JBiSE> Vol.6 No.1, January 2013

OPEN ACCESS

Myocardial insulin resistance does not always parallel skeletal muscle and whole body insulin resistance: A mini review —Myocardial Insulin Resistance

PDF (Size:380KB) PP. 31-35 DOI: 10.4236/jbise.2013.61005

Author(s)
Ikuo Yokoyama

ABSTRACT
Insulin resistance (IR) is recognized to be of critical importance in a variety of metabolic diseases and coronary artery disease (CAD). Impaired skeletal muscle glucose utilization (SMGU) plays an important role in the pathogenesis of IR, whereas it is controversial whether myocardial IR is similar in this respect. Methods: Twenty-two studies of myocardial IR and skeletal muscle IR using positron emission tomography (PET) and/or whole body IR were reviewed. Heart and skeletal muscle IR were measured with PET and ^{18}F-FDG under hyperinsulinemic euglycemic insulin clamp technique. Whole body IR was also determined at the time of PET under hyperinsulinemic euglycemic insulin clamp technique. Results: One study reported that heart and skeletal muscle IR is present in untreated type 2 diabetes mellitus (T2DM), hypertension and CAD (as reflected in a myocardial glucose utiliation rate (MGU) in T2DM vs control [$p < 0.01$], and an SMGU in T2DM vs control [$p < 0.01$]). A significant negative relationship between MGU and FFA ($r = -0.665$, $p < 0.01$) and a significant positive relationship between MGU and whole body IR ($r = 0.855$, $p < 0.01$) was also observed in T2DM. Significantly reduced MGU and SMGU and a positive correlation between them ($r = 0.78$, $P < 0.0001$) were noted in the normal myocardial segments of patients with CAD. Another study showed that heart and skeletal muscle IR was present in T2DM both with CAD (MGU):, $p < 0.01$; SMGU: $p < 0.01$) or without CAD (MGU: $p < 0.01$; SMGU: $p = 0.06$). A significant positive relationship between the whole body glucose disposal rate and MGU ($r = 0.60$, $p < 0.01$) as well as SMGU ($r = 0.76$, $p < 0.01$) was also reported. Much more severe myocardial IR in T2DM and hypertriglyceridemia ($p < 0.05$) due to increased plasma free fatty acids, FFA ($r = -0.60$, $p < 0.01$) and plasma triglycerides levels ($r = -0.74$, $p < 0.001$) was reported. A significant negative relationship between MGU and plasma triglycerides ($r = -0.74$, $p < 0.001$) was also noted. However, other studies reported that MGU was increased in essential hypertension despite the fact that SMGU and whole body IR were present. Furthermore, it has also been reported that MGU in hypertensive T2DM without medication for diabetes was similar to controls (p = ns) despite the presence of skeletal muscle IR ($p < 0.01$) and whole body IR. Myocardial IR was not detected in non-diabetic non-hypertensive hypertriglyceridemia (Myocardial ^{18}F-FDG Uptake (MFU) in hypertriglyc-eridemics was (p = ns) despite findings of reduced skeletal muscle ^{18}F-FDG uptake (SMFU in hyper-triglyceridemia ($p < 0.01$) and whole body IR (GDR) in hypertriglyceridemia ($p < 0.01$). Myocardial IR was not detected under very high dose insulin clamping (about 10 times higher than the usual dose) with MGU in T2DM vs control p = ns, while whole body IR in T2DM was still present, ($p < 0.02$) and skeletal muscle ($p < 0.01$). Preserved MGU under chronic use of sul-fonylurea drugs in T2DM has been reported (T2DM with SU vs control, p = ns). Moreover, myocardial IR was not seen in hypertensive T2DM despite the existence of skeletal muscle and whole body IR. Myocardial IR in T2DM with CAD could be improved by thiazolidinediones (MGU before rosiglitazone, vs after rosiglitazone $p < 0.05$) and in mixed combined hyperlipidemia with CAD before-vs-after pioglitazone ($p < 0.01$). This suggests that myocardial IR in T2DM and CAD paralleled an improvement of skeletal muscle and whole body IR. However, troglitazone failed to improve myocardial IR in T2DM without CAD within 12 weeks therapy (MGU before therapy vs after, p = ns). Nonetheless, it did improve myocardial IR in T2DM without CAD after 12 months' therapy (before therapy vs after 12 months, $p < 0.05$). Conclusion: The myocardium possesses mechanisms to resist IR different from those in the rest of the body. Therefore, myocardial IR does not always parallel skeletal muscle and whole body IR.

• Open Special Issues
• Published Special Issues
• Special Issues Guideline

JBiSE Subscription

Most popular papers in JBiSE

About JBiSE News

Frequently Asked Questions

Recommend to Peers

Recommend to Library

Contact Us

Downloads:	437,459
Visits:	1,228,423

Sponsors >>

International Conference on Bioinformatics and Biomedical Engineering(iCBBE)

KEYWORDS
Insulin Resistance; Myocardial Insulin Resistance; Coronary Artery Disease; Metabolic Syndrome; Diabetes; Hyperlipidemia; PET; FDG

Cite this paper

Yokoyama, I. (2013) Myocardial insulin resistance does not always parallel skeletal muscle and whole body insulin resistance: A mini review
—Myocardial Insulin Resistance
. *Journal of Biomedical Science and Engineering*, 6, 31-35. doi: 10.4236/jbise.2013.61005.

