

**WILD NUTRITION®**

# Clinical Solutions for Dysmenorrhoea in Adolescent (teen) Females



**Presented by  
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Wild Nutrition.  
Clinical Nutritional Therapist**

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# WILD NUTRITION®



FOOD-GROWN®



## Lorna Driver-Davies, Clinical Nutritional Therapist



With 14 years specialist experience in women's gynaecological and hormone nutrition. Functional medicine and naturopathic practice.

**Head of Practitioners, Wild Nutrition** (*previously Head of Nutrition*)

- Significant clinical experience in adolescent females, menstrual cycle irregularities, expertise in endometriosis and perimenopause. Particular interest in endocrine-gynaecological immunology.
- Case contributor to evidence-based peer reviewed functional medicine textbook on hormones, endometriosis and Hashimoto's thyroid disease.
- Regular nutrition speaker for consultant surgeons and nurses: BSGE (British Society of Gynaecological Endoscopy).
- Sits in on live theatre for general gynaecological and endometriosis laparoscopic surgery.
- Regularly lectures and mentors NT's and medics/other health practitioners.

# Introduction: Adolescent Dysmenorrhoea

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**This workshop explores why dysmenorrhoea can be worse in an adolescent females when compared to matured adult females of healthy menstrual fertile years.**

*Dysmenorrhoea is the medical term for painful periods caused by menstrual cramps of uterine origin.*

Affects approximately 70-90% of teenagers who menstruate; one-third report marked pain and 20-30% regularly miss school. Impact on education, sporting activities, lifestyle\*(1).

**Primary dysmenorrhoea:** No organic disease established. No pelvic pathologies are diagnosed.

**Secondary dysmenorrhoea:** Pelvic pathologies are diagnosed, e.g endometriosis. Endometriosis **is the most common cause** of secondary dysmenorrhea in adolescence.

*Caution: Secondary dysmenorrhoea can be easily missed in teens.*

# Established Pathogenesis (Mechanism) of Dysmenorrhoea

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Adult women with dysmenorrhoea have higher levels of prostaglandins, which are highest during the first two days of menses \*(2)

Pain associated with dysmenorrhoea: hyperproduction of uterine prostaglandins, particularly of PGF<sub>2</sub>α and PGE<sub>2</sub>. Resulting in increased uterine tone and high-amplitude contractions. \*(3)

Prostaglandin production is controlled by progesterone: when progesterone levels drop, immediately prior to menstruation, prostaglandin levels increase \*(4)

*Decline in progesterone leading to the generation of arachidonic acid, and therefore, the production of prostaglandins.*

**Primary dysmenorrhoea is also associated with pro-inflammatory cytokines (interleukin-6 [IL-6]).**

# Why are teens so disadvantaged in this pathogenesis picture (1)?

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**Adolescent females have an ‘immature cycle’ – irregular cycles absence of ovulation. The HPO axis is not fully mature at menarche.**

Long and irregular menstrual cycles are common in the first 1–2 years after menarche, but the majority of girls settle into a regular pattern of cycles (24–38-day interval) by late adolescence. \*(5)

*“Several studies that measured prostaglandin concentrations in the luteal phase, through endometrial biopsies and menstrual fluids, revealed that dysmenorrheic females have higher levels of prostaglandins than eumenorrheic females” \*(6)*

# Why are teens so disadvantaged in this pathogenesis picture (2)?

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## **Immature cycles lack progesterone - Progesterone mediates inflammatory prostaglandins \*(7)**

*“Many adolescent cycles previously classified as anovulatory may have instead been ovulatory with luteal insufficiency” \*(8).* Yes, there is ovulation, but progesterone would be insufficient. Again, ‘immature’ HPO function.

When there’s less progesterone produced, also less GABA available for pain management (GABA also works as an anti inflammatory) \*(9).

Oestrogen is potentially more inflammatory when not mediated by progesterone.

Lower levels of progesterone – not the same beneficial effects in adult female for sleep (deeper, good quality sleep being a way to de-flame).

**In addition, adolescent chronotype is later to bed (immune shift time) and melatonin ‘drop’ is later too. Melatonin immune system modulator/antioxidant.**

# Why are teens so disadvantaged in this pathogenesis picture (3)?

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This very special period of human existence, where **nutritional requirements are intensified** at the same time as **nutrition gaps statistically widen**.

***“Growth and development are rapid during teenage years, and the demand for most nutrients is relatively high”***. British Nutrition Foundation.

