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The Role of Smoking and Alcohol in Arthritis Progression in East Africa

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ABSTRACT

Arthritis represents a growing non-communicable disease (NCD) burden in East Africa, driven by demographic transition, urbanization, lifestyle changes, and persistent inflammatory triggers. Tobacco smoking and alcohol consumption are modifiable behavioral exposures implicated in arthritis incidence, severity, and progression, particularly in rheumatoid arthritis (RA), osteoarthritis (OA), and gout. While extensive data exist from Europe and North America, evidence from East Africa remains fragmented. This review synthesizes current knowledge on the mechanistic and epidemiological links between smoking, alcohol use, and arthritis progression in East African populations. Smoking promotes citrullination, autoantibody formation, oxidative stress, and pro-inflammatory cytokine activation, accelerating RA severity and structural joint damage. Alcohol exhibits complex dose-dependent effects: moderate consumption may show immunomodulatory properties, whereas heavy use exacerbates gout, systemic inflammation, liver dysfunction, and medication toxicity. Sociocultural practices, co-infections, malnutrition, occupational exposures, and limited rheumatology services modify these associations in East Africa. We highlight regional knowledge gaps and propose research and public health priorities to mitigate arthritis progression through targeted behavioral interventions.

Keywords: Smoking, alcohol, rheumatoid arthritis, osteoarthritis, gout, inflammation, East Africa, disease progression

INTRODUCTION

Arthritis represents a heterogeneous spectrum of inflammatory and degenerative joint disorders characterized by chronic pain, stiffness, structural joint damage, and progressive functional impairment. The most prevalent forms include rheumatoid arthritis (RA), osteoarthritis (OA), and gout. RA is a systemic autoimmune disorder marked by persistent synovitis and progressive joint erosion; OA is primarily degenerative, involving cartilage breakdown and subchondral bone remodeling; and gout is an inflammatory arthritis driven by monosodium urate crystal deposition secondary to hyperuricemia [1]. Collectively, these conditions contribute substantially to global disability and reduced quality of life. Globally, musculoskeletal disorders rank among the leading causes of disability-adjusted life years (DALYs), with arthritis being a major contributor. While historically considered diseases of high-income countries (HICs), arthritis and other non-communicable diseases (NCDs) are increasingly recognized as significant public health concerns in low- and middle-income countries (LMICs), including those in Sub-Saharan Africa (SSA) [2]. Epidemiological transitions driven by urbanization, increased life expectancy, and lifestyle changes have accelerated the burden of NCDs in SSA. East African countries like Uganda, Kenya, Tanzania, Rwanda, Burundi, Ethiopia, and South Sudan are simultaneously confronting persistent infectious diseases and rising chronic inflammatory conditions, creating a dual burden of disease [3].

Recent data indicate that musculoskeletal disorders now account for a growing proportion of DALYs in SSA, with substantial impacts on workforce productivity and household economic stability. Despite this rising burden, arthritis remains underdiagnosed and undertreated in many East African settings due to limited specialist care, inadequate diagnostic facilities, and constrained access to disease-modifying therapies such as DMARDs and biologics [4]. Behavioral risk factors are increasingly shaping the epidemiology of chronic inflammatory diseases in the region. Tobacco use and harmful alcohol consumption have risen in tandem with rapid urbanization, globalization, and socioeconomic transitions. Tobacco smoking is a well-established risk factor for RA development and severity,

influencing immune tolerance, autoantibody production (e.g., anti-citrullinated protein antibodies), and systemic inflammation [5]. Alcohol consumption demonstrates complex, dose-dependent effects: while moderate intake has been associated with reduced RA risk in some HIC studies, harmful consumption contributes to metabolic dysregulation, hyperuricemia, oxidative stress, and immune dysfunction—pathways implicated in arthritis progression [6].

Smoking and alcohol consumption exert systemic biological effects that are relevant to arthritis onset and progression. Smoking promotes protein citrullination, particularly in genetically susceptible individuals carrying HLA-DRB1 shared epitope alleles, and amplifies inflammatory signaling via cytokines such as TNF-α and IL-6. It also accelerates oxidative stress, contributing to joint tissue damage. Alcohol affects multiple pathways linked to arthritis, including bone metabolism, immune regulation, gut microbiome composition, and purine metabolism—mechanisms especially important in gout and inflammatory arthritis [7].

