Original article

Role and mechanism of rosiglitazone on the impairment of insulin secretion induced by free fatty acids on isolated rat islets

TIAN Jing-yan, LI Guo, GU Yan-yun, ZHANG Hong-li, ZHOU Wen-zhong, WANG Xiao, ZHU Hong-da, LUO Tian-hong and LUO Min

Keywords: *rosiglitazone; fatty acids; uncoupling protein 2*

Background Prolonged exposure of pancreatic β-cells to fatty acids increases basal insulin secretion but inhibits glucose-stimulated insulin secretion. Rosiglitazone is a new antidiabetic agent of the thiazolidinediones. However, the relationship between thiazolidinediones and insulin secretion is highly controversial. The aim of this study is to explore the effect and mechanism of rosiglitazone on insulin secretion of islets under chronic exposure to free fatty acids (FFA).

Methods Pancreatic islets were isolated from the pancreata of male Sprague-Dawley rats by the collagenase digestion and by the dextran gradient centrifugation method. The purified islets were cultured in the presence or absence of rosiglitazone and palmitate for 48 hours. The insulin secretion was measured by radioimmunoassay. The mRNA level of peroxisome proliferator-activated receptor (, uncoupling protein 2 (UCP-2) and insulin were determined by real-time polymerase chain reaction (PCR). The cell cytotoxicity assay was measured by cell counting kit-8.

Results Islets exposed to elevated palmitate for 48 hours showed an increased basal and a decreased glucose-stimulated insulin secretion (P<0.01). The mRNA level of UCP-2 was increased by 3.7 fold in the 0.5 mmol/L concentration of palmitate. When islets were cultured with palmitate (0.5 mmol/L) in the presence of rosiglitazone (1.0 μmol/L), both basal and glucose-stimulated insulin secretion reversed to a pattern of control islets (P<0.05, P<0.01). The addition of rosiglitazone in the culture medium decreased the mRNA level of UCP-2 by 2.2 fold, having a statistically significant difference (P<0.05) as compared with islets cultured with palmitate alone. The cell viability was not affected.

Conclusion The protective effects of rosiglitazone on insulin secretion of isolated pancreatic islets under chronic exposure to palmitate might be mediated through the downregulation of UCP-2 expression.

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Type 2 diabetes is a disease characterized by hyperglycemia and is caused by absolute or relative insulin deficiency, sometimes associated with insulin resistance. The insulin secretion of β-cell in type 2 diabetes is also adversely affected by chronic elevation of free fatty acids (FFA). Uncoupling protein 2 (UCP-2) may be one negative modulator of insulin secretion.[1] Accumulating evidence shows that β-cell UCP-2 expression is upregulated by glucolipotoxic conditions and that increased activity of UCP-2 decreases insulin secretion, suggesting that activation of UCP-2 could play a role in type 2 diabetes.[2] Rosiglitazone is a new antidiabetic agent of the thiazolidinediones, which shows the enhancement of sensitivity of insulin in the muscle, adipose tissue, and liver by activating peroxisome proliferator-activated receptor (

Shanghai Institute of Endocrinology and Metabolism, Shanghai Clinical Center of Endocrine and Metabolic Diseases, Ruijin Hospital, Shanghai Second Medical University, Shanghai 200025, China (Tian JY, Li G, Gu YY, Zhang HL, Zhou WZ, Wang X, Zhu HD, Luo TH and Luo M)

Correspondence to: Dr. LI Guo, Shanghai Institute of Endocrinology and Metabolism, Shanghai Clinical Center of Endocrine and Metabolic Diseases, Ruijin Hospital, Shanghai Second Medical University, Shanghai 200025, China (Tel: 86-21-64370045 ext 663327. Fax: 86-21-64749885. Email: liguopbl2002@yahoo.com.cn)

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(PPAR().[3,4] However, the relationship between thiazolidinediones and insulin secretion is highly controversial.[5,6] In the present report, we studied the effect of elevated palmitate on isolated rat pancreatic islets and examined the effect of rosiglitazone on insulin secretion and UCP-2 expression under chronic exposure to palmitate.

