Primary Aldosteronism (PA) is the most frequent cause of secondary hypertension. Hyperaldosteronism is associated with cardiovascular disease in a blood pressure-dependent and independent manner [1-3]. In addition, PA is considered to be an endocrine cause of diabetes mellitus [4]. Hyperaldosteronism may contribute to abnormalities in glucose metabolism in a multifactorial manner. Hypokalemia induced by hyperaldosteronism leads to impaired insulin secretion and sensitivity. Moreover, aldosterone itself is shown to cause β-cell dysfunction and insulin resistance. Recent basic research reveals that aldosterone impairs β-cell function via glucocorticoid receptors (GR) but not via mineralocorticoid receptors (MR) [5].

Previous clinical studies show discrepant results about whether specific treatments used in PA (adrenalectomy or MR-antagonist treatment) improve glucose metabolism. In the analyses of most of these studies, no differentiation was made between aldosterone producing adenomas (APA) and idiopathic adrenal hyperplasia. However, surgical treatment for unilateral APA can completely correct the hyperaldosteronism, whereas MR-antagonist treatment for idiopathic adrenal hyperplasia cannot. As mentioned above, aldosterone impairs β-cell function, but not via the MR-receptor. Thus, MR-antagonist treatment may not attenuate the effect of hyperaldosteronism on β-cells. Hence, evaluating the effect of aldosterone excess on β-cell function by analyzing data only from patients with APA before and after adrenalectomy...
would probably yield more accurate results. However, many previous studies comparing glucose metabolism before and after adrenalectomy had limited numbers of patients [6, 7].

In the present study, we precisely localized APA by means of the segment-selective adrenal venous sampling method. Then, we attempted to evaluate glucose metabolism by performing a 75g-oral glucose tolerance test (OGTT) before and one year after adrenalectomy in 61 patients with unilateral APA.

**Materials and Methods**

**Study population**

The clinical records of 61 patients were retrospectively analyzed. The subjects were definitively diagnosed with PA during screening of hypertensive patients who visited our outpatient clinic between 2007 and 2014, according to the Japan Endocrine Society (JES) guidelines [8]. The diagnosis was confirmed by endocrinologic examinations, such as the furosemide plus upright test, saline-loading test, and captopril-loading test [8-10]. All subjects were diagnosed with unilateral APA by computed tomography imaging and segment-selective ACTH loading adrenal venous sampling (SS-ACTH-AVS), as reported previously [9-12]. All subjects underwent unilateral adrenalectomy. A 75-OGTT was performed on all patients before surgery and six months to one year after surgery. Exclusion criteria were as follows: diabetes mellitus (diagnosed according to the Japan Diabetes Society guidelines [13]), renal insufficiency with estimated glomerular filtration rate (eGFR) < 30 mL/min/1.73m², and excess of cortisol (determined as > 3 μg/dL cortisol level after a 1 mg dexamethasone suppression test [DST] [14]). The study protocol was approved by the research ethics committee of Yokohama Rosai Hospital. The requirement of informed consent was waived because of no intervention and no further examinations. The outline of this study is shown on the website of Yokohama Rosai Hospital (https://www.yokohamah.johas.go.jp/).

**Oral glucose tolerance test**

After baseline blood sampling, a standard 75g-OGTT was performed [15]. Plasma samples for glucose and immunoreactive insulin (IRI) (Roche diagnostics, Basel, Switzerland) were obtained at 0, 30, 60, 90, and 120 minutes after glucose loading.

