

STRESS DURING MENSTRUAL CYCLE IN ADOLESCENTS

Ms. Divya Jacob* | Dr. Shama Lohumi**

* Research Scholar, Himalayan University, Itanagar, Arunachal Pradesh, India.

** Principal, Shivalik Institute of Nursing, Sanjauli, Shimla, Himachal Pradesh, India.

ABSTRACT

Adolescence is a time of life with specific health and needs and rights for development. It is also a time to develop awareness and skills, learn to manage emotions and relationships, and acquire attributes and abilities that will be critical for enjoying the adolescent years and assuming adult roles. Adolescent girls constitute one fifth of the female population in the world. Generally this group is considered healthy and has not been given adequate attention in health programmes. The reason is age specific mortality is comparatively low in this age group as compared to others. In countries like India, adolescent girls face serious health problem due to socio-economic and environmental conditions as well as gender discrimination.

Key Words: Stress, Menstrual cycle, Adolescent girls.

About Authors

Author Ms. Divya Jacob is a Ph.D. Scholar at Himalayan University in Itanagar in the Indian state of Arunachal Pradesh.



Author Dr Shama Lohumi is Principal at Shivalik College of Nursing, Shimla, Himachal Pradesh, India. She has various national and international publications in her name.

INTRODUCTION

Adolescence is a time of life with specific health and needs and rights for development. It is also a time to develop awareness and skills, learn to manage emotions and relationships, and acquire attributes and abilities that will be critical for enjoying the adolescent years and assuming adult roles. All societies recognise that there is a difference between being a child and becoming an adult. How this transition from childhood to adulthood is defined and recognised differs between cultures and over time. In the past, it has often been relatively rapid, and in some societies it still is. In many countries, however, this is changing. (WHO)

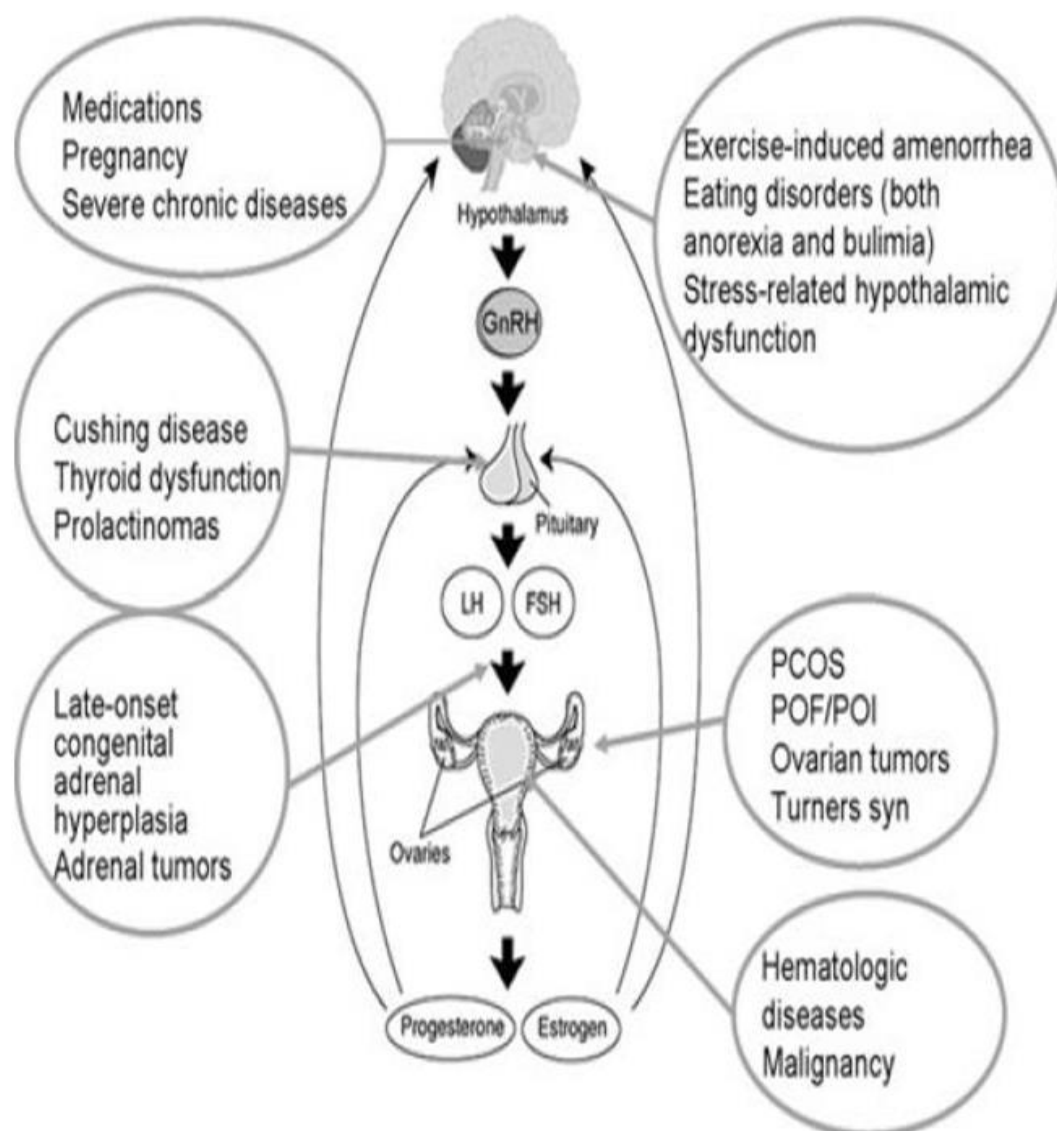
Adolescent girls constitute one fifth of the female population in the world. Generally this group is considered healthy and has not been given adequate attention in health programmes. The reason is age specific mortality is comparatively low in this age group as compared to others. In countries like India, adolescent girls face serious health problems due to socio-economic and environmental conditions as well as gender discrimination. These factors make them more vulnerable to health risks. Globally adolescents account for 1/5th of the population that is more than 1 billion. 4 out of 5 adolescents live in developing countries. According to Population Bureau statistics, in 1996, 30% of the total population was that of adolescents (284.02 million). Due to gradual decrease in the growth rate of the overall population, there is little increase in the number of adolescents in population projections till the year 2016 (Population projection 1996-2016) census of India.

