

# Estimation of Global and Regional Incidence and Prevalence of Abdominal Aortic Aneurysms 1990 to 2010

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## ABSTRACT

The global burden of abdominal aortic aneurysm (AAA) has not been studied previously. Such information is important given the emergence of cardiovascular diseases in developing countries. We conducted a systematic literature review and estimated the global and regional incidence and prevalence of AAA in 21 world regions by age and sex. The search for prevalence and incidence of AAA using standard clinical and epidemiological terms was conducted using MEDLINE (1950 to 2010), EMBASE (1980 to 2010), AMED (1985 to 2010), CINAHL (1982 to 2010), and LILACS (2008 to 2010). Data abstracted from the systematic review served as priors for Bayesian meta-regression analyses. The analysis drew from 26 high-quality studies to estimate AAA prevalence and incidence. In 1990, the global age-specific prevalence rate per 100,000 ranged from 8.43 (95% CI: 7.03 to 10.14) in the 40 to 44 years age group to 2,422.53 (95% CI: 2,298.63 to 2,562.25) in the 75 to 79 years age group; the corresponding range in 2010 was 7.88 (95% CI: 6.54 to 9.59) to 2,274.82 (95% CI: 2,149.77 to 2,410.17). Prevalence was higher in developed versus developing nations, and the rates within each development stratum decreased between 1990 and 2010. Globally, the age-specific annual incidence rate per 100,000 in 1990 ranged from 0.89 (95% CI: 0.66 to 1.17) in 40 to 44 years age group to 176.08 (95% CI: 162.72 to 190.28) in the 75 to 79 years age group. In 2010, this range was 0.83 (95% CI: 0.61 to 1.11) to 164.57 (95% CI: 152.20 to 178.78). The highest prevalence in 1990 was in Australasia and North America high income regions: 382.65 (95% CI: 356.27 to 410.88) and 300.59 (95% CI: 280.93 to 321.54), respectively. Australasia had the highest prevalence in 2010, although the prevalence decreased to 310.27 (95% CI: 289.01 to 332.94). Regional prevalence increased in Oceania, tropical Latin America, Asia Pacific high income, Southern Sub-Saharan Africa (SSA), Central SSA, South Asia, Western SSA, and Central Asia. AAA global prevalence and incidence rates have decreased over the last 20 years. However, rising rates in some regions highlight the need for policies to enhance global disease surveillance and prevention.

Abdominal aortic aneurysm (AAA) is a focal dilation of the abdominal aorta of at least 1.5 times the normal diameter or an absolute value of 3.0 cm or greater [1]. Known risk factors for AAA include male sex, atherosclerosis, smoking, hypertension, and history of AAA in a first-degree relative [2–4]. During the 20th century, many developed nations reported a consistent increase in the incidence and mortality associated with AAA [5–9]. The reported overall prevalence in developed countries was approximately 5%, and it was shown to be 4 times higher in men than in women [10,11], although women diagnosed with AAA appear to have a worse prognosis than men [12,13]. AAA mortality is greatest among those previously undiagnosed who may present with rupture; such

individuals have up to 90% mortality rate if rupture occurs outside of the hospital [2,3,14–16].

The observed adverse epidemiological trends [5–9] led to targeted screening efforts as an important component of the management of AAA, and the benefit of screening was supported by systematic evidence review and synthesis of data from randomized controlled trials [17]. In this context, recent reports on the basis of data over the past 2 decades suggest that the mortality associated with AAA has declined in the United States, England, Wales, Australia, New Zealand, and Sweden [18–22]. However, these country-level reports from developed nations are not enough to provide reliable global estimates of incidence and prevalence of AAA. To aid in the planning of policies and programs for the global

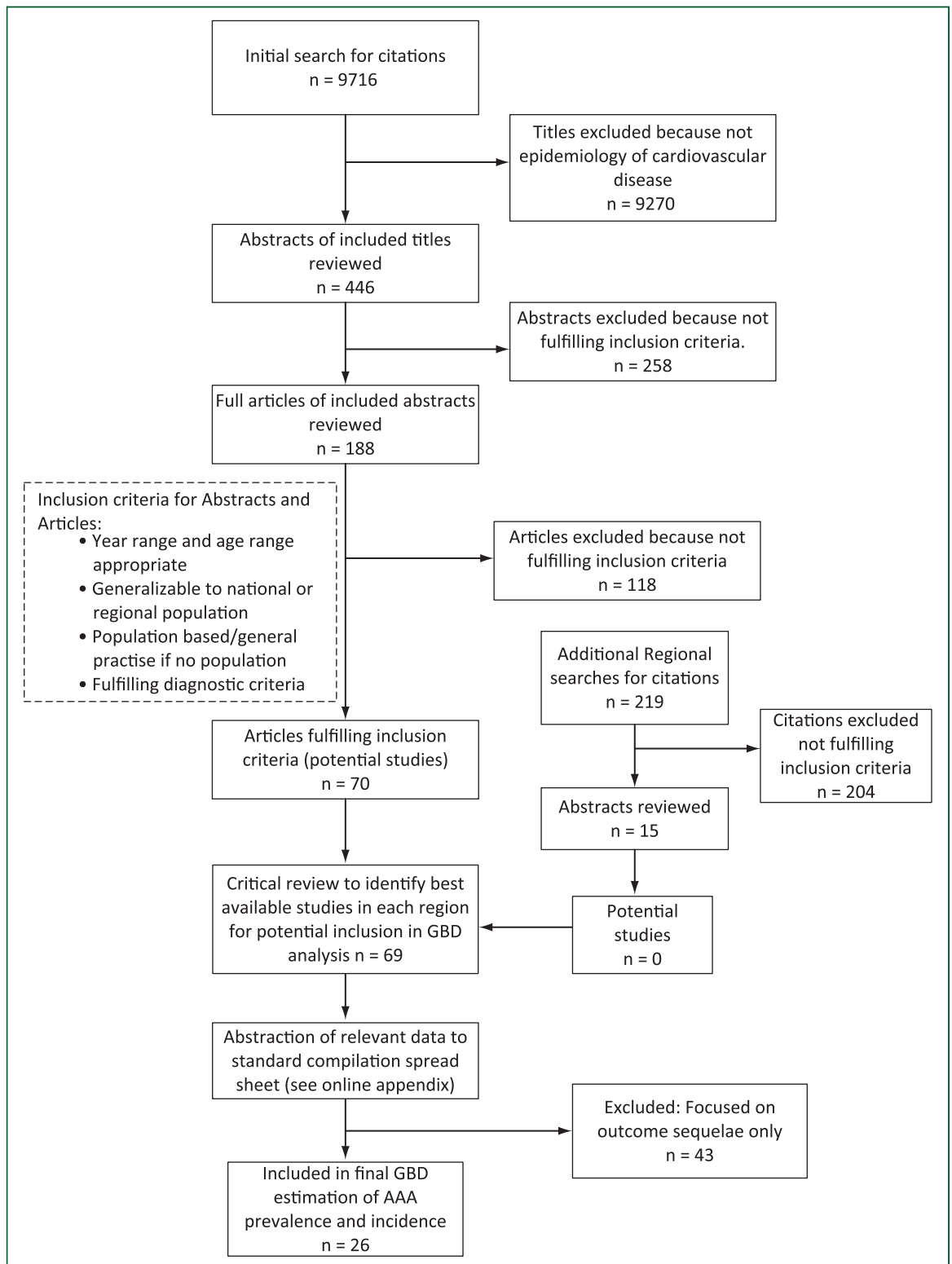
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**FIGURE 1. Literature review and data abstraction flow chart for AAA systematic analysis.** Abdominal aortic aneurysm (AAA) studies included the definition of important clinical sequelae for AAA, which were intact AAA (infra renal aortic diameter  $\geq 30$  mm), fatal ruptured AAA, emergency surgical repair for ruptured AAA, and elective surgical repair of AAA (open or endovascular). GBD, global burden of disease.

