large molecular weight, it is unlikely to enter milk or be orally absorbed by the infant. However, caution is recommended until we have more data on milk levels following the use of this hypoglycemic agent.

# “What about Ozempic, doc?”

Table 1. Summary of milk analysis results.

Participant	SubQ Dose (mg/Week)	Semaglutide (0 h)	Semaglutide (12 h)	Semaglutide (24 h)
1	0.5	n.d.	n.d.	n.d.
2	0.5	n.d.	n.d.	n.d.
3	0.25	n.d.	n.d.	n.d.
4	0.5	n.d.	n.d.	n.d.
5	0.25	n.d.	n.d.	n.d.
6	1	n.d.	n.d.	n.d.
7	0.5	n.d.	n.d.	n.d.
8 *	1	n.d.	n.d.	n.d.

\* The participant reported using a semaglutide product from a compounding pharmacy rather than a product prepared by the manufacturer; n.d. means not detected at lower limit of detection (LLOD) of 1.7 ng/mL.



Article

## Subcutaneous Semaglutide during Breastfeeding: Infant Safety Regarding Drug Transfer into Human Milk

Hanin Diab <sup>1</sup>, Taylor Fuquay <sup>2</sup>, Palika Datta <sup>2</sup>, Ulrich Bickel <sup>3</sup>, Jonathan Thompson <sup>1,†</sup> and Kaytlin Krutsch <sup>2,\*,†</sup>

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† These authors contributed equally to this work.

- “Born out of an immediate clinical need for data in order to make evidence based decisions when treating breastfeeding women for T2DM and/ or weight loss”
- Samples from 8 women at 0, 12 and 24h following self-administered sc semaglutide 0.25-1mg doses
- **Semaglutide NOT detected in ANY samples** – below limits of detection of the LCMS assay used in all cases
- “these findings support that the direct infant risk due to semaglutide in milk is likely negligible.... Fears concerning infant exposure to maternal semaglutide via breastmilk are likely overestimated.”

## *Another perspective...*

- 
- Need calories to make milk! Semaglutide markedly reduces food intake, and to varying degrees depending on individual – potential for impact on supply (and milk nutrient composition?) when exclusively breastfeeding
  - Nausea, vomiting and reduced PO intake may influence hydration status
  - In **early** post-partum, recommend a more gentle approach
    - Just made a human!
    - Gentle movement, sustainable dietary changes
    - Support for mood and social connections
    - These medications are increasingly considered longterm/ indefinite – so no rush to recommence?



Infant Risk Center

AT TEXAS TECH UNIVERSITY HEALTH SCIENCES CENTER

*Another perspective...*



# Key Points

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- Obesity is a chronic, complex disease.
- Once weight is lost, powerful hormonal and central mechanisms – not lack of motivation – drive (near-universal) weight regain.
- Weight stigma is pervasive, and a person-first approach is essential.
- Obesity is associated with earlier BF cessation. Underlying mechanisms are likely physiological, clinical and psychosocial.
- Weight loss during lactation (for all women) should be slow and sustainable, with caution around pharmacological agents or overly-restrictive diets in the early post-partum period.