# ORIGINAL ARTICLE
# IMPACT OF FAST FOOD CONSUMPTION ON LIPID PROFILE, GONADOTROPIN, AND PROLACTIN LEVELS IN YOUNG FEMALES

## Mehir un Nisa Iqbal, Aqsa Khan, Taseer Ahmed Khan
**Department of Physiology, University of Karachi, Karachi Pakistan**

**Background:** Fast food consumption is detrimental to the physical health of human. A daily high intake of fast food alters lipid profile results in obesity which leads to development of multiple health hazards including hormone imbalances. The aim of this study was to determine the effect of fast food consumption on the lipid profile, gonadotropins and prolactin levels. **Method:** This study was a pilot study on fast food consumer (n=35) and non-consumer (n=15) ladies in age range of 18 to 25 years. A self-structured food frequency questionnaire was used to assess the fast food consumption. Fasting serum cholesterol, High Density Lipids (HDL), Low Density Lipids (LDL), and Triglyceride (TG) levels were estimated through Randox kit method. Serum Leutinizing Hormone (LH), Follicular Stimulating Hormone (FSH), and Prolactin were estimated using solid phase chemiluminescent enzyme immunoassay. **Results:** Significant differences were observed in LDL ($p<0.05$) among consumers and non-consumers. Significant differences were also found in LH level ($p<0.05$). **Conclusion:** Fast food altered HDL/LDL ratio which may alter the LH/FSH ratio. Further work is needed to determine the role of fast food consumption and hormone imbalance.
**Keywords:** Fast food, Lipid profile, Gonadotropins, Hormone, WHR, Obesity, Nutrition, Coronary Heart Disease, PCOS

## INTRODUCTION

Fast foods are prepared and served promptly and usually contain high calories. Fast food consumption has noticeably increased worldwide. Pakistan has higher rates of fast food consumption among Asian countries.[1]

A large amount of fast food consumption can cause many perilous health related problems. Fast foods can contribute to a greater risk of obesity.[2] It can also contribute to heart disease[3], breast cancer[4], insulin resistance[5], diabetes[3], and infertility in women[6]. Obesity is one of the disorders caused by excessive food intake, lack of physical activity, and genetic susceptibility. Habitual dietary practice is likely to be high in salt and is provoked by growing commercialization of fast foods and processed foods.

Eating habits are influenced by lack of time to prepare meals at home, accessibility of fast food chains and food vendor machines. The excessive consumption of fast food has greater impact on health and may lead to an increased risk of coronary heart disease (CHD).[3] Altered lipid profile increases the risk of myocardial infarction.[7] Fast foods containing trans fatty acids (TFAs) increase the risk of ovulatory infertility.[6] It may also develop the risk of increased foetal loss.[8]

This study was carried out to effects of fast food consumption on lipid profile, gonadotropins, and prolactin levels in young females.

## MATERIAL AND METHODS

Fifty young females (35 fast food consumers and 15 non-consumers) were recruited from Department of Physiology, University of Karachi. Apparently healthy females in age ranges of 18 to 25 years with no history of metabolic or hormonal disorders or other similar disease were included in the study. About 5 ml of blood sample was collected from the antecubital vein after 12 hour overnight fasting. Serum was separated by with high speed centrifugation and stored at -86 ℃ till analysis.

A self-structured questionnaire was designed to collect information about an individual's demographic data, lifestyle and dietary profile. The diet pattern was determined using structured questionnaire consisting of 44 food items. All food items had three divisions and these divisions were based on scoring 0–14 for rare consumption, 15–24 for average, and ≥25 for high consumption of fast food.

Lipid profile, i.e., Total Cholesterol, Triglycerides (TG), Low Density Lipids (LDL), and High Density Lipids (HDL), and Follicular Stimulating Hormone (FSH), Leutinizing Hormone (LH), and Prolactin were estimated using the Randox kit method and solid phase competitive chemiluminescent enzyme immunoassay respectively. The hormone levels were estimated during the follicular phase of menstrual cycle of all subjects. The reference values used were cholesterol <200 mg/dl, TG <150 mg/dl, HDL >60 mg/dl, LDL <150 mg/dl, FSH 1.24–7.8 MIU/ml, LH 1.42–15.4 MIU/ml, and Prolactin 2–17 ηg/ml.

Statistical analysis was carried out using IBM-SPSS-22. Data were expressed as percentages and Mean±SE. Student's *t*-test was used to compare the means of the two groups, and $p<0.05$ was taken as significant.

## RESULTS

The mean age of the subjects was 23.5±2.5 years (range= 18–25 years). Fifteen percent (15%) of fast food consumers were overweight and 33.3% had abnormal Weight Hip Ratio (WHR) (Table-1).

**Table-1: Demographic and anthropometric profile of the subjects**

| Test Parameters | Consumers (%) | Non-consumers (%) |
|---|---|---|
| **BMI Category** | | |
| Severely underweight | 12.12 | 6 |
| Underweight | 39.4 | 13 |
| Normal | 27.33 | 66 |
| Overweight | 15.15 | 13 |
| Obese | 6 | 2 |
| Total | 100 | 100 |
| **WHR** | | |
| Normal | 66.7 | 100 |
| Abnormal | 33.3 | 0 |
| Total | 100 | 100 |

No significant differences were observed among consumers and non-consumers in cholesterol ($p>0.05$), TG ($p>0.05$), and HDL ($p>0.05$), whereas significant difference was observed in LDL ($p<0.05$). The hormone levels were measured during the follicular phase of all participants. Significant differences were observed in LH ($p<0.05$) and non-significant differences were found in FSH ($p>0.05$) and Prolactin ($p>0.05$) among consumers and non-consumers (Table-2).

