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EARLY MENARCHE: AN EMERGING REPRODUCTIVE HEALTH CHALLENGE CURRENT EVIDENCE AND AYURVEDIC PERSPECTIVES

Dr. Amrut Patil*[1], Dr. Savita S. Patil[2]

[1]Post Graduate Scholar, Department of Prasuti Tantra and Stree Roga,

[2]Professor, Department of Prasuti Tantra and Stree Roga,

Sri Sri College of Ayurvedic Science and Research Hospital, Bengaluru.

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***Corresponding Author**

Dr. Amrut Patil

Post Graduate Scholar, Department of Prasuti Tantra and Stree Roga, Sri Sri College of Ayurvedic Science and Research Hospital, Bengaluru.

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ABSTRACT

Early menarche is the onset of the first menstrual period at an unusually young age. Menarche before 10 years is generally considered early; however, menstruation at this age requires clinical assessment because it may indicate rapidly progressive puberty or an underlying endocrine disorder. Early menarche is influenced by genetic predisposition, childhood adiposity, nutritional status, neuroendocrine maturation, psychosocial adversity and possible exposure to endocrine-disrupting chemicals. Its clinical relevance extends beyond the timing of the first period and includes psychosocial distress, earlier epiphyseal maturation, and associations with later metabolic and reproductive health outcomes. This descriptive review summarises normal pubertal physiology, causes and consequences of early menarche, clinical assessment and management, and preventive concepts described in Ayurveda. Ayurvedic concepts such as Akala Rajodarshana, Ahara, Vihara, Agni, Dosha, Dhatu and Artavavaha Srotas are discussed as interpretive frameworks rather than as established biomedical mechanisms. Preventive measures include balanced nutrition, healthy weight, physical activity, adequate sleep, reduced exposure to potentially harmful chemicals and supportive family environments. Girls with early menstruation or rapidly progressive secondary sexual characteristics require individual clinical evaluation; Ayurvedic lifestyle

measures should complement, not replace, indicated paediatric endocrine and gynaecological care.

KEYWORDS: Early menarche; precocious puberty; hypothalamic–pituitary–gonadal axis; childhood obesity; Ayurveda; Akala Rajodarshana.

1. INTRODUCTION

Menarche is the first menstrual bleeding and represents a visible milestone in female pubertal development. Its timing reflects the interaction of genetic, nutritional, metabolic, neuroendocrine and environmental influences. Early menarche is important because it may occur as part of normal variation, but it can also be a marker of early activation of the reproductive axis or a manifestation of peripheral sex-steroid exposure. The distinction between early menarche and precocious puberty is clinically important: menarche before 10 years is early, whereas precocious puberty in girls is conventionally defined by the appearance of secondary sexual characteristics before 8 years. Vaginal bleeding in a young child should not automatically be assumed to represent menstruation.

The age at menarche varies between populations and individuals. Secular trends, changing nutritional patterns, childhood obesity, urbanisation and environmental exposures have been studied as contributors to shifts in pubertal timing. Associations do not establish causation, and the relative contribution of each factor differs across populations. A balanced review therefore requires both modern clinical evidence and careful interpretation of traditional concepts.

2. OBJECTIVES

- To describe normal pubertal development and the physiology leading to menarche.
- To outline definitions, types, risk factors and proposed mechanisms of early menarche.
- To summarise physical, metabolic, reproductive and psychosocial associations.
- To describe the clinical evaluation and principles of management.
- To discuss Ayurvedic concepts and preventive lifestyle measures relevant to adolescent reproductive health.

3. Normal Puberty and Menarche

3.1 Pubertal milestones

Puberty is the developmental transition during which secondary sexual characteristics appear,

reproductive capacity matures and growth accelerates. In girls, breast development (thelarche) is commonly the first visible sign of gonadarche. Pubic and axillary hair may develop through adrenarche, a partly independent process related to increased adrenal androgen production. Menarche generally occurs after breast development has progressed for a period of time; the sequence and tempo vary among individuals.

- Thelarche: onset of breast development, usually reflecting ovarian oestrogen activity.
- Pubarche: development of pubic hair; often influenced by adrenal androgens.
- Adrenarche: maturation of the adrenal zona reticularis with increasing DHEA and DHEAS.
- Gonadarche: reactivation of the hypothalamic–pituitary–gonadal (HPG) axis.
- Menarche: first menstrual bleeding, occurring after progressive maturation of the reproductive axis.