METHODS

Reagents and chemicals

Culture medium CMRL-1066 and TRIzol were purchased from Gibco (USA). Palmitate, Collagenase XI, fatty-acid free BSA and rosiglitazone from Sigma (USA). Dextran 70 from Amersham Pharmacia Biosciences (Uppsala, Sweden). SuperScript First-Strand Synthesis System for reverse transcription polymerase chain reaction (RT-PCR) from Invitrogen Life Technologies (USA). Hot start fluorescent PCR core reagent kit from Shanghai Transhold Tech, China. Insulin assay kit from Shanghai Instate of Biology Products, China. Cell counting kit-8 (CCK-8) obtained from Dojin Laboratories, Kumamoto (Japan). The FFA concentration assay kit from Wako (Japan). Protein concentration assay kit from Jiancheng Biology Institute, Nanjing, China. All other chemicals were of analytical grade.

Animals

Male Sprague-Dawley (SD) rats were obtained from Shanghai Laboratory Animal Center, Chinese Academy of Sciences. The animals were given free access to water and a standard diet until the start of the experiments. All experiments were carried out on rats aged 8－12 weeks.

Islet isolation and culture condition

Pancreatic islets were isolated from the pancreata of male SD rats by the collagenase digestion and the dextran gradient centrifugation method.[7] Briefly, after ligation of the pancreatic duct, the pancreata were distended by injection of 10 ml Hanks containing 12.5 mg/ml collagense. The glands were removed, cleaned from lymph nodes and fat tissue, and digested at 37˚C for 17 minutes. The supernatant fluid was discarded and the tissue resuspended and sedimented by Hanks for 3 times. The digest was filtered through a 800 μm steel screen and the filtrate washed by Hanks for another 2 times. Then the islets were partly separated from exocrine tissue by gradient centrifugation (1000 *g,* 20 minutes, 4˚C). The islets were hand-picked individually and about 300－500 islets were isolated from one rat. Purified islets were first cultured overnight at 37˚C in a 5% CO_2－95% air atmosphere in CMRL-1066 medium (11.2 mmol/L glucose) with 0.5% fatty-acid free BSA, then for 48 hours in various culture media with or without palmitate and rosiglitazone.

Fatty-acid solutions

The fatty-acid solutions were prepared as described previously.[8] In brief, the stock solutions of palmitate was dissolved in ethanol:H_2O (1:1, vol:vol) at 56˚C at a final concentration of 150 mmol/L. Aliquots of stock solutions were mixed with fatty-acid free BSA (10% solution in H_2O) by being stirred for 1 hours at 37˚C and then diluted in culture media. The final molar ratio of fatty-acid: BSA was 5:1. The final ethanol concentration was ≤0.33% (vol:vol). All control conditions included a solution of vehicle (ethanol: H_2O) mixed with fatty-acid free BSA at the same concentration as the fatty-acid solution. All FFA concentrations in media were examined with the kit.

Glucose-stimulated insulin secretion

At the end of the culture, the islets were washed twice with Krebs-Ringer bicarbonate solution [KRB, containing 119 mmol/L NaCl, 4.74 mmol/L KCl, 2.54 mmol/L $CaCl_2$, 1.19 mmol/L $MgCl_2$, 1.19 mmol/L Na_2HPO_4, 25 mmol/L $NaHCO_3$, 10 mmol/L HEPES, and 0.5% fatty-acid free BSA (pH 7.4)]. Following a 45 minutes pre-incubation period at 2.8 mmol/L glucose, batches of 10 islets of comparable size were kept at 37˚C for 60 minutes in KRB and 0.5% fatty-acid free BSA containing 2.8 mmol/L glucose. At the end of this period, the medium was completely removed and replaced with KRB and 0.5% fatty-acid free BSA containing 16.7 mmol/L glucose. After another 60 minutes incubation, 100 μl medium was removed and the insulin was measured by radioimmunoassay.[9] To each well, 100 μl of 0.1 mmol/L NaOH was added to solubilize islets for determination of islets protein content according to the Bradford protein concentration assay methods. All the results were adjusted for protein concentration.

