Impact of Microalbuminuria on Incident Stroke  
A Meta-Analysis

Meng Lee, MD; Jeffrey L. Saver, MD; Kuo-Hsuan Chang, MD; Hung-Wei Liao, MD;  
Shen-Chih Chang, PhD; Bruce Ovbiagele, MD, MS

Background and Purpose—Microalbuminuria, a marker of both kidney disease and endothelial dysfunction, may be associated with global vascular risk, but the nature and magnitude of the link between microalbuminuria and incident stroke has not been clearly defined. The purpose of this study was to assess the consistency and strength of the association of microalbuminuria with risk of stroke in prospective studies using meta-analysis.

Methods—We conducted a systematic search of electronic databases and bibliographies for studies reporting a multivariate-adjusted estimate, represented as relative risk with 95% CI, of the association between microalbuminuria and stroke risk. Studies were excluded if a majority of study participants had established kidney disease or pre-eclampsia. Estimates were combined using a random-effect model.

Results—We identified 12 studies, with a total of 48 596 participants and 1263 stroke events. Overall, presence of microalbuminuria was associated with greater stroke risk (relative risk, 1.92; 95% CI, 1.61 to 2.28; \( P < 0.001 \)) after adjustment for established cardiovascular risk factors. There was evidence of significant heterogeneity in the magnitude of the association across studies (\( P \) for heterogeneity <0.001, \( I^2 = 68\% \)), which was partially explained by differences in study population, microalbuminuria definition, and different microalbuminuria-related risk among stroke subtypes. However, in stratified analyses, microalbuminuria was associated with increased risk of subsequent stroke in all subgroups (general population, diabetics, those with known stroke).

Conclusions—Microalbuminuria is strongly and independently associated with incident stroke risk. Future studies should explore whether microalbuminuria is just a risk marker or a modifiable risk factor for stroke.  

Key Words: endothelial dysfunction ■ incidence ■ meta-analysis ■ microalbuminuria ■ stroke

O

ver the last 4 decades, several prospective clinical studies have identified a series of independent risk factors for symptomatic vascular events, including stroke.\(^1\)\(^2\) However, a better understanding of the multifactorial pathogenesis of atherosclerosis, the underlying entity behind most vascular events, and the fact that many of these events occur in persons who do not harbor conventional vascular risk factors has prompted a search for novel risk factors for prediction of cardiovascular disease.\(^3\) Nonetheless, the clinical value of many of these emerging risk factors remains uncertain largely due to inconsistency of data, paucity of prospective studies, or lack of evidence that their predictive ability is independent of conventional risk factors.\(^3\)

One such emerging vascular risk factor is microalbuminuria.\(^4\) Microalbuminuria is generally defined as a urinary albumin excretion rate (or albumin excretion rate) of 30 to 299 mg/day or an albumin:creatinine ratio of 2.5 to 25 mg/mmol in men and 3.5 to 25 mg/mmol in women.\(^4\) Although often seen as a sign of early kidney disease (ie, impairment in glomerular filtration barrier), microalbuminuria interacts with several conventional vascular risk factors and is an independent marker of endothelial dysfunction.\(^4\)

Indeed, it is thought that assessing kidney structure using this relatively simple test could be a window to the systemic vasculature, that is, leaky renal vessels reflecting the permeability of the vasculature in general and an individual’s susceptibility to target organ damage.\(^5\)

There is convincing evidence of an independent positive relationship between overt proteinuria and stroke risk,\(^6\) but the nature and magnitude of the link between microalbuminuria and incident stroke has so far not been systematically investigated. In this study, we aimed to assess the consistency and strength of the association of microalbuminuria with risk of stroke in prospective cohort studies using meta-analysis.

Methods

Literature Search

The search strategy was conducted according to the recommendations of the Meta-analysis of Observational Studies in Epidemiology.\(^7\) We performed a systematic search of PubMed (1966 to October 2009), EMBASE (1947 to October 2009), the Cochrane library (including CENTRAL), MEDLINE, and LILACS using the search strategy:
“proteinuria” or “albuminuria” or “microalbuminuria” or “macroalbuminuria” crossed with “stroke” or “cerebrovascular disease” or “cerebrovascular attack” or “cerebral ischemia” or “brain ischemia” or “intracranial hemorrhage.” We restricted the search to human studies. There were no language restrictions. Manual searches of bibliographies of all relevant studies and review articles were reviewed and identified by 2 investigators (M.L. and K.H.C.).