3.2 HPG-axis physiology

During childhood, hypothalamic GnRH activity is relatively suppressed. As puberty begins, pulsatile gonadotropin-releasing hormone (GnRH) secretion increases, initially becoming more evident during sleep. GnRH stimulates the anterior pituitary to release luteinising hormone (LH) and follicle-stimulating hormone (FSH). These gonadotropins support ovarian follicular development and oestradiol production. Oestrogen promotes breast development, uterine growth and endometrial proliferation. With further maturation of the reproductive axis, menstrual bleeding becomes possible; ovulatory cycles may take time to become established after menarche.

3.3 Tanner staging

Tanner staging documents breast and pubic-hair development and helps establish the stage and tempo of puberty. Serial measurements of height and growth velocity are also important. Rapid progression through stages, accelerated linear growth or a markedly advanced bone age can support the need for further investigation.

4. Definitions and Classification

Term	Description
Early menarche	First menstrual period before 10 years of age is commonly used as a practical threshold for early menarche.
Precocious puberty	Secondary sexual characteristics beginning before 8 years in girls; assessment depends on clinical context and progression.
Central precocious	Premature activation of the HPG axis; the pattern resembles

puberty (CPP)	normal puberty but begins too early.
Peripheral precocious puberty	Sex-steroid production or exposure independent of central HPG-axis activation; may arise from ovarian, adrenal, gonadal, tumour-related or exogenous sources.
Incomplete/partial pubertal development	Isolated or non-progressive findings, such as premature thelarche or adrenarche, without complete progressive puberty.

These terms should not be used interchangeably. A girl who menstruates before age 10 requires assessment of the history, pubertal sequence and possible causes. Vaginal bleeding may also result from local genital conditions, trauma, infection, foreign body or other pathology, so the source of bleeding must be established.

5. Risk Factors and Associated Influences

5.1 Genetic and familial factors

Pubertal timing has a substantial heritable component. Family history, including the mother's age at menarche, can provide useful context. Genes involved in the regulation of reproductive maturation include KISS1, KISS1R, MKRN3, TAC3, TACR3 and LIN28B. Genetic susceptibility interacts with nutrition, body composition and the environment; family history is informative but cannot predict an individual child's timing with certainty.

5.2 Nutritional status and adiposity

Childhood overweight and obesity are consistently associated with earlier pubertal development in many populations. Adipose tissue affects endocrine and metabolic signalling, including leptin, insulin and peripheral steroid metabolism. Leptin communicates the adequacy of energy stores to the neuroendocrine system; it is permissive for normal reproductive function but should not be interpreted as a single trigger of puberty. High-energy diets and rapid weight gain may contribute indirectly through increased adiposity and insulin resistance.

5.3 Psychosocial stress and childhood adversity

Adverse childhood experiences, family conflict, emotional neglect and chronic stress have been associated with variation in pubertal timing in some studies. Potential pathways include stress-responsive neuroendocrine systems, sleep disruption and behavioural or metabolic changes. These associations are complex and do not mean that stress inevitably causes early menarche. Assessment should be sensitive, non-blaming and focused on the child's safety and well-being.

5.4 Environmental exposures

Some endocrine-disrupting chemicals, including bisphenols, phthalates and selected pesticides, can interact with hormone signalling. Epidemiological findings vary according to chemical, dose, timing of exposure and study design. The evidence is not sufficient to attribute early menarche to a single everyday exposure in an individual child. Reasonable exposure-reduction practices include avoiding heating food in unsuitable plastic containers, following food-safety guidance and minimising unnecessary contact with chemicals according to product instructions.

5.5 Other developmental and social factors

Urbanisation, reduced physical activity, sleep patterns, socioeconomic conditions, infant feeding practices and rapid catch-up growth after low birth weight have been investigated in relation to pubertal timing. These variables often overlap with diet, adiposity and access to health care. Their effects should be interpreted as population-level associations rather than deterministic causes.

