Abdominal Aortic Aneurysm: Systematic Review with ☸SAIMSARA.

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Abstract: The aim of this paper is to synthesize current research on abdominal aortic aneurysm (AAA) by systematically extracting and structuring information from a diverse body of scientific literature, identifying key themes, and highlighting clinical and research implications. The review utilises 410 studies with 150739 total participants (naïve ΣN). The prevalence of abdominal aortic aneurysm (AAA) in general and screened populations varies, with a median prevalence of 3.05% and a range from 0.7% to 5.1%. While screening programs demonstrate a mortality benefit, and endovascular repair shows promise for ruptured AAAs, the heterogeneity of study designs and variable follow-up periods limit definitive conclusions. The most significant limitation affecting certainty is the Heterogeneity of Study Designs, which impedes direct comparisons and broad generalizability. A concrete next step is to conduct sex-specific clinical trials to evaluate AAA interventions and therapies in women, addressing current gaps in evidence.

Keywords: Abdominal Aortic Aneurysm; Endovascular Aneurysm Repair; Aneurysm Rupture; Aneurysm Growth; Inflammation; Genetic Factors; Smooth Muscle Cells; Endoleak; Open Surgical Repair; Cardiometabolic Traits

Review Stats

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- Scope: All fields
- Keyword Gate: Fuzzy (≥60% of required terms, minimum 2 terms matched in title/abstract)
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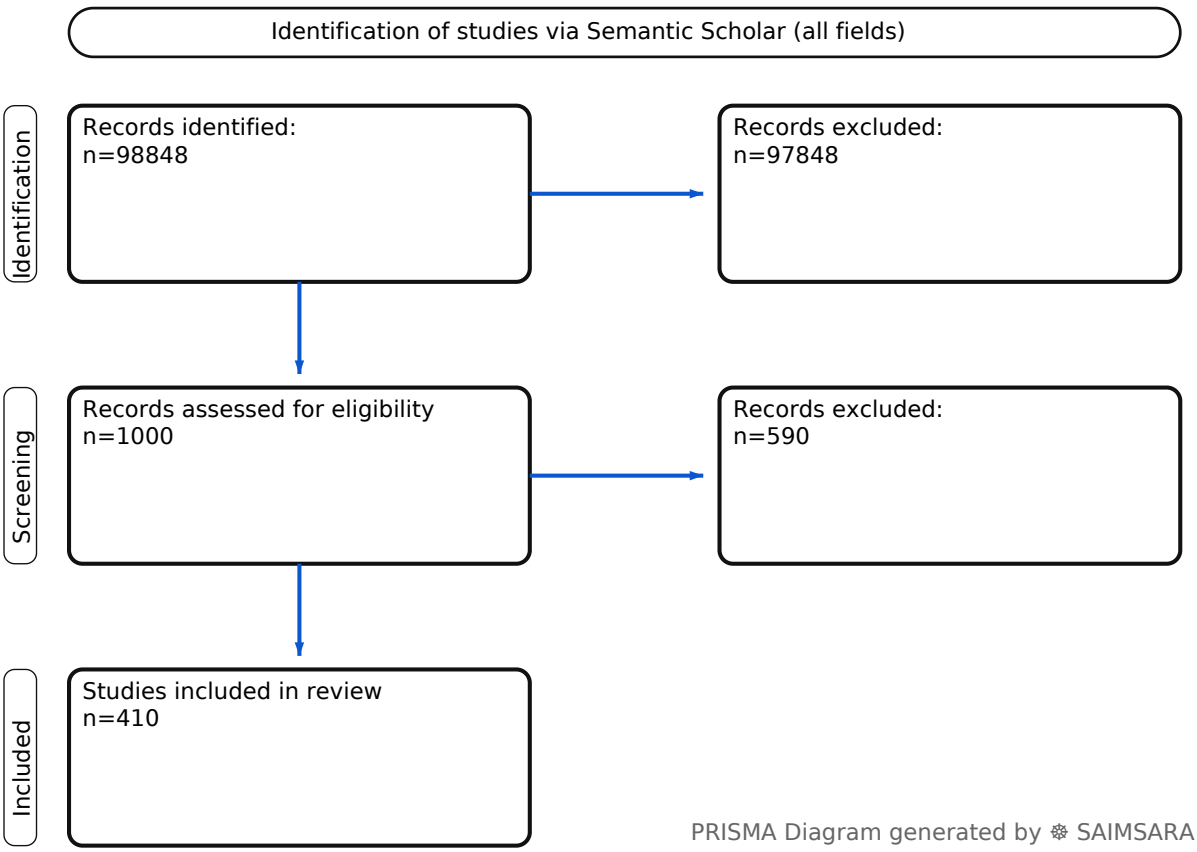
- Downloaded Abstracts/Papers: 1000
- Included original Abstracts/Papers: 410
- Total study participants (naïve ΣN): 150739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Records excluded:
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Screening
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Records excluded:
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Included
Studies included in review
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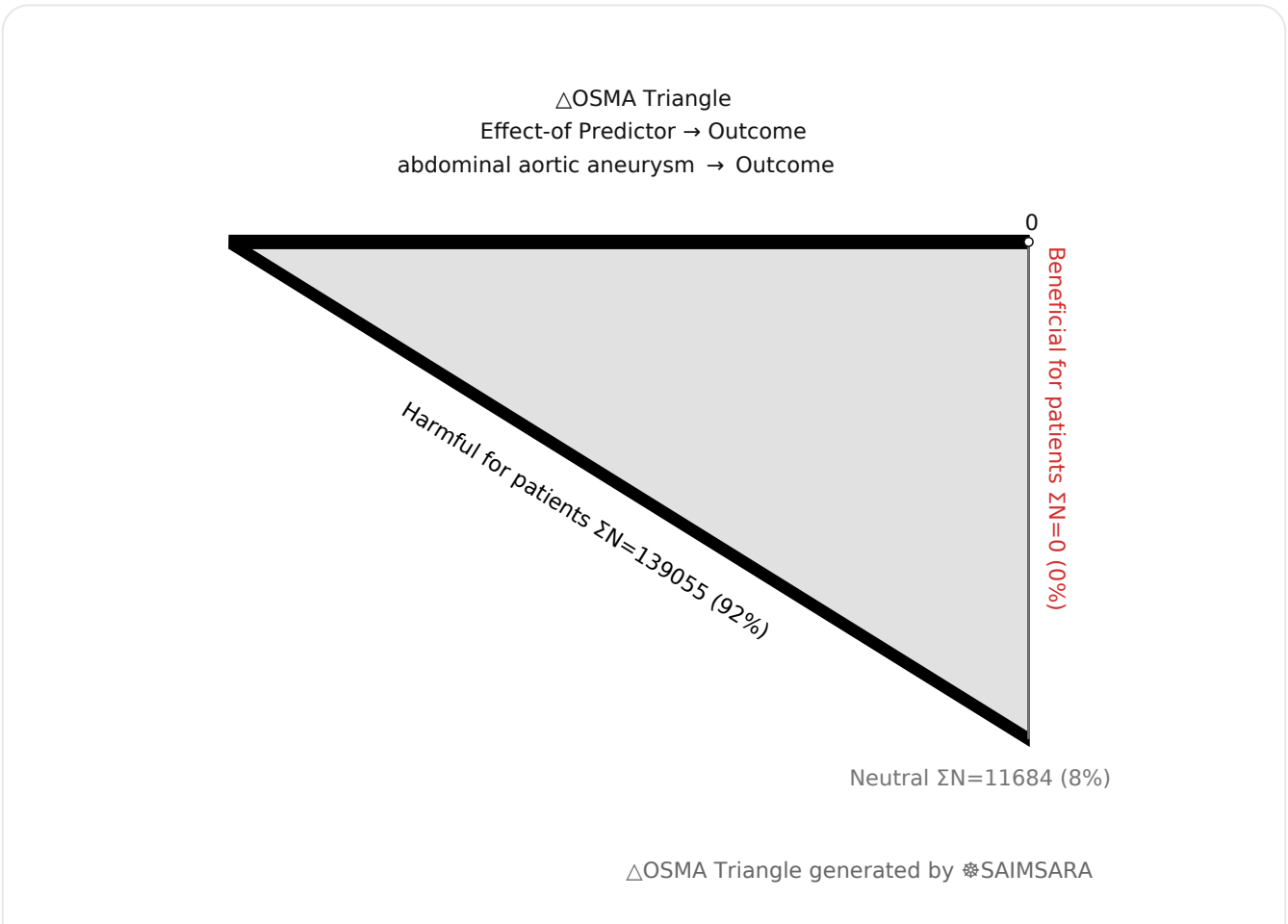
PRISMA Diagram generated by ☸ SAIMSARA


△OSMA Triangle
Effect-of Predictor → Outcome
abdominal aortic aneurysm → Outcome
0
Beneficial for patients ΣN=0 (0%)
Harmful for patients ΣN=139055 (92%)
Neutral ΣN=11684 (8%)
△OSMA Triangle generated by ☸SAIMSARA

Outcome-Sentiment Meta-Analysis (OSMA): *(LLM-only)*

Frame: Effect-of Predictor → Outcome • *Source*: Semantic Scholar

Outcome: Outcome Typical timepoints: peri/post-op, 30-day. Reported metrics: %, CI, p.