Sheldon states, in the result of a research on 4000 women, that it is only 3% of them who have regular menstrual cycles. Almost all women experience irregular menstruation cycles from a month to another and there must be little changes (Beri-Beri, 2010). Irregular menstrual cycle does not have a particular pattern like extended menstrual cycle of more than 35 days (*oligomenorrhoea*), short menstrual cycle of less than 21 days or experiencing menstruation more than once in a month (*polimenorrhoea*) or even not menstruating for 3 months (*amenorrhoea*). Short and long cycles, both show unwell metabolism and hormonal system. The impact is – it becomes more difficult to be pregnant (*infertility*). Short cycle occurs when a woman can experience an ovulation but ovum does not really ripen; hence it is difficult to be fertilized. Long cycle in women indicates that ovum is rarely produced or that a woman experiences a quite long infertility. If ovum is rarely produced, it means fertilisation will be rare to occur. Irregular menstrual cycle also gives women difficulty to find out when the fertile period is and when it is not. Women who have 28 day-cycle are only about 10-15% (Hestiantoro, 2007).

WHY SHOULD THE MENSTRUAL CYCLES BE CONSIDERED A VITAL SIGN?

Evaluation of abnormal menstrual patterns throughout adolescence may permit early identification of potential health concerns for adulthood, just as abnormal blood pressure, heart rate, or respiratory rate may be keys to the diagnosis of potentially serious health conditions. The figure underneath demonstrates the number of conditions that may result in irregularities or loss of the menstrual cycle. By including an evaluation of the menstrual cycle as an important vital sign, clinicians gain an additional tool in assessing overall health status for both patients and parents.

Two main systems make up the physiological stress response: the autonomic nervous system and the HPA axis. The autonomic nervous system is comprised of the parasympathetic nervous system and the sympathetic nervous system. According to Polyvagal theory, the ventral branch of the vagus (the primary component of the parasympathetic nervous system) is responsible for maintaining homeostasis during rest, thereby keeping heart rate low. When an organism is confronted with a stressor, the most immediate response involves vagal withdrawal, which leads to an increase in heart rate, indicating the organism's preparedness to respond to an anticipated stressor. If this response is insufficient, the phylogenetically older sympathetic nervous system is activated, entailing the fight-or-flight response which elevates heart rate (further). RSA (Respiratory sinus arrhythmia) is frequently assessed and is considered a valid index of vagal tone.