References

[1] Ueshima, K., Oba, K., Yasuno, S., Fujimoto, A., Tanaka, S., Ogihara, T., Saruta, T. and Nakao, K. (2011) Influence of coronary risk factors on coronary events in japanese high-risk hypertensive patients. Primary and secondary prevention of ischemic heart disease in a subanalysis of the candesartan antihypertensive survival evaluation in Japan (CASE-J) trial. Circulation Journal, 75, 2411- 2416. doi:10.1253/circj.CJ-10-1161

[2] Steven, M. and Haffner, M.D. (2006) Risk constellations in patients with the metabolic syndrome: Epidemiology, diagnosis, and treatment patterns. The American Journal of Medicine, 119, S3-S9. doi:10.1016/j.amjmed.2006.01.008

[3] Steven, M. and Haffner, MD. (2006) Relationship of metabolic risk factors and development of cardiovascular disease and diabetes. Obesity, 14, 121S-127S. doi:10.1038/oby.2006.291

[4] Yokoyama, I., Inoue, Y., Moritan, T., Ohtomo, K. and Nagai, R. (2003) Simple quantification of skeletal muscle glucose utilization by static 18F-FDG PET. Journal of Nuclear Medicine, 44, 1592-1598. doi:10.1097/00006231-200501000-00006

[5] Yokoyama, I., Inoue, Y., Moritan, T., Ohtomo, K. and Nagai, R. (2005) Measurement of skeletal muscle glucose utilization by dynamic 18F-FDG PET without arteial blood sampling. Nuclear Medicine Communications, 26, 31-37.

[6] Ohtake, T., Kosaka, N., Watanabe, T., Yo-koyama, I., Moritan, T., Masuo, M., Iizuka, M., Kozeni, K., Momose, T., Oku, S., Nishikawa, J., Sasaki, Y. and Iio, M. (1991) Noninvasive method to obtain input function for measuring tissue glucose utilization of thoracic and abdominal organs. Journal of Nuclear Medicine, 32, 1432-1438.

[7] Voipio-Pulkki, L.M., Nuutila, P., Knuuti, M.J., Ruotsalainen, U., Haaparanta, M., Ter?s, M., Wegelius, U. and Koivisto, V.A. (1993) Heart and skeletal muscle glucose disposal in type 2 diabetic patients as determined by positron emission tomography. Journal of Nuclear Medicine, 34, 2064-2067.

[8] Yokoyama, I., Yonekura, K., Ohtake, T., Kawamura, H., Matsumoto, A., Inoue, Y., Aoyagi, T., Sugiura, S., Omata, M., Ohtomo, K. and Nagai, R. (2000) Effect of insulin resistance on Heart and skeletal muscle FDG uptake in type II diabetics. Journal of Nuclear Cardiology, 7, 242-248. doi:10.1016/S1071-3581(00)70013-4

[9] Nuutila, P., M?ki, M., Laine, H., Knuuti, M.J., Ruotsalainen, U., Lu-otolahti, M., Haaparanta, M., Solin, O., Jula, A., Koivisto, V.A., et al. (1995) Insulin action on heart and skeletal muscle glucose uptake in essential hypertension. Journal of Clinical Investigation, 96, 1003- 1009. doi:10.1172/JCI118085

[10] Yokoyama, I., Ohtake, T., Momomura, S., Yonekura, K., Kobayakawa, N., Aoyagi, T., Sugiura, S., Yamada, N., Ohtomo, K., Sasaki, Y., Omata, M. and Yazaki, Y. (1999) Insulin action on Heart and skeletal muscle FDG uptake in patients with hyper-triglyceridemia. Journal of Nuclear Medicine, 40, 1116-1121.

[11] Yokoyama, I., Moritan, T. and Inoue, Y. (2012) Functional imaging of skeletal muscle glucose metabolism by 18FDG PET to characterize insulin resistance in patients at high risk for coronary artery dis-ease. Journal of Biomedical Science and Engineering, 5, 819-825. doi:10.4236/jbise.2012.512A103

[12] Yokoyama, I., Yonekura, K., Moritan, T., Tateno, M., Momose, T., Oh-tomo, K., Inoue, Y. and Nagai, R. (2001) Troglitazone can improve impaired femoral muscle glucose utilization in type II diabetics with or without hypertension. Journal of Nuclear Medicine, 42, 1005-1010.