Common endpoints: Common endpoints: mortality, complications, survival.

Predictor: abdominal aortic aneurysm — exposure/predictor. Routes seen: subcutaneous.

Typical comparator: no p-t2el. older age, placebo, open repair, open repair in patients with….

- **1) Beneficial for patients** — Outcome with abdominal aortic aneurysm — — — ΣN=0
- **2) Harmful for patients** — Outcome with abdominal aortic aneurysm — [2], [26], [29], [50], [53], [83], [165], [262], [341], [344], [402] — ΣN=139055
- **3) No clear effect** — Outcome with abdominal aortic aneurysm — [1], [3], [4], [5], [6], [7], [8], [9], [10], [11], [12], [13], [14], [15], [16], [17], [18], [19], [20], [21], [22], [23], [24], [25], [27], [28], [30], [31], [32], [33], [34], [35], [36], [37], [38], [39], [40], [41], [42], [43], [44], [45], [46], [47], [48], [49], [51], [52], [54], [55], [56], [57], [58], [59], [60], [61], [62], [63], [64], [65], [66], [67], [68], [69], [70], [71], [72], [73], [74], [75], [76], [77], [78], [79], [80], [81], [82], [84], [85], [86], [87], [88], [89], [90], [91], [92], [93], [94], [95], [96], [97], [98], [99], [100], [101], [102], [103], [104], [105], [106], [107], [108], [109], [110], [111], [112], [113], [114], [115], [116], [117], [118], [119], [120], [121], [122], [123], [124], [125], [126], [127], [128], [129], [130], [131], [132], [133], [134], [135], [136], [137], [138], [139], [140], [141], [142], [143], [144], [145], [146], [147], [148], [149], [150], [151], [152], [153], [154], [155], [156], [157], [158], [159], [160], [161], [162], [163], [164], [166], [167], [168], [169], [170], [171], [172], [173], [174], [175], [176], [177], [178], [179], [180], [181], [182], [183], [184], [185], [186], [187], [188], [189], [190], [191], [192], [193], [194], [195], [196], [197], [198], [199], [200], [201], [202], [203], [204], [205], [206], [207], [208], [209], [210], [211], [212], [213], [214], [215], [216], [217], [218], [219], [220], [221], [222], [223], [224], [225], [226], [227], [228], [229], [230], [231], [232], [233], [234], [235], [236], [237], [238], [239], [240], [241], [242], [243], [244], [245], [246], [247], [248], [249], [250], [251], [252], [253], [254], [255], [256], [257], [258], [259], [260], [261], [263], [264], [265], [266], [267], [268], [269], [270], [271], [272], [273], [274], [275], [276], [277], [278], [279], [280], [281], [282], [283], [284], [285], [286], [287], [288], [289], [290], [291], [292], [293], [294], [295], [296], [297], [298], [299], [300], [301], [302], [303], [304], [305], [306], [307], [308], [309], [310], [311], [312], [313], [314], [315], [316], [317], [318], [319], [320], [321], [322], [323], [324], [325], [326], [327], [328], [329], [330], [331], [332], [333], [334], [335], [336], [337], [338], [339], [340], [342], [343], [345], [346], [347], [348], [349], [350], [351], [352], [353], [354], [355], [356], [357], [358], [359], [360], [361], [362], [363], [364], [365], [366], [367], [368], [369],

[370], [371], [372], [373], [374], [375], [376], [377], [378], [379], [380], [381], [382], [383], [384], [385], [386], [387], [388], [389], [390], [391], [392], [393], [394], [395], [396], [397], [398], [399], [400], [401], [403], [404], [405], [406], [407], [408], [409], [410] — ΣN=11684

1) Introduction

Abdominal aortic aneurysm (AAA) is a localized dilatation of the abdominal aorta, characterized by progressive weakening and expansion of the arterial wall. Its rupture is a life-threatening event, necessitating a comprehensive understanding of its pathophysiology, risk factors, diagnosis, and management strategies. Research into AAA spans from elucidating complex molecular mechanisms and genetic predispositions to evaluating the efficacy and cost-effectiveness of various screening and therapeutic interventions.

2) Aim

The aim of this paper is to synthesize current research on abdominal aortic aneurysm (AAA) by systematically extracting and structuring information from a diverse body of scientific literature, identifying key themes, and highlighting clinical and research implications.

3) Methods

Systematic review with multilayer AI research agent: keyword normalization, retrieval & structuring, and paper synthesis (see SAIMSARA About section for details).

