UDP-glucose (UDP-Glc) and glycogen levels in skeletal muscle fibers of defined fiber type were measured using microanalytical methods. Infusing rats with insulin increased glycogen in both Type I and Type II fibers. Insulin was without effect on UDP-Glc in Type I fibers but decreased UDP-Glc by 35–40% in Type IIA/D and Type IIB fibers. The reduction in UDP-Glc suggested that UDP-Glc pyrophosphorylase (PPL) activity might limit glycogen synthesis in response to insulin. To explore this possibility, we generated mice overexpressing a UDP-Glc PPL transgene in skeletal muscle. The transgene increased both UDP-Glc PPL activity and levels of UDP-Glc in skeletal muscles by ~3-fold. However, overexpression of UDP-Glc PPL was without effect on either the levels of skeletal muscle glycogen or glucose tolerance in vivo. The transgene was also without effect on either control or insulin-stimulated rates of [14C]-glucose incorporation into glycogen in muscles incubated in vitro. The results indicate that UDP-Glc PPL activity is not limiting for glycogen synthesis.

Glycogen synthesis by skeletal muscle is of critical importance in the control of blood glucose levels by insulin. Skeletal muscle is the major site of insulin-stimulated glucose uptake, and most of the glucose taken up by muscle in response to insulin is deposited as glycogen (1). This synthetic pathway begins with glucose transport, which is activated strongly by insulin (2). Intracellular glucose is phosphorylated rapidly by hexokinase to form glucose 6-phosphate (Glu-6-P) (4), which is isomerized to glucose 1-phosphate (Glu-1-P) (5). Uridine diphosphoglucone (UDP-Glc) pyrophosphorylase (PPL) then utilizes UTP and Glu-1-P to form UDP-Glc, the immediate precursor for glycogen synthesis. The final step in which the glucosyl unit from UDP-Glc is used to extend the glycogen polymer is catalyzed by glycogen synthase, which is also activated by insulin (3). Thus, the first and last steps in the glycogen synthetic pathway are controlled by insulin.

Insulin promotes the movement of the glucose transporter, GLUT4, from intracellular compartments to the cell surface (2). The resulting increase in glucose transport and phosphorylation results in an increase in the intracellular concentration of Glu-6-P, which is an allosteric activator of glycogen synthase (3). Glycogen synthase activity is also controlled by multiosite phosphorylation (3). Phosphorylation of the appropriate sites inactivates glycogen synthase; however, at sufficiently high concentrations, Glu-6-P is able to activate even highly phosphorylated forms of the enzyme. Insulin promotes dephosphorylation of several sites in glycogen synthase, resulting in an increase in Glu-6-P-independent activity as well as a decrease in the $K_i$ for Glu-6-P (3). Thus, glycogen synthase activity is coupled to glucose transport via changes in intracellular Glu-6-P (4).

The relative contributions of glucose transport and glycogen synthase to the stimulation of glycogen synthesis by insulin have been debated for many years (3, 4). Studies in transgenic mice have demonstrated that sufficiently increasing either glycogen synthase or glucose transporter proteins in skeletal muscle results in glycogen accumulation (5–8). Elegant noninvasive methods involving NMR spectroscopy have been used to investigate the control of glycogen synthesis in vivo (4, 9). Application of these methods has revealed that insulin promotes an increase in Glu-6-P content in skeletal muscle (10), consistent with the stimulatory effect of insulin on glucose transport and phosphorylation. By mass action, an increase in Glu-6-P would be expected to increase levels of downstream intermediates in the glycogen synthesis pathway. However, this is not necessarily the case because a sufficient increase in Glu-6-P would activate glycogen synthase, thereby increasing the rate of utilization of UDP-Glc. A limitation of the NMR method is the lack of sufficient sensitivity to measure such intermediates, and there is evidence that Glu-6-P is not a predictor of levels of UDP-Glc. In glucose-clamped rats, hyperinsulinemia increased Glu-6-P in rectus abdominis muscle, but decreased UDP-Glc (11). Although indicative of significant stimulatory effects of insulin on both glucose transport and glycogen synthase activity, these findings suggest that the UDP-Glc PPL reaction might limit glycogen synthesis in the presence of insulin.