**TABLE 1.** Age-specific AAA prevalence rates per 100,000 population by country development status, 1990 and 2010

Region	Age group, yrs											
	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	≥80
<b>1990</b>												
Developed												
Estimate	2.71	3.60	5.90	10.16	28.69	156.51	440.16	894.50	1,501.89	2,171.32	2,666.91	3,404.25
CI lower	2.03	2.72	4.74	8.09	25.04	145.54	411.89	849.48	1,429.27	2,068.36	2,536.17	3,243.10
CI upper	3.66	4.85	7.39	12.73	33.42	169.34	472.52	943.63	1,579.25	2,281.32	2,799.43	3,579.85
Developing												
Estimate	2.04	2.78	4.62	7.70	21.07	117.95	332.82	685.27	1,165.47	1,709.27	2,190.06	2,882.98
CI lower	1.55	2.10	3.62	6.01	17.79	105.43	298.04	621.62	1,060.83	1,547.18	1,995.39	2,621.50
CI upper	2.73	3.78	6.03	10.04	25.06	131.86	373.11	754.73	1,286.28	1,893.16	2,413.17	3,158.70
Global												
Estimate	2.19	2.98	4.95	8.43	23.33	130.46	368.44	764.26	1,301.44	1,890.59	2,422.53	3,174.85
CI lower	1.75	2.39	4.10	7.03	20.80	121.21	343.78	720.75	1,227.29	1,779.46	2,298.63	3,021.94
CI upper	2.81	3.84	6.10	10.14	26.35	140.66	396.87	812.47	1,379.98	2,013.09	2,562.25	3,339.34
<b>2010</b>												
Developed												
Estimate	2.43	3.24	5.31	9.06	25.71	142.12	397.73	827.97	1,402.69	1,943.94	2,478.62	3,158.28
CI lower	1.83	2.42	4.26	7.10	22.34	131.78	370.37	783.24	1,336.95	1,852.43	2,361.96	3,006.07
CI upper	3.14	4.23	6.64	11.56	30.11	153.14	424.92	871.27	1,474.43	2,038.61	2,603.68	3,314.10
Developing												
Estimate	1.94	2.63	4.43	7.59	20.66	116.63	333.64	678.22	1,146.26	1,654.68	2,139.31	2,836.16
CI lower	1.50	2.01	3.53	5.96	17.64	104.44	296.61	613.62	1,042.59	1,497.68	1,943.86	2,599.24
CI upper	2.60	3.49	5.62	9.66	24.39	130.72	376.47	749.21	1,261.74	1,820.22	2,350.28	3,103.74
Global												
Estimate	2.02	2.74	4.60	7.88	21.85	123.25	350.94	725.66	1,229.12	1,761.18	2,274.82	3,002.78
CI lower	1.62	2.17	3.79	6.54	19.42	113.63	322.47	677.33	1,154.16	1,658.85	2,149.77	2,861.13
CI upper	2.57	3.47	5.66	9.59	24.77	133.98	383.45	776.35	1,312.17	1,871.27	2,410.17	3,150.92

AAA, abdominal aortic aneurysm.

prevention and management of AAA, comprehensive epidemiological surveillance is warranted, especially in light of the current worldwide epidemiological transition and emergence of cardiovascular diseases in developing countries. We conducted a systematic analysis of all the evidence available in the public domain to identify high-quality studies of AAA epidemiology. Using the information from those studies and Bayesian meta-regression methods, we provide the first comprehensive estimates of global and regional incidence and prevalence of AAA in 21 regions worldwide from 1990 to 2010.

## METHODS

The methods consisted of 2 main stages: 1) literature search and extraction of data from studies on AAA incidence and prevalence and 2) statistical modeling of regional and global incidence and prevalence of AAA on the basis of the extracted data.

### Literature search and data extraction

Important clinical sequelae (conditions) were defined for AAA a priori. Sequelae for AAA were intact AAA (infrarenal

aortic diameter  $\geq 30$  mm), fatal ruptured AAA, emergency surgical repair for ruptured AAA, and elective surgical repair of AAA (open or endovascular). We conducted a systematic review of published literature on AAA. The initial search for prevalence and incidence of sequelae using standard clinical and epidemiological terms was conducted using the following databases: MEDLINE (1950 to 2010), EMBASE (1980 to 2010), AMED (1985 to 2010), CINAHL (1982 to 2010), and LILACS (2008 to 2010). Due to a paucity of papers covering some regions and countries, a broader search using simply AAA was conducted for these areas. Experts on AAA were approached, and reference lists of included papers were scanned for additional papers. Papers considered for inclusion were evaluated for their quality and relevance prior to a final decision on which to include in the review. For each of the 21 global regions, a “best available data” approach was employed.

Initial screening retrieved 9,716 titles of articles (Fig. 1), of which 446 abstracts were reviewed, resulting in full assessment of 188 full-text papers and final abstraction of data from 69 studies (Online Appendix). The extracted data included study characteristics (e.g., setting, duration, design), diagnostic criteria, mean age of study participants,

**TABLE 2.** Age-specific AAA annual incidence rates per 100,000 population by country development status, 1990 and 2010

Region	Age group, yrs											
	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	≥80
<b>1990</b>												
Developed												
Estimate	0.15	0.31	0.70	1.10	13.94	44.70	81.74	125.12	157.93	177.30	189.26	223.17
CI lower	0.09	0.24	0.51	0.75	12.77	41.04	75.95	115.22	145.14	162.24	172.97	203.75
CI upper	0.22	0.41	0.96	1.55	15.34	49.22	87.92	135.66	171.95	194.57	208.39	244.62
Developing												
Estimate	0.12	0.24	0.53	0.81	10.01	33.56	61.26	94.90	123.28	144.78	163.55	197.89
CI lower	0.08	0.18	0.37	0.54	8.94	29.54	54.70	84.01	109.63	127.86	144.50	174.76
CI upper	0.18	0.31	0.76	1.20	11.34	38.24	69.16	107.46	139.65	164.50	187.90	225.52
Global												
Estimate	0.12	0.26	0.57	0.89	11.17	37.17	68.06	106.31	137.28	157.54	176.08	212.04
CI lower	0.09	0.20	0.44	0.66	10.34	34.23	63.45	98.43	127.18	144.97	162.72	197.04
CI upper	0.17	0.32	0.75	1.17	12.14	40.65	73.02	114.59	147.57	170.08	190.28	227.47
<b>2010</b>												
Developed												
Estimate	0.13	0.28	0.62	0.96	12.57	40.60	74.05	116.52	147.64	158.37	175.12	203.38
CI lower	0.09	0.21	0.45	0.65	11.40	36.61	68.67	106.67	134.92	145.02	159.43	185.00
CI upper	0.20	0.36	0.87	1.41	13.88	44.75	80.16	127.04	160.84	171.67	191.64	223.97
Developing												
Estimate	0.11	0.22	0.51	0.79	9.94	33.39	61.70	94.29	121.12	139.26	157.56	188.59
CI lower	0.07	0.17	0.36	0.53	8.80	29.21	54.68	82.65	106.79	124.01	139.33	169.09
CI upper	0.16	0.29	0.70	1.15	11.24	37.93	69.55	107.16	137.17	157.23	178.77	211.89
Global												
Estimate	0.12	0.23	0.53	0.83	10.56	35.27	65.03	101.33	129.69	146.30	164.57	196.24
CI lower	0.08	0.19	0.40	0.61	9.65	32.07	59.81	92.95	119.83	135.47	152.20	182.94
CI upper	0.16	0.29	0.68	1.11	11.60	38.92	71.10	110.98	141.65	158.83	178.78	211.53