**Measurements and calculation of indices**

The following indices were utilized in this study: Insulinogenic index = ΔIRI₀-₃₀ / Δplasma glucose (PG)₀-₃₀ [16]; homeostasis model assessment of insulin resistance (HOMA-IR) = [fasting plasma glucose (FPG) × fasting IRI (FIRI)] / 405 [17]; ISIₘatsu ¬da = 10,000 / [sqrt (FPG × FIRI × mean PG × mean IRI)] [18], where mean PG and mean IRI represent mean glucose and insulin concentrations during the OGTT; and the disposition index = Stumvoll-1 / HOMA-IR, which was shown to have the best-fit hyperbolic line for insulin sensitivity and insulin secretion in Japanese [19]. Stumvoll-1 = 1,283 + 1.829 × IRI₃₀ – 138.7 × PG₃₀ + 3.772 × FIRI [20]. For the insulinogenic index, HOMA-IR, and ISIₘatsu ¬da, the units used for PG and IRI were mg/dL and μU/mL, respectively. For Stumvoll-1, the units used for PG and IRI were pmol/mL and mmol/L, respectively. The area under the curve (AUC) was calculated using a trapezoidal method. Negative or unusual values for insulinogenic index were obtained in 3 patients; the values for these results were assumed absent.

**Statistical analysis**

The Shapiro-Wilk test was performed to assess the normal distribution of the quantitative variables. Data are given as mean ± standard deviation for parameters with a normal distribution or the median (25-75th percentile) for parameters with a non-normal distribution. The paired t-test for parametric data or the Wilcoxon signed-rank test for non-parametric data were used to compare the parameters before and after surgery. The paired t-test for parametric data or the Wilcoxon signed-rank test for non-parametric data were used to compare the parameters before and after surgery. The statistical analysis was performed using JMP® software (SAS Institute Inc., Cary, NC, USA) for all analyses.

**Results**

**Clinical characteristics before and after adrenalectomy**

The characteristics of the 31 male and 30 female participants before and after surgery are shown in Table 1. The mean age at the time of surgery was 49.2 ± 10.6 years. After surgery, plasma aldosterone concentrations (PAC) (278 [174-397] to 76.5 [58.7-92] pg/mL, p < 0.001) and urinary aldosterone excretion...
Glucose metabolism post adrenalectomy

OGTT increased significantly at 0 min (5.3 ± 3.0 to 7.7 ± 5.3 μU/mL, \(p < 0.001\)), 30 min (38.8 ± 21.8 to 57.0 ± 32.8 μU/mL, \(p < 0.001\)), 60 min (53.3 ± 38.8 to 76.1 ± 53.5 μU/mL, \(p < 0.001\)), and 90 min (60.4 ± 42.5 to 79.3 ± 60.8 μU/mL, \(p = 0.02\)). The AUC-IRI increased ~1.5 times after surgery (77.3 [51.6-118.0] to 99.3 [64.2-156.2] μU/mL × hr, \(p < 0.001\)).

Insulin secretion and insulin resistance after adrenalectomy

We calculated the indices of insulin secretion and insulin resistance (or sensitivity) using the results of the 75g-OGTT before and after surgery (Fig. 2). The insulinogenic index increased significantly after surgery (0.5 [0.4-0.8] to 0.8 [0.4-1.1], \(p < 0.001\)). On the other hand, the disposition index, the product of measures of insulin secretion and insulin sensitivity, did not change (806.2 [489.4-1,138.9] to 686.6 [479.4-922.1], \(p = 0.25\)). HOMA-IR, an index of hepatic insulin resistance, increased significantly after surgery (0.5 [0.4-0.8] to 0.8 [0.4-1.1], \(p < 0.001\)). In addition, ISI_matsuda, an index of whole body insulin sensitivity, decreased significantly (6.9 [4.5-10.4] to 5.2 [3.4-7.9], \(p < 0.001\)).

Table 1  Comparison of the clinical characteristics of the participants before and after adrenalectomy

