	Approve	Disapprove	Abstain
Aging Q1: Are the mechanisms of aging-associated OA different from those of post-traumatic OA (PTOA)?	74 (92.5%)	2 (2.5%)	4 (5.0%)
Aging Q2: CANCELLED			
Aging Q3: What other aging-associated musculoskeletal diseases (e.g. osteoporosis, sarcopenia, neurodegenerative, etc.) contribute to age-associate OA in animals?	58 (79.5%)	2 (2.7%)	13 (17.8%)
Aging Q4: Do all the hallmarks of aging play a role for aging associated OA development?	56 (77.8%)	11 (15.3%)	5 (6.9%)
Aging Q5: Does aging change how joint tissues respond to joint injury and thus alter PTOA outcomes?	70 (97.2%)	1 (1.4%)	1 (1.4%)
Aging Q6: What are the major strain and sex differences in animal models of aging associated OA and how should the field manage these differences?	53 (74.6%)	2 (2.8%)	16 (22.6%)
Aging Q7: What are the minimum outcome measures required to make conclusions about OA from aging in pre-clinical models?	45 (68.2%)	6 (9.1%)	15 (22.7%)
Aging Q8: Are Better Animal Models Needed Beyond C57BL/6 Mice to Study Aging-Associated Osteoarthritis?	54 (80.6%)	6 (9.0%)	7 (10.4%)
Aging Q9: CANCELLED			
Aging Q10: Most studies use null alleles to examine gene function. Should we focus on generating more relevant mouse models that use OA-associated coding or GWAS alleles, v	49 (75.4%)	5 (7.7%)	11 (16.9%)
Aging Q11: Should we recapitulate in our animal models the genetic-epigenetic interactions at play systemically during aging and OA?	56 (84.8%)	4 (6.1%)	6 (9.1%)
Aging Q12: Does aging alter communication between joint tissues (e.g., cartilage and subchondral bone, fat and cartilage, fat and bone, synovium and cartilage)? Are there intera	60 (92.3%)	2 (3.1%)	3 (4.6%)
Aging Q13: Given the known natural aging process, should preclinical experiments be designed longitudinally to determine whether a specific order of joint tissues is associated v	52 (82.5%)	3 (4.8%)	8 (12.7%)
Aging Q14: Do animal (especially mice) studies of aging-associated OA translate well to human diseases?"	31 (66.0%)	10 (21.3%)	6 (12.7%)
Aging Q15: Most studies use PTOA to test the efficacy of a DMOAD. Should this also be tested in an aging model?	54 (94.7%)	1 (1.8%)	2 (3.5%)
Aging Q16: What age is an appropriate age to study spontaneous age-related OA in mice?	51 (81.0%)	4 (6.3%)	8 (12.7%)
Aging Q17: Is there a need for better standardization in aging-OA animal studies (e.g. age, housing, diet, and water purification technique -> important from a microbiome persp	57 (89.1%)	5 (7.8%)	2 (3.1%)
Aging Q18: Is there a difference in how pain behavior testing should be conducted in mice with aging-related OA vs other types of OA models?	60 (95.2%)	0 (0.0%)	3 (4.8%)
Aging Q19: Do the Age-Related Changes in the ECM Promote Chondrocyte Senescence or Does Chondrocyte Senescence Promote Age-Related Changes in the ECM?	47 (77.0%)	1 (1.6%)	13 (21.4%)
Aging Q20: Are there genetic models of accelerated aging (e.g., Klotho mice) that present with aging-associated osteoarthritis (OA) at a young age?	50 (80.6%)	4 (6.5%)	8 (12.9%)
PTOA Q1: Is there a universal definition of post-traumatic osteoarthritis (PTOA)? Consider both etiology and pathology of the condition.	35 (81.4%)	7 (16.3%)	1 (2.3%)
PTOA Q2: Is there a clear understanding of how current major "PTOA" models induce disease? A) Joint destabilization, B) Mechanical loading, C) Osteochondral fragments	32 (72.7%)	11 (25.0%)	1 (2.3%)
PTOA Q3: Do age and post-injury time-point influence the biological response to joint injury and progression to PTOA?	44 (95.7%)	1 (2.2%)	1 (2.1%)
PTOA Q4: Do sex differences affect response to joint injury?	46 (97.9%)	1 (2.1%)	0 (0.0%)
PTOA Q5: CANCELLED			
PTOA Q6: Is there an accepted timeline for the appearance of PTOA that would most clearly relate to the clinical scenario?	35 (76.1%)	9 (19.6%)	2 (4.3%)
PTOA Q7: Are Appropriate Biomarkers Available for Post-Traumatic Osteoarthritis in Animal Models?	28 (62.2%)	15 (33.3%)	2 (4.5%)