- **Bias:** Qualitatively inferred from study design fields. Randomized controlled trials (RCTs) generally offer lower risk of bias for intervention efficacy, while cohort and case-control studies are more susceptible to confounding. Animal models and studies with unspecified design or population may have limited generalizability to human clinical practice.

4) Results

4.1 Study characteristics

The reviewed literature comprises a wide array of study designs, including numerous randomized controlled trials (RCTs) evaluating screening programs and surgical interventions, alongside cohort studies, case-control studies, and mixed-design investigations exploring pathophysiology, risk factors, and outcomes. Many studies employed murine models to investigate molecular mechanisms and potential therapeutic targets, while others focused on human populations ranging from specific

patient cohorts (e.g., those undergoing endovascular repair) to large population-based screenings. Follow-up periods varied significantly, from short-term (e.g., 30 days) to long-term (e.g., up to 14 years for surgical outcomes or 15 years for screening efficacy).

4.2 Main numerical result aligned to the query

The prevalence of abdominal aortic aneurysm (AAA) in general and screened populations varies, with a median prevalence of 3.05% and a range from 0.7% to 5.1% [29, 341]. Specifically, one study reported a prevalence of 5.1% in men aged 65-74 years with an aortic diameter ≥30 mm [29], while another found an overall prevalence of 2.1% in a total population, with 4.0% in men and 0.7% in women [341].

4.3 Topic synthesis

- **Genetic and Cardiometabolic Predisposition:** Genetic correlations exist between AAA and 21 cardiometabolic traits, suggesting shared causal variants and pathways, particularly in cholesterol metabolism and inflammation [1]. Specific genetic variants like those on 9p21 [25] and within the DAB2IP gene [48] confer susceptibility, and familial aggregation is recognized [62, 295, 400, 401, 409].
- **Pathophysiology and Molecular Mechanisms:** AAA development involves complex processes including upregulation of long noncoding RNA H19 [5], activation of matrix metalloproteinase-3 (MMP3) by Netrin-1 [6], and increased levels of proteins associated with proteolysis, oxidative stress, lipid metabolism, and inflammation [8]. MicroRNAs (e.g., miR-29b, miR-21, miR-24, miR-181b) play regulatory roles [22, 24, 27, 36, 329], and inhibition of histone demethylase JMJD3 [337] or maintenance of VSMC homeostasis by unspliced XBP1 [338] can protect against AAA. Intraluminal thrombus contributes to local hypoxia and wall weakening [46, 196, 199, 215, 228, 302].
- **Risk Factors and Epidemiology:** Key risk factors include older age, male sex, smoking, and hypercholesterolemia [2, 29, 33, 136, 225, 341, 402]. Chronic kidney disease and dilated proximal neck are independent predictors of sac enlargement [2]. Diabetes has shown a negative association with AAA events [136] and lower growth rates [344, 253]. Aortic rupture in murine models can occur in 25% of mice within 7 days of angiotensin II infusion [50].
- **Diagnosis and Monitoring:** Ultrasonography is a primary screening method [63, 65, 83, 131, 162, 212, 252, 271, 290, 396]. Biomarkers like growth/differentiation factor 15 (GDF15) and cystatin B show diagnostic potential, while myeloperoxidase has prognostic value for growth [8]. Imaging techniques, including CT and MRI, are used for assessment, rupture prediction, and follow-up [31, 45, 175, 237, 310, 316, 374, 392]. Wall stress calculations, influenced by diameter, asymmetry, and intraluminal thrombus, are important for rupture

risk assessment [31, 45, 157, 159, 188, 192, 250, 262, 379].