Abbreviation as in Table 1.

and sex ratio of patients. The final global burden of disease (GBD) estimation of prevalence and incidence was executed with data from 26 studies [23–48], which spanned 6 GBD regions: Europe (Western), Australasia, North Africa/Middle East, Asia Pacific (high income), Latin America (tropical), and North America (high income).

### Epidemiological modeling

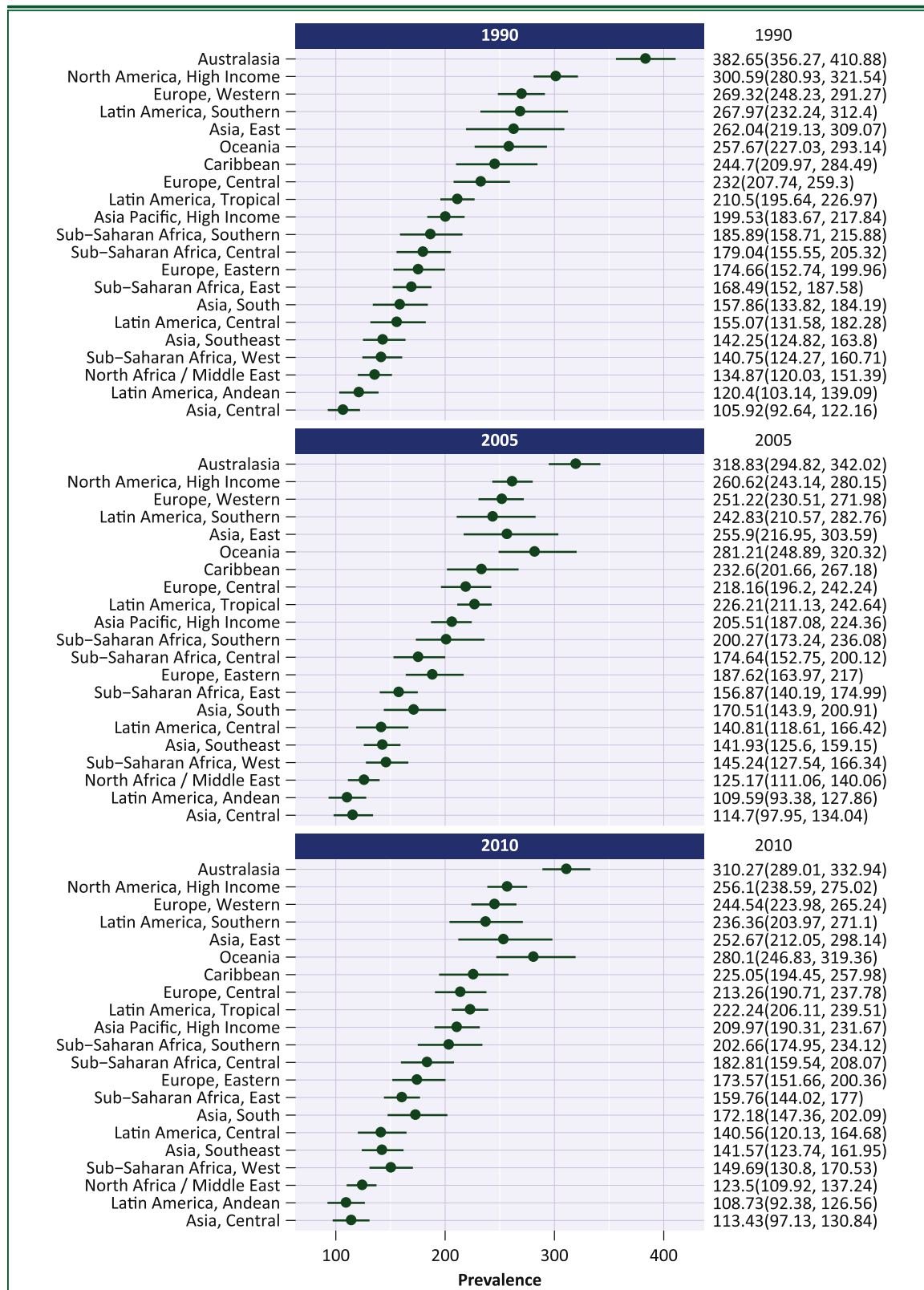
Estimates were made from data gathered in systematic reviews of epidemiological data using the previously described Bayesian meta-regression method (DisMod-MR) developed for the GBD 2010 [49]. In addition to assumptions of the generic model of DisMod-MR, we assumed that 1) remission of AAA (regardless of any treatments such as repair) was zero for all ages modeled; 2) incidence and prevalence before age 25 was zero; 3) relative difference of prevalence between men and women was used to adjust incidence in women; and 4) level of prevalence in different regions was proportional to number of deaths due to ruptured aneurysm in the regions in ages 50 to 65 years.

The DisMod-MR uses 2 types of covariates: those that explain true variation in prevalence and those that explain variation due to case definitions or diagnostic technology.