Development is rapid, and highly compressed – faster than any other lifestage. This is partly what creates the need for intensified nutrition.

**National Diet & Nutrition Survey – official statistics on teen females\*(10):**

1. Adolescent females lack the dietary intake of foods that would mitigate the over production of inflammatory prostaglandins.

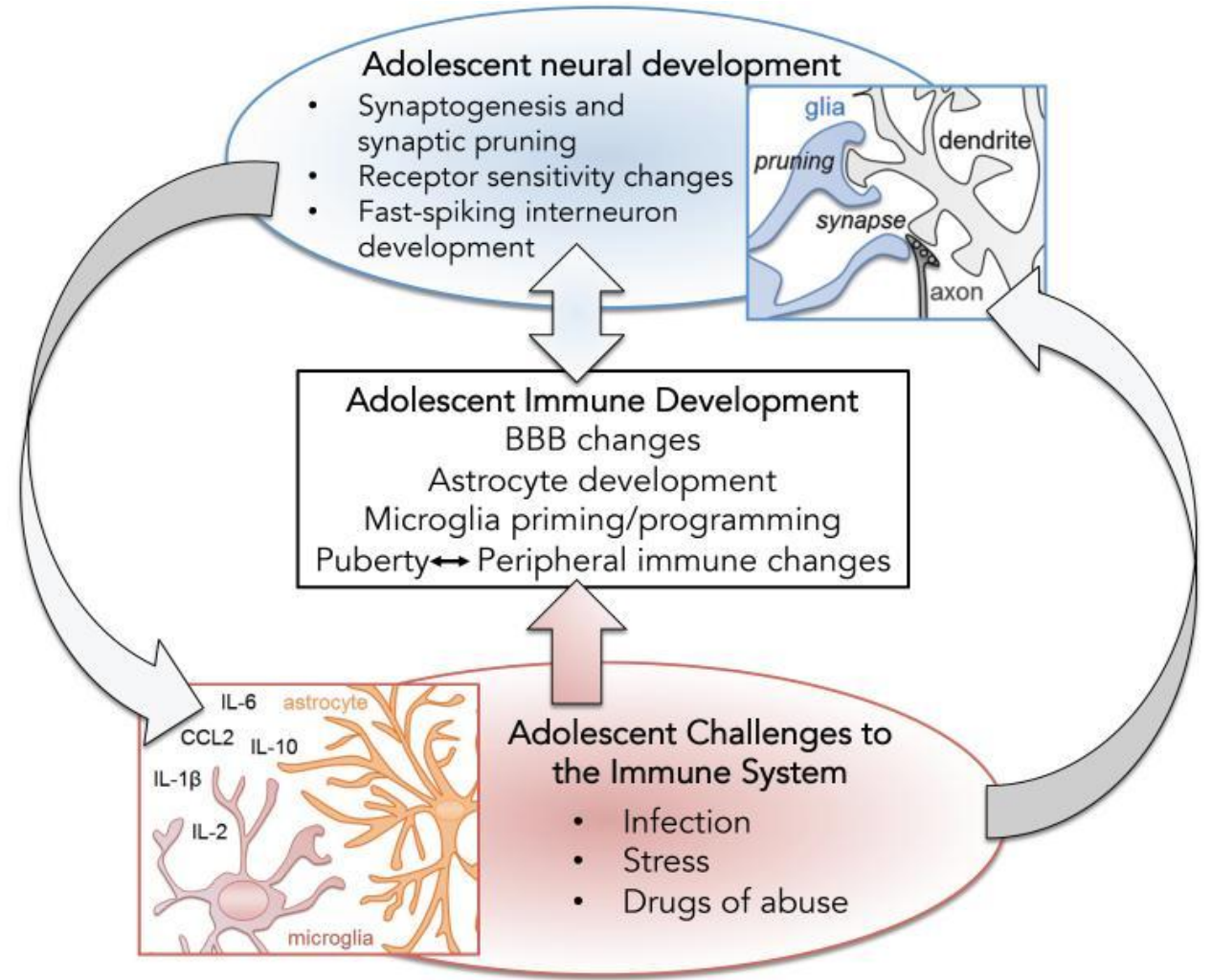
2. Increased intake foods that lead to higher levels of arachidonic acid (precursor to inflammatory prostaglandins).

# Why are teens so disadvantaged in this pathogenesis picture (4)?

Puberty & Menarche = Immune system changes as part the normal developmental course.

More predisposed to immune stability, inflammation.