[13] Peltoniemi, P., Yki-J?rvinen, H., Oikonen, V., Oksanen, A., Takala, T.O., R?nnemaa, T., Erkinjuntti, M., Knuuti, M.J. and Nuutila, P. (2001) Resistance to exercise-induced increase in glucose uptake during hyperinsulinemia in insulin-resistant skeletal muscle of patients with type 1 diabetes. Diabetes, 50, 1371-1377. doi:10.2337/diabetes.50.6.1371

[14] Nuutila, P., Koivisto, V.A., Knuuti, J., Ruotsalainen, U., Ter?s, M., Haaparanta, M., Bergman, J., Solin, O., Voipio- Pulkki, L.M., Wegelius, U., et al. (1992) Glucose-free fatty acid cycle operates in human heart and skeletal muscle in vivo. Journal of Clinical Investigation, 89, 1757-1744. doi:10.1172/JCI115780

[15] Paternostro, G., Camici, P.G., Lammerstma, A.A., Marinho, N., Baliga, R.R., Kooner, J.S., Radda, G.K. and Ferrannini, E. (1996) Cardiac and skeletal muscle insulin resistance in patients with coronary heart disease a study with positron emission tomography. Journal of Clinical Investigation, 98, 2094-2099. doi:10.1172/JCI119015

[16] Iozzo, P., Cha-reonthaitawee, P., Dutka, D., Betteridge, D.J., Ferrannini, E. and Camici, P.G. (2002) Independent association of type 2 diabetes and coronary artery disease with myocardial insulin resistance. Diabetes, 51, 3020-3024. doi:10.2337/diabetes.51.10.3020

[17] Monti, L.D., Lan-doni, C., Setola, E., Galluccio, E., Lu- cotti, P., Sandoli, E.P., Origgi, A., Lucignani, G., Piatti, P. and Fazio, F. (2004) Myocardial insulin resistance associated with chronic hypertriglyceridemia and increased FFA levels in type 2 diabtic patients. American Journal of Physiology—Heart and Circulatory Physiology, 287, H1225-H1231. doi:10.1152/ajpheart.00629.2003

[18] Yokoyama, I., Ohtake, T., Momomura, S., Yonekura, K., Yamada, N., Nishikawa, J., Sasaki, Y. and Omata, M. (1998) Organ specific insulin resistance in patients with non-insulin dependent diabetes mellitus and hypertension. Journal of Nuclear Medicine, 39, 884-889.

[19] Utriainen, T., Takala, T., Luotolahti, M., R?nnemaa, T., Laine, H., Ruotsalainen, U., Haaparanta, M., Nuutila, P. and Yki-J?rvinen, H. (1998) Insulin resistance characterizes glucose uptake in skeletal muscle but not in the heart in NIDDM. Diabetologia, 41, 555-559. doi:10.1007/s001250050946

[20] Yokoyama, I., Inoue, Y., Moritan, T., Ohtomo, K. and Nagai, R. (2006) Myocardial glucose utilization in type II diabetes mellitus patients treated with sulphonylurea drugs. European Journal of Nuclear Medicine and Molecular, 33, 703-708. doi:10.1007/s00259-005-0042-x

[21] Nuutila, P., Knuuti, J., Ruotsalainen, U., Koivisto, V.A., Eronen, E., Ter?s, M., Bergman, J., Haaparanta, M., Voipio-Pulkki, L.M., Viikari, J. et al. (1993) Insulin resistance is localized to skeletal but not heart muscle in type 1 diabetes. American Journal of Physiology, 264, E756-E762.

[22] Lautamaki, R., Airaksinen, K.E., Seppanen, M., Toikka, J., Luotolahti, M., Ball, E., Borra, R., Harkonen, R., Iozzo, P., Stewart, M., Knuuti, J. and Nuutila, P. (2005) Rosiglitazone improves myocardial glucose uptake in patients with type 2 diabetes and coronary artery disease a 16-week randomized, double-blind, placebo-controlled study. Diabetes, 54, 2787-2794. doi:10.2337/diabetes.54.9.2787

[23] Naoumova, R.P., Kindler, H., Leccisotti, L., Mongillo, M., Khan, M.T., Neuwirth, C., Seed, M., Holvoet, P., Betteridge, J. and Camici, P.G. (2007) Pioglitazone improves myocardial blood flow and glucose utilization in nondiabetic patients with combined hyperlipidemia. A randomized, double-blind, placebo-controlled study. Journal of the American College of Cardiology, 50, 2051- 2058. doi:10.1016/j.jacc.2007.07.070

[24] Yokoyama, I., Moritan, T. and Inoue, Y. (2012) Heart and skeletal muscle insulin resistance but not myocardial blood flow reserve could be related to chronic use of thiazolidione in type-2 diabetics. Journal of Biomedical Science and Engineering, 5, 829-835. doi:10.4236/jbise.2012.512A105

[25] Yokoyama, I., Inoue, Y. and Moritan, T. (2012) Recovery of coronary microangiopathy in patients with type 2 diabetes. Journal of Hypo & Hyperglycemia, in press.

[26] Yokoyama, I., Ohtake, T., Momomura, S., Yonekura, K., Woo-Soo, S., Nishikawa, J., Sasaki, Y. and Omata, M. (1998) Hyper-glycemia rather than insulin resistance is related to coronary flow reserve in patients with non-insulin dependent diabetes mellitus. Diabetes, 47, 119-124. doi:10.2337/diabetes.47.1.119

Copyright © 2006-2013 Scientific Research Publishing Inc. All rights reserved.