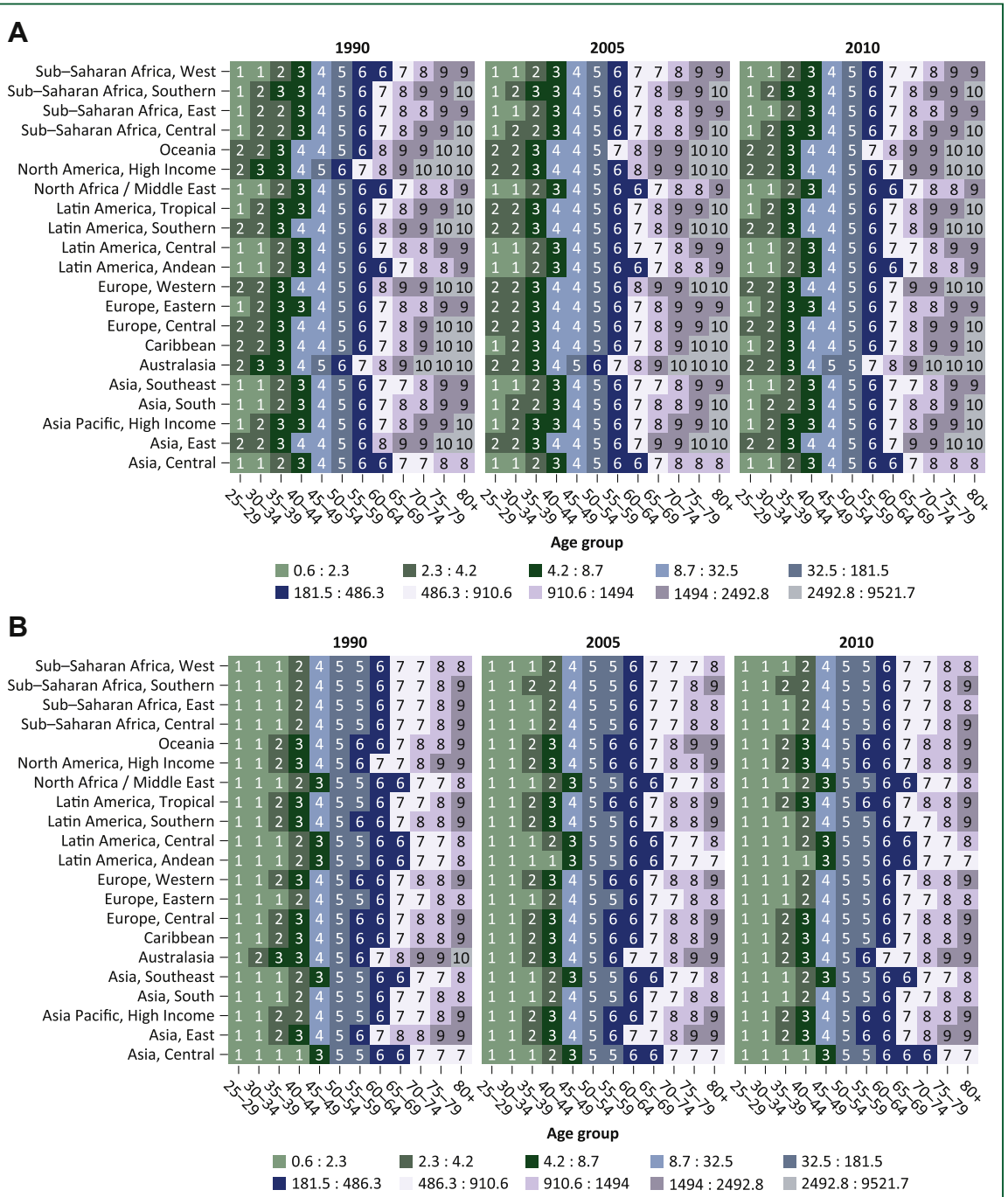
This tool estimates a generalized negative binomial model for all of the epidemiological data with various types of fixed and random effects. DisMod-MR can be used to estimate age-sex-population specific prevalence from heterogeneous and often sparse data sets. The GBD dealt with the problem of absent or low-quality epidemiological data from some regions or countries by incorporating covariates (e.g., cardiovascular risk factors, national income, differences in measurement method) and “borrowing strength” from nearby regions and years of observation in DisMod-MR models and by using prior standard assumptions about the relationship among disease-specific incidence, prevalence, case fatality, and mortality in DisMod-MR models [49–51].

### RESULTS

The AAA prevalence rates per 100,000 population by country development status are detailed in Table 1. In 1990, the global prevalence rate ranged from 8.43 (95% CI: 7.03 to 10.14) in the 40 to 44 years age group to 2,422.53 (95% CI: 2,298.63 to 2,562.25) in the 75 to 79 years age group, whereas the corresponding rates in 2010 were 7.88 (95% CI: 6.54 to 9.59) to 2,274.82 (95% CI: 2,149.77 to 2,410.17). In



**FIGURE 2. AAA prevalence by GBD region in 1990, 2005, and 2010.** The dots denote estimates of AAA prevalence rates per 100,000 population in all GBD regions. The bars around the estimates are the corresponding 95% uncertainty intervals. Abbreviations as in [Figure 1](#).



**FIGURE 3. AAA prevalence by GBD region and age group in 1990, 2005, and 2010.** (A) Total population. (B) Men. (C) Men. Each color-coded box represents a range of age-specific prevalence rates per 100,000 population for a GBD region in 1990, 2005, and 2010. Color gradations (also delineated by numbers within the color-coded boxes) represent different tiers of prevalence rates. The color gradient from green to blue to purple to gray (or increasing numbers) observed with increasing age indicates an increase in AAA prevalence by age within each region in 1990, 2005, and 2010. Age groups are in years. Abbreviations as in Figure 1.

both years, the global prevalence rate increased with age, and there was a consistent decrease in AAA prevalence from 1990 to 2010 across all age groups. In developed nations, the 1990

rates were 10.16 (95% CI: 8.09 to 12.73) in the 40 to 44 years age group and 2,666.91 (95% CI: 2,536.17 to 2,799.43) in the 75 to 79 years age group; in 2010, they

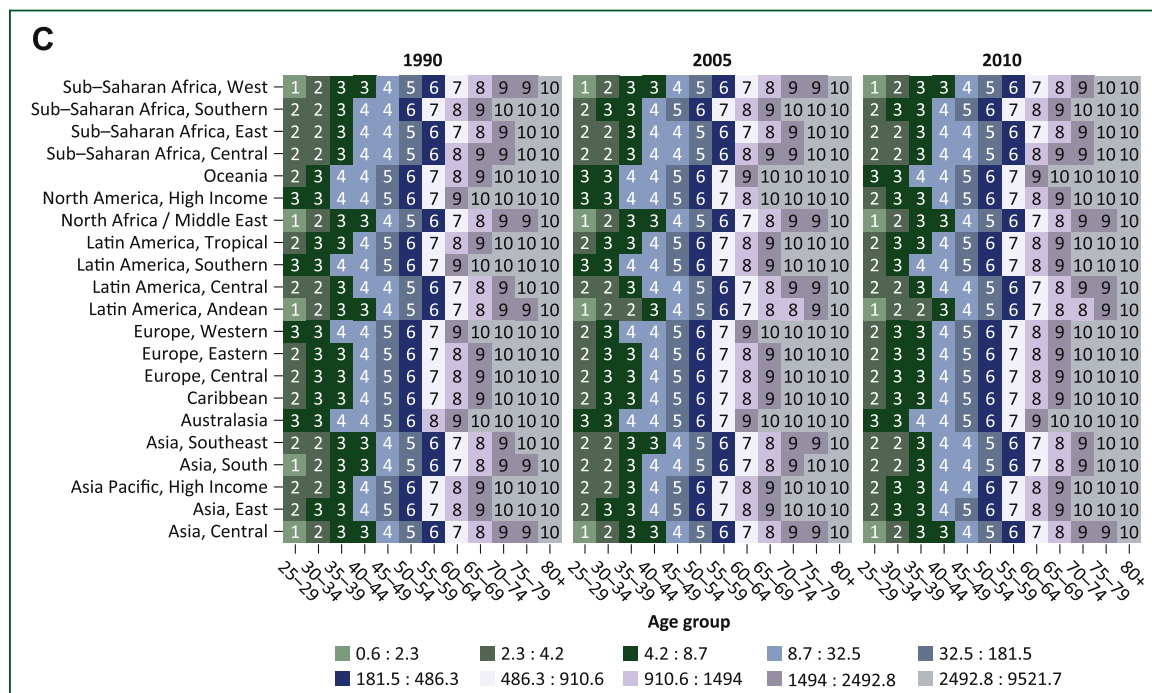


FIGURE 3. (continued).

were 9.06 (95% CI: 7.10 to 11.56) and 2,478.62 (95% CI: 2,361.96 to 2,603.68), respectively. In developing nations, these rates were 7.70 (95% CI: 6.01 to 10.04) to 2,190.06 (95% CI: 1,995.39 to 2,413.17) in 1990 and 7.59 (95% CI: 5.96 to 9.66) to 2,139.31 (95% CI: 1,943.86 to 2,350.28), respectively. Across all age groups, the rates in developed nations were higher than in developing countries in 1990 and 2010.