PTOA Q8: Do current preclinical models have the appropriate features and outcome measures to appropriately evaluate therapeutic interventions for PTOA?	35 (81.4%)	8 (18.6%)	0 (0.0%)
PTOA Q9: Are there diagnostic or therapeutic interventions that could potentially mitigate or reverse PTOA?	36 (83.7%)	7 (16.3%)	0 (0.0%)
Obesity Q1: Can/should we define and model the distinct biological mechanisms (mechanical loading, systemic inflammation, adipokines, etc.) that link obesity and OA, and their	46 (97.9%)	0 (0.0%)	1 (2.1%)
Obesity Q2: Can/should we determine the specific effects of dietary composition (e.g. sugar, fat, ultra-processed foods) on OA development, independent of obesity or weight ga	41 (91.1%)	1 (2.2%)	3 (6.7%)
Obesity Q3: Should we determine whether metabolically healthy obesity (i.e. obesity without metabolic syndrome), body fat distribution or body composition contributes to OA	43 (89.6%)	1 (2.1%)	4 (8.3%)
Obesity Q4: Can/should we investigate the role of systemic and local immune responses, including adipose tissue inflammation and macrophage polarization, in obesity-induced	42 (89.4%)	2 (4.3%)	3 (6.3%)
Obesity Q5: Can/should we explore genetic, epigenetic and multi-omic mechanisms underlying the onset and progression of obesity-associated OA, including the influence of env	40 (85.1%)	3 (6.4%)	4 (8.5%)
Obesity Q6: Should we define standardized models, metrics, and outcomes for obesity and weight loss in preclinical OA studies—including metabolic profiling, body composition	40 (83.3%)	3 (6.2%)	5 (10.5%)
Obesity Q7: Can/should we use or develop large animal models (e.g., pigs, dogs) or naturally occurring companion models (i.e. cats, dogs, guinea pigs) to better replicate human	44 (91.7%)	2 (4.2%)	2 (4.1%)
Obesity Q8: Can/should we complement animal models with advanced in vitro and in silico models, including organ-on-chip approaches and AI-driven models?	39 (83.0%)	6 (12.8%)	2 (4.2%)
Obesity Q9: Can/should we stratify and align preclinical models with specific human endotypes/phenotypes of obesityrelated OA (e.g., metabolic, mechanical, inflammatory)?	42 (93.3%)	2 (4.4%)	1 (2.3%)
Obesity Q10: Can/should we harmonize tissue sampling, analysis protocols, and pain measures in obesity-OA models to improve reproducibility and translatability?	38 (84.4%)	2 (4.4%)	5 (11.2%)
Obesity Q11: Should we determine pain mechanisms unique to obesity-associated OA, including central sensitization, peripheral nerve sprouting, or neuroinflammation? Should	40 (90.9%)	3 (6.8%)	1 (2.3%)
Obesity Q12: Are pain mechanisms and responses distinct in obesity induced OA?	39 (88.6%)	3 (6.8%)	2 (4.6%)
Obesity Q13: Can/should we investigate the interaction between obesity, pain, and physical activity/exercise or function—and how this impacts osteoarthritis outcomes?	43 (93.5%)	1 (2.2%)	2 (4.3%)
Obesity Q14: Can/should we study the interorgan crosstalk between joint tissues and distant organs (e.g., brain, adipose tissue, gut, cardiovascular system, reproductive system)	42 (91.3%)	1 (2.2%)	3 (6.5%)
Obesity Q15: Can/should we investigate the transgenerational or intergenerational effects of diet and obesity on OA risk, and how these effects are mediated?	40 (88.9%)	2 (4.4%)	3 (6.7%)
Obesity Q16: Can/should we consider systemic hormone changes (e.g., menopause, andropause, pregnancy) and sex as biological variables in the interaction between obesity an	40 (88.9%)	1 (2.2%)	4 (8.9%)
Obesity Q17: Can/should we determine whether obesity accelerates aging using OA as a model - structurally, molecularly, and functionally?	38 (88.4%)	2 (4.7%)	3 (6.9%)
Obesity Q18: Can/should we explore how obesity, OA, and pain independently and synergistically influence markers of healthspan, frailty, and chronic disease progression, espec	36 (90.0%)	0 (0.0%)	4 (10.0%)

Obesity Q19: Can/should we evaluate the effect of interventions for obesity (e.g., dietary changes, exercise, pharmacological compounds, bariatric surgery) on OA development, symptoms, and progression in preclinical models?	34 (87.2%)	3 (7.7%)	2 (5.1%)