Conditions associated with amenorrhea or disordered menses

DETERMINANTS OF THE STRESS RESPONSE

Individual factors:

In our study we found that sex was related to stress reactivity, with girls showing higher heart rate and cortisol reactivity, which is consistent with earlier findings pertaining to heart rate, however sex differences in cortisol reactivity are generally not found in children. *Oral contraceptive use* and *menstrual cycle phase* were unrelated to stress reactivity. These factors were shown to be of influence in adults, but not in adolescents, perhaps because the menstrual cycle of girls is less stable, or a different ratio of sex hormones contributes differently to cortisol reactivity. PPS reactivity was found in our study not to vary according to sex which is consistent with previous studies.

In line with theoretical propositions, we found effects of activity level, emotionality, shyness and sociability on cortisol, heart rate, RSA and PPS reactivity. Specifically, in children, decreased shyness was related to lower PPS reactivity. In adolescents, a higher sociability score was related to lower cortisol and heart rate reactivity. A higher emotionality score was also related to lower cortisol reactivity, and a higher activity level was related to lower

RSA reactivity. Previous research would suggest a strong relation between temperament measures and physiological stress reactivity, which we found, interestingly, for adolescents but not children, as only *shyness* was related to PPS reactivity in children. Possibly, in children, the effects of temperament on stress responses were outweighed by other determinants included in the models. This underlines the importance of examining multiple determinants of physiological reactivity in a single study. Previous studies in young children and adults have also reported significant effects of temperament on physiological stress reactivity. It is unclear why these relations should be of greater importance in adolescence as opposed to late childhood; and this warrants further research. The other individual factors that were examined in this study (i.e. *ethnicity*, *BMI* and *birth weight*) were not related to any of the physiological or PPS responses.

Developmental factors:

The physiological stress systems are proposed to undergo developmental changes between childhood and adolescence, and indeed, our data showed distinct determinants influencing stress reactivity in childhood versus adolescence. Only the influence of urbanicity and SES on heart rate reactivity was uniform in both samples. In general, pertaining to physiological stress alone, the physiological reactivity indices (*heart rate with cortisol and RSA*) were less strongly correlated in adolescents as compared to children. Also, the manipulation check showed that the stress response patterns were as expected in children, but not in adolescents. Pertaining to the determinants, we observed that temperament seemed to be a more important influence in adolescents than in children. Age remained a significant factor in children, but not in adolescents. In children, *menstruation* in girls was examined as a proxy for pubertal development, but this was also not related to stress reactivity. Significant effects of parenting were examined in both children and adolescents, albeit different parenting styles. In sum, the differential response patterns and differential determinants of stress reactivity in children and adolescents found in our study confirm previous propositions of developmental changes in physiological stress reactivity in humans during adolescence.

Environmental factors:

Environmental determinants of the stress response have been neither frequently examined nor controlled for in past studies. However, there also seems to be evidence in animal and human research that early environmental adversity is linked to subsequent blunted physiological stress responses. The findings from a study generally support as we found that a lower *SES* and living in a more *urban* area were related to blunted heart rate reactivity in children and adolescents, blunted RSA reactivity in children and blunted cortisol reactivity in adolescents, confirming earlier findings. As lower *SES* environments and *city-dwelling* are viewed as a more demanding and socially stressful, these could be considered more adverse environments to grow up in, thus blunting stress responses. Alternatively, given that physiological stress systems are partially genetically determined, it is possible that (the parents of) individuals who are inherently hypo-aroused, move to, for example, more populated areas or attain a lower *SES*.