Study Selection and Data Abstraction

Studies were selected if they met the following entry criteria: (1) prospectively collected data obtained within cohort studies or clinical trials; (2) reported quantitative estimates of the multivariate-adjusted relative risk (RR) and 95% CI for stroke associated with microalbuminuria; and (3) follow-up duration was at least 1 year. Studies were excluded if (1) the study design was cross-sectional, case–control, or retrospective cohort studies; (2) the majority of the participants had chronic kidney disease (ie, estimated glomerular filtration rate <60 mL/min/1.73 m², kidney transplant, Fabry disease, eclampsia, or pre-eclampsia; (3) only reported unadjusted or age-and sex-adjusted RR; and (4) did not report 95% CI. The prespecified definitions of microalbuminuria are listed in Table 1. Studies that used slightly varying definitions were included if they were otherwise comparable. All data from eligible studies were abstracted independently by 2 investigators (M.L. and K.H.C.). Discrepancies were resolved by discussion with a third investigator (B.O.) and by referencing the original report.

Assessment of Study Quality

We assessed quality of all articles that met the selection criteria with the following 8 characteristics: (1) prospective study design; (2) maintenance of comparable groups; (3) adjustment of potential confounders; (4) documented loss of follow-up rate; (5) outcome assessed blind to exposure status; (6) clear and proper definition of exposures (microalbuminuria) and outcomes (stroke); (7) temporality (microalbuminuria measured at baseline, not at time of outcomes assessment) and (8) follow-up of at least 1 year. Studies were graded as good quality if they met 6 to 8 criteria; fair if they met 3 to 5; and poor if they met <3 criteria.

Statistical Analysis

Data analysis used multivariate-adjusted outcome data (expressed as RRs and 95% CIs). We converted these values in every study by using their natural logarithms, and the SEs were calculated from these logarithmic numbers and their corresponding 95% CIs. The statistical analysis used the inverse variance approach to combine log RRs and SEs. We used a random-effect model and explored for heterogeneity in the magnitude of the association across studies (P for heterogeneity <0.001, I²=68%). The exclusion of any single study from the analysis did not alter the overall finding in a sensitivity test (data not shown). There was mild asymmetrical appearance lacking studies on the left lower part of the funnel plot (Figure 3). The RR from a fixed-effect model was 1.82 (95% CI, 1.65 to 1.99; P<0.001).

Table 3 shows microalbuminuria was associated with greater subsequent stroke risk (RR, 1.92; 95% CI 1.61 to 2.28; P<0.001) after adjustment for established cardiovascular risk factors (Figure 2). There was evidence of significant heterogeneity in the magnitude of the association across studies (P for heterogeneity <0.001, I²=68%). The exclusion of any single study from the analysis did not alter the overall finding in a sensitivity test (data not shown). There was mild asymmetrical appearance lacking studies on the left lower part of the funnel plot (Figure 3). The RR from a fixed-effect model was 1.82 (95% CI, 1.65 to 1.99; P<0.001).

Results

The literature review identified 160 full articles for detailed assessment, among which 124 were excluded for no stroke estimate, 6 for no adjusted estimate or only age-and sex-adjusted estimate, and 18 for only proteinuria and stroke risk estimate. Our final primary analysis included 12 prospective cohort studies12–23 with 13 estimates because 1 study reported diabetes mellitus (DM) and non-DM population separately (Figure 1).16 The study characteristics are shown in Table 2. There were a total 48 596 participants and 1263 stroke events in the current meta-analysis. Among 13 estimates, 6 derived from the general population,15–18,22,23 5 from a Type 2 DM population,13,14,16,19,20 and 2 from a population with a stroke history.12,21 Nine studies were from a white-dominant population12–19,22 2 from an Asian population,20,21 and 1 from an American Indian population.23 The participant number ranged from 37021 to 23 433.22 The follow-up duration ranged from 1.1 years21 to 13.4 years.23 Three of the 12 studies used UACR12,16,19 for microalbuminuria measurement, whereas 9 used UACR.13–15,17,18,20–23 Eleven studies reported fatal plus nonfatal stroke as a primary end point, whereas 1 reported fatal stroke as a primary end point.19 Three studies used ischemic stroke as a primary end point15,18,20 and others used all reported stroke as a primary end point.12–14,16,17,19,21–23 Overall quality of studies was good (median, 6; range, 5 to 7).

