L6-Glut4MYC大鼠肌细胞系

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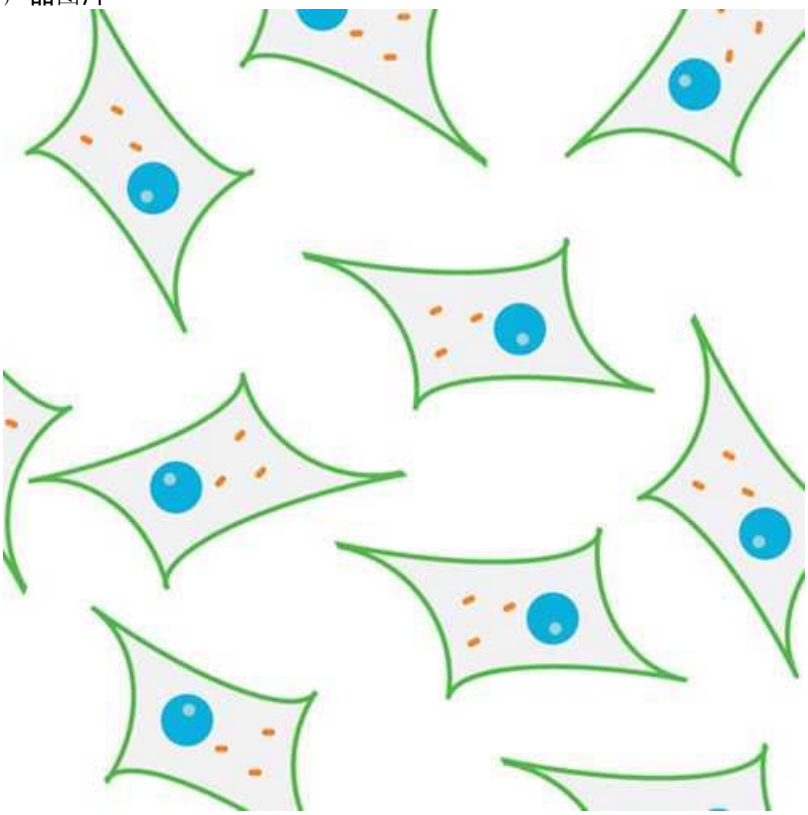

产品英文名称
L6-GLUT4myc Rat Myoblast Cell Line
产品别名
Kerafast独特的生物试剂
货号/SKU
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产品基础信息
From the laboratory of Amira Klip, PhD, Hospital For Sick Children.
产品描述信息

Product Type:	Cell Line

Name:	L6-GLUT4myc
Cell Type:	Skeletal muscle
Accession ID:	CVCL_0P25
Organism:	Rat
Morphology:	Myoblast/Myotube
Source:	Quadriceps
Biosafety Level:	II
Growth Conditions:	MEM-α (with Ribonucleosides and Deoxyribonucleosides) +10% FBS, 1% Antimycotic +5 ug/ml blasticidin
Subculturing:	2-3 days
Cryopreservation:	MEM-α +10% FBS+ 10% DMSO
Comments:	The myc-epitope in the GLUT4myc has the amino acid sequence of AEEQKLISEEDLLK
Storage:	Liquid nitrogen
Shipped:	Dry ice