- **Medical Management and Stabilization:** Doxycycline, a matrix metalloproteinase inhibitor, did not significantly reduce AAA growth over 2 years in small AAAs (difference, 0.0 cm; 95% CI, -0.07 to 0.07 cm) [4], but preclinical studies suggest potential stabilization [219, 251, 254, 353]. Angiotensin receptor blockers like telmisartan suppressed experimental aneurysms [11, 223]. Statins are associated with reduced growth [193, 273, 356, 369]. Other targets include c-Jun N-terminal kinase [60], glycolysis restriction [30], and microRNA modulation [22, 24, 36, 329].
- **Surgical and Endovascular Repair:** For asymptomatic AAAs, long-term overall survival is similar between endovascular repair (EVAR) and open repair (hazard ratio 0.96, 95% CI 0.82 to 1.13 for all-cause mortality) [3], though EVAR is associated with more secondary procedures [3]. For ruptured AAAs, an endovascular strategy was associated with a survival advantage, gain in quality-adjusted life years (QALYs), similar reintervention rates, and reduced costs at three years compared to open repair [7]. However, 30-day mortality for suspected ruptured AAA was not significantly reduced (35.4% EVAR vs 37.4% open) [14]. Technical success for EVAR is high (e.g., 93.8% for AAA/TAA [324], 97.7% for large bore arteriotomy closure [330]), but persistent type II endoleaks are associated with significantly higher rates of AAA-related mortality, rupture, sac enlargement, and reintervention [2].
- **Small AAA Management:** Surveillance intervals for small AAAs (3.0-5.4 cm) are recommended based on size, with several years for 3.0-4.0 cm, around 1 year for 4.0-4.9 cm, and 6 months for 5.0-5.4 cm [344]. Rupture rates are almost fourfold higher in women than men, and current smokers have doubled rupture rates [344]. Men with aortic diameters of 25-29 mm have an increased risk of mortality and subsequent hospital admissions compared to those with ≤24 mm [29].
- **Associated Conditions:** AAA is associated with other vascular conditions such as peripheral vascular disease [63, 66, 123] and carotid stenosis [132]. Patients with aortic aneurysms have an overall prevalence of intracranial aneurysms of 11.8%, with female patients having a higher risk (OR=2.08, 95% CI 1.49–3.03) [325]. Splanchnic artery aneurysms can also be associated with infrarenal AAAs [335].

5) Discussion

5.1 Principal finding

The prevalence of abdominal aortic aneurysm (AAA) in general and screened populations varies, with a median prevalence of 3.05% and a range from 0.7% to 5.1% [29, 341].

5.2 Clinical implications

- **Screening Benefits:** Population-based screening for AAA, particularly in men aged 65-74, demonstrates a maintained mortality benefit and favorable cost-effectiveness over 10 years [53, 238].
- **Tailored Surveillance:** Surveillance intervals for small AAAs should be individualized based on size, with shorter intervals for larger aneurysms (e.g., 6 months for 5.0-5.4 cm) and longer intervals for smaller ones (e.g., several years for 3.0-4.0 cm) [344].
- **Ruptured AAA Management:** An endovascular strategy for ruptured AAA is associated with a survival advantage, improved quality of life, and reduced costs at 3 years compared to open repair [7], despite similar 30-day mortality rates [14].
- **Post-EVAR Monitoring:** Persistent type II endoleaks after endovascular aneurysm repair (EVAR) are associated with significantly higher rates of AAA-related mortality, rupture, sac enlargement, and reintervention, necessitating careful follow-up [2].
- **Comorbidity Awareness:** Clinicians should be aware of the increased risk of intracranial aneurysms in patients with aortic aneurysms, particularly in women, and consider screening for other vascular diseases [325].

5.3 Research implications / key gaps

- **Optimal Medical Therapies** — Further randomized controlled trials are needed to definitively establish the efficacy of medical therapies (e.g., doxycycline, telmisartan, statins) in limiting AAA growth and rupture risk across diverse patient cohorts [4, 11, 160, 193].
- **Sex-Specific Pathophysiology** — Research should further investigate the distinct pathophysiological mechanisms and risk factor profiles for AAA in women, given their lower prevalence but higher rupture rates for small aneurysms [2, 202, 260, 341, 344].
- **Biomarker Integration into Practice** — Prospective studies are required to validate and integrate novel diagnostic and prognostic biomarkers (e.g., GDF15, cystatin B, myeloperoxidase, microRNAs) into clinical decision-making algorithms for AAA surveillance and intervention [8, 27, 36, 329].
- **Long-term EVAR Outcomes for Ruptured AAA** — Extended follow-up data from randomized trials comparing EVAR and open repair for ruptured AAA are needed to confirm the durability of initial survival advantages and evaluate long-term reintervention burdens [7, 14].
- **Personalized Rupture Risk Prediction** — Development and validation of advanced biomechanical models incorporating patient-specific factors (e.g., wall thickness, geometry asymmetry, intraluminal thrombus properties) are crucial for more accurate rupture risk prediction beyond simple diameter [31, 45, 157, 190, 192, 250, 262, 379].

5.4 Limitations

- **Heterogeneity of Study Designs** — The diverse range of study designs limits the ability to perform quantitative meta-analysis and generalize findings consistently.
- **Variable Follow-up Periods** — Inconsistent follow-up durations across studies make it challenging to compare long-term outcomes for interventions and natural history.
- **Missing Quantitative Data** — Many summary entries lack specific numerical data for sample sizes, statistics, or follow-up, hindering comprehensive quantitative synthesis.
- **Qualitative Bias Inference** — Bias was inferred qualitatively, which may not fully capture the methodological rigor or limitations of individual studies.
- **Focus on Male Populations** — A significant portion of screening and epidemiological studies primarily focused on men, limiting direct applicability to women.