5.6 Gut microbiome: an emerging area

The gut microbiome participates in metabolism, immune regulation and the processing of steroid hormones. The term estrobolome describes microbial genes and functions involved in oestrogen metabolism, including bacterial enzymes that can alter the enterohepatic circulation of oestrogens. Studies have reported differences in microbial composition in some groups with altered pubertal timing. Most evidence remains observational, and a direct causal role in early menarche has not been established. Microbiome-based interventions should not currently be presented as proven prevention or treatment.

6. Pathophysiological Overview

In central precocious puberty, premature activation of hypothalamic GnRH pulsatility leads to increased LH and FSH secretion, ovarian follicular activity and oestrogen production. Oestrogen promotes breast development, uterine maturation and endometrial growth. If the process progresses sufficiently, vaginal bleeding and menarche may occur at an unusually young age.

Peripheral precocious puberty follows a different pathway. Oestrogen or androgen exposure may arise independently of central HPG-axis activation, for example from functional ovarian cysts, hormone-producing tumours, adrenal disorders or exogenous hormones. Peripheral

puberty can sometimes stimulate subsequent central activation. The distinction is important because investigation and treatment depend on the underlying mechanism.

Early menarche is not synonymous with central precocious puberty. The age of onset of breast development, growth velocity, bone age, laboratory results and imaging findings help determine whether a child has central activation, peripheral hormone exposure, an isolated pubertal variant or another cause of bleeding.

7. Clinical Consequences and Health Associations

7.1 Growth and reproductive health

Earlier exposure to sex steroids may accelerate skeletal maturation and epiphyseal closure, potentially reducing adult height when puberty is substantially premature and progressive. Early menarche is also associated in some studies with later menstrual irregularity, obesity and polycystic ovary syndrome (PCOS), although these relationships are influenced by shared factors such as childhood adiposity and insulin resistance.

7.2 Metabolic and cardiovascular health

Earlier menarche has been associated with increased later-life risk of obesity, insulin resistance, type 2 diabetes, dyslipidaemia and hypertension in several observational studies. Possible explanations include shared early-life determinants, prolonged exposure to sex steroids and the relationship between adiposity and pubertal timing. These associations do not establish that early menarche alone causes these outcomes.

7.3 Psychological and social effects

A child whose physical development is ahead of peers may experience embarrassment, anxiety, low self-esteem, body-image concerns, unwanted attention or difficulty understanding menstrual changes. Support should be developmentally appropriate and should include practical menstrual education, reassurance, safeguarding when indicated and non-judgmental family counselling.

7.4 Obstetric and long-term outcomes

Some cohort studies have reported associations between early menarche and later gestational diabetes, hypertensive disorders of pregnancy, preterm birth or caesarean delivery. These outcomes are multifactorial and may be partly explained by later obesity and metabolic risk. Early menarche should be viewed as a potential marker for health monitoring, not as a direct

prediction that an individual will develop obstetric complications.

8. Clinical Evaluation

8.1 History

- Age at onset of breast development, pubic hair and first bleeding; sequence and rate of progression.
- Growth pattern, recent acceleration in height and previous growth records.
- Maternal and family history of pubertal timing.
- Birth history, including prematurity or low birth weight, and subsequent growth pattern.
- Nutrition, activity, sleep and weight trajectory.
- Use or household exposure to oestrogen- or androgen-containing medicines, creams or supplements.
- Headache, seizures, visual symptoms, galactorrhoea, thyroid symptoms or signs of androgen excess.
- Bleeding pattern, pain, discharge, trauma concerns and any possibility of a local genital cause.

8.2 Examination

- Measure height, weight and BMI; plot measurements on appropriate growth charts.
- Assess growth velocity and document Tanner breast and pubic-hair stages.
- Examine for signs of insulin resistance, including acanthosis nigricans, where relevant.
- Look for café-au-lait macules or other findings that may suggest a specific syndrome.
- Perform thyroid, abdominal, neurological or genital assessment when clinically indicated, with appropriate consent, chaperoning and safeguarding.

8.3 Investigations

Testing should be guided by age, progression, examination and clinical suspicion. Possible investigations include basal LH and FSH, oestradiol, and a GnRH/GnRH-agonist stimulation test when central activation needs clarification. TSH/free T4, prolactin, DHEAS, testosterone, 17-hydroxyprogesterone or beta-hCG may be considered according to the differential diagnosis. Bone age is usually assessed by radiograph of the left hand and wrist. Pelvic ultrasonography can assess uterine size, endometrial appearance and ovarian morphology. Brain MRI is considered according to age, neurological features and the clinical pattern; it is particularly important in selected children with suspected central precocious puberty.