Globally, the age-specific annual incidence rate per 100,000 in 1990 ranged from 0.89 (95% CI: 0.66 to 1.17) in the 40 to 44 years age group to 176.08 (95% CI: 162.72 to 190.28) in the 75 to 79 years age group, whereas this range in 2010 was 0.83 (95% CI: 0.61 to 1.11) to 164.57 (95% CI: 152.20 to 178.78). Both globally and by country development status, the incidence rates detailed in Table 2 mirror the observed prevalence rate pattern; that is, the rates increased with age and were higher in 1990 than 2010 across all age groups. Furthermore, the incidence rates in developed nations were consistently higher than in developing nations.

Differences in regional prevalence rates of AAA are illustrated in Figure 2. The highest prevalence rates in 1990 were in Australasia and North America high income: 382.65 (95% CI: 356.27 to 410.88) and 300.59 (95% CI: 280.93 to 321.54), respectively (Fig. 2). Australasia remained at the top of the list in 2010, although the rate dropped to 310.27 (95% CI: 289.01 to 332.94); however, rates in the Oceania region rose from 257.67 (95% CI: 227.03 to 293.14) in 1990, a fourth place position, to 280.10 (95% CI: 246.83 to 319.36) in 2010, a second place position. Rates also declined in Western Europe from

269.32 (95% CI: 248.23 to 291.27) in 1990 to 244.54 (95% CI: 223.98 to 265.24) in 2010.

Other instances of decline in regional rates were in Southern Latin America, East Asia, Caribbean, Central Europe, Eastern Europe, East Sub-Saharan Africa (SSA), Central Latin America, Southeast Asia, North Africa/Middle East, and Andean Latin America. In addition to Oceania, other increases in regional rates were observed in tropical Latin America, Asia Pacific high income, Southern SSA, Central SSA, South Asia, West SSA, and Central Asia. The pattern of increasing prevalence with age was preserved regionally (Fig. 3A) and within sex, as depicted by the color chart in Figures 3B and 3C. However, across all ages, the rates were distinctly higher among men across all age groups. Regional differences in incidence rates (Fig. 4) were similar to differences in prevalence. Figures 5A, 5B, and 5C depict the increasing incidence rates with age across all regions, which were consistent within sex, albeit with higher rates among men compared with women of similar age groups.

## DISCUSSION

This systematic evaluation of the global epidemiology of AAA provides evidence that the prevalence and incidence of AAA have declined over the past 2 decades, which is consistent with reports from individual countries [18–22]. This study provides further evidence that the burden of AAA increases with age and that men are affected more frequently than women. At the global level, the incidence and prevalence rates are higher in developed versus

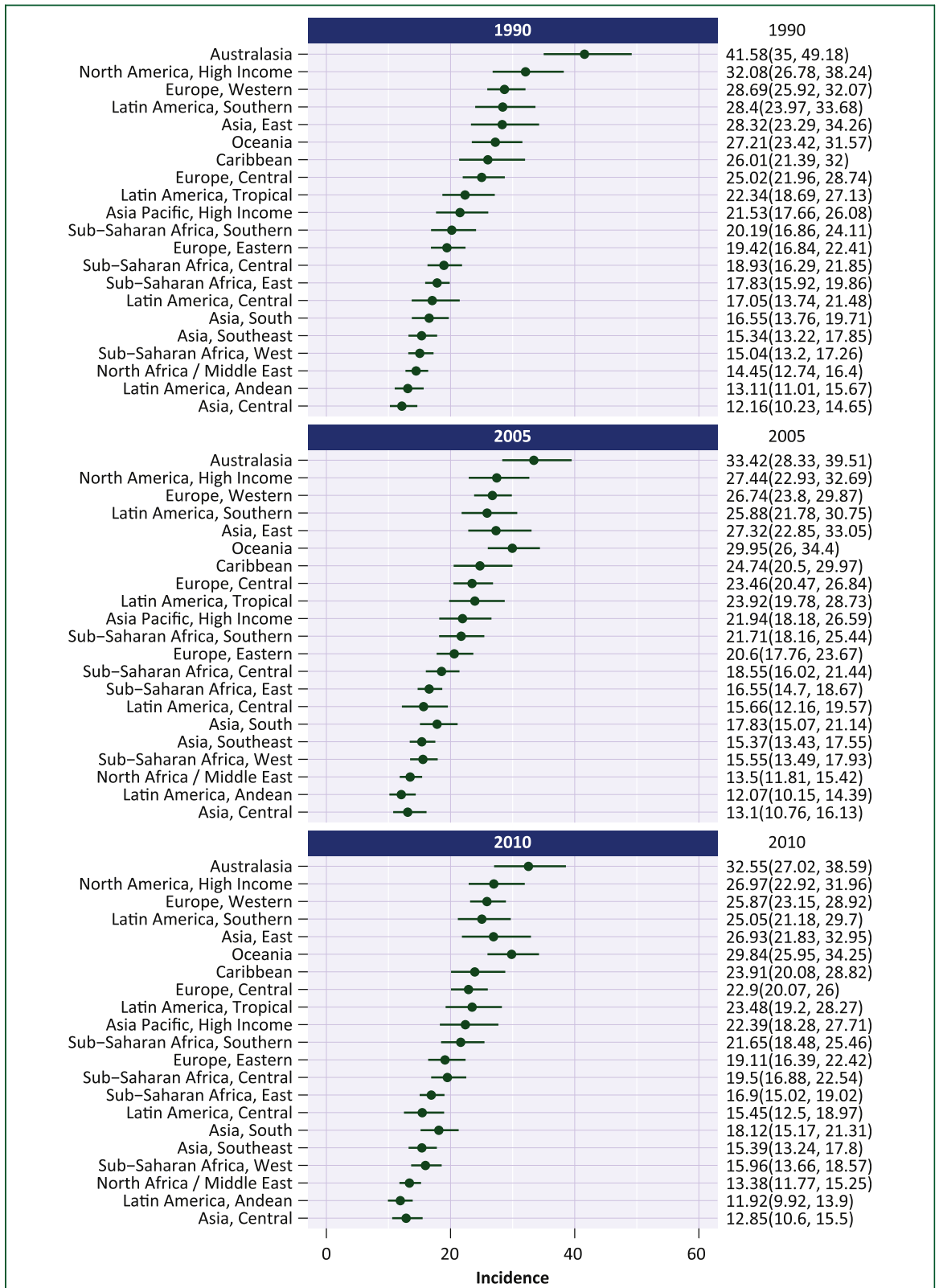
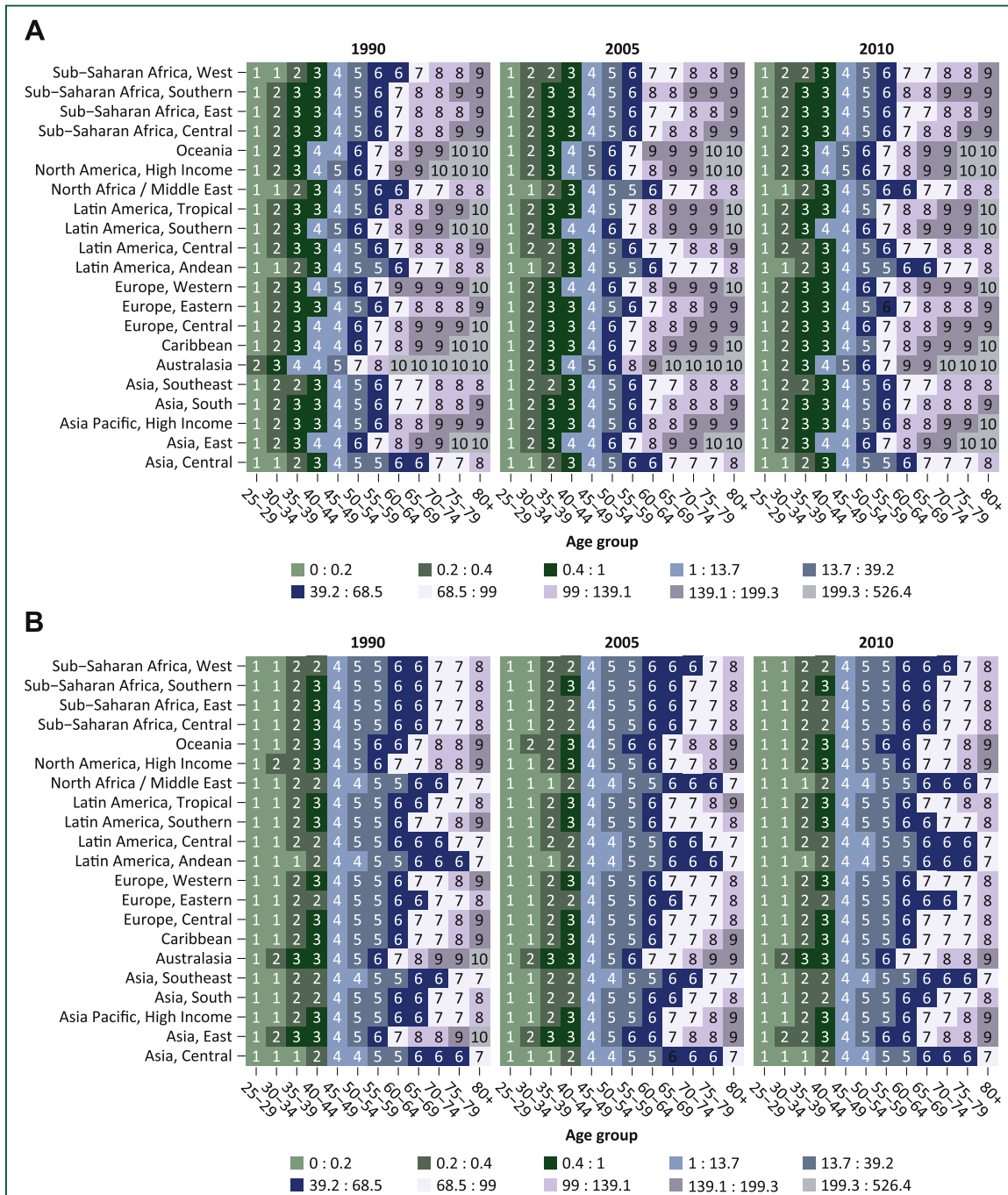