Overall, presence of microalbuminuria was associated with greater subsequent stroke risk (RR, 1.92; 95% CI 1.61 to 2.28; P<0.001) after adjustment for established cardiovascular risk factors (Figure 2). There was evidence of significant heterogeneity in the magnitude of the association across studies (P for heterogeneity <0.001, I²=68%). The exclusion of any single study from the analysis did not alter the overall finding in a sensitivity test (data not shown). There was mild asymmetrical appearance lacking studies on the left lower part of the funnel plot (Figure 3). The RR from a fixed-effect model was 1.82 (95% CI, 1.65 to 1.99; P<0.001).

Table 3 shows microalbuminuria was associated with increased risk of subsequent stroke in all subgroups when we stratified the estimates by population, study type, ethnicity, microalbuminuria prevalence at entry, follow-up duration, participant number, measurement methods of microalbuminuria, and end point. Significant heterogeneity was found between the DM population and the population with a stroke.
history (RR, 1.70; 95% CI, 1.43 to 2.04 versus RR, 2.92; 95% CI, 1.12 to 7.64; P for heterogeneity among groups = 0.04, $I^2=77\%$). Also, the association with ischemic stroke risk (RR, 2.21; 95% CI, 1.44 to 3.38) was significantly higher than hemorrhagic stroke risk (RR, 1.03; 95% CI, 0.68 to 1.55) and unspecified stroke risk (RR, 1.66; 95% CI, 1.48 to 1.87; P for heterogeneity among groups <0.01, $I^2=82\%$). Heterogeneity was noted for studies, which completely (RR, 1.61; 95% CI, 1.43 to 1.81) versus partially (RR, 2.26; 95% CI, 1.66 to 3.07; P for heterogeneity among groups <0.001, $I^2=91\%$) corresponded to our prespecified microalbuminuria definition.

Discussion

In this meta-analysis of 12 prospective cohort studies, among almost 49 000 individuals experiencing >1200 stroke events, we found that persons with baseline microalbuminuria have a risk of future stroke that is approximately 90% greater than those without microalbuminuria. This relationship was consistent across diverse population subgroups (ie, general populations, diabetic populations, and populations with a stroke history). Furthermore, the size and inclusion of only prospective data strengthen the robustness of our findings, because issues of selection bias, recall bias, and reverse causality are unlikely. In addition, all studies included in our meta-analysis reported a multivariate-adjusted RR, which probably mitigated the possibility of known confounding influencing our results.

We observed that the impact of microalbuminuria was greatest in the population with a history of stroke and relatively modest in the diabetic population. It is conceivable that this distinction is because among the diabetics, microalbuminuria may have more likely been a reflection of “earlier stage” nephropathy and not necessarily widespread vascular disease that could have precipitated future strokes. On the other hand, microalbuminuria among those with prior cerebrovascular bed damage (stroke) may have signified persons with extensive interaction of vascular factors and vascular instability at highest risk for further cerebrovascular events.

We found microalbuminuria had the greatest impact on ischemic stroke, which would be expected given the aforementioned postulated underlying mechanisms for this association, followed by unspecified stroke type, and then an almost neutral effect on hemorrhagic stroke, further underscoring the likelihood that atherosclerosis might be the pathological link between microalbuminuria and stroke.

Our meta-analysis, based on observational studies, cannot prove causality and mechanistically it is unclear how albuminuria would directly cause stroke. However, there was some evidence implying that stroke risk might be reduced when we reduced microalbuminuria. A study showed that short-term reduction in albuminuria after initiation of blood pressure-lowering treatment has been associated with lower long-term stroke risk. Among various blood pressure-lowering agents, for a similar level of attained blood pressure, modulators of the renin–angiotensin system lower urine albumin excretion or decrease incidence of new-onset microalbuminuria more effectively than other agents among patients with diabetes. In fact, among microalbuminuric subjects, treatment with fosinopril had a significant effect on lessening urinary albumin excretion and was associated with a trend in reducing cardiovascular events, results that could not completely be attributed to the reduction in arterial blood pressure. Future studies are needed to investigate whether limiting urinary albumin excretion with angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers may reduce cardiovascular events, including strokes, beyond their blood pressure-lowering effects.