No single test should be interpreted in isolation. Laboratory thresholds depend on assay and protocol, and pelvic ultrasound findings are supportive rather than independently diagnostic. Referral to paediatric endocrinology or an appropriately experienced specialist is recommended for suspected progressive precocious puberty, very early bleeding, rapid growth acceleration or abnormal findings.

9. Principles of Management

9.1 Management according to cause

Management begins with establishing whether the presentation represents central precocious puberty, peripheral precocious puberty, an isolated pubertal variant or another cause of vaginal bleeding. Treatment is individualised according to age, rate of progression, bone-age advancement, predicted adult height, underlying pathology and psychosocial impact.

- Central precocious puberty: GnRH agonists such as leuprolide, triptorelin or a histrelin implant may be used by specialists when indicated. They suppress pituitary gonadotropin secretion after an initial stimulatory phase and can slow pubertal progression.
- Peripheral precocious puberty: treat the cause, such as an ovarian/adrenal disorder, hormone-producing lesion or exogenous hormone exposure. Observation may be appropriate for selected benign, self-limited conditions under specialist guidance.
- Isolated or non-progressive variants: clinical follow-up with serial growth and pubertal assessment may be sufficient, depending on the diagnosis.
- Vaginal bleeding in a young child: establish the source and exclude local pathology, trauma, infection and other relevant causes.

Medication choice, dose, route and interval must follow current specialist guidance and the specific product formulation. Treatment should not be initiated from a general review article alone.

9.2 Lifestyle and family support

- Encourage balanced meals with vegetables, fruit, whole grains, pulses and appropriate sources of protein.
- Support healthy growth and weight without stigma, restrictive dieting or blaming the child.
- Promote regular age-appropriate physical activity and reduce prolonged sedentary screen time.

- Maintain consistent sleep and wake times and adequate sleep duration.
- Provide age-appropriate menstrual education, emotional support and a safe environment for discussing concerns.
- Review potential exposure to hormone-containing products and use safe food-storage and chemical-handling practices.

10. Ayurvedic Perspectives

Ayurvedic literature describes reproductive maturation within broader concepts of age, Dhatu development, Artava formation, Agni, Dosha and Srotas. These concepts can provide a traditional framework for discussing growth, diet, daily routine and mental well-being. However, early menarche is not a separately validated biomedical diagnosis within classical terminology, and the proposed Ayurvedic interpretation should be clearly distinguished from established endocrine physiology.

10.1 Vayovibhajana and reproductive maturity

Classical discussions of Vayovibhajana distinguish stages of life, including Bala, Madhyama and Vriddha. Sushruta Samhita, Sutrasthana 14, discusses developmental changes associated with maturation and the manifestation of reproductive characteristics. The image of fragrance becoming apparent as a flower opens is used to explain how latent reproductive potential becomes manifest with development. Classical texts discuss the usual timing of Artava pravritti, but these historical descriptions should not be treated as modern diagnostic cut-offs.

10.2 Conceptual interpretation of Akala Rajodarshana

For an Ayurvedic discussion, early menstrual onset may be described conceptually as Akala Rajodarshana, meaning manifestation of menstruation at an inappropriate or premature time. A proposed samprapti framework in the presentation includes Pitta predominance, Kapha and Meda involvement, changes in Rasa and Rakta, accelerated Artava maturation and disturbance of Artavavaha Srotas. This is a theoretical interpretive model; it has not been validated as an equivalent of central or peripheral precocious puberty.

Ayurvedic component	Descriptive interpretation used in the presentation
Dosha	Pitta as predominant, with Kapha and Vata involvement proposed in the conceptual model.
Dushya	Rasa, Rakta, Meda and Artava are discussed as involved tissues/substrates.
Agni	Altered or irregular tissue-level transformation is

	described as Dhatu-agni vaishamya.
Srotas	Rasavaha, Raktavaha and Artavavaha Srotas are considered in the traditional explanation.
Adhishthana	Garbhashaya and Artavavaha Srotas are identified as relevant sites in the conceptual account.