FIGURE 4. AAA annual incidence rates by GBD region in 1990, 2005, and 2010. The dots denote estimates of the annual incidence rates of AAA per 100,000 population in all GBD regions. The bars around the estimates are the corresponding 95% uncertainty intervals. Abbreviations as in Figure 1.





**FIGURE 5. AAA annual incidence rates by GBD region and age group in 1990, 2005, and 2010. (A) Total population. (B) Women. (C) Men.** The charts delineate estimates of age-specific annual incidence rates of AAA per 100,000 population for all GBD regions. Each color-coded box represents a range of age-specific incidence rates for a GBD region. Color gradations (also delineated by numbers within the color-coded boxes) represent different tiers of incidence rates. The color gradient from green to blue to purple to gray (or increasing numbers) observed with increasing age indicates an increase in AAA incidence by age within each region in 1990, 2005, and 2010. Age groups are in years. Abbreviations as in Figure 1.

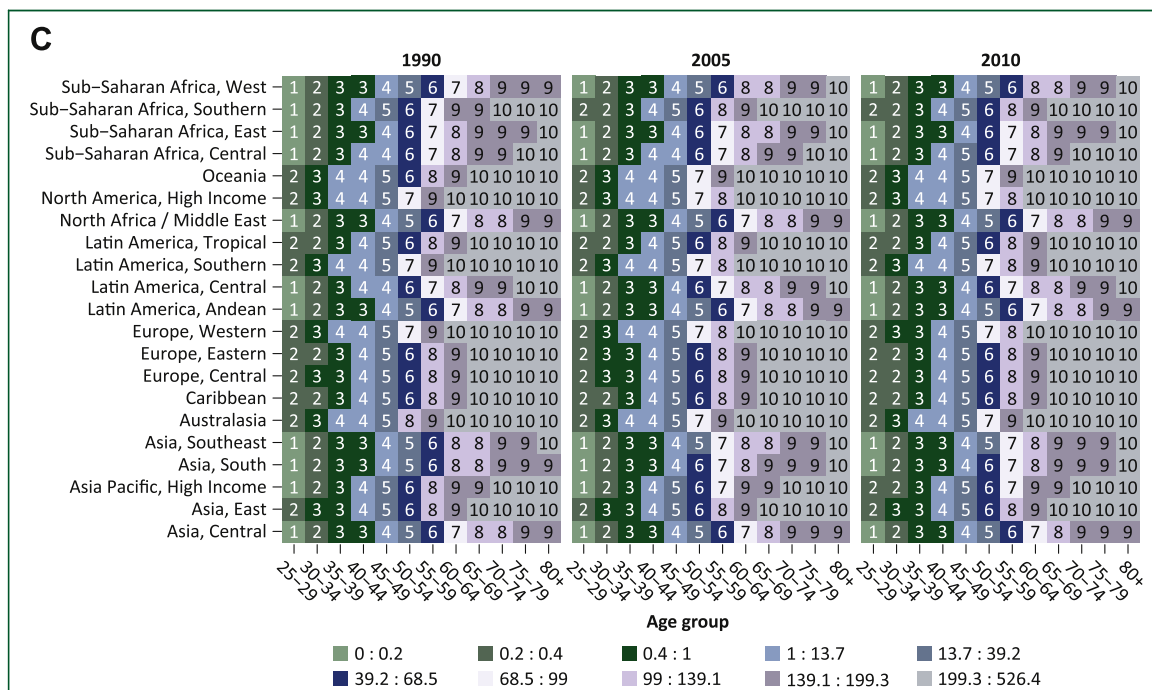


FIGURE 5. (continued).

developing nations. However, regional assessments reveal increases in prevalence for some regions, notably Oceania, tropical Latin America, Asia Pacific high income, Southern SSA, Central SSA, South Asia, West SSA, and Central Asia. The insight that this study provides may have implications for future surveillance efforts and intervention.