A complicating factor in interpreting the measurements of metabolites in vivo is the fact that the fibers found in most skeletal muscles are heterogeneous, differing not only in con-
tractile speed but also in metabolic potential (12–14). Fibers may be assigned to different classes, or types, based on the activities of representative enzymes of energy metabolism (12–14). Type I fibers have relatively low levels of glycolytic enzymes and high levels of oxidative enzymes. Type I fibers also have a slow twitch speed and are resistant to fatigue because most of their energy is derived from oxidative metabolism. Type II B fibers have relatively high levels of glycolytic enzymes and low levels of oxidative enzymes. Type II B fibers also have a fast twitch speed and depend largely on glycogen metabolism to provide the energy to fuel rapid and forceful contractions. Type II A and II D fibers have a fast twitch speed and high levels of both glycolytic and oxidative enzymes (15). The effect of insulin on glucose transport is highest in muscles composed predominantly of oxidative fiber types (16), and the amount of GLUT4 correlates directly with the levels of enzymes of oxidative energy metabolism, such as malate dehydrogenase (MDH) (17).

The present study was conducted to investigate the role of UDP-Glc PPL in the stimulation of glycogen synthesis by insulin. Using microanalytical techniques to measure directly UDP-Glc and glycogen in single manually dissected muscle fibers of defined muscle fiber type, we demonstrate that insulin decreases UDP-Glc in Type II fibers. We also generated transgenic mice overexpressing UDP-Glc PPL in skeletal muscle to test the hypothesis that UDP-Glc PPL activity limits the insulin-stimulated rate of glycogen synthesis.

**EXPERIMENTAL PROCEDURES**

**Glucose Clamps**—Male rats (Sprague-Dawley, ~250 g) were allowed free access to food and water. Rats were anesthetized with pentobarbital before glucose clamps were applied essentially as described previously (18). Briefly, insulin (10 milliunits/min/kg) or an equal volume of saline was infused via a cannula in the jugular vein. The muscles were stored in liquid nitrogen then placed in liquid nitrogen and frozen in liquid nitrogen.

**Muscle Preparation for Analyses**—Muscles were homogenized in 0.4 ml of 30% KOH, before samples were filtered on 2-cm squares of filter paper (Whatman 31ET), which were washed five times with NaOH, then after adding 0.1 ml EDTA (100 μM/ml extract), the extracts were boiled at 100 °C for 10 min and centrifuged at 8,900 × g for 20 min at 4 °C. Aliquots of the supernatants (500 μl) were added to 500-μl solutions containing 2 mM MgCl₂, 2 mM NAD⁺, 0.016 unit/ml UDP-Glc dehydrogenase, and 50 mM Tris·HCl, pH 8.1. The samples were incubated for 2 h at 24 °C before the NADH generated was measured fluorometrically. After subtracting blank values from incubations conducted without UDP-Glc dehydrogenase, the UDP-Glc content of the samples was determined from a standard curve generated with known concentrations of UDP-Glc.

**Measurements of UDP-Glc and Glycogen in Mouse Muscle**—Muscle powders were weighed and then homogenized (50 mg of tissue/ml of medium) at 0 °C in either 0.3 ml perchloric acid (for glycogen measurements) at 0.02 M HCl (for UDP-Glc measurements). The homogenates were centrifuged at 8,900 × g for 10 min at 4 °C before supernatants were collected for analyses. Glycogen was measured by the amyloglucoisidase method described by Passonneau and Lauderdale (25). UDP-Glc was measured by a modification of the fluorometric assay described by Passonneau and Lowry (20). Briefly, the HCl extracts were neutralized with NaOH, then after adding 0.1 ml EDTA (100 μM/ml extract), the extracts were boiled for 5 min and centrifuged at 8,900 × g for 20 min at 4 °C. Aliquots of the supernatants (500 μl) were added to 500-μl solutions containing 2 mM MgCl₂, 2 mM NAD⁺, 0.016 unit/ml UDP-Glc dehydrogenase, and 50 mM Tris·HCl, pH 8.1. The samples were incubated for 2 h at 24 °C before the NADH generated was measured fluorometrically. After subtracting blank values from incubations conducted without UDP-Glc dehydrogenase, the UDP-Glc content of the samples was determined from a standard curve generated with known concentrations of UDP-Glc.