**Table-2: Comparison of means of the two groups**

| Test Parameters | Consumers (Mean±SE) | Non-Consumers (Mean±SE) | *p* |
|---|---|---|---|
| **Lipid Profile** | | | |
| Cholesterol | 306.3±115.8 | 189.1±16 | >0.05 |
| Triglycerides | 108.6±11.3 | 117±20 | >0.05 |
| HDL | 36±3.1 | 29±3.7 | >0.05 |
| LDL | 249±117 | 137.2±17.4 | <0.05 |
| **Hormone Profile** | | | |
| LH | 9.2±2 | 2.55±0 | <0.05 |
| FSH | 8.84±2.55 | 9.41±0.7 | >0.05 |
| Prolactin | 14.6±1.44 | 9.46±0.1 | >0.05 |

*p*<0.05=significant

## DISCUSSION

The current study showed detrimental effects of fast food consumption on weight. Some of them were overweight and their waist hip ratio was abnormal. A previous study suggested a strong relationship between fast food consumption and obesity.[2] It has been documented that increased waist to hip ratio is considered to be a risk of developing Coronary Heart Disease.[9]

The results demonstrated that cholesterol, TG, HDL, FSH and Prolactin had no significant difference among consumers and non-consumers whereas LDL and LH were found having significant differences. High intake of fast food consumption containing fats alters HDL/LDL ratio.[10] It augments LDL and reduces HDL

that causes weight gain leading to overweight status and obesity. Altered ratio of LDL and HDL shows increased risk of developing Coronary Heart Disease[11] and type 2 diabetes mellitus (T2DM)[12]. Presently we investigated the reproductive hormone profile which is the indicators of ovulation (like LH, FSH, and Prolactin) to ensure that risk of infertility was there. But it was noted that LH was significantly high whereas FSH and Prolactin were not significant. A study among Pakistani women documented that impaired LH/FSH ratio is an important diagnostic parameter of Polycystic Ovary Syndrome (PCOS).[13]

In Pakistan, not only the urban but also the rural population has a high risk potential for cardiovascular diseases (CVD), endocrine diseases and non-communicable diseases. Unfortunately the identification of risk factors in the rural population and the rural health education regarding endocrine diseases has not been a hallmark of our health promoting efforts. As a matter of fact, there is little population-based data available in Pakistan on fast food consumption and risks of insulin resistance, diabetes mellitus, breast cancer and ovulatory infertility. Further research on these aspects is needed to have a better insight to the problem.

## CONCLUSION

Fast food alters HDL/LDL ratio and may alter the LH/FSH ratio. They may be predictors of and risk factors for CHD and PCOS respectively.

## REFERENCES

1. Baig AK, Saeed M. Review of trends in fast food consumption. Eur J Econ Finance Adm Sci 2012;48:77–85.
2. Payab M, Kelishadi R, Qorbani M, Motlagh ME, Ranjbar SH, Ardalan G, *et al*. Association of junk food consumption with high blood pressure and obesity in Iranian children and adolescents: the CASPIAN-IV Study. J Pediatr (Rio J) 2015;91(2):196–205.
3. Odegaard AO, Koh WP, Yuan JM, Gross MD, Pereira MA. Western-style fast food intake and cardiometabolic risk in an eastern country. Circulation 2012;126(2):182–8.
4. Chajes V, Thiebaut AC, Rotival M, Gauthier E, Maillard V, Boutron-Ruault MC, *et al*. Association between serum trans-monounsaturated fatty acids and breast cancer risk in the E3N-EPIC Study. Am J Epidemiol 2008;167:1312–20.
5. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, *et al*. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet 2005;365(9453):36–42.
6. Chavarro JE, Rich-Edwards JW, Rosner BA, Willett WC. Dietary fatty acid intakes and the risk of ovulatory infertility. The Am J Clin Nutr 2007;85:231–7.
7. Baylin A, Kabagambe EK, Ascherio A, Spiegelman D, Campos H. High 18:2 trans-fatty acids in adipose tissue are associated with increased risk of nonfatal acute myocardial infarction in Costa Rican adults. J Nutr 2003;133:1186–91.
8. Morrison JA, Glueck CJ, Wang P. Dietary trans fatty acid intake is associated with increased fetal loss. Fertil Steril 2008;90:385–90.
9. Gelber RP, Gaziano JM, Orav EJ, Manson JE, Buring JE, Kurth T. Measures of obesity and cardiovascular risk among men and women. J Am Coll Cardiol 2008;52:605–15.
10. Müller H, Lindman AS, Brantsaeter AL, Pedersen JI. The serum LDL/HDL cholesterol ratio is influenced more favorably by

exchanging saturated with unsaturated fat than by reducing saturated fat in the diet of women. J Nutr 2003;133:78–83.

11. Kannel WB. Metabolic risk factors for coronary heart disease in women: Perspective from the Framingham Study. Am Heart J 1987;114(2):413–9.

12. Habib SS, Aslam M, Hameed W. Gender differences in lipids and lipoprotein (a) profiles in healthy individuals and patients with type 2 Diabetes Mellitus. Pak J Physiol 2005;1(1–2):2–6.

13. Akram M, Roohi N. Endocrine correlates of polycystic ovary syndrome in Pakistani women. J Coll Physicians Surg Pak 2015;25(1):22–6.

**Address for Correspondence:**

**Mehir un Nisa Iqbal,** Department of Physiology, University of Karachi, Karachi, Pakistan. **Cell:** +92-345-3133309
**Email:** mehirunisa@uok.edu.pk