Australasia, North America high income, and Western Europe had the most appreciable decline in AAA prevalence and incidence rates. Such improvements in epidemiological pattern may be attributed to increases in risk factor control such as smoking cessation programs afforded by the health systems in these regions. Evidence of a decline in AAA in the high-income regions plus the scarcity of data in developing regions had the potential to create a false impression of overall global decline. However, our data and methods were robust enough to detect an increasing trend in AAA in a number of regions. Adverse trends in these regions are concerning and raise 2 fundamental questions. What is responsible for the worsening picture, and what is a reasonable response strategy? Increasing burden [49,52] of risk factors for atherosclerotic cardiovascular disease is worth considering. In this context, smoking cessation programs or policies to lower tobacco consumption may eventually provide good return on investment—akin to a World Health Organization “best buy” [53]—in regions with adverse trends in AAA prevalence and incidence, mostly in developing countries.

Smoking is an important target because multiple studies have identified smoking as the most important modifiable risk factor for AAA [44,47,54,55]. Current smoking,

specifically, appears to have the greatest effect, with evidence of a dose response whereby higher daily cigarette intake is associated with higher risk of AAA [56]. Although the mechanisms through which smoking increases AAA formation are unclear, multifactorial effects on atherosclerosis and overall vascular homeostasis may be implicated [57–63]. It is important to note that approximately 900 million smokers live in developing nations, accounting for 70% of global consumption, which stems from the aggressive marketing activities of tobacco companies in these areas [64]. Other strategies for reducing the burden of AAA could include improvements in diagnosis, treatment, and overall disease surveillance. However, these are resource intense and may not be routinely feasible in low- to middle-income regions.

It is important to recognize a major limitation of this study, which is the fact that we may have underestimated the global and regional trends in the incidence and prevalence of AAA due to scarcity of data in some regions. Given limited data for specific regions, it is uncertain as to how reasonable it is to project from other regions. Furthermore, the strength of our systematic analysis rests on the quality of the included studies. These are all factors that make apparent the need for improved global surveillance data to improve future assessments of disease burden.

### SUMMARY

AAA global prevalence and incidence rates have decreased over the last 20 years. However, regional evaluation revealed rising AAA rates in many regions of the world,

which highlights the need to improve prevention, diagnosis and treatment, and collection of disease surveillance data globally.

## REFERENCES

- Aggarwal S, Qamar A, Sharma V, Sharma A. Abdominal aortic aneurysm: a comprehensive review. *Exp Clin Cardiol* 2011;16:11–5.
- Kniemeyer HW, Kessler T, Reber PU, Ris HB, Hakki H, Widmer MK. Treatment of ruptured abdominal aortic aneurysm, a permanent challenge or a waste of resources? Prediction of outcome using a multi-organ-dysfunction score. *Eur J Vasc Endovasc Surg* 2000;19:190–6.
- Gillum RF. Epidemiology of aortic aneurysm in the United States. *J Clin Epidemiol* 1995;48:1289–98.
- Blanchard JF, Armenian HK, Friesen PP. Risk factors for abdominal aortic aneurysm: results of a case-control study. *Am J Epidemiol* 2000;151:575–83.
- Fowkes FG, Rudan D, Rudan I, et al. Comparison of global estimates of prevalence and risk factors for peripheral artery disease in 2000 and 2010: a systematic review and analysis. *Lancet* 2013;382:1329–40.
- Filipovic M, Goldacre MJ, Roberts SE, Yeates D, Duncan ME, Cook-Mozaffari P. Trends in mortality and hospital admission rates for abdominal aortic aneurysm in England and Wales, 1979–1999. *Br J Surg* 2005;92:968–75.
- Eickhoff JH. Incidence of diagnosis, operation and death from abdominal aortic aneurysms in Danish hospitals: results from a nation-wide survey, 1977–1990. *Eur J Surg* 1993;159:619–23.
- Castleden WM, Mercer JC. Abdominal aortic aneurysms in Western Australia: descriptive epidemiology and patterns of rupture. *Br J Surg* 1985;72:109–12.
- Melton LJ 3rd, Bickerstaff LK, Hollier LH, et al. Changing incidence of abdominal aortic aneurysms: a population-based study. *Am J Epidemiol* 1984;120:379–86.
- Derubertis BG, Trocciola SM, Ryer EJ, et al. Abdominal aortic aneurysm in women: prevalence, risk factors, and implications for screening. *J Vasc Surg* 2007;46:630–5.
- Scott RA, Bridgewater SG, Ashton HA. Randomized clinical trial of screening for abdominal aortic aneurysm in women. *Br J Surg* 2002;89:283–5.
- Norman PE, Powell JT. Abdominal aortic aneurysm: the prognosis in women is worse than in men. *Circulation* 2007;115:2865–9.
- Guirguis-Blake J, Wolff TA. Screening for abdominal aortic aneurysm. *Am Fam Physician* 2005;71:2154–5.
- Upchurch GR Jr, Schaub TA. Abdominal aortic aneurysm. *Am Fam Physician* 2006;73:1198–204.
- van der Vliet JA, Boll AP. Abdominal aortic aneurysm. *Lancet* 1997;349:863–6.
- Blanchard JF. Epidemiology of abdominal aortic aneurysms. *Epidemiol Rev* 1999;21:207–21.
- Cosford PA, Leng GC. Screening for abdominal aortic aneurysm. *Cochrane Database Syst Rev* 2007;(2):CD002945.
- About Compressed Mortality. Centers for Disease Control and Prevention WONDER. Available at: <http://wonder.cdc.gov/cm/icd10.html>. Accessed January 17, 2014.
- About Compressed Mortality, 1979–1998. Centers for Disease Control and Prevention WONDER. Available at: <http://wonder.cdc.gov/cm/icd9.html>. Accessed January 17, 2014.
- Anjum A, von Allmen R, Greenhalgh R, Powell JT. Explaining the decrease in mortality from abdominal aortic aneurysm rupture. *Br J Surg* 2012;99:637–45.
- Anjum A, Powell JT. Is the incidence of abdominal aortic aneurysm declining in the 21st century? Mortality and hospital admissions for England & Wales and Scotland. *Eur J Vasc Endovasc Surg* 2012;43:161–6.
- Choke E, Vijaynagar B, Thompson J, Nasim A, Bown MJ, Sayers RD. Changing epidemiology of abdominal aortic aneurysms in England and Wales: older and more benign? *Circulation* 2012;125:1617–25.
- Freiberg MS, Arnold AM, Newman AB, Edwards MS, Kraemer KL, Kuller LH. Abdominal aortic aneurysms, increasing infrarenal aortic diameter, and risk of total mortality and incident cardiovascular disease events: 10-year follow-up data from the Cardiovascular Health Study. *Circulation* 2008;117:1010–7.
- Newman AB, Arnold AM, Burke GL, O’Leary DH, Manolio TA. Cardiovascular disease and mortality in older adults with small abdominal aortic aneurysms detected by ultrasonography: the Cardiovascular Health Study. *Ann Intern Med* 2001;134:182–90.
- Scott RA, Ashton HA, Lamparelli MJ, Harris GJ, Stevens JW. A 14-year experience with 6 cm as a criterion for surgical treatment of abdominal aortic aneurysm. *Br J Surg* 1999;86:1317–21.
- Kim LG, P Scott RA, Ashton HA, Thompson SG. A sustained mortality benefit from screening for abdominal aortic aneurysm. *Ann Intern Med* 2007;146:699–706.
- Lindholt JS, Vammen S, Juul S, Fasting H, Henneberg EW. Optimal interval screening and surveillance of abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 2000;20:369–73.
- Norman PE, Flicker L, Almeida OP, Hankey GJ, Hyde Z, Jamrozik K. Cohort profile: the Health in Men Study (HIMS). *Int J Epidemiol* 2009;38:48–52.
- Morris GE, Hubbard CS, Quick CR. An abdominal aortic aneurysm screening programme for all males over the age of 50 years. *Eur J Vasc Surg* 1994;8:156–60.
- Wilkinson AB, Hubbard CS, Day NE, Quick CR. The incidence of small abdominal aortic aneurysms and the change in normal infrarenal aortic diameter: implications for screening. *Eur J Vasc Endovasc Surg* 2001;21:165–70.
- Heather BP, Poskitt KR, Earnshaw JJ, Whyman M, Shaw E. Population screening reduces mortality rate from aortic aneurysm in men. *Br J Surg* 2000;87:750–3.
- Scott RA, Vardulaki KA, Walker NM, Day NE, Duffy SW, Ashton HA. The long-term benefits of a single scan for abdominal aortic aneurysm (AAA) at age 65. *Eur J Vasc Endovasc Surg* 2001;21:535–40.
- Lindholt JS, Juul S, Fasting H, Henneberg EW. Preliminary ten year results from a randomised single centre mass screening trial for abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 2006;32:608–14.
- Ashton HA, Buxton MJ, Day NE, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. *Lancet* 2002;360:1531–9.
- Norman PE, Jamrozik K, Lawrence-Brown MM, et al. Population based randomised controlled trial on impact of screening on mortality from abdominal aortic aneurysm. *BMJ* 2004;329:1259.
- Bengtsson H, Bergqvist D, Ekberg O, Janzon L. A population based screening of abdominal aortic aneurysms (AAA). *Eur J Vasc Surg* 1991;5:53–7.
- Scott RA, Wilson NM, Ashton HA, Kay DN. Influence of screening on the incidence of ruptured abdominal aortic aneurysm: 5-year results of a randomized controlled study. *Br J Surg* 1995;82:1066–70.
- al-Zahrani HA, Rawas M, Maimani A, Gasab M, Aba al Khail BA. Screening for abdominal aortic aneurysm in the Jeddah area, western Saudi Arabia. *Cardiovasc Surg* 1996;4:87–92.
- Adachi K, Iwasawa T, Ono T. Screening for abdominal aortic aneurysms during a basic medical checkup in residents of a Japanese rural community. *Surg Today* 2000;30:594–9.
- Puech-Leão P, Molnar LJ, Oliveira IR, Cerri GG. Prevalence of abdominal aortic aneurysms—a screening program in São Paulo, Brazil. *São Paulo Med J* 2004;122:158–60.
- Sariosmanoglu N, Ugurlu B, Karacelik M, et al. A multicentre study of abdominal aorta diameters in a Turkish population. *J Int Med Res* 2002;30:1–8.
- Boll AP, Verbeek AL, van de Lisdonk EH, van der Vliet JA. High prevalence of abdominal aortic aneurysm in a primary care screening programme. *Br J Surg* 1998;85:1090–4.
- Kyriakides C, Byrne J, Green S, Hulton NR. Screening of abdominal aortic aneurysm: a pragmatic approach. *Ann R Coll Surg Engl* 2000;82:59–63.
- Simoni G, Pastorino C, Perrone R, et al. Screening for abdominal aortic aneurysms and associated risk factors in a general population. *Eur J Vasc Endovasc Surg* 1995;10:207–10.