Cell cytotoxicity assay

After the treatments, cell viability was assessed with CCK-8 to count living cells by combining WST-8 [2-(2-methoxy-4-nitrophenyl)-3-(4-nitrophenyl)-5-(2,4

-disulfophenyl)-2H-tetrazolium] and 1-methoxy PMS [1-methoxy-phenazine methosulfate].[10] Briefly, after the medium was removed, 10 μl of CCK-8 solution was added to each well of the plate. The plate was incubated for 4 hours. The absorbance was measured at 450 nm using a microplate reader. Data were expressed as relative viability as accumulated as following: relative viability=[A(subance)- A(blank)] /[A(subtance)-A(blank)]×100%.

Real-time quantitative RT-PCR

Total RNA was extracted by the TRIzol isolation method from 100 islets following a 48 hours culture. First strand cDNA was generated from 2 μg RNA in a 10 ml volume using the oligo (dT) primer.[11,12] After reverse transcription, cDNA was used for real-time PCR using a SYBR Green PCR. The β-actin was co-amplified as an endogenous control to standardize the amount of the sample RNA added to the reaction. Real-time PCR was used for the following parameters: 50˚C for 2 minutes, 9 ˚C for 20 minutes, 95˚C for 30 seconds, and 55˚C for 1 minute, 50 cycles. Primers used in the analysis of the genes are shown in the Table. Results are expressed as relative mRNA express level according to the $2^{\text{delta-delta Ct}}$ variant.

Table. Primers used in the analysis of the genes

Primer (bp)	Sequence
Rat β-actin	Sense: 5'-CACGATGGAGGGGCCGGACTCATC-3'
(167)	Antisense: 5'-AAAGACCTCTATGCCAACACAGT-3'
UCP-2 (149)	Sense: 5′-GCATTGGCCTCTACGACTCT-3′
	Antisense: 5′-CTGGAAGCGGACCTTTACC-3′
PPAR8 (137)	Sense: 5'- AGGGCGATCTTGACAGGAA-3′
	Antisense: 5'- GATGGCCACCTCTTTGCTC-3′
Insulin (119)	Sense: 5'-CACCTTTGTGGTCCTCACCT-3′
	Antisense: 5'- CAGCTCCAGTTGTGGCACT-3′

Statistical analysis

The data were expressed as mean ± standard deviation (SD). Data were analysed by SPSS10.0 software. $P<0.05$ was considered statistically significant.

RESULTS

Insulin secretion

Basal insulin secretion (glucose concentration 2.8 mmol/L) of the rat islets was (18.5±2.4) μU • h^{-1} • 10 $islets^{-1}$ and significantly increased to (73.5±8.1) μU • h^{-1} • 10 $islets^{-1}$ in response to 16.7 mmol/L glucose. Compared with the group of 0 mmol/L palmitate, islets exposed to palmitate (0.25, 0.5, 1.0 mmol/L) for 48 hours showed an increased basal and a decreased glucose-stimulated insulin secretion ($P<0.01$, Fig. 1). Rosiglitazone was dissolved in dimethyl sulfoxide (DMSO) with a final concentration of DMSO of 0.1% in the medium. The direct effects of rosiglitazone in various concentrations (0.01, 0.1, 1.0, 5.0 μmol/L) was examined. Both basal and glucose-stimulated insulin secretions in 0.01, 0.1, 1.0 μmol/L concentration were not affected ($P>0.05$), but in the 5.0 μmol/L concentration of rosiglitazone, basal and glucose-stimulated insulin secretions were