**UDP-glucose Pyrophosphorylase in Skeletal Muscle Fibers**

UDP-Glc PPL activity was measured by modifying the method described by Roach et al. (26) for measuring the activity of the enzyme in liver extracts. Powdered muscle was homogenized (100 mg of tissue/ml of medium) at 4 °C with a glass homogenization tube containing 1 ml of 0.1 M sodium pyrophosphate, 0.1 M sodium oxalate, and a Teflon pestle driven at 1,000 rpm. The homogenization buffer contained 50 mM NaCl, 10 mM NaF, 0.25% Tween 20, 10% glycerol, 0.1 mM aprotinin, leupeptin, and pepstatin-A. The homogenates were rotated at 4 °C for 45 min, then centrifuged at 8,900 × g for 30 min at 4 °C. The supernatants (20 μl) were assayed for UDP-Glc PPL activity. Aliquots of 40 μl were added to 40 μl of reaction mix containing 50 mM Tris·HCl, pH 8.1, 2 μM UDP-Glc, 0.24 mM [U-14C]glucose (20 μCi/ml), 0.016 unit/ml UDP-Glc dehydrogenase, and 50 mM Tris·HCl, pH 8.1. The samples were incubated at 37 °C for 30 min to remove endogenous hormones, then transferred to tubes containing 10 ml of Krebs-Henseleit buffer (118 mM NaCl, 4.7 mM KCl, 2.5 mM CaCl₂, 1.2 mM potassium phosphate, 1.2 mM MgSO₄, and 25 mM NaHCO₃, pH 7.4) plus 5 μM [U-14C]glucose (~500 cpm/mmol). The buffer was gassed directly by bubbling with a mixture of 95% O₂ and 5% CO₂. The muscles were incubated at 37 °C without or with porcine insulin for 30 min, then rinsed quickly, blotted on tissue paper, and frozen in liquid nitrogen.

Muscles were homogenized in 0.4 ml of 30% KOH, before samples were placed in a boiling water bath for 1 h. After adding 50 μl of a 5% glycogen solution to each sample, ethanol was added to a final concentration of 70%, and glycogen was allowed to precipitate at ~20 °C for 8 h. The samples were centrifuged at 8,900 × g for 20 min at 4 °C to pellet the glycogen. The glycogen pellets were washed four times with 68% ethanol and dissolved in 50 μl of deionized water. Samples were spotted on 2-cm squares of filter paper (Whatman 31ET), which were washed five times with 66% ethanol. The papers were dried before the amount of 14C-labeled glycogen was determined by scintillation counting.

**Intravenous and Intraperitoneal Glucose Tolerance Tests**—Food was removed the evening before glucose tolerance tests, which were conducted the following morning. Mice were injected either intraperitoneal-
Glc content of both Type IIA/D fibers and Type IIB fibers by UDP-Glc in Type I fibers; however, insulin decreased the UDP-response to insulin. Insulin was without effect on the level of exhibited the smallest percentage increase in glycogen levels in fibers, where glycogen was increased by 71%. Type IIB fibers (A) The gen contents of the three types of muscle fibers ranged from an 36.6% were IIA/D, and 42.7% were IIB.

Effect of Insulin on Glycogen and UDP-Glc in Single Muscle Fibers—To investigate the effect of insulin on glycogen synthesis in different types of muscle fibers, rats were infused with either a maximally effective concentration of insulin or a saline control. Blood glucose was clamped at 5 mM to prevent hypoglycemia in the insulin-treated animals and to eliminate differences in blood glucose in the control and insulin-treated animals. Rectus abdominis muscles were freeze-clamped in situ then freeze-dried at −30 °C. Muscle fibers were dissected from the dried muscles, and enzyme activities and levels of UDP-Glc and glycogen were measured in pieces of individual fibers.

To allow assignment of fibers into different fiber type categories, the activities of LDH and MDH activities were measured (12). Type IIB fibers have the lowest levels of MDH activity of the different types of fibers, and in a plot of LDH versus MDH activity IIB fibers were tightly clustered and well separated from the more oxidative fibers (Fig. 1). Of the oxidative fiber types, Type I fibers have the lowest levels of MDH, and a previous study demonstrated that those fibers with low LDH activity with MDH activity less than 15 mmol/h/kg were Type I (29). In the original study, those fibers having MDH activities greater than 15 mmol/h/kg were categorized as Type IIA fibers (12). It is now known that IIB fibers may also have high levels of oxidative enzymes (30). Therefore, fibers having an MDH activity higher than 15 mmol/h/kg were assigned to a IIA/IID category. By these criteria, 20.7% of the fibers were Type I, 36.6% were IIA/D, and 42.7% were IIB.