5.5 Future directions

- **Standardized Outcome Reporting** — Implement standardized reporting of key clinical and molecular outcomes in AAA research.
- **Sex-Specific Clinical Trials** — Conduct clinical trials specifically designed to evaluate AAA interventions and therapies in women.
- **Multi-Omics Biomarker Panels** — Develop and validate multi-omics biomarker panels for early detection and personalized risk stratification.
- **Advanced Imaging for Risk** — Utilize advanced imaging techniques combined with computational fluid dynamics for precise rupture risk assessment.
- **Targeted Drug Delivery** — Explore nanotherapy and targeted drug delivery systems for localized therapeutic intervention in AAA.

6) Conclusion

The prevalence of abdominal aortic aneurysm (AAA) in general and screened populations varies, with a median prevalence of 3.05% and a range from 0.7% to 5.1% [29, 341]. While screening programs demonstrate a mortality benefit, and endovascular repair shows promise for ruptured AAAs, the heterogeneity of study designs and variable follow-up periods limit definitive conclusions. The most significant limitation affecting certainty is the **Heterogeneity of Study Designs**, which impedes direct comparisons and broad generalizability. A concrete next step is to conduct sex-specific clinical trials to evaluate AAA interventions and therapies in women, addressing current gaps in evidence.

References

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Figure 1. Publication-year distribution of included originals