<table>
<thead>
<tr>
<th></th>
<th>Before surgery</th>
<th>After surgery</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (male/female)</td>
<td>31 / 30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>49.2 ± 10.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.1 (21.4-26.8)</td>
<td>23.0 (21.1-27.2)</td>
<td>0.11</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>170 (158-189.5)</td>
<td>128 (122-136)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>104 (92.5-117.2)</td>
<td>80 (74-85)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Number of antihypertensive drugs used</td>
<td>2 (1-2)</td>
<td>0 (0-1)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Plasma aldosterone (pg/mL)</td>
<td>278 (174-397)</td>
<td>76.5 (58.7-92)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Plasma renin activity (ng/mL/hr)</td>
<td>0.3 (0.1-0.4)</td>
<td>0.5 (0.2-1.1)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Urinary aldosterone (μg/day)</td>
<td>17.3 (12.5-32.5)</td>
<td>3.25 (1.4-5.1)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Serum potassium (mEq/L)</td>
<td>3.1 (3.0-3.5)</td>
<td>4 (3.9-4.2)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Cases being treated with potassium replacement</td>
<td>8 (13.1%)</td>
<td>0 (0%)</td>
<td>0.006</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>0.7 (0.6-0.9)</td>
<td>0.8 (0.6-0.9)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>eGFR (mL/min/1.73m²)</td>
<td>78.0 (68.3-100.6)</td>
<td>73.3 (65.7-87.0)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>84 (61-132.5)</td>
<td>88 (66-154)</td>
<td>0.15</td>
</tr>
<tr>
<td>HDL-cholesterol (mg/dL)</td>
<td>51 (44.5-57)</td>
<td>49 (42-60.5)</td>
<td>0.74</td>
</tr>
<tr>
<td>LDL-cholesterol (mg/dL)</td>
<td>111 (96-134.5)</td>
<td>116 (94.5-129.5)</td>
<td>0.92</td>
</tr>
<tr>
<td>AST (IU/L)</td>
<td>18 (16-23)</td>
<td>19 (15-22)</td>
<td>0.8</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>17 (13-23.5)</td>
<td>17 (11-22)</td>
<td>0.17</td>
</tr>
<tr>
<td>γ-GTP (IU/L)</td>
<td>21 (13-31.5)</td>
<td>17.5 (13-37.5)</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Data are given as mean ± standard deviation or as the median (25-75th percentile). BMI, body mass index; eGFR, estimated glomerular filtration rate; HDL, high density lipoprotein; LDL, low density lipoprotein; AST, aspartate amino transferase; ALT, alanine amino transferase; γ-GTP, γ-glutamyltranspeptidase.

(17.3 [12.5-32.5] to 3.25 [1.4-5.1] μg/day, \(p < 0.001\)) decreased significantly. Systolic blood pressure (170 [158-189.5] to 128 [122-136] mmHg, \(p < 0.001\)) and diastolic blood pressure (104 [92.5-117.2] to 80 [74-85] mmHg, \(p < 0.001\)) were significantly improved. Serum potassium levels increased significantly (3.1 [3.0-3.5] to 4 [3.9-4.2] mEq/L, \(p < 0.001\)), and the number of patients being treated with potassium replacement decreased significantly (8 [13.1%] to 0 [0%]). Serum creatinine levels increased slightly, but significantly (0.7 [0.6-0.9] mg/dL to 0.8 [0.6-0.9] mg/dL, \(p < 0.001\)), possibly due to the improvement of glomerular filtration.

Plasma glucose and IRI levels after surgery, assessed by 75g-OGTT

As shown in Fig. 1, a 75g-OGTT was performed before and one year after adrenalectomy. Fasting plasma glucose levels increased slightly (88.7 ± 9.3 to 91.5 ± 10.0 mg/dL, \(p = 0.024\)), however, post-challenge glucose levels did not change. The AUC-PG was similar before and after surgery (287.5 [255.9-320.0] to 263.5 [239.3-325.1] mg/dL × hr, \(p = 0.08\)). On the other hand, after surgery IRI levels measured during OGTT increased significantly at 0 min (5.3 ± 3.0 to 7.7 ± 5.3 μU/mL, \(p < 0.001\)), 30 min (38.8 ± 21.8 to 57.0 ± 32.8 μU/mL, \(p < 0.001\)), 60 min (53.3 ± 38.8 to 76.1 ± 53.5 μU/mL, \(p < 0.001\)), and 90 min (60.4 ± 42.5 to 79.3 ± 60.8 μU/mL, \(p = 0.02\)). The AUC-IRI increased ~1.5 times after surgery (77.3 [51.6-118.0] to 99.3 [64.2-156.2] μU/mL × hr, \(p < 0.001\)).

