OPEN ACCESS Freely available online

Journal of Cell Science & Therapy

ISSN: 2157-7013

Short Communication

Insulin Resistance Syndrome: Dysfunctional Endothelial Progenitor Cells

Sergio Spaans*

Department of Molecular Systems, Eindhoven University of Technology, Eindhoven, The Netherlands

DESCRIPTION

Insulin Resistance (IR) and obesity are the two main disorders that make up the Metabolic Syndrome (MetS) [1]. The National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) has identified five diagnostic criteria, and the presence of any three of the features (central obesity, dyslipidemia (high triglycerides, low HDL), hypertension, and impaired fasting glucose) is thought to be sufficient to make the diagnosis. The MetS affects about 35% of US people, and it seems to be a relatively widespread illness worldwide. Additionally, the occurrence rises with age [2] MetS increases the risk of diabetes by five times and cardiovascular disease by two to four times. [3].

Endothelial cell dysfunction, a crucial early manifestation of atherosclerosis, is brought on by a number of harmful insults, including obesity, hypertension, dyslipidemia, and hyperglycemia, all characteristics of MetS. Many studies have found endothelial dysfunction in MetS patients.

Endothelial progenitor cells are a subtype of progenitor cells isolated from adult bone marrow, umbilical arteries, and peripheral blood that have the ability to circulate, multiply, and develop into mature Endothelial Cells (EPCs). EPCs are blood-circulating cells that appear to target vascular or tissue damage sites preferentially, making a considerable contribution to neoangiogenesis and reendothelialization. It is important to note right away that there is considerable debate over the proper definition of EPCs [4-6].

In general, it is acknowledged that the evaluation of surface markers like CD34 and vascular endothelial growth factor receptor-2, or VEGFR-2, can identify EPCs (KDR). Importantly, the only putative EPC phenotype that has consistently and conclusively been shown to be an independent predictor of cardiovascular outcomes is the CD34+KDR+ combination.

There is little information available on the MetS's EPC number and capabilities. There seem to be two studies that have specifically examined EPC number in MetS patients and matched controls (without other confounders, comorbidities such as diabetes or cardiovascular disease).

Regarding coexisting illnesses and morbidity, such as diabetes and peripheral vascular disease, or concurrent medications in these two subgroups, not enough information is given. Since the primary goal of this study was to examine EPC status in diabetic patients with peripheral vascular disease, the information provided for patients in MetS is less thorough than that provided in the two studies that only looked at MetS.

In a later study, Fadini et al. demonstrated that there was a decrease in progenitor cells (CD34+ cells) in patients with MetS. It appears that many of these patients may also have diabetes and be taking medications like statins, ACE-I inhibitors, ARBs, and anti-diabetic treatments like pioglitazone, which could have affected the data.

CONCLUSION

In individuals with MetS who obviously show apparent endothelial dysfunction, EPC quantity and functionality may represent a potential biological biomarker of endothelium integrity and defective neoangiogenesis. Prospective research should show that they can forecast CVD. Statins, ACE-I, ARBs, PPAR-gamma agonists, and INCRETIN-based therapies, which have been shown to upregulate and enhance EPC number and functionality, need to be studied more thoroughly with respect to both number and functionality of EPCs because this could inform us of their direct beneficial effects on the vulnerable vasculature of Mets. As a result, EPC number and/or functionality may become a new cellular biomarker of CVD risk, which may help clinicians plan more effective pharmacotherapy for patients with MetS.

REFERENCES

1. Ernst ND, Cleeman JI. National cholesterol education program keeps a priority on lifestyle modification to decrease cardiovascular disease risk. Curr Opin Lipidol. 2002; 13(1):69-73.
2. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation;

Correspondence to: Sergio Spaans, Department of Molecular Systems, Eindhoven University of Technology, Eindhoven, The Netherlands, E-mail: s.e.g.spaan@tue.nl

Received: 11-Jan-2023; Manuscript No. JCEST-23-21367; **Editor assigned:** 13-Jan-2023; Pre-Qc No JCEST-23-21367 (PQ); **Reviewed:** 25-Jan-2023; Qc No JCEST-23-21367; **Revised:** 01-Feb-2023, Manuscript No. JCEST-23-21367 (R); **Published:** 10-Feb-2023, DOI: 10.35248/2157-7013.23.14.380

Citation: Spaans S (2023) Insulin Resistance Syndrome: Dysfunctional Endothelial Progenitor Cells. J Cell Sci Therapy.14:380.

international atherosclerosis society; and international association for the study of obesity. Circulation. 2009; 120(16):1640-1645.

3. Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. Jama. 2002;288(21):2709-2716.
4. Hirschi KK, Ingram DA, Yoder MC. Assessing identity, phenotype, and fate of endothelial progenitor cells. ATVB. 2008; 28(9): 1584-1895.
5. Timmermans F, Plum J, Yöder MC, Ingram DA, Vandekerckhove B, Case J. Endothelial progenitor cells: identity defined? J Cell Mol Med. 2009; 13(1):87-102.
6. Fadini GP, Baesso I, Albiero M, Sartore S, Agostini C, Avogaro A. Technical notes on endothelial progenitor cells: ways to escape from the knowledge plateau. Atherosclerosis. 2008; 197(2):496-503.