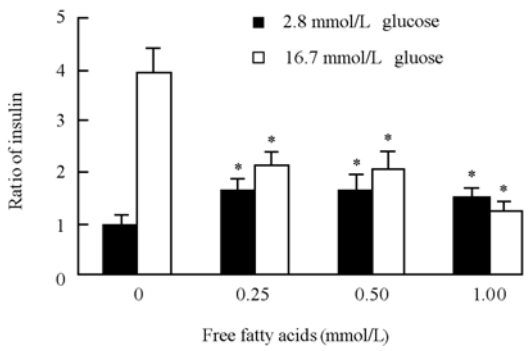

Fig. 1. Islets were cultured with palmitate (0.25, 0.5, 1.0 mmol/L) for 48 hours. Insulin secretion is shown as the ratio of the value compared with the group of 2.8 mmol/L glucose and 0 mmol/L palmitate. Each value represents mean ± SD (n=6). *$P<0.01$ compared with 0 mmol/L palmitate group.

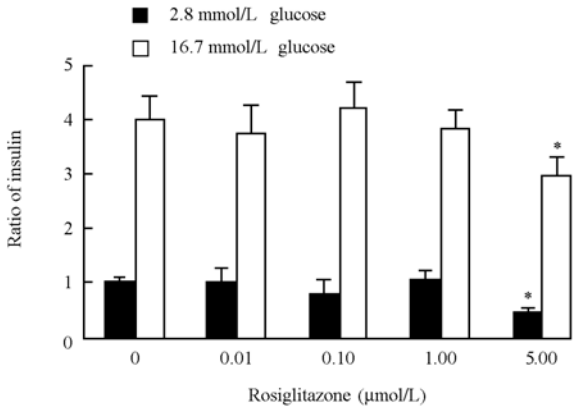

Fig. 2. Islets cultured with various concentrations of rosiglitazone (0.01, 0.1, 1.0, 5.0 μmol/L, for 48 hours. Insulin secretion is shown as the ratio of the value compared with the group of 2.8 mmol/L glucose and 0 mmol/L rosiglitazone. Each value represents mean ± SD (n=6). *$P<0.01$ compared with 0 μmol/L rosiglitazone group.

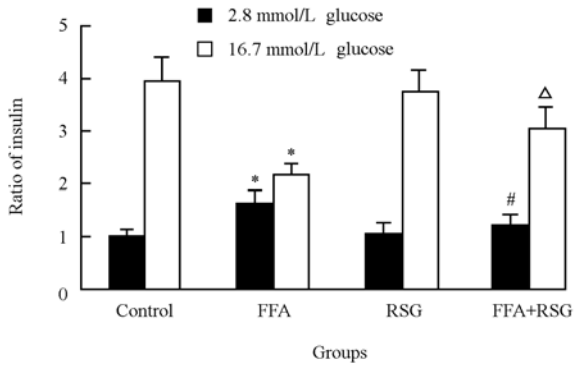

Fig. 3. Islets cultured with FFA (palmitate 0.5 mmol/L) and/or rosiglitazone (1.0 μmol/L) for 48 hours. Insulin secretion shown as the ratio of the value compared with the control of 2.8 mmol/L glucose. Each value represents mean ± SD (n=6). *P<0.01 compared with the control. $^{\#}P$ <0.05 and $^{\triangle}P$ <0.01 compared with FFA group. FFA: free fatty acids; RSG: rosiglitazone.

decreased (P<0.01, Fig. 2). When islets were cultured with palmitate (0.5 mmol/L) in the presence of rosiglitazone (1.0 μmol/L), basal insulin secretion was reduced (P<0.05) and glucose-stimulated insulin secretion was increased (P<0.01) compared with islets cultured with palmitate alone (Fig. 3).