产品安全信息

Cell Line ReferencesWang Q, Khayat Z, Kishi K, Ebina Y, Klip A. (1998) GLUT4 translocation by insulin in intact muscle cells: detection by a fast and quantitative assay. FEBS Letters 427: 193-7;Ueyama A, Yaworsky KL, Wang Q, Ebina Y, Klip A. (1999) GLUT-4myc ectopic expression in L6 myoblasts generates a GLUT-4-specific pool conferring insulin sensitivity. Am. J. Physiol. Endocrinol. Metab. 277: E572-8;Ishikura S, Antonescu CN, Klip A. (2010) Documenting GLUT4 Exocytosis and Endocytosis in Muscle Cell Monolayers. Curr. Protoc. Cell Biol., John Wiley & Sons, Inc., 46: Unit 15.15: 1-9;Rudich A, Konrad D, Török D, Ben-Romano R, Huang C, Niu W, Garg RR, Wijesekara N, Germinario RJ, Bilan PJ, Klip A. (2003)Indinavir uncovers different contributions of GLUT4 and GLUT1 towards glucose uptake in muscle and fat cells and tissues. Diabetologia 46: 649-58.Huang C, Somwar R, Patel N, Niu W, Török D, Klip A. Sustained exposure of L6 myotubes to high glucose and insulin decreases insulin-stimulated GLUT4 translocation but upregulates GLUT4 activity. Diabetes. 2002 Jul;51(7):2090-8.Application ReferencesPillon NJ, Li YE, Fink LN, Brozinick JT, Nikolayev A, Kuo MS, Bilan PJ, KlipA. (2014) Nucleotides released from palmitate-challenged muscle cells through pannexin-3 attract monocytes. Diabetes. 63: 3815-26.Li Q, Zhu X, Ishikura S, Zhang D, Gao J, Sun Y, Contreras-Ferrat A, Foley KP, Lavandero S, Yao Z, Bilan PJ, Klip A, Niu W. (2014) Ca²? signals promote GLUT4 exocytosis and reduce its endocytosis in muscle cells. Am J Physiol Endocrinol Metab. Jul 15;307(2):E209-24.Sun Y, Chiu TT, Foley KP, Bilan PJ, Klip A. (2014) Myosin Va mediates Rab8A-regulated GLUT4 vesicle exocytosis in insulin-stimulated muscle cells. Mol Biol Cell. 25: 1159-70.Foley KP, Klip A. (2014) Dynamic GLUT4 sorting through a syntaxin-6 compartment in muscle cells is derailed by insulin resistance-causing ceramide. Biol Open. 3: 314-25.Chiu TT, Sun Y, Koshkina A, Klip A. (2013) Rac-1 superactivation triggers insulin-independent glucose transporter 4 (GLUT4) translocation that bypasses signaling defects exerted by c-Jun N-terminal kinase (JNK)- and ceramide-induced insulin resistance. J Biol Chem. 288:17520-31.Pillon NJ, Arane K, Bilan PJ, Chiu TT, Klip A. (2012) Muscle cells challenged with saturated fatty acids mount an autonomous inflammatory response that activates macrophages. Cell Commun Signal. 10: 30.Boguslavsky S, Chiu T, Foley KP, Osorio-Fuentealba C, Antonescu CN, Bayer KU, Bilan PJ, Klip A. (2012) Myo1c binding to submembrane actin mediates insulin-induced tethering of GLUT4 vesicles. Mol Biol Cell. 23: 4065-78.Kewalramani G, Fink LN, Asadi F, Klip A. (2011) Palmitate-activated macrophages confer insulin resistance to muscle cells by a mechanism involving protein kinase C ? and ?. PLoS One. 6: e26947.Sun Y, Bilan PJ, Liu Z, Klip A. (2010) Rab8A and Rab13 are activated by insulin and regulate GLUT4 translocation in muscle cells. Proc Natl Acad Sci U S A. 107: 19909-14.Ishikura S, Antonescu CN, Klip A. (2010) Documenting GLUT4 Exocytosis and Endocytosis in Muscle Cell Monolayers. Curr. Protoc. Cell Biol., John Wiley & Sons, Inc., 46: Unit 15.15: 1-9.Samokhvalov V, Bilan PJ, Schertzer JD, Antonescu CN, Klip A. (2009) Palmitate- and lipopolysaccharide-activated macrophages evoke contrasting insulin responses in muscle cells. Am J Physiol Endocrinol Metab. 296: E37-46.Antonescu CN, Díaz M, Femia G, Planas JV, Klip A. (2008) Clathrin-dependent and independent endocytosis of glucose transporter 4 (GLUT4) in myoblasts: regulation by mitochondrial uncoupling. Traffic. 9: 1173-90.Thong FS, Bilan PJ, Klip A. (2007) The Rab GTPase-activating protein AS160 integrates Akt, protein kinase C, and AMP-activated protein kinase signals regulating GLUT4 traffic. Diabetes. 56: 414-23.Wijesekara N, Tung A, Thong F, Klip A. (2006) Muscle cell depolarization induces again in surface GLUT4 via reduced endocytosis independently of AMPK. Am J Physiol Endocrinol Metab. 290: E1276-86.Ishiki M, Randhawa VK, Poon V, Jebailey L, Klip A. (2005) Insulin regulates the membrane arrival, fusion, and C-terminal unmasking of glucose transporter-4 via distinct phosphoinositides. J Biol Chem. 280: 28792-802.Rudich A, Konrad D, Török D, Ben-Romano R, Huang C, Niu W, Garg RR, Wijesekara N, Germinario RJ, Bilan PJ, Klip A. (2003) Indinavir uncovers different contributions of GLUT4 and GLUT1 towards glucose uptake in muscle and fat cells and tissues. Diabetologia 46: 649-58.Wang Q, Somwar R, Bilan PJ, Liu Z, Jin J, Woodgett JR, Klip A. (1999) Protein kinase B/Akt participates in GLUT4 translocation by insulin in L6 myoblasts. Mol Cell Biol. 19: 4008-18.Wang Q, Khayat Z, Kishi K, Ebina Y, Klip A. (1998) GLUT4 translocation by insulin in intact muscle cells: detection by a fast and quantitative assay. FEBS Letters 427: 193-7.Gao MH, Giamouridis D, Lai NC, Walenta E, Paschoal VA, Kim YC, Miyanohara A, Guo T, Liao