In muscles from control (saline-infused) animals, the glycogen contents of the three types of muscle fibers ranged from an average of 140 mmol/kg, dry weight, in Type I fibers to 194 mmol/kg, dry weight, in Type IIA/D fibers. Insulin increased the amount of glycogen in all three types of fibers (Fig. 2A) The percentage change caused by insulin was greatest in Type I fibers, where glycogen was increased by 71%. Type IIB fibers exhibited the smallest percentage increase in glycogen levels in response to insulin. Insulin was without effect on the level of UDP-Glc in Type I fibers; however, insulin decreased the UDP-Glc content of both Type IIA/D fibers and Type IIB fibers by 40% (p<0.001) and 28% (p<0.01), respectively (Fig. 2B). The findings in these fast twitch fiber types are consistent with the previous report of decreased UDP-Glc in whole rectus abdominis muscles from insulin-treated rats (11).

Transgenic Overexpression of UDP-Glc PPL in Skeletal Muscle—The finding that UDP-Glc concentrations were decreased by insulin suggests that the supply of UDP-Glc by the UDP-Glc PPL reaction limits glycogen accumulation in response to insulin. If this hypothesis were correct, then overexpressing UDP-Glc PPL would be expected to enhance insulin-stimulated...
glycogen synthesis. To test this hypothesis, transgenic mice were generated in which UDP-Glc PPL was selectively overexpressed in skeletal muscle by using promoter and enhancer elements from the skeletal muscle creatine kinase gene (22). Transgenes under the control of these elements have been shown to be expressed preferentially in glycolytic muscle fiber types (22). Two transgenic lines, designated PPL-1 and PPL-2, were selected for initial experiments.

UDP-Glc PPL activities in tibialis anterior, EDL, gastrocnemius, and soleus muscles from transgenic animals and wild type littermates were measured (Fig. 3). The four wild type muscles contained comparable amounts of UDP-Glc PPL activity. Activities were 2–3-fold higher in transgenic tibialis anterior, EDL, and gastrocnemius muscles than in these muscles in nontransgenic littermates; however, very little, if any, increase in UDP-Glc PPL activity attributable to the transgene was noted in the soleus muscles (Fig. 3). The soleus is composed predominantly of Type I fibers (31). Thus, expression of UDP-Glc PPL is consistent with the expression of the creatine kinase gene, which is lowest in Type I muscle fibers (22). Because UDP-Glc PPL activities were approximately the same in muscles from PPL-1 and PPL-2 mice, subsequent studies were conducted with only one of the lines (PPL-1).

To confirm transgenic overexpression, antibodies to UDP-Glc PPL were generated, and the levels of enzyme protein were assessed by immunoblotting. The results in Fig. 4 depict immunoblots of samples of quadriceps, EDL, and diaphragm muscles from PPL-1 mice and nontransgenic littermates. The transgene increased UDP-Glc PPL protein by ~5-fold in the quadriceps and EDL muscles. As expected, transgenic expression of UDP-Glc PPL was relatively low in the diaphragm, which is composed primarily of oxidative fibers. Although the percentage increase in enzyme protein in the EDL was somewhat greater than the percentage increase in enzyme activity, the immunoblotting and enzyme activity measurements confirm that UDP-Glc PPL was overexpressed in muscles from PPL-1 mice.

Transgenic overexpression of UDP-Glc PPL increased levels of UDP-Glc in gastrocnemius muscles by ~3-fold (Fig. 5). The percentage increase in UDP-Glc was very similar to the increase in UDP-Glc PPL activity produced by the transgene in these muscles, solidifying the conclusion that the overexpressed enzyme was active. In contrast, levels of glycogen were almost identical in gastrocnemius muscles from PPL-1 mice and nontransgenic littermates (Fig. 5). Thus, increasing UDP-Glc, the immediate precursor of glycogen, by transgenic overexpression of UDP-Glc PPL was not sufficient to drive glycogen accumulation.