Puberty is a time when the immune system is more prone to autoimmunity development \*(11)

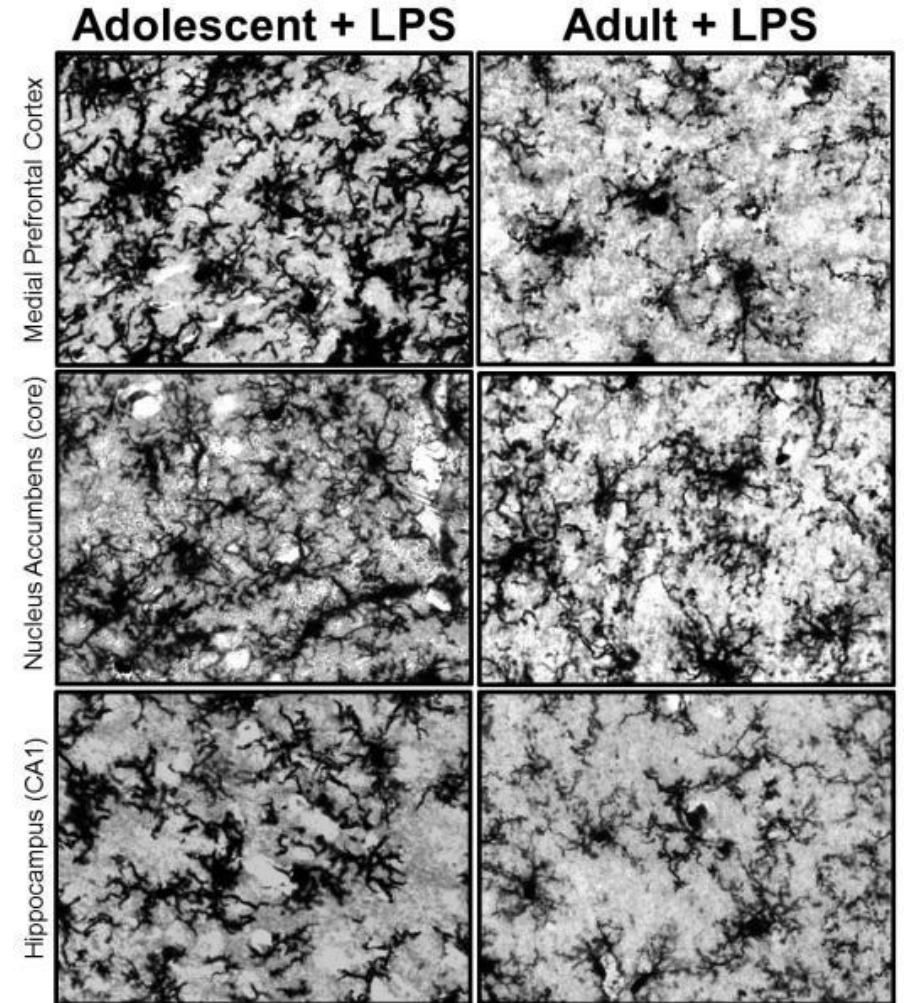


# Why are teens so disadvantaged in this pathogenesis picture (5)?

Teens LOVE sugary foods, junk food = more opportunity for gut dysbiosis/infection = inflammation. Link between gut dysbiosis and dysmenorrhoea \*(12).

Increased lipopolysaccharide (LPS) in teens: LPS, a component of gram-negative bacteria cell walls, increases the production of inflammatory prostaglandins \*(13).

Teens = more glycaemic instability = more inflammation.



# Why are teens so disadvantaged in this pathogenesis picture (6)?

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Teen brain (well established in research): Heightened amygdala (immature, and excited) activity, especially in response to social distress and ambiguity, is associated with **increased cortisol levels** and a stronger HPA axis (hypothalamic-pituitary-adrenal axis) response to stress.

Increased cortisol = inflammation.

Some studies also show the degradation of cortisol is SLOWER in teens

Histamine: Histamine = more inflammation and pain.

Histamine connection cross over with dysmenorrhoea:

- ADHD/neurodivergent/Tourette's
- OCD/anxiety/sleep issues
- multiple allergies/atopic skin issues

(and more)

# Key Supportive Supplement Formulas



FOOD-GROWN®

# Teen Girl & Premenstrual Support





Caution: Vegan and vegetarian adolescent females

# Immune Support & Multi Strain Biotic



Vitamin D also ESSENTIAL

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area.**

You can also ask Lorna more questions about her lecture topic, supplements and Wild Nutrition.

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<https://www.wildnutrition.com/pages/practitioner-community>