Cell cytotoxicity assay

In the group of 0.25 and 0.5 mmol/L palmitate, the cell viabilities were not affected while 1.0 mmol/L palmitate caused a 35% decrease compared with the group of 0 mmol/L palmitate. Rosiglitazone (0.01, 0.1, 1.0 μmol/L) did not affect the cell viability. In contrast, in the group of 5.0 μmol/L rosiglitazone, the cell viability was decreased by 70% compared with the group of 0 μmol/L rosiglitazone. The cell viability was not affected in the group of palmitate (0.5 mmol/L) with rosiglitazone (1.0 μmol/L) (P>0.05, Fig. 4).

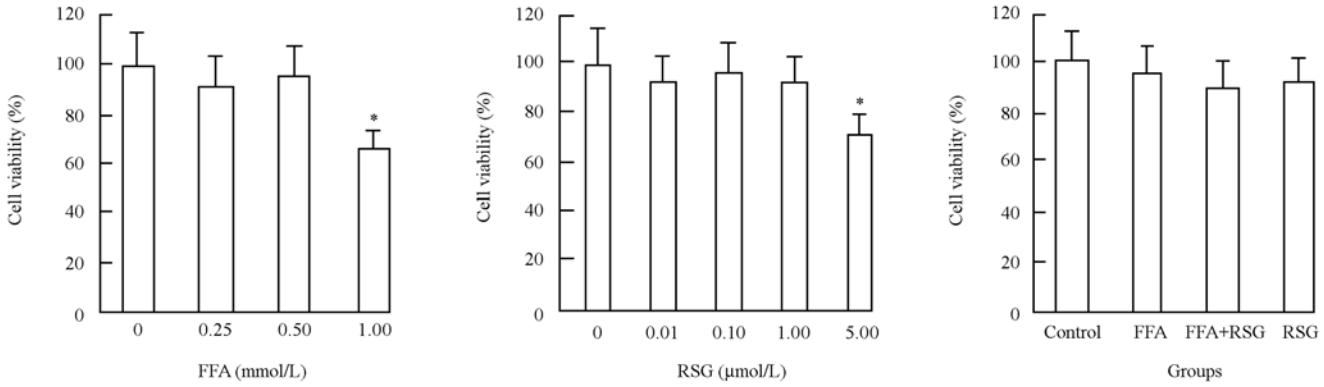

Fig. 4. After cultured with FFA and/or rosiglitazone for 48 hours, cell viability was assessed with a CCK-8. Data were expressed as relative viability. Each value represents mean ± SD (n=6). * P<0.01 compared with the group of 0 concentration. FFA: free fatty acids; RSG: rosiglitazone.

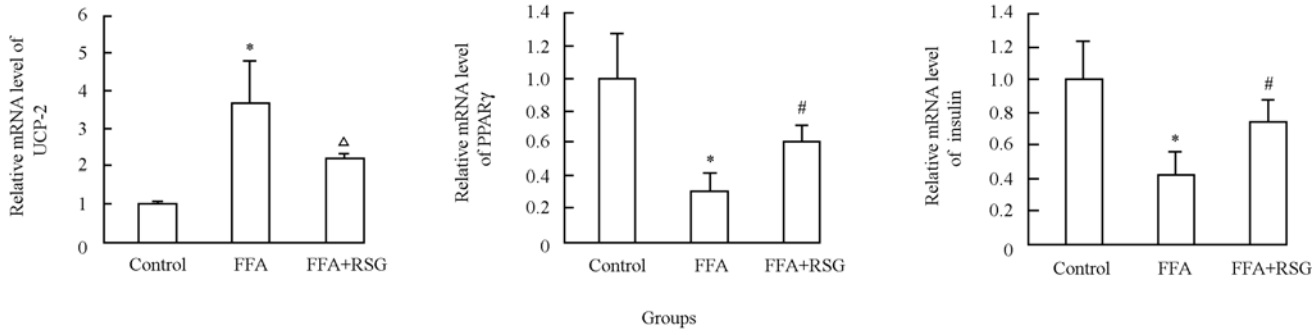

Fig. 5. Islets were cultured with FFA (palmitate 0.5 mmol/L) and /or rosiglitazone (1.0 μmol/L) for 48 hours. Results are expressed as relative mRNA express level. Such expressions as UCP-2 mRNA were measured by real-time PCR and quantified as the ratio of UCP-2/β-actin. The values of UCP-2/β-actin are normalized to that of the control. Data (n=9) are expressed as mean±SD. *P<0.01 compared with the control. $^{\#}P$ <0.05 and $^{\triangle}P$ <0.01 compared with FFA group. FFA: free fatty acids; RSG: rosiglitazone.