Compared with studies whose definitions completely corresponded to our prespecified microalbuminuria definition, those that only partially did so appeared to have a stronger link to stroke risk. Most of the studies in the latter category did not set an upper limit of UACR, which means participants with macroalbuminuria were also included. As such, it is not surprising that stroke risk was higher with data likely comprising the entire albuminuria range (microalbuminuria + macroalbuminuria).
Table 2. Study Characteristics

<table>
<thead>
<tr>
<th>Study Design and Population</th>
<th>Study</th>
<th>Country</th>
<th>No. of Participants (Women, %)</th>
<th>No. of Patients</th>
<th>Prevalence of Microalbuminuria at Entry</th>
<th>No. of Stroke</th>
<th>Age, Mean±SD, Years</th>
<th>Follow-Up, Years</th>
<th>Definition of Microalbuminuria</th>
<th>Adjusted Variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke cohort from a hospital</td>
<td>Beamer, 1999&lt;sup&gt;12&lt;/sup&gt;</td>
<td>USA</td>
<td>121 (11)</td>
<td>21%</td>
<td>26</td>
<td>62±8</td>
<td>1.5</td>
<td>All recurrent stroke</td>
<td>UAC 20–200 mg/L</td>
<td>HTN, DM, smoking status</td>
</tr>
<tr>
<td>Type 2 diabetic cohort, no stroke history at entry</td>
<td>Gillett, 2003&lt;sup&gt;13&lt;/sup&gt;</td>
<td>Australia</td>
<td>1083 (52)</td>
<td>40%</td>
<td>89</td>
<td>66±10</td>
<td>4.9</td>
<td>All stroke</td>
<td>UACR ≥3.0 mg/mmol</td>
<td>Age, sex, BMI, waist circumference, diabetes duration, glycemic control, BP, BP treatment, lipid-lowering therapy, aspirin, atrial fibrillation/flutter, smoking, alcohol, exercise status, and carotid bruit status</td>
</tr>
<tr>
<td>Type 2 diabetic cohort without CVD history at entry</td>
<td>Hitman, 2000&lt;sup&gt;19&lt;/sup&gt;</td>
<td>UK</td>
<td>2838 (32)</td>
<td>24%</td>
<td>60</td>
<td>62±8</td>
<td>3.9</td>
<td>All stroke</td>
<td>UACR &gt;2.5 mg/mmol or an albumin excretion rate on timed collection ≥20 μg/min</td>
<td>Age, sex, current smoking, DM, HTN, AF, LVEF &lt;50%, LVH, total cholesterol, and creatinine</td>
</tr>
<tr>
<td>Population-based cohort without CVD history at entry</td>
<td>Kistorp, 2004&lt;sup&gt;22&lt;/sup&gt;</td>
<td>Denmark</td>
<td>537 (58)</td>
<td>NR</td>
<td>21</td>
<td>68±11</td>
<td>5</td>
<td>Ischemic stroke</td>
<td>UACR &gt;18.4 mg/g</td>
<td>Age, sex, retinopathy, proteinuria, DM duration, systolic BP, HbA1C, atorvastatin use</td>
</tr>
<tr>
<td>Nondiabetic cohort, stroke history at entry was 1%</td>
<td>Miettinen, 1996a&lt;sup&gt;16&lt;/sup&gt;</td>
<td>Finland</td>
<td>1375 (52)</td>
<td>NR</td>
<td>30</td>
<td>58±0.2</td>
<td>7</td>
<td>All stroke</td>
<td>UAC 150–299 mg/L</td>
<td>Sex, area, age, history of stroke, total cholesterol, LDL cholesterol, TG, smoking, HTN</td>
</tr>
<tr>
<td>Type 2 diabetic cohort, stroke history at entry was 6%</td>
<td>Miettinen, 1996b&lt;sup&gt;16&lt;/sup&gt;</td>
<td>Finland</td>
<td>1056 (45)</td>
<td>NR</td>
<td>125</td>
<td>58±0.2</td>
<td>7</td>
<td>All stroke</td>
<td>UAC 150–299 mg/L</td>
<td>Sex, area, age, history of stroke, total cholesterol, LDL cholesterol, TG, smoking, HTN</td>
</tr>
<tr>
<td>HTN, nondiabetic cohort with all received ACEI, stroke history at entry was 2%</td>
<td>Schrader, 2006&lt;sup&gt;17&lt;/sup&gt;</td>
<td>Germany</td>
<td>2582 (62)</td>
<td>32%</td>
<td>15</td>
<td>63±8</td>
<td>3.5</td>
<td>All stroke</td>
<td>UAC 20–300 mg/g</td>
<td>Age, sex, HF, CHD, MI, hyperlipidemia, hyperuricemia</td>
</tr>
<tr>
<td>Population-based, nondiabetic cohort, no CVD history at entry</td>
<td>Solbu, 2009&lt;sup&gt;18&lt;/sup&gt;</td>
<td>Norway</td>
<td>5215 (49)</td>
<td>33%</td>
<td>225</td>
<td>60±10</td>
<td>9.7</td>
<td>Ischemic stroke</td>
<td>UACR 0.75–30 mg/mmol</td>
<td>Age, sex, metabolic syndrome, current smoking, hard physical activity ≥1 hour per week, eGFR</td>
</tr>
<tr>
<td>Population-based, older-onset diabetic cohort</td>
<td>Valmadrid, 2000&lt;sup&gt;19&lt;/sup&gt;</td>
<td>USA</td>
<td>668 (57)</td>
<td>31%</td>
<td>65</td>
<td>67±11</td>
<td>12</td>
<td>Fatal stroke</td>
<td>UAC 30–299 mg/L</td>
<td>Age, sex, glycemic control, insulin use, alcohol intake, physical activity, history of CVD, intake of anti-HTN agents, presence and severity of diabetic retinopathy</td>
</tr>
<tr>
<td>Type 2 diabetic clinic-based cohort, no stroke history at entry</td>
<td>Yang, 2008&lt;sup&gt;20&lt;/sup&gt;</td>
<td>China</td>
<td>5403 (55)</td>
<td>25%</td>
<td>91</td>
<td>57 (interquartile range, 46–67 years)</td>
<td>5.4</td>
<td>Ischemic stroke</td>
<td>UACR 2.5–25 mg/mmol in men and 3.5–25 mg/mmol in women</td>
<td>Age, sex, smoking status (current and former), HTN, BMI, LDL cholesterol, duration of DM, eGFR, and use of drugs at baseline (lipid-lowering drugs, oral antidiabetic drugs, and insulin)</td>
</tr>
<tr>
<td>Ischemic stroke cohort from a hospital</td>
<td>Yokota, 2009&lt;sup&gt;21&lt;/sup&gt;</td>
<td>Japan</td>
<td>370 (34)</td>
<td>NR</td>
<td>49</td>
<td>70±11</td>
<td>1.1</td>
<td>All recurrent stroke</td>
<td>UAC 20–300 mg/g</td>
<td>Sex, DM, high BP (factors with P&lt;0.05 in univariate analysis)</td>
</tr>
<tr>
<td>Population-based cohort, no stroke history at entry</td>
<td>Yuyun, 2004&lt;sup&gt;22&lt;/sup&gt;</td>
<td>UK</td>
<td>23433 (53)</td>
<td>12%</td>
<td>237</td>
<td>58±9</td>
<td>7.2</td>
<td>All stroke and stroke subtypes</td>
<td>UACR 2.5–25 mg/mmol</td>
<td>Age, sex, smoking, hypertension treatment, systolic BP, total cholesterol, DM, BMI, physical activity, family history of stroke, and baseline CHD</td>
</tr>
<tr>
<td>Population-based cohort, stroke history at entry was 1%</td>
<td>Zhang, 2008&lt;sup&gt;23&lt;/sup&gt;</td>
<td>USA (American Indians)</td>
<td>3915 (60)</td>
<td>22%</td>
<td>228</td>
<td>57±9</td>
<td>13.4</td>
<td>All stroke</td>
<td>UAC 30–299 mg/L</td>
<td>Age, sex, systolic and diastolic BP, BMI, waist circumference, LDL and HDL cholesterol, TG, physical activity, smoking, alcohol use, and fasting glucose</td>
</tr>
</tbody>
</table>