However, most mechanistic and longitudinal evidence on these exposures comes from high-income countries (HICs), raising concerns about whether the findings apply to East African populations. East Africa is shaped by distinct contextual factors that may modify exposure–disease relationships, including genetic variation in immune-regulatory polymorphisms, high prevalence of chronic infections (e.g., TB, HIV, parasitic diseases) that alter immune function, and substantial nutritional variability (micronutrient deficiencies and protein-energy malnutrition) [8]. In addition, delayed diagnosis, constrained access to DMARDs/biologics, and widespread use of herbal/traditional therapies can influence arthritis trajectories and potentially magnify or obscure the effects of smoking and alcohol.

Despite a rising regional arthritis burden, there is limited East Africa–specific evidence on how tobacco use and harmful alcohol consumption affect disease severity and progression. Extrapolating from HIC studies may be inappropriate due to differences in genetics, infection profiles, nutrition, healthcare access, and cultural/socioeconomic determinants of substance use. Moreover, tobacco and alcohol control programs in SSA often prioritize cardiovascular disease and cancer, with less attention to musculoskeletal outcomes, limiting guidance for clinicians and policymakers. This review therefore aims to examine the biological mechanisms linking smoking and alcohol to arthritis progression; synthesize epidemiologic evidence across rheumatoid arthritis (RA), osteoarthritis (OA), and gout with emphasis on Sub-Saharan Africa (SSA) and other low- and middle-income country (LMIC) data; evaluate East Africa–specific modifiers; identify key research gaps; and outline the resulting clinical and policy implications. Its significance lies in supporting context-relevant prevention, integrating behavioral risk modification into arthritis care, and strengthening equitable evidence generation for the region.

Overview of Arthritis in East Africa

Arthritis is an increasingly important public health problem in East Africa, contributing to chronic pain, disability, lost productivity, and rising healthcare costs. The region is undergoing an epidemiological transition marked by increasing non-communicable diseases, longer life expectancy, and lifestyle changes, while still facing major constraints in specialist care and diagnostic capacity [9]. Consequently, many arthritis cases remain underdetected, and patients frequently present with advanced disease. Rheumatoid arthritis (RA), an autoimmune inflammatory arthritis, is estimated to affect ~0.1–1% of populations in Sub-Saharan Africa, although true prevalence is likely higher due to misdiagnosis and limited access to confirmatory testing (e.g., RF/ACPA) and imaging. In East Africa, delayed presentation is common, with patients often reaching health facilities after prolonged symptoms and established joint deformities [9]. Limited numbers of rheumatologists, inconsistent DMARD availability, and minimal access to biologic therapies contribute to persistent inflammation and accelerated disability.

Osteoarthritis (OA), the most common degenerative arthritis, increases with age and obesity, but is also strongly shaped by context-specific occupational exposures. Manual labor, subsistence farming, repetitive kneeling/squatting, and heavy load-carrying place chronic mechanical stress on knees, hips, spine, and hands, potentially accelerating joint degeneration even in younger adults.

Gout is also rising, particularly in urban centers, driven by dietary transitions, alcohol intake, obesity, and metabolic syndrome [10]. However, gout is frequently under-recognized, and limited access to urate-lowering therapy and long-term follow-up leads to recurrent flares, tophi, and preventable joint damage.

Smoking and Arthritis Progression

Smoking is an important, modifiable driver of arthritis progression in East Africa, where tobacco use varies widely across countries but remains highest among men, with rising uptake among women and youth. In rural settings, smokeless tobacco and locally rolled cigarettes are common, increasing exposure in populations with limited access to preventive health services. Biologically, smoking promotes arthritis through multiple interconnected pathways [10]. It induces peptidylarginine deiminase (PAD) activity in the lungs, leading to protein citrullination and the generation of anti-citrullinated protein antibodies (ACPA) in genetically susceptible individuals, particularly HLA-DRB1 shared epitope carriers—mechanisms strongly linked to aggressive rheumatoid arthritis (RA). Smoking also elevates pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6, and CRP, accelerating synovial inflammation, cartilage degradation, and osteoclastogenesis. In settings with limited biologic therapy access, this cytokine amplification may hasten radiographic progression [11]. Additionally, tobacco smoke generates reactive oxygen

species that damage cartilage and synovium and impair chondrocyte repair, while reducing bone mineral density via RANKL-mediated osteoclast activation. Clinically, smoking is associated with seropositive RA, higher disease activity, poorer treatment response, and potentially worsened oxidative cartilage damage in osteoarthritis; its role in gout is less direct but may operate through systemic inflammation and vascular dysfunction [12].