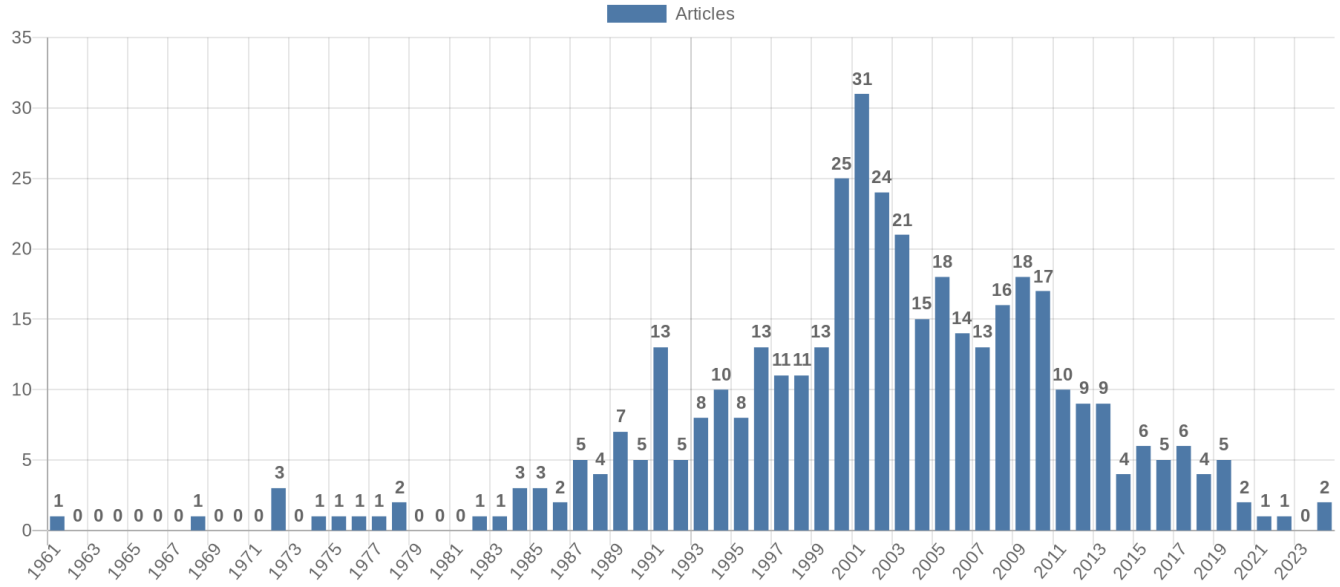

Figure 2. Study-design distribution of included originals

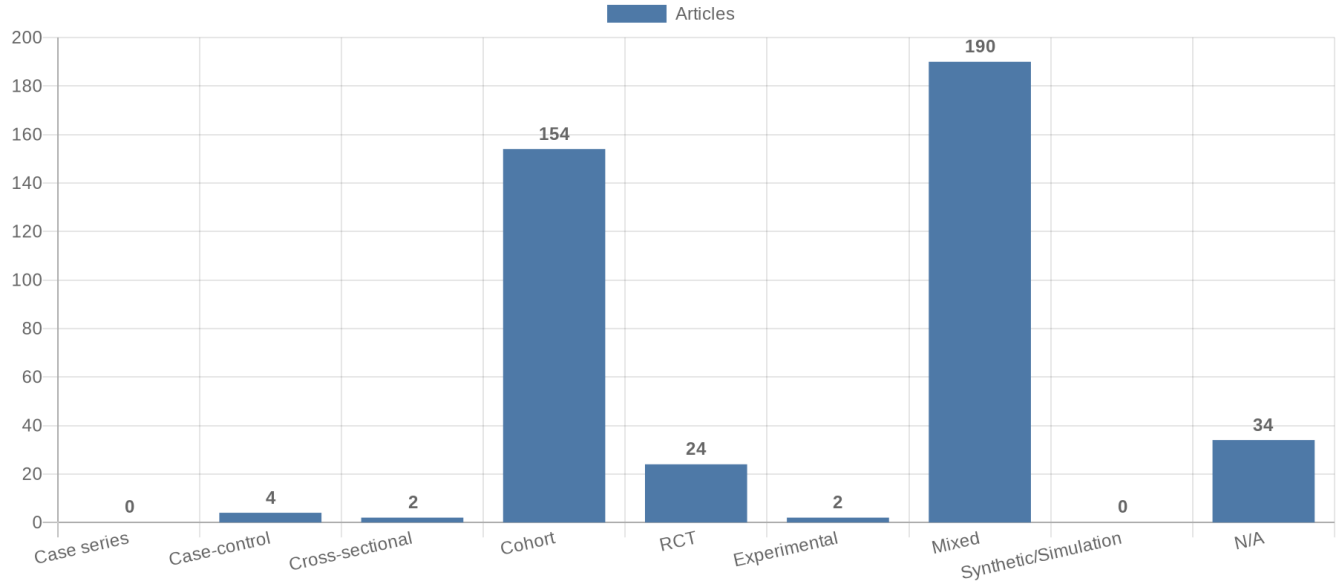

Figure 3. Study-type (directionality) distribution of included originals

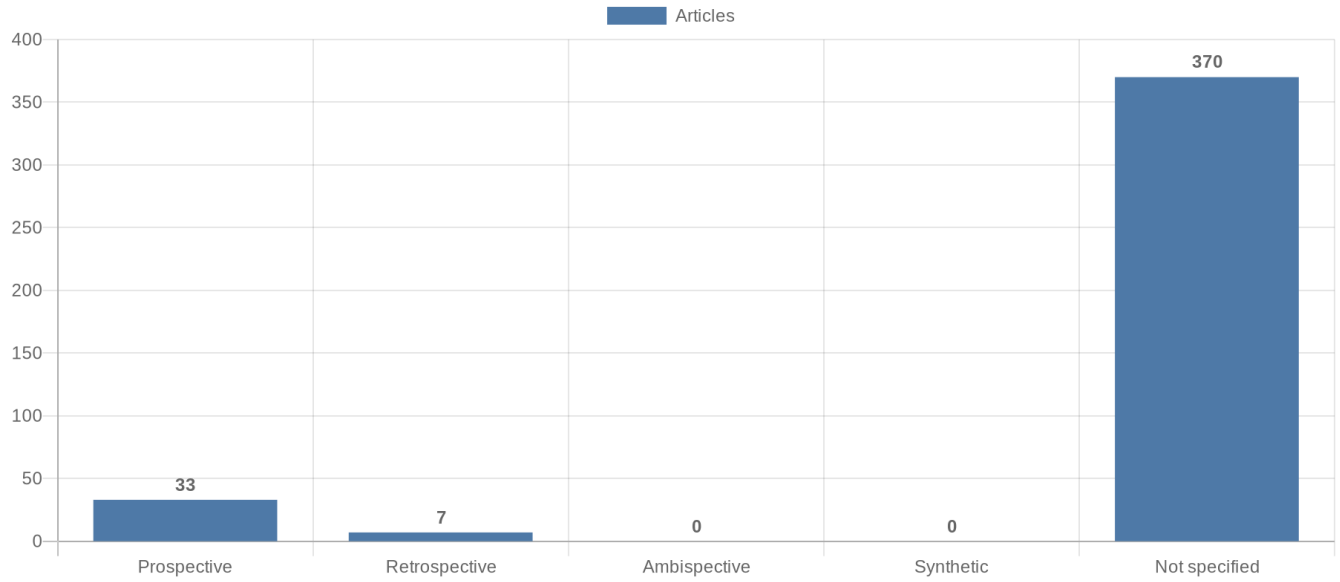

Figure 4. Main extracted research topics

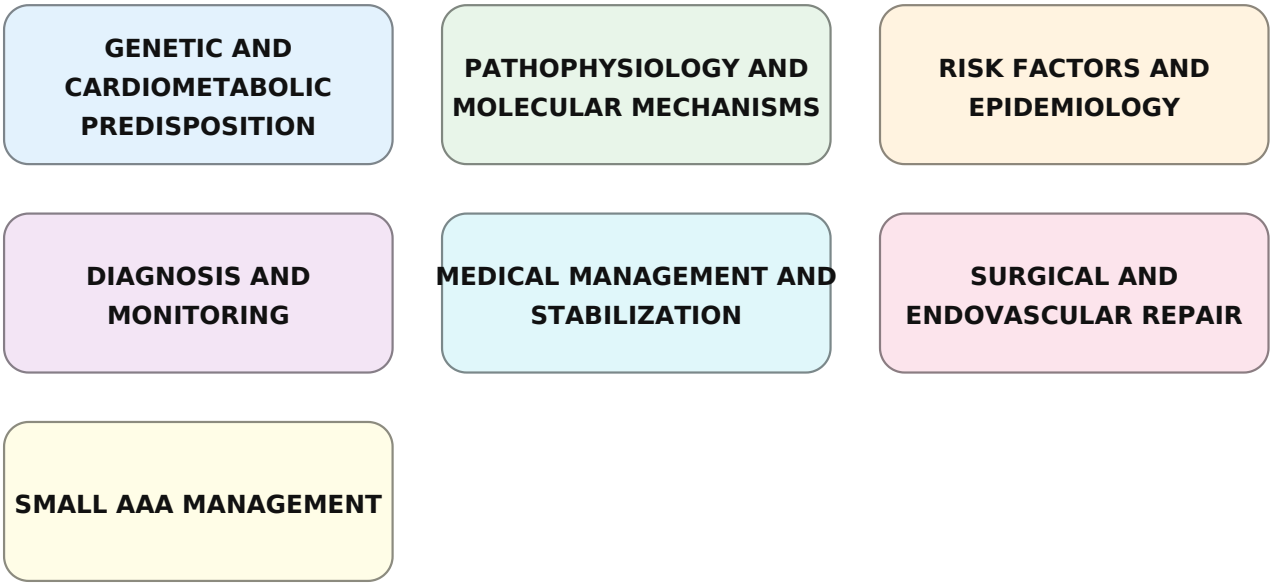

