
**Omission of co-authors.** The author byline and affiliations should read:

**Distribution and Determinants of Coronary Artery Disease in an Urban Pakistani Setting**

Sunita Dodani, MD, FCPS, MS; David D MacLean, MD; Ronald E LaPorte, PhD; and Michel Joffres, PhD

**Objective:** We assessed the distribution of coronary artery disease (CAD) and its association with the major biological risk factors and behaviors among Pakistanis presenting at a tertiary care hospital in Karachi, Pakistan.

**Method:** An epidemiologic cross-sectional study was conducted at the Aga Khan University Hospital (a teaching hospital) in Karachi, Pakistan. A total of 600 adult (≥18 years of age) patients visiting family practice clinics for general check-up were included. The association of biological risk factors with CAD (smoking, obesity [body mass index (BMI)], hypertension, family history of ischemic heart diseases [IHD], sedentary lifestyle, diabetes mellitus, total cholesterol, low density lipoprotein [LDL] levels, high density lipoprotein [HDL] levels, and triglycerides) were assessed.

**Results:** On univariate analysis, age ≥40 years, early menopause, BMI ≥29.9 kg/m², diabetes, high cholesterol, and positive family history of IHD were independently associated with CAD. We found age ≥40 years, diabetes, and positive family history of IHD strongly related with CAD on multivariate analysis.

**Conclusion:** Looking at the strong association of major risk factors with CAD, the unique characteristics of Pakistanis must be studied in depth, with focus on high-risk groups. *(Ethn Dis. 2005;15:429–435; erratum: Ethn Dis. 2006;16:309.)*

**Key Words:** Coronary Artery Disease, Developing Countries, Hypertension, Risk Factors

From the Department of Epidemiology, University of Pittsburgh, Pittsburgh, Pennsylvania (SD, REL); The Institute for Health Research and Education, Simon Fraser University, Burnaby, BC, Canada (DDM); and the Department of Community Health and Epidemiology, Dalhousie University, Halifax, NS, Canada (MJ).

Address correspondence and reprint requests to Sunita Dodani, MD, FCPS, MS; Department of Epidemiology; University of Pittsburgh; 3512 Fifth Avenue; Pittsburgh, PA 15101; 412-383-1038; sud0@pitt.edu.

The new citation for this article should read:


**Incorrect Byline.** The author byline for the article, “Culturally sensitive smoking cessation intervention program redesign for Arab-American youth” should read, “Al-Faouri I, Weglicki L, Rice VH, et al.”


**Incorrect author name.** The name of co-author Harold Snieder, PhD was inadvertently misspelled in the byline, page 568, and the Author Contribution section, page 577.

Incorrect title and abstract. The article title and abstract should read:

Analysis of Exhaled Breath: The Promise of Painless Blood Glucose Testing

Student Researcher: Winston Hu; Mentor: Pietro Galassetti, MD, PhD

Recent advances in analytical technology have revealed that human exhaled breath may contain many more gases than previously estimated and up to several hundred. Many of them, called volatile organic compounds, may potentially track the endogenous process currently monitored with invasive and often painful techniques. Glucose homeostasis is one of these processes and, we hypothesized that the integrated analysis of the exhaled gases ethanol and acetone (derived from intestinal alcoholic fermentation of glucose and oxidation of free fatty acids, respectively), would allow estimation of blood glucose profiles during the transient hyperglycemia following ingestion of a sugar-rich drink. The diagnosis and follow-up care of diabetes is based on blood glucose measurements. Glucose loading is also used for the diagnosis of gestational diabetes. Millions of these measurements are performed daily, with significant discomfort or pain for the patients, and at considerable expense. In our research, we attempted to create the conceptual basis for glucose levels measurement.


Addendum. The following statement serves as an addendum to the article, “Testosterone Inhibits Adipogenic Transcription Factors PPAR-γ2 and C/EBP-α through TCF4 Transcription Factor” and should read:

The authors would like to acknowledge Dr. Rajan Singh as a coauthor, and to also thank Melissa Braga, Dr. Nestor Gonzalez-Cadavid, and Dr. Eric Fearon for material help. This student abstract is a preliminary report for the research paper, which has been published in Endocrinology, listed as follows: “Singh R, Artaza JN, Taylor WE, Braga M, Yuan X, Gonzalez-Cadavid NF, Bhasin S. Testosterone inhibits adipogenic differentiation in 3T3-L1 cells: nuclear translocation of androgen receptor complex with beta-catenin and TCF4 may bypass canonical Wnt signaling to downregulate adipogenic transcription factors. Endocrinology. 2005 Oct 6 [Epub ahead of print] PMID: 16210377 [PubMed – as supplied by publisher].