CVD indicates cardiovascular disease; ACEI, angiotensin-converting enzyme inhibitor; NR, not reported; BMI, body mass index; BP, blood pressure; AF, atrial fibrillation; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; HDL, high-density lipoprotein; TG, triglycerides; HF, heart failure; CHD, coronary heart disease; MI, myocardial infarction; eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein.
The current systematic review found the prevalence of microalbuminuria ranged from 12% to 40%. The high prevalence rate of microalbuminuria raises a question whether it is cost-effective to screen microalbuminuria in diabetic and general populations. Health economic analyses have shown that screening for albuminuria in the Type 2 diabetic patients and subsequent initiation of angiotensin II antagonists treatment in those found positive contributed to better outcomes, including reduction of cardiovascular event, and may represent an excellent value. On the other hand, some have argued that simply treating all middle-aged diabetic patients with angiotensin-converting enzyme inhibitors is a simple strategy that provides additional benefit at modest additional cost. For the general population, a strategy of annual dipstick screening for gross proteinuria with follow-up testing and treatment with an angiotensin-converting enzyme inhibitor may not be cost-effective with regard to slowing progression of kidney disease or decreasing mortality. This is because the yield is so low due to the low prevalence (<1%) of gross proteinuria in the general population. However, screening for the general population for microalbuminuria and subsequently treating those found positive with fosinopril may be more cost-effective compared with no screening and adopting an ordinary healthcare perspective given the substantially higher rates of microalbuminuria in the general population.