Insulin secretion and insulin resistance after adrenalectomy

We calculated the indices of insulin secretion and insulin resistance (or sensitivity) using the results of the 75g-OGTT before and after surgery (Fig. 2). The insulinogenic index increased significantly after surgery (0.5 [0.4-0.8] to 0.8 [0.4-1.1], \(p < 0.001\)). On the other hand, the disposition index, the product of measures of insulin secretion and insulin sensitivity, did not change (806.2 [489.4-1,138.9] to 686.6 [479.4-922.1], \(p = 0.25\)). HOMA-IR, an index of hepatic insulin resistance, increased significantly after surgery (1.0 [0.6-1.5] to 1.5 [1.0-2.2], \(p < 0.001\)). In addition, ISI_matsuda, an index of whole body insulin sensitivity, decreased significantly (6.9 [4.5-10.4] to 5.2 [3.4-7.9], \(p < 0.001\)).
**Fig. 1** Plasma glucose and IRI levels before and after adrenalectomy in patients with APA

Plasma glucose levels (A) and IRI levels (C) were measured during 75g-OGTT before and after adrenalectomy. AUC-PG (B) and AUC-IRI (D) were calculated using a trapezoidal method. Data of plasma glucose levels and IRI levels are expressed as the mean ± standard deviation. The box-and-whisker plot shows the median, upper and lower quartiles, and the range of the data. *p < 0.05, ***p < 0.001 vs before surgery. IRI, immunoreactive insulin; OGTT, oral glucose tolerance test; AUC, area under the curve; PG, plasma glucose.

**Fig. 2** Changes in insulin secretion and insulin resistance after adrenalectomy

Using the data of 75g-OGTT, the insulinogenic index (A), disposition index (B), HOMA-IR (C), and ISI_matsuda (D) were calculated. The box-and-whisker plot shows the median, upper and lower quartiles, and the range of the data. HOMA-IR, homeostasis model assessment of insulin resistance.
**Metabolic parameters after adrenalectomy**

We found that HOMA-IR increased and ISI\textsubscript{matsuda} decreased after adrenalectomy, thus we compared metabolic parameters before and after surgery. As shown in Table 1 and Fig. 3, no change in BMI was observed (23.1 [21.4-26.8] kg/m\textsuperscript{2} to 23.0 [21.1-27.2] kg/m\textsuperscript{2}, \(p = 0.11\)). Liver enzymes concentrations (levels of aspartate amino transferase, alanine amino transferase and \(\gamma\)-glutamyltranspeptidase) and lipid profiles (levels of serum triglyceride, high density lipoprotein-cholesterol and low density lipoprotein-cholesterol) were similar before and after surgery.

**Correlations between changes in indices of insulin secretion or insulin resistance and changes in the level of potassium, aldosterone and eGFR**

To clarify which factors contributed to the increases in insulin secretion or insulin resistance, we analyzed correlations with changes in the level of potassium and aldosterone. In addition, we analyzed the correlation with eGFR, as decreased glomerular filtration may decrease the renal clearance of insulin. As shown in Table 2, changes in the AUC-IRI, insulinogenic index, disposition index, HOMA-IR, and ISI\textsubscript{matsuda} showed no correlation with changes in the levels of serum potassium, PAC, urinary aldosterone, or the eGFR.

**Discussion**

In the present study, we found that not only insulin secretion but also insulin resistance increased after adrenalectomy in patients with unilateral APA, resulting in similar glucose levels pre- and post-surgery. The improvement observed in insulin secretion after surgery indicates that aldosterone excess has a negative effect on \(\beta\)-cell function. In addition, the present study showed that insulin resistance also increases after adrenalectomy.