Parenting styles index the family environment of youth and in this way may influence the developing stress systems. Protective parenting factors were related to increased cortisol, heart rate and PPS reactivity in the present study. This is consistent with earlier findings of less optimal parenting behaviours being related to HPA hypo-reactivity. In a study, less *emotional warmth* was related to blunted cortisol reactivity in children, less *involvement* was related to blunted cortisol and heart rate reactivity, and less *overprotection* was related to blunted PPS reactivity in adolescents. Comparably, a higher degree of *inconsistent discipline* was related to blunted PPS reactivity in adolescents. The less optimal perceived parental *rejection* was positively related to PPS reactivity in children, which does not fit with this pattern. However, this pertains to perceived *parental rejection*, and likewise, perceived *physiological stress*. That this relation is positive, may indicate that some children are more sensitive to external influences, and for this reason reported a higher degree of both parental *rejection* and PPS during the psychosocial stress procedure.

(Early) adversity may also be indexed by number of *adverse life events*. Previous studies found that having experienced more adverse life events was related to blunted cortisol and heart rate reactivity in boys and young

adults. In contrast to these findings, we observed no relation between adverse life events and physiological stress responses in adolescents. Internal reliability was, however, quite low in the adolescent sample, which could account for these findings.

Substance use-related factors:

In our study, in the adolescent sample, *tobacco use* was found to be associated with blunted cortisol and PPS reactivity. Previous studies also found *alcohol* and *cannabis use* to be related to blunted cortisol and heart rate reactivity, though these relations were not evident in the present study. An effect of *tobacco use* on physiological reactivity was expected, yet the finding of its effect on PPS seems less intuitive, and has not been previously examined to our knowledge. It is not entirely clear why we observed this relation in the present study, and further research will be needed to confirm or disprove this finding.

The results of a study should be considered in light of some limitations. First, the study was cross-sectional; therefore no conclusions can be drawn as to the direction of influence of the determinants and the stress reactivity variables. Second, information on pubertal stage was only available for the adolescent sample. We were able to assess puberty-related changes in girls in the child sample with *menstruation*; however, it would have been preferable to have information on pubertal stage in children as well. Developmental differences within the child and adolescent samples are likely to have still been influential, as the age ranges within these samples were still quite large. In order to fully examine developmental differences in relation to stress reactivity, it is necessary to have a sufficiently large sample to divide it into groups of smaller age ranges. Third, *SES* was operationalised based on the higher occupational level of either parent and coded into only three levels. Furthermore, only a small percentage of participants had a low *SES* background. A more comprehensive definition of *SES*, including financial, educational as well as occupational information is necessary to fully examine the relation of *SES* with stress response indices. Fourth, though our PPS questionnaire had been used in previous studies, it has not yet been formally validated. Fifth, our measures of stress reactivity were based on difference scores. Such measures are still widely used and clearly superior to single measurements, however, more comprehensive measures that utilise all of the measurement points, such as area under the curve estimates or growth curve modelling, are more sophisticated than difference scores and preferable in stress reactivity research.

Current research has focussed increasingly on physiological stress reactivity as a vulnerability marker for (mental) health problems in children and adolescents. However, such studies are inconsistent in the inclusion of potential covariates that may influence the developing stress systems. Determinant factors of stress reactivity were outlined in several theories (our sample of children and adolescents from the general population provided an exceptional opportunity to systematically test such factors, also as reports in the literature pertaining to many of these factors are either lacking or inconclusive. In children and adolescents, individual, developmental, environmental and substance use-related factors influenced stress reactivity measures. Furthermore, we investigated the hypothesis that PPS reactivity was positively related to cortisol, heart rate and RSA reactivity, but found that, for the most part, this did not hold in our samples, in contrast to a recent study in adolescents. In sum, our study showed that it is imperative that future studies take into consideration determinants of stress reactivity that may account for found relations. This study provides an indication of which determinants should be considered in children and adolescents.

REFERENCES

1. Alpert BS, Wilson DK (1992) Stress reactivity in childhood and adolescence. In: Turner JR, Sherwood A, Light KC, editors. Individual differences in cardiovascular response to stress. New York, NY: Plenum Press. pp. 187–198.
2. Bouma EMC, Riese H, Ormel J, Verhulst FC, Oldehinkel AJ (2009) Adolescents' cortisol responses to awakening and social stress; Effects of gender, menstrual phase and oral contraceptives. The TRAILS study. *Psychoneuroendocrinology* 34: 884–893.
3. Childs E, Dlugos A, De Wit H (2010) Cardiovascular, hormonal, and emotional responses to the TSST in relation to sex and menstrual cycle phase. *Psychophysiology* 47: 550–559.