Figure 5. Limitations of current studies (topics)

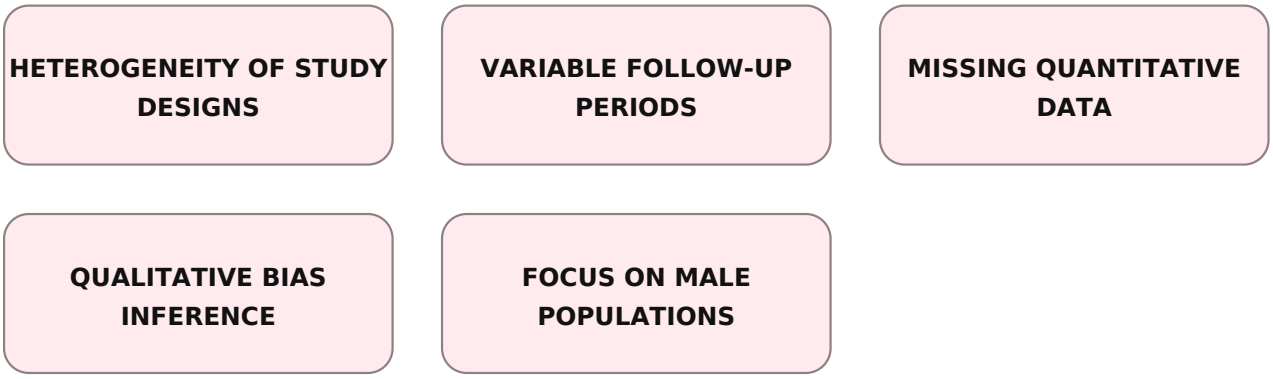

Figure 6. Future research directions (topics)

OPTIMAL MEDICAL THERAPIES

SEX-SPECIFIC PATHOPHYSIOLOGY

BIOMARKER INTEGRATION INTO PRACTICE

LONG-TERM EVAR OUTCOMES FOR RUPTURED AAA

PERSONALIZED RUPTURE RISK PREDICTION

STANDARDIZED OUTCOME REPORTING

SEX-SPECIFIC CLINICAL TRIALS