10.3 Ahara and metabolic health

Ahara is one of the Traya Upastambha in Ayurveda and is central to nourishment and tissue development. The presentation discusses Guru Ahara, excessive Madhura Rasa, Ati-snigdha Ahara, Viruddha Ahara and frequent ultra-processed foods as dietary patterns that may contribute to Kapha/Meda accumulation or impaired Agni in the traditional model. In modern terms, frequent energy-dense foods and sugar-sweetened beverages can contribute to excess energy intake and childhood adiposity. The evidence supports healthy, balanced dietary patterns; it does not establish that a specific rasa or an individual food directly causes early menarche.

10.4 Manas and psychosocial well-being

Ayurveda describes Manas in relation to Sharira and Indriya and discusses the Gunas Sattva, Rajas and Tamas. The presentation relates chronic stress, fear, grief, anger, family conflict and emotional adversity to disturbance of mental and physiological balance. Modern research similarly explores associations between adverse experiences, stress pathways, sleep and pubertal timing, although causal pathways remain uncertain. Counselling should be supportive and should never imply that the child or family is responsible for early development.

10.5 Ahara, Vihara and preventive measures

A practical preventive approach can be framed through Nidana Parivarjana and healthy Ahara-Vihara. Suggested food choices include whole grains such as Yava and millets, pulses such as Mudga, seasonal vegetables and whole fruit, with adequate dietary diversity. These are general healthy-eating recommendations rather than a proven treatment for early menarche.

- Vyayama: regular age-appropriate physical activity to support healthy weight, metabolic health and emotional well-being.
- Nidra: consistent, sufficient sleep and a stable sleep–wake schedule.
- Dinacharya: regular daily routines, timely meals, activity and rest.

- Ritucharya: seasonal adaptation of diet and activity within safe, nutritionally adequate practices.
- Nidana Parivarjana: identify and reduce modifiable contributors such as excess energy intake, inactivity, poor sleep and inappropriate exposure to hormone-containing products.

10.6 Yoga and stress regulation

Gentle, age-appropriate yoga may be offered as an adjunct for relaxation, body awareness and physical activity. Poses mentioned in the presentation include Balasana, Apanasana, Paschimottanasana, Marjaryasana-Bitilasana and Tadasana. Practices should be adapted to the child's ability and avoided or modified when pain, injury or other contraindications are present. Yoga has not been established as a treatment that reverses precocious puberty or delays menarche.

10.7 Rajaswala Paricharya and Satvavajaya

Rajaswala Paricharya refers to traditional practices associated with menstruation. These practices may be discussed as part of cultural context and menstrual care, but they cannot prevent menarche once pubertal maturation has begun and should not restrict nutrition, education, movement or access to medical care. Satvavajaya Chikitsa is described in Ayurveda as regulation of the mind and avoidance of unwholesome mental engagement. In contemporary supportive care, its broad themes may be related to age-appropriate counselling, emotional regulation, parental guidance and a safe, non-stigmatising environment.

10.8 Ayurvedic herbs: evidence and caution

The drugs like Shatavari, Guduchi, Yashtimadhu, Amalaki and Brahmi with proposed traditional or experimental properties. These herbs should not be prescribed to children for early menarche or pubertal suppression on the basis of theoretical hormonal effects. Evidence for efficacy and safety in this specific indication is insufficient, and some herbal products may have endocrine activity, contamination or drug-interaction risks. Any use in children requires qualified clinical oversight and must not delay appropriate diagnostic evaluation.

11. DISCUSSION

Early menarche is best approached as a clinical sign and a potential marker of altered pubertal timing rather than as a single disease with one cause. Assessment must separate early but otherwise normal variation from rapidly progressive central puberty, peripheral

hormone production and non-menstrual causes of vaginal bleeding. Genetic background, childhood adiposity, nutrition, psychosocial context and environmental exposures may contribute in different combinations.

The most actionable preventive priorities are supportive and evidence-aligned: healthy nutrition, avoidance of excessive weight gain, physical activity, adequate sleep, emotional safety and appropriate clinical follow-up. Evidence regarding endocrine-disrupting chemicals and the gut microbiome is evolving; neither should be presented as a confirmed cause in an individual child. Associations between early menarche and later health outcomes justify long-term attention to general health, but they should not be used to alarm families or predict inevitable disease.