Alcohol Consumption and Arthritis Progression

Alcohol consumption includes commercially produced beverages and locally brewed drinks (e.g., waragi, chang'aa, busaa). Unregulated alcohol may contain contaminants affecting systemic health.

Table 1: Level of Consumption	Immunologic Effect
Low–Moderate	Possible suppression of certain pro-inflammatory pathways
Heavy/Chronic	Immune dysregulation, ↑ IL-6, TNF-α, gut permeability

In East Africa, harmful drinking patterns are more common than moderate controlled intake, increasing the likelihood of pro-inflammatory consequences.

Alcohol use has complex, arthritis-specific effects, and its impact in East Africa may differ from patterns reported in Western cohorts. In rheumatoid arthritis (RA), some studies from high-income settings report an inverse association between *moderate* alcohol intake and RA incidence, possibly reflecting anti-inflammatory or lifestyle confounding effects. However, heavy or harmful drinking clearly disrupts immune regulation, promotes systemic inflammation, and worsens comorbid risk profiles [13]. Clinically, alcohol is particularly important because it compromises RA treatment safety: hepatotoxicity and alcohol–drug interactions can limit methotrexate eligibility, increase liver enzyme abnormalities, and contribute to poor adherence and treatment interruption. These risks are amplified in East Africa, where background liver vulnerability is relatively common due to chronic viral hepatitis, potential aflatoxin exposure, and limited routine biochemical monitoring factors that can worsen treatment outcomes and accelerate disability [13]. Alcohol is also a major trigger for gout. It raises serum urate by increasing purine turnover, reducing renal urate excretion, and promoting lactic acidosis that enhances urate retention. Beer and spirits, widely consumed in many East African settings, are strongly associated with hyperuricemia, and when combined with urban dietary shifts toward processed foods, they can increase flare frequency and progression to chronic tophaceous gout [14]. For osteoarthritis (OA), alcohol appears to act mainly through indirect pathways such as obesity, muscle weakness, and increased risk of falls or joint trauma, while evidence for direct cartilage toxicity remains limited.

Contextual Modifiers in East Africa

In East Africa, the relationship between smoking, alcohol use, and arthritis progression is shaped by several contextual modifiers that may intensify disease severity compared with high-income settings. HIV remains prevalent in many communities, and in some contexts smoking and harmful alcohol use are more common among people living with HIV [15]. Persistent immune activation, along with antiretroviral therapy–related metabolic changes, may synergize with tobacco- and alcohol-driven inflammation to worsen joint damage, pain, and disability [16]. Tuberculosis and other chronic infections further complicate this landscape: smoking increases TB risk, and TB-associated immune dysregulation can obscure rheumatologic diagnosis and constrain treatment options, especially when immunosuppressive agents or biologics are considered. Nutritional challenges, including vitamin D, calcium, and antioxidant deficiencies, may amplify oxidative stress and bone loss linked to smoking and alcohol, accelerating structural deterioration [17]. Health-system constraints delayed diagnosis, limited specialist care, inconsistent DMARD supply, and minimal biologic access- mean that behavioral risks often operate against a backdrop of poorly controlled inflammation, leading to faster progression and worse functional outcomes. Gender and socioeconomic factors also matter: smoking is typically higher among men, yet arthritis disproportionately affects women, who may face second-hand smoke exposure and reduced financial capacity for sustained treatment [18]. Clinically, these realities support integrating smoking cessation and alcohol screening (e.g., AUDIT) into arthritis care to reduce flare risk, improve adherence, and prevent complications such as methotrexate hepatotoxicity. At the policy level, stronger tobacco and alcohol regulation, plus inclusion of musculoskeletal health in NCD strategies, is essential, while research priorities include registries, prospective cohorts, biomarker profiling, and culturally tailored cessation interventions [19].

CONCLUSION

Smoking and harmful alcohol consumption are significant modifiable drivers of arthritis progression in East Africa. Smoking accelerates RA through citrullination, cytokine amplification, oxidative stress, and bone loss. Alcohol demonstrates dose-dependent effects, with heavy consumption exacerbating gout, impairing immune regulation, and complicating pharmacologic management. Regional contextual factors including infectious diseases, delayed diagnosis, nutritional deficiencies, and limited rheumatologic infrastructure likely magnify these effects. Targeted

behavioral interventions, improved surveillance, and region-specific research are urgently needed to mitigate disability from arthritis in East Africa.

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