Some limitations need to be mentioned. First, meta-analyses can be constrained by comprehensiveness of searches, methodological rigor of the included studies, and publication bias, especially when the meta-analysis was conducted of epidemiological studies rather than randomized controlled trials. Second, the studies varied with respect to the characteristics of participants, definition of stroke in outcome assessment, follow-up duration, among others and indeed, heterogeneity was found by formal analysis. Still, our sensitivity analysis showed that removing any 1 study did not alter the main meta-analysis findings. Finally, there was evidence of a publication bias as...
seen in the funnel plot (Figure 3). Some studies did not report RR with 95% CI when an insignificant result was found after adjusting for known cardiovascular risk factors and, as such, could not be included in our meta-analysis. This issue probably resulted in an overestimation of the association between microalbuminuria and stroke risk. However, when we exclude 3 estimates with larger SEs, the overall RR between microalbuminuria and stroke risk decreased to 1.61 (95% CI, 1.39 to 1.85).

In conclusion, our formal meta-analysis found a significant and strong association between microalbuminuria and subsequent stroke risk across various population subtypes after adjustment of established cardiovascular risk factors. Future studies, preferably randomized controlled studies of agents that lower or prevent microalbuminuria, should explore whether microalbuminuria is just a risk marker or a potentially modifiable risk factor for stroke.

### Acknowledgments

We thank Yueh Lee, MS, for article retrieval.

### Sources of Funding

Supported by CMPRG 660311, Taiwan (M.L.), National Institutes of Health Specialized Program for Translational Research in Acute Stroke (SPOTRIAS; J.L.S.), and University of California–Los Angeles RCMAR under National Institutes of Health/National Institute on Aging Grant P30-AG021684 (B.O.).

### Disclosures

J.L.S. has received honoraria from universities as a visiting professor; is an employee of the University of California, which holds a patent on retriever devices for stroke; is a scientific consultant regarding trial design and conduct to Concentric Medical, Talecris, and Ev3; is a site investigator in multicenter trials sponsored by Lundbeck for which the UC Regents received payments based on the clinical trial contracts for the number of subjects enrolled; is a site investigator in the National...
Institutes of Health Interventional Management of Stroke (IMS) 3 and Combination Therapy of rt-PA and Epifibatide to Treat Acute Ischemic Stroke (CLEAR-ER) multicenter clinical trials for which the UC Regents receive payments based on the clinical trial contracts for the numbers of subjects enrolled; has declined consulting/honoraria monies from Genentech since 2002; and is funded by National Institutes of Health–National Institute of Neurological Disorders and Stroke Awards P50 NS044378 and U01 NS 44364.

References
Impact of Microalbuminuria on Incident Stroke: A Meta-Analysis
Meng Lee, Jeffrey L. Saver, Kuo-Hsuan Chang, Hung-Wei Liao, Shen-Chih Chang and Bruce Ovbiagele

Stroke. 2010;41:2625-2631; originally published online October 7, 2010;
doi: 10.1161/STROKEAHA.110.581215

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/41/11/2625

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2013/10/02/STROKEAHA.110.581215.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/
Abstract

Impact of Microalbuminuria on Incident Stroke — A Meta-Analysis

Meng Lee, MD1,2; Jeffrey L. Saver, MD1; Kuo-Hsuan Chang, MD3; Hung-Wei Liao, MD4; Shen-Chih Chang, PhD5; Bruce Ovbiagele, MD, MS1

1Stroke Center and Department of Neurology, University of California, Los Angeles, 2Department of Neurology, Chang Gung Memorial Hospital at Chiayi, and 3Linkou, Chang Gung University College of Medicine, Taiwan; Ching-Ten Clinic, Taiwan; 4Department of Epidemiology, School of Public Health, University of California, Los Angeles, Calif.

Stroke 2010; 41: 2625-2631