The present study demonstrates that the insulinogenic index and AUC-IRI during 75g-OGTT significantly increased after adrenalectomy. Previous clinical studies have shown controversial results regarding whether treatment of PA improves insulin secretion. Catena \textit{et al.} showed that the AUC-insulin decreased but AUC-glucose remained unchanged during 75g-OGTT after treatment of PA, where treatment included both adrenalectomy and MR-antagonist treatment [6]. As mentioned in the introduction, a recent...
report showed that aldosterone impairs β-cell function, but not through MR [5]. Thus, including MR-antagonist treatment into the analysis may be inadequate for evaluating the effect of aldosterone excess on β-cell function. Fischer et al. showed that first-phase insulin secretion in response to intravenous glucose infusion increased significantly after adrenalectomy, in a study with limited number of patients (n = 9) [7]. Compared with previous studies, the present study, with relatively large number of patients (n = 61), clearly demonstrated that insulin secretion increases after adrenalectomy in patients with unilateral APA.

In this study, we attempted to clarify what factors contributed to increased insulin secretion. It is thought that insulin secretion in patients with PA is impaired through hyperaldosteronism itself; not only through hypokalemia [21, 22]. However, in this study, no significant correlation was observed between changes in the indices of insulin secretion and changes in the level of serum potassium, PAC, or urinary aldosterone. A possible reason is that the number of patients was insufficient to detect a statistically significant correlation. Especially, changes in the levels of PAC and urinary aldosterone demonstrated much variability. Moreover, potassium replacement and use of antihypertensive drugs before surgery may make statistical analysis difficult. Thus, we think that the results in this study do not exclude the possibility of a negative effect of potassium and aldosterone on insulin secretion. Further investigations with larger numbers of patients are required.

We attempted to evaluate insulin resistance before and after adrenalectomy by calculating the indices of HOMA-IR and ISImatsuda. HOMA-IR reflects hepatic insulin resistance in the fasting state. ISImatsuda reflects whole body (including liver, muscle, and fat) insulin sensitivity in both fasting and post-glucose loading states. ISImatsuda was shown to have a high correlation (r = 0.73) with the rate of whole-body glucose disposal during the euglycemic insulin clamp, which is hardly performed in routine medical care [18]. We found that insulin resistance increased after adrenalectomy in patients with APA. Previous studies have shown that patients with PA have increased insulin resistance compared with normotensive controls and patients with essential hypertension [6, 7]. However, it is not well understood whether surgical treatment for PA improves insulin resistance. For example, Catena et al. showed that HOMA-IR declined in the first 6 months after surgical or MR-antagonist treatment for PA, but this decline was not observed at the end of follow-up [6]. Fischer et al. showed that adrenalectomy for APA (n = 9) did not affect the index of insulin resistance, assessed using the insulin clamp method [7]. Two other studies reported that improvements in insulin sensitivity were observed in patients with APA after adrenalectomy, but not in patients with idiopathic adrenal hyperplasia treated with MR-antagonists [23, 24]. The limited number of patients and the different study protocols used may explain the conflicting results.

### Table 2

Correlation between changes in indices of insulin secretion or insulin resistance and changes in the level of potassium, aldosterone, and eGFR