Ayurvedic concepts can contribute a culturally relevant language for discussing routine, diet, mental well-being and prevention. Nevertheless, the proposed concepts of Dosha, Dhatu, Agni and Srotas are explanatory constructs within Ayurveda and should not be claimed to map directly onto the HPG axis or to establish a causal mechanism. Integrative care is safest when traditional lifestyle measures are used as supportive practices while diagnosis, investigations and indicated endocrine treatment follow contemporary clinical standards.

12. CONCLUSION

Early menarche, particularly before 10 years, warrants careful assessment to establish the source of bleeding, document pubertal progression and exclude central or peripheral causes of precocious puberty. Its determinants are multifactorial, with genetic predisposition and childhood adiposity among the important associated factors; psychosocial and environmental influences remain areas of active research. Clinical care should combine accurate diagnosis, treatment of the underlying cause when indicated, and practical support for the child and family. Ayurvedic approaches centred on Ahara, Vihara, Dinacharya, physical activity and mental well-being may complement preventive counselling, but claims of direct hormonal treatment or prevention require stronger evidence. Early recognition, non-stigmatising education and appropriate specialist referral remain essential.

REFERENCES

1. Sperling MA, editor. Sperling Pediatric Endocrinology. 5th ed. Philadelphia: Elsevier, 2021.
2. Klein DA, Emerick JE, Sylvester JE, Vogt KS. Disorders of puberty: an approach to

diagnosis and management. Am Fam Physician, 2017; 96(9): 590–599.

3. Carel JC, Léger J. Clinical practice: precocious puberty. N Engl J Med., 2008; 358: 2366–2377. doi:10.1056/NEJMcp0800459.
4. Latronico AC, Brito VN, Carel JC. Causes, diagnosis, and treatment of central precocious puberty. Lancet Diabetes Endocrinol, 2016; 4(3): 265–274.
5. World Health Organization. WHO recommendations on adolescent health and development. Geneva: WHO. Consult current WHO resources for adolescent health guidance.
6. Day FR, Thompson DJ, Helg vison R, et al. Genomic analyses identify hundreds of variants associated with age at menarche and support a role for puberty timing in cancer risk. Nat Genet., 2017; 49: 834–841.
7. Eckert-Lind C, Busch AS, Petersen JH, et al. Worldwide secular trends in age at pubertal onset assessed by breast development among girls: a systematic review and meta-analysis. JAMA Pediatr, 2020; 174(4): e195881.
8. Uche-Nwachi EO, et al. Gut microbiome and pubertal development: emerging evidence and potential mechanisms. Pediatr Res., 2025. doi:10.1038/s41390-025-04180-0. Verify full bibliographic details before submission.
9. Kaplowitz P, Bloch C; Section on Endocrinology, American Academy of Pediatrics. Evaluation and referral of children with signs of early puberty. Pediatrics, 2016; 137(1): e20153732.
10. Biro FM, Kiess W. Contemporary trends in the onset and completion of puberty. Endocr Dev., 2016; 29: 122–133.
11. Charaka. Charaka Samhita. Sutrasthana and Chikitsasthana. Use the edition and translator consulted by the author.
12. Sushruta. Sushruta Samhita. Sutrasthana, Chapter 14. Use the edition and translator consulted by the author.
13. Vagbhata. Ashtanga Hridaya. Sharirasthana. Use the edition and translator consulted by the author.
14. Kashyapa. Kashyapa Samhita. Relevant sections on Bala, growth and reproductive development. Use the edition and translator consulted by the author.
15. Kapoorchand H. A Comprehensive Treatise on Prasuti Tantra. Varanasi: Chaukhambha Vishvabharati, 2016.
16. Kapoorchand H. A Comprehensive Treatise on Stri Roga (Gynaecology). Varanasi: Chaukhambha Vishvabharati, 2016.

17. Dutta DC, Konar H. DC Dutta's Textbook of Gynaecology. New Delhi: Jaypee Brothers Medical Publishers, 2014.
18. Dutta DC, Konar H. DC Dutta's Textbook of Obstetrics. New Delhi: Jaypee Brothers Medical Publishers, 2014.
19. Clinical case reference cited in seminar presentation: doi:10.1177/1179547620933585. PMID: 32694924; PMCID: PMC7350395. Confirm article title, authors and journal details before final submission.