Real-time quantitative RT-PCR

Compared with the control, the mRNA level of UCP-2 increased 3.7 fold when islets were exposed to palmitate (0.5 mmol/L) for 48 hours. In the mean time, the mRNA levels of PPAR(and Insulin decreased by 30% and 43% respectively. The addition of rosiglitazone (1.0 μmol/L) in the culture medium significantly increased the mRNA level of PPAR(and decreased the mRNA level of UCP-2, while at the same time, the mRNA level of Insulin was increased as compared with islets cultured with palmitate alone (Fig. 5).

DISCUSSION

Lipid metabolism plays an important role in the regulation of insulin secretion. Chronic hyperlipidemia can exert deleterious effects on β-cell function, which contributes to the progressive deterioration of glucose homeostasis characteristic of type 2 diabetes.[13] Prolonged exposure of pancreatic β-cells to fatty acid increases basal insulin secretion but inhibits glucose-stimulated insulin secretion.[14] UCP-2 is coming to be recognized as a possible link between fatty acid excess and impaired glucose-stimulated insulin secretion.[15] Proteins of the uncoupling protein family are located in the inner mitochondrial membrane and act as proton channels or transporters to uncouple the electrochemical gradient produced by the respiratory chain.[16] The UCP family consists of four main isoforms with different tissue distribution and UCP-2 is the only known uncoupling protein expressed in pancreatic β-cells.[17] UCP-2 knockout mice display an increased β-cell mass and retained insulin secretion capacity in the face of glucolipotoxicity.[18] Fatty acids or dietary fats increase UCP-2 mRNA and protein levels in several tissues and chronic exposure of the β-cells to FFA increases the expression level of the UCP-2 gene and decreases the effects of glucose on the ATP-to-ADP ratio and on the mitochondrial membrane potentials.[19]

The UCP-2 promoter contains a PPAR response element.[20] The observation that FFA increases UCP-2 mRNA expression independent of their oxidation suggests an implication of PPAR in the induction of UCP-2. Rosiglitazone, an agonist of PPAR(, has been shown to have a protective effect on pancreatic islet cell structure and function in animal models of type 2 diabetes and human isolated islets. But the mechanisms triggered by PPAR(were not investigated in that study.[5] Therefore, our hypothesis is that the protective effects of rosiglitazone on insulin secretion of isolated pancreatic islets under chronic exposure to FFA might be mediated through the downregulation of UCP-2 expression.

In our study we successfully built the model of isolated rat pancreatic islets, which had a better response to high glucose. The basal insulin secretion was (18.5 ±2.4) μU •h^{-1} •10 islets^{-1} when glucose concentration was 2.8 mmol/L and significantly increased about 4 times in response to 16.7 mmol/L glucose. After that, we studied the effect of insulin secretion and cell cytotoxicity of palmitate on isolated rat pancreatic islets. As supposed, it caused an increased basal and a decreased glucose-stimulated insulin secretion (P<0.01). The cell viability on palmitate 1.0 mmol/L was decreased by 65% while on other palmitate concentrations were not effected. We also examined the direct effects of various concentration (0.01, 0.1, 1.0, 5.0 μmol/L) rosiglitazone. Both basal and glucose-stimulated insulin secretions in 0.01, 0.1, 1.0 μmol/L concentration were not affected (P>0.05), but in the 5.0 μmol/L concentration of rosiglitazone, basal and glucose-stimulated insulin secretions were decreased (P<0.01). According to the cell cytotoxicity assay, in the group of rosiglitazone (5.0 μmol/L), the cell viability was decreased by 70%, compared with the group of 0 μmol/L rosiglitazone. We believed that the cell cytotoxicity caused the decrease of basal and glucose-stimulated insulin secretion.