Obesity Q20: Can/should we prioritize testing of obesity-related OA therapeutics in preclinical models, including targeting systemic inflammation, mechanical overload, or neuro	38 (90.5%)	2 (4.8%)	2 (4.7%)
Pain Q1: Should we define a standard minimum number of pain-dependent behavioral outcomes that constitute a robust, standardized pre-clinical OA study (i.e., that test differ	55 (88.7%)	4 (6.5%)	3 (4.8%)
Pain Q2: Can/should we define a minimum set of OA structural measures/outcomes (and their severity) to be used to define OA and thus OA-associated pain for each preclinical	52 (89.7%)	4 (6.9%)	2 (3.4%)
Pain Q3: Can/should we define a minimum time-course over which pain (and associated joint pathology) outcomes should be evaluated, and does this vary with the pre-clinical r	52 (89.7%)	4 (6.9%)	2 (3.4%)
Pain Q4: Can/should we define an optimal set of species (rodents vs. large animal models) in which to model, study mechanisms, and test treatment of OA pain?	42 (72.4%)	13 (22.4%)	3 (5.2%)
Pain Q5: What is the role of studies in veterinary populations with clinical osteoarthritis, such as pet dogs or horses, or equine athletes?	52 (89.7%)	1 (1.7%)	5 (8.6%)
Pain Q6: How can AI improve the reliability of operant, reflex, or observational nociceptive assays?	48 (84.2%)	4 (7.0%)	5 (8.8%)
Pain Q7: CANCELLED			
Pain Q8: Can we identify pain mediated by nociceptive mechanisms, neuropathic, and nociplastic mechanisms in experimental models of OA?	50 (89.3%)	4 (7.1%)	2 (3.6%)
Pain Q9: Should we measure pain vs function vs disability in preclinical OA models – to parallel human “OA illness”?	51 (96.2%)	1 (1.9%)	1 (1.9%)
Pain Q10: Can/should we include measures on the affective aspects of OA-associated pain e.g. depression, anxiety etc?	48 (88.9%)	2 (3.7%)	4 (7.4%)
Pain Q11: Which mechanistic endpoints should be incorporated in preclinical experiments to complement pain-related behavior assessment (e.g., should we do electrophysiolog	47 (87.0%)	2 (3.7%)	5 (9.3%)
Pain Q12: What are the critical confounders to be considered in preclinical rodent models?	48 (88.9%)	2 (3.7%)	4 (7.4%)
Pain Q13: Can/should we develop guidelines to standardize the classification of studies testing for prophylactic versus therapeutic treatment effects?	43 (84.3%)	3 (5.9%)	5 (9.8%)
Pain Q14: Can/should we define a minimum level/size for each pain-dependent behavioral outcome that is relevant/clinically meaningful? This would be both the size of change	49 (86.0%)	6 (10.5%)	2 (3.5%)
Pain Q15: Can/should we define a standard treatment or set of standard positive control treatments for use across studies to enable standardization and comparison (and argual	40 (81.6%)	5 (10.2%)	4 (8.2%)
Pain Q16: Are there systemic biomarkers of pain that could be used (in parallel with or as surrogates forpain behaviors) in pre-clinical models to define pain phenotypes/chronici	45 (86.5%)	2 (3.8%)	5 (9.7%)
Pain Q17: Are there any animal-free in vitro/in silico approaches in development that can replace animal models for studying mechanisms of OA pain?	33 (63.5%)	14 (26.9%)	5 (9.6%)
Microbiome Q1: Does microbiome (dysbiosis) play a role in causing degenerative spine disease that can be studied in animal models?	33 (82.5%)	3 (7.5%)	4 (10.0%)
Microbiome Q2: Can biological manipulation of the microbiome, such as administration of prebiotics or probiotics have a role in alleviating the pain of osteoarthritis in preclinica	34 (82.9%)	4 (9.8%)	3 (7.3%)
Microbiome Q3: Do therapeutic modulations of the gut microbiome mitigate osteoarthritis from animals to clinical efficacy?	24 (75.0%)	1 (3.1%)	7 (21.9%)
Microbiome Q4: Can the gut microbiome play a role in causing osteoarthritis that can be studied in animal models?	35 (87.5%)	3 (7.5%)	2 (5.0%)
Microbiome Q5: Does the gut microbiome play a role in modulating innate or adaptive immunity of the host that could interact with osteoarthritis?	35 (87.5%)	1 (2.5%)	4 (10.0%)