<table>
<thead>
<tr>
<th></th>
<th>Δ Serum potassium</th>
<th>Δ PAC</th>
<th>Δ Urinary aldosterone</th>
<th>Δ eGFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ AUC-IRI</td>
<td>ρ = 0.025 (p = 0.85)</td>
<td>ρ = -0.058 (p = 0.67)</td>
<td>ρ = -0.120 (p = 0.38)</td>
<td>ρ = 0.120 (p = 0.38)</td>
</tr>
<tr>
<td>(n = 59)</td>
<td>(n = 56)</td>
<td>(n = 55)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td>Δ Insulinogenic index</td>
<td>ρ = 0.082 (p = 0.55)</td>
<td>ρ = 0.056 (p = 0.69)</td>
<td>ρ = 0.044 (p = 0.75)</td>
<td>ρ = 0.013 (p = 0.92)</td>
</tr>
<tr>
<td>(n = 59)</td>
<td>(n = 56)</td>
<td>(n = 55)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td>Δ Disposition index</td>
<td>ρ = 0.044 (p = 0.75)</td>
<td>ρ = 0.054 (p = 0.70)</td>
<td>ρ = 0.07 (p = 0.61)</td>
<td>ρ = -0.161 (p = 0.25)</td>
</tr>
<tr>
<td>(n = 59)</td>
<td>(n = 56)</td>
<td>(n = 55)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td>Δ HOMA-IR</td>
<td>ρ = 0.001 (p = 0.99)</td>
<td>ρ = 0.095 (p = 0.49)</td>
<td>ρ = 0.013 (p = 0.93)</td>
<td>ρ = 0.169 (p = 0.22)</td>
</tr>
<tr>
<td>(n = 59)</td>
<td>(n = 56)</td>
<td>(n = 55)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td>Δ ISImatsuda</td>
<td>ρ = -0.137 (p = 0.30)</td>
<td>ρ = 0.045 (p = 0.74)</td>
<td>ρ = -0.220 (p = 0.11)</td>
<td>ρ = 0.016 (p = 0.90)</td>
</tr>
<tr>
<td>(n = 59)</td>
<td>(n = 56)</td>
<td>(n = 55)</td>
<td>(n = 55)</td>
<td></td>
</tr>
</tbody>
</table>

Spearman correlation coefficient (ρ) were used to evaluate correlations between variables. PAC, plasma aldosterone concentration; eGFR, estimated glomerular filtration rate; AUC, areas under the curve; IRI, immunoreactive insulin; HOMA-IR, homeostasis model assessment of insulin resistance.
The reason for increases in insulin resistance in the present study is unknown. In this study, no significant changes were observed in the metabolic factors, such as body mass index, lipid profile, and liver enzymes before and after adrenalectomy. A possible reason is that decreased blood flow into muscle induced increases in insulin resistance. In this study, systolic and diastolic blood pressure in the participants decreased dramatically after adrenalectomy. Decreased blood pressure may impair the blood flow into muscle. Skeletal muscle insulin-mediated glucose uptake is equal to the product of arteriovenous glucose difference across and blood flow into muscle [25, 26]. Hence, it is possible that decreased blood pressure induced impaired glucose uptake into muscle. However, this is only hypothesis because there is no study that examined whether acute change in blood pressure affect insulin resistance in muscle. Another possible reason is that insulin resistance increases as a compensatory reaction against increased insulin secretion, in order to maintain glucose homeostasis, by unknown mechanisms.

The disposition index, the product of measures of insulin secretion and insulin sensitivity, did not change after adrenalectomy in the present study. This result means that glucose homeostasis was maintained before and after surgery. It is difficult to determine which change occurs first after adrenalectomy; an increase in insulin secretion or increase in insulin resistance. An increases in insulin secretion may be just as a result of compensatory reaction to increase in insulin resistance. However, taking into consideration previous reports that show aldosterone excess suppresses insulin secretion in vivo and in vitro, it is plausible that improved insulin secretion is as a result of not only compensatory reaction but also improvement in aldosterone excess.

There were several limitations to this study. First, insulin resistance was assessed by the indices of HOMA-IR or ISI_matsuda, whereas the gold standard in analysis of insulin resistance is the insulin clamp method. Second, the number of participants was small, despite it being greater than in previous studies. In this study, no statistically significant relationships were observed between changes in indices of insulin secretion or insulin resistance and changes in the levels of potassium and aldosterone. This result may have been caused by insufficient statistical power because of the limited number of patients. Third, a 75g-OGTT was performed before and only one year after adrenalectomy. By assessing the change in insulin secretion and insulin resistance at early time after adrenalectomy, it may be possible to clarify which is the first change after adrenalectomy; an increase in insulin secretion or increase in insulin resistance.

In conclusion, our data demonstrate that insulin secretion increases after adrenalectomy in patients with unilateral APA. However, plasma glucose levels remained unchanged, by parallel increase in insulin resistance. These findings provide new insights into the relationship between aldosterone and glucose metabolism.

Acknowledgement

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Disclosure

None of the authors have any potential conflicts of interest associated with this research.

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