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主要内容

永生化的大鼠骨骼肌细胞（L6）用Myc-epitope过度表达Glut4，这对于测量Glut4易位是有用的。亮点：L6肌细胞线稳定地表达含有14个氨基酸表位的人C-Myc的Glut4蛋白在其第一个外部外圈仅可用于研究葡萄糖摄取和Glut4易位的细胞模型，而不会透化或分馏可用于新型抗糖尿病的细胞复合筛选可用于通过基于质粒的基因转移和病毒感染方案（逆转录病毒和腺病毒）胰岛素刺激骨骼肌中的胰岛素刺激的胰岛素肌肉的结果主要是由于葡萄糖转运蛋白的转移到细胞表面。 L6细胞最初来自大鼠骨骼肌，作为单核肌细胞繁殖，但可以分化为多核原发性肌管。 Myotubes表达了几种典型的骨骼肌，包括Glut4葡萄糖转运蛋白。胰岛素刺激具有高灵敏度和最大反应性的葡萄糖摄取，并且在分化的L6 myotubes和Glut4表达方中，随着L6细胞分化，可以获得这些特性。 L6 myotubes的这些特征是重要的，因为Glut4负责成熟的骨骼肌中的胰岛素依赖性葡萄糖摄取。也可用：L6大鼠肌细胞细胞系列从amira Klip，Phd，Sick Childs医院的实验室。

厂牌介绍

关于**Kerafast Inc.**

Kerafast 是一家位于波士顿的试剂公司，其主要使命是为QuanQiu科学界提供易于使用的独特实验室研究工具。我们的产品组合包括细胞系、抗体、小分子、染料等，其中许多在其他地方无法获得。自 2011 年成立以来，来自全球 190 多个机构的研究人员通过我们的在线平台提供了他们的创新试剂，无需通过传统的材料转让协议流程即可快速获取材料。

我们处理提供实验室的所有销售和运输物流，并从每次销售中返还丰厚的特许权使用费。因此，我们帮助提供实验室节省时间和资源，同时为进一步研究提供额外资金。采购科学家可以更轻松地发现和获取其他地方通常无法获得的独特试剂，同时还可以资助其他研究人员的工作。这创建了一个QuanQiu科学家社区，他们贡献和获取*Reagent for the Greater Good*，以加速他们自己的研究以及整体科学进步。

2018 年，Kerafast 与Absolute Antibody合并，后者是一家总部位于英国的公司，其愿景是为所有研究人员提供重组抗体技术。此次合并将两家公司聚集在一起，共同致力于改善科学界可用的研究工具的选择。

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