4. Del Giudice M, Ellis BJ, Shirtcliff EA (2011) The Adaptive Calibration Model of stress responsivity. *Neuroscience and Bio-behavioral Reviews* 35: 1562–1592.
5. Derryberry D, Rothbart MK (1988) Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology* 55: 958–966.
6. Dockray S, Susman EJ, Dorn LD (2009) Depression, cortisol reactivity, and obesity in childhood and adolescence. *Journal of Adolescent Health* 45: 344–350.
7. Evans BE, Greaves-Lord K, Euser AS, Tulen JHM, Franken IHA, et al. (2012) Alcohol and tobacco use and heart rate reactivity to a psychosocial stressor in an adolescent population. *Drug and Alcohol Dependence* 126: 296–303.
8. Goldsmith HH, Buss AH, Plomin R, Rothbart MK, Thomas A, et al. (1987) Round table: What is temperament: 4 approaches. *Child Development* 58: 505–529.
9. Gunnar MR, Wewerka S, Frenn K, Long JD, Griggs C (2009) Developmental changes in hypothalamus-pituitary-adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology* 21: 69–85.
10. Hestiantoro & Andon. 2007. Why is Menstruation Irregular? Cause and Solution. Department of Obstetrics and Gynecology RSUPN Cipto Mangunkusumo. Jakarta.
11. Huizink AC, Ferdinand RF, Ormel J, Verhulst FC (2006) Hypothalamic-pituitary-adrenal axis activity and early onset of cannabis use. *Addiction* 101: 1581–1588.
12. Jessop DS, Turner-Cobb JM (2008) Measurement and meaning of salivary cortisol: A focus on health and disease in children. *Stress: The International Journal on the Biology of Stress* 11: 1–14.
13. Kagan J (1997) Temperament and the reactions to unfamiliarity. *Child Development* 68: 139–143.
14. Kagan J, Reznick JS, Snidman N (1988) Biological bases of childhood shyness. *Science* 240: 167–171.
15. Kirschbaum CP, Kudielka BMMS, Gaab JMS, Schommer NCMS, Hellhammer DHP (1999) Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *SO - Psychosomatic Medicine* March/April 1999 61 (2) 154–162.
16. Kirschbaum C, Hellhammer DH (1989) Salivary cortisol in psychobiological research - an overview. *Neuropsychobiology* 22: 150–169.
17. Kudielka BM, Wüst S (2010) Human models in acute and chronic stress: Assessing determinants of individual hypothalamus–pituitary–adrenal axis activity and reactivity. *Stress: The International Journal on the Biology of Stress* 13: 1–14.
18. Kudielka BM, Hellhammer DH, Wüst S (2009) Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology* 34: 2–18.
19. Kudielka BM, Buske-Kirschbaum A, Hellhammer DH, Kirschbaum C (2004) HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: Impact of age and gender. *Psychoneuroendocrinology* 29: 83–98.
20. Lupien SJ, McEwen BS, Gunnar MR, Heim C (2009) Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience* 10: 434–445.
21. McEwen BS (1998) Protective and damaging effects of stress mediators. *New England Journal of Medicine* 338: 171–179.
22. Porges SW (1995) Cardiac vagal tone: A physiological index of stress. *Neuroscience & Bio-behavioral Reviews* 19: 225–233.
23. Rohleder N, Wolf JM, Piel M, Kirschbaum C (2003) Impact of oral contraceptive use on glucocorticoid sensitivity of pro-inflammatory cytokine production after psychosocial stress. *Psychoneuroendocrinology* 28: 261–273.
24. Romeo RD (2010) Adolescence: A Central Event in Shaping Stress Reactivity. *Developmental Psychobiology* 52: 244–253.
25. Tarullo AR, Gunnar MR (2006) Child maltreatment and the developing HPA axis. *Hormones and Behavior* 50: 632–639.
26. Tyrka AR, Wier LM, Anderson GM, Wilkinson CW, Price LH, et al. (2007) Temperament and response to the Trier Social Stress Test. *Acta Psychiatrica Scandinavica* 115: 395–402.