Thus, we selected the concentration of palmitate (0.5 mmol/L) with rosiglitazone (1.0 μmol/L) to observe the combined effect. When islets were cultured with palmitate (0.5 mmol/L) in the presence of rosiglitazone (1.0 μmol/L), both basal and glucose-induced insulin secretion reversed to a pattern of control islets (P<0.05 and P<0.01, respectively). In addition, the cell viability was not affected. The above results suggest that rosiglitazone plays a protective effect on insulin secretion of isolated pancreatic islets under chronic exposure to palmitate.

Furthermore, we explored the possible mechanism

through the real-time PCR. When islets were exposed to palmitate (0.5 mmol/L) for 48 hours, the mRNA level of PPAR(decreased by 30%, the mRNA level of UCP-2 increased 3.7 fold and that of Insulin decreased by 43% compared with the control. The addition of rosiglitazone (1.0 μmol/L) in the culture medium significantly increased the mRNA level of PPAR(and decreased the mRNA level of UCP-2, and as a result, the mRNA level of Insulin has a statistically significant difference as compared with islets cultured with palmitate alone.

Our finding confirmed that the blunted insulin secretion as a result of chronic exposure of islets to high palmitate levels is associated with a UCP-2 mRNA upregulation. Moreover, we investigated the role and mechanism of PPAR(agonist, such as rosiglitazone. In the present study, we have demonstrated for the first time that rosiglitazone added to the culture medium was able to prevent UCP-2 expression increase through the activation of PPAR(and to restore both the mRNA level of Insulin and insulin secretion. This protective effect of rosiglitazone on islet β-cells implies that part of the therapeutic action of thiazolidinediones in human type 2 diabetes could be the result of restoration of insulin secretion in addition to its well-known effects on the peripheral insulin target tissues.

In conclusion, we demonstrated that the protective effects of rosiglitazone on insulin secretion of isolated pancreatic islets under chronic exposure to palmitate might be mediated through the downregulation of UCP-2 expression. Our data indicate that rosiglitazone may reverse the insulin secretion defection caused by chronic exposure to FFA, representing a new therapeutic strategy to type 2 diabetes.

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News

Chinese warned of rising rate in hepatitis diseases

Wealthy Chinese are being warned by doctors to note a growing number of hepatitis diseases, as nearly one in every 10 Chinese is a hepatitis B virus carrier.

The high incidence of hepatitis is attributed by doctors to an increase in intake of alcohol and animal fat in diet, which has made hepatitis a so-called “Rich man’s disease”, said Prof. FAN Jian-gao, with a hospital affiliated to the Shanghai University of Communications. “Wealth is not to be blamed here, but the lack of knowledge about a healthy life,” said doctor YANG Bing-hui, when giving lectures in Shanghai for public education marking Love Livers Day, which falls on March 18, 2006.

Chinese Ministry of Health said earlier in February that it is going to launch a nationwide survey on the current epidemic situation of hepatitis B, as the control of hepatitis B has been listed as one of the priorities of the ministry on its infectious disease control agenda. It is estimated that China has some 120 million hepatitis B virus carriers, more than half of the world’s total. The disease has caused a huge financial burden on patients’ families.

According to a study by the Shanghai Academy of Contagious Diseases, the medical expense of a chronic hepatitis B patient averages 20 477 yuan (2559 U.S. dollars) a year and more for hepatocirrhosis and liver cancer treatment. The country’s total spending on treatment of the disease is estimated at 900 billion yuan (112.5 billion U.S. dollars) every year.

Over 80 percent of the hepatitis B virus infection in China is contracted through child birth, said doctors. They called for people not to discriminate against hepatitis virus carriers.

(Source: Xinhuanet)