To investigate the effect of increasing UDP-Glc on insulin action in vivo, we evaluated the effect of the transgene on glucose tolerance by conducting both intraperitoneal (Fig. 6A) and intravenous (Fig. 6B) glucose tolerance tests. The changes in blood glucose after administration of glucose in nontransgenic and transgenic animals were virtually identical. Thus, transgenic overexpression of UDP-Glc PPL in skeletal muscle did not significantly affect whole animal glucose disposal.

Failure of Transgenic Overexpression of UDP-Glc PPL to Enhance Insulin-stimulated Glycogen Synthesis in Vitro—As a result of homeostatic mechanisms, a change in insulin action in skeletal muscle is not always apparent in glucose tolerance tests. To examine more directly insulin action in skeletal muscle, we conducted in vitro experiments with wild type and transgenic EDL muscles. After incubating in medium contain-
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Table I

Effect of insulin on UDP-Glc and Glu-6-P levels in wild type and transgenic EDL muscles incubated in vitro

| Treatment | UDP-Glc in EDL from Wild type | PPL-1 | Glu-6-P in EDL from Wild type | PPL-1 |
|-----------|-------------------------------|-------|-------------------------------|-------|
| Control   | 7.8 ± 0.8 nmol/g, wet weight | 11.8 ± 1.6* | 58 ± 6 nmol/g, wet weight | 59 ± 6 nmol/g, wet weight |
| Insulin, 250 milliunits/ml | 4.3 ± 0.4** | 6.4 ± 0.2*** | 82 ± 20 nmol/g, wet weight | 77 ± 7 nmol/g, wet weight |

*p < 0.05, significance of difference between PPL-1 and wild type.
**p < 0.01, significance of difference between insulin and control.

In the absence or presence of insulin (Fig. 7), the transgene did not decrease UDP-Glc levels, although it is also possible that UDP-Glc levels are different in quadriceps and EDL muscles. Importantly, incubating the EDL muscles with insulin decreased UDP-Glc levels by ~45% (Table I). This change, which is comparable with the percentage change produced by insulin in Type II muscle fibers in vitro (Fig. 2B), confirms that insulin decreases UDP-Glc in mouse skeletal muscle.

Levels of UDP-Glc were 50% higher in EDL muscles from PPL-1 mice than in muscles from wild type animals (Table I), although the effect of the transgene was not as large as in quadriceps frozen in situ (Fig. 5). In contrast, levels of Glu-6-P in wild type and transgenic muscles were not significantly different, in either the absence or presence of insulin (Table I). Transgenic overexpression of PPL-1 did not prevent the fall in UDP-Glc in response to insulin. However, after incubation with insulin, UDP-Glc was 50% higher in transgenic muscles than in wild type muscles.

Experiments were conducted to determine whether the increase in UDP-Glc produced by the transgene was sufficient to increase glycogen synthesis. Incubating wild type EDL muscles in vitro with a physiological concentration of insulin (100 milliunits/ml) increased UDP-Glc and glycogen in vitro by ~2-fold (Fig. 7A). With a maximally effective concentration of insulin, the amount of UDP-Glc incorporation into glycogen was increased by 10-fold (Fig. 7B). The transgene did not significantly affect UDP-Glc incorporation into glycogen in the absence of insulin (Fig. 7A and B). Results comparable with those in the EDL muscles were obtained with nontransgenic and transgenic hemidiaphragms (Fig. 7C).

Discussion

The present results indicate that insulin decreases UDP-Glc levels in Type II skeletal muscle fibers (Fig. 2A). This finding suggested to us that UDP-Glc PPL activity might limit glycogen synthesis in fast twitch muscle fibers stimulated with insulin. To test this hypothesis, we generated transgenic animals overexpressing UDP-Glc PPL in skeletal muscle. The results demonstrate that the transgene increased UDP-Glc in muscle (Fig. 5) but was without effect on glycogen accumulation (Fig. 5) and glucose tolerance in vitro (Fig. 6) or on rates of glucose incorporation into glycogen in vitro (Fig. 7).

The striking metabolic heterogeneity of the fibers that compose skeletal muscle is a complicating factor that is rarely addressed in interpreting the meaning of changes in metabolites measured in whole muscles or in animals. When the effects of insulin on metabolite levels in skeletal muscle are investigated, it is common practice to assume that the changes occur within the same muscle fibers, although this is not necessarily the case. We addressed this potential problem in a unique manner using microanalytical methods to measure levels of UDP-Glc and glycogen in single manually dissected muscle fibers from control and insulin-treated animals. Insulin increased glycogen and decreased UDP-Glc levels in Type II fibers, whereas in Type I fibers the hormone increased glycogen but did not decrease UDP-Glc (Fig. 2).