45. Chichester Aneurysm Screening Group, Viborg Aneurysm Screening Study; Western Australian Abdominal Aortic Aneurysm Program; Multicentre Aneurysm Screening Study. A comparative study of the prevalence of abdominal aortic aneurysms in the United Kingdom, Denmark, and Australia. *J Med Screen* 2001;8:46–50.
46. Pleumeekers HJ, Hoes AW, van der Does E, et al. Aneurysms of the abdominal aorta in older adults. The Rotterdam Study. *Am J Epidemiol* 1995;142:1291–9.
47. Singh K, Bonna KH, Jacobsen BK, Bjork L, Solberg S. Prevalence of and risk factors for abdominal aortic aneurysms in a population-based study: the Tromso Study. *Am J Epidemiol* 2001;154:236–44.
48. Salem MK, Rayt HS, Hussey G, et al. Should Asian men be included in abdominal aortic aneurysm screening programmes? *Eur J Vasc Endovasc Surg* 2009;38:748–9.
49. Vos T, Flaxman AD, Naghavi M, et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2163–96.
50. Murray CJ, Vos T, Lozano R, et al. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2197–223.
51. Forouzanfar MH, Moran AE, Flaxman AD, et al. Assessing the global burden of ischemic heart disease, part 2: analytic methods and estimates of the global epidemiology of ischemic heart disease in 2010. *Glob Heart* 2012;7:331–42.
52. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2224–60.
53. From burden to “best buys”: reducing the economic impact of non-communicable diseases in low- and middle-income countries. World Health Organization. Available at: [http://www.who.int/nmh/publications/best\\_buys\\_summary.pdf](http://www.who.int/nmh/publications/best_buys_summary.pdf). Accessed January 17, 2014.
54. Lindblad B, Borner G, Gottsater A. Factors associated with development of large abdominal aortic aneurysm in middle-aged men. *Eur J Vasc Endovasc Surg* 2005;30:346–52.
55. Wanhainen A, Bergqvist D, Boman K, Nilsson TK, Rutegard J, Bjorck M. Risk factors associated with abdominal aortic aneurysm: a population-based study with historical and current data. *J Vasc Surg* 2005;41:390–6.
56. Iribarren C, Darbinian JA, Go AS, Fireman BH, Lee CD, Grey DP. Traditional and novel risk factors for clinically diagnosed abdominal aortic aneurysm: the Kaiser Multiphasic Health Checkup Cohort Study. *Ann Epidemiol* 2007;17:669–78.
57. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. *J Am Coll Cardiol* 2004;43:1731–7.
58. Napoli C, Ignarro LJ. Nitric oxide and atherosclerosis. *Nitric Oxide* 2001;5:88–97.
59. Morrow JD, Frei B, Longmire AW, et al. Increase in circulating products of lipid peroxidation (F2-isoprostanes) in smokers. Smoking as a cause of oxidative damage. *N Engl J Med* 1995;332:1198–203.
60. Smith CJ, Fischer TH. Particulate and vapor phase constituents of cigarette mainstream smoke and risk of myocardial infarction. *Atherosclerosis* 2001;158:257–67.
61. Craig WY, Palomaki GE, Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. *BMJ* 1989;298:784–8.
62. Stefanadis C, Vlachopoulos C, Tsiamis E, et al. Unfavorable effects of passive smoking on aortic function in men. *Ann Intern Med* 1998;128:426–34.
63. MacSweeney ST, Young G, Greenhalgh RM, Powell JT. Mechanical properties of the aneurysmal aorta. *Br J Surg* 1992;79:1281–4.
64. Trade foreign policy, diplomacy and health: tobacco. Available at: <http://www.who.int/trade/glossary/story089/en/>. Accessed January 17, 2014.