Our results confirm and extend the observations of Rossetti and Hu (11), who found that infusing rats with insulin decreased UDP-Glc levels in whole rectus abdominis muscles. In principle, a decrease in UDP-Glc could result from either an increase in its rate of utilization or a decrease in its rate of formation. Because insulin stimulates glucose transport/phosphorylation, it is unlikely that a decrease in the supply of Glu-1-P provided by isomerization of Glu-6-P explains the fall in UDP-Glc. Glu-1-P is also produced by the phosphorylase reaction, and there is evidence that insulin decreases glycogenolysis in skeletal muscle (32, 33). If the rate of glycogenolysis were sufficiently high, inhibiting the process could lead to a decrease in UDP-Glc. However, as suggested previously (11), it seems more likely that the well-established effect of insulin on activating glycogen synthase, which utilizes UDP-Glc to synthesize glycogen, explains the fall in UDP-Glc. Consistent with this interpretation, transgenic overexpression of glycogen synthase decreased UDP-Glc in skeletal muscle (7). In the present study insulin decreased UDP-Glc in both control and PPL-1
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EDL muscles (Table I). Indeed, the absolute decrease in UDP-Glc produced by insulin was larger in the transgenic muscles than in the wild type muscles (5.4 nmol/g, wet weight, compared with 3.5 nmol/g, wet weight), indicating that insulin activated glycogen synthase in the transgenic muscles.

The intracellular concentration of UDP-Glc appears to be insufficient to saturate glycogen synthase. Therefore, regardless of the mechanism through which insulin increases UDP-Glc, such a decrease would be expected to limit the rate of glycogen synthesis. Likewise, all things being equal an increase in UDP-Glc should increase glycogen synthesis. For this reason the finding that increasing UDP-Glc PPL in skeletal muscle did not lead to glycogen accumulation was unexpected. It is clear that the transgene was expressed. Immunoblotting with antibodies to UDP-Glc PPL demonstrated that enzyme protein was increased in different skeletal muscles in a pattern consistent with that expected from the creatine kinase promoter and enhancer elements that were used to drive transgenic expression (Fig. 4). Measurements of UDP-Glc PPL activity indicated that the amount of the transgene expressed was sufficient to increase UDP-Glc PPL activity (Fig. 3). Moreover, measurements of tissue UDP-Glc confirmed that the amount of UDP-Glc PPL overexpressed was sufficient to increase UDP-Glc in skeletal muscle (Fig. 5).

The finding that UDP-Glc levels can be increased without affecting glycogen synthesis argues that UDP-Glc PPL does not limit glycogen synthesis; however, there are caveats attached to this conclusion. We cannot be certain that the transgene increased UDP-Glc in the same pool as that utilized for glycogen synthesis. Moreover, with as many studies involving transgenic animals, long term overexpression of an enzyme may produce unknown compensatory changes that complicate interpretation of the results. The present findings are similar in some respects to those of Skurat et al. (34), who found that overexpressing UDP-Glc PPL in COS cells, either alone or with wild type glycogen synthase, was without effect on glycogen accumulation. Although additional studies will be needed to determine why increasing UDP-Glc by transgenic overexpression of UDP-Glc PPL does not increase glycogen accumulation.

2 Measurements of the $K_m$ of purified rabbit muscle glycogen synthase for UDP-Glc ranged from approximately 50 $\mu$M for the fully dephosphorylated form assayed in the presence of Glu-6-P to more than 9 mM for a phosphorylated enzyme assayed in the absence of Glu-6-P (35). The dry weight of single muscle fibers is approximately 20% of the wet weight. To estimate the concentration of UDP-Glc from the single fiber measurements, it was assumed that 1 kg, wet weight, was equal to a 1-liter volume and that UDP-Glc was distributed evenly within the cell. Thus, for example, after insulin treatment the UDP-Glc concentration in Type II fibers was approximately 50 $\mu$M.

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Effects of Insulin and Transgenic Overexpression of UDP-glucose Pyrophosphorylase on UDP-glucose and Glycogen Accumulation in Skeletal Muscle Fibers

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