MSTS 2026 Abstract Submission Guidelines

The MSTS Program Committee welcomes abstracts related to all aspects of Musculoskeletal Tumors.

If you are an MSTS member, or if you have submitted an abstract to MSTS in the past, please begin your 2026 submission by entering your username and email address. This is necessary to tie your submission to your existing record – please **do not** create a duplicate record in your name. If you need to confirm your username, please contact MSTS staff at info@msts.org.

If you are not a member of MSTS and have not previously submitted an abstract, please enter your email address in the New User section of the form. You will then be able to provide the information needed to create a non-member user record and to submit your abstract.

We require ELECTRONIC SUBMISSION of your abstract through the MSTS Abstract Submission program. Abstracts will not be accepted by email.

Note: Please be sure you have all necessary information, including your blinded and unblinded abstract files, ready before beginning the abstract submission. Once you submit your abstract, you will not have the option to add/change information, change authors, or make any edits.

Important Submission Information:

1. **MSTS Membership:** We encourage the submitter, or at least one listed co-author, to be a current member of MSTS (all membership categories qualify). Non-MSTS authors are also eligible for the submission of abstracts.
2. **Co-Author Information:** You must have the full name and email address of every co-author on the abstract. A maximum of fifteen (15) co-authors can be included.
3. **Industry Representatives:** Industry representatives are not eligible to submit abstracts.
4. **Financial Disclosures:** Financial Disclosure is required for the submitter, presenter, and each co-author: The disclosure must be on file with AAOS with a disclosure submission date of October 29, 2024, or later. To verify if an existing disclosure on file is current, or to create a disclosure file, please click here for the AAOS Disclosure Program.
5. **Presenter Information:**
    - If your abstract is selected for presentation, the full name and email address of the person who will present your abstract at the 2026 MSTS Annual Meeting is required.
    - All presenters must register to attend the 2026 MSTS Annual Meeting.
    - Presenters should hold an MD or PhD Degree. If non-MD/PhD trainees present from the podium, the corresponding author/principal investigator should be available for discussion.
    - Industry representatives are not permitted to present either a poster or podium presentation at the MSTS Annual Meeting.
6. **Abstract Category** *New*: Note new categories for 2026 are listed on page 3 of these guidelines. Only one category can be selected for each abstract.
7. **Uploading Abstracts**: you are required to upload **both** a blinded and unblinded version of your abstract. (Blinded - No Authors/Co-Authors or Institution names should be included. Unblinded - ALL Authors/Co-Authors and Institution names should be included).

Abstract Structure for Submission *New*: Please see the following information regarding the organization and content of your abstract:

1. **Structure** *New*:
    a. **Significance & Hypothesis/Techniques**: Why should the study be done? The importance of the proposed study in the field; Knowledge or technical barrier. Technology-driven research describes the purpose. A hypothesis-driven research clearly states a hypothesis. What is the goal of the study?
    b. **Innovation**: What is new? Are there technical, conceptual, or clinical Innovation?
    c. **Approach** (Materials & Methods): How was this study done? Rigor (control/intervention groups; sample size/statistics; biological variables; methods).
    d. **Results**: Clinical or Scientific Data, Figure panels show pre, intra, post-operative, follow-up images, or clinical photos; diagrams, graphs, tables.
    e. **Discussion & Impact**: How does the study influence or change current knowledge, techniques, or practice.
2. **Length**: Please submit a Microsoft Word document that **does not exceed 500 words.** Abstracts greater than 500 words will not be reviewed. Figures and tables, authors, affiliations, references, and abstract title do not count towards the 500-word limit.
3. **Figure Panels and Tables**: Limit 3 (Do not count toward the 500-word limit). A composition of Figures in one Figure Panel is counted as one Figure.
4. **Font:** Standard Calibri font, type size 10pt.
5. **Format:** Abstract should be single spaced with a 1-inch margin both on the top and bottom as well as the left and right sides.
6. **References**: Please omit any reference to authorship and/or institution within the body of the abstract.

Posters

MSTS recognizes poster presentation highly valuable for interactive learning and networking. All posters will be electronically displayed on the LED screen and the 2023 Annual Meeting App.

Poster Award Finalists: Twelve (12) top-scored posters from MSTS ESIs will be selected. Twenty-four (24) Posters (Paper or Fabric-Traditional Posters) will be displayed. In addition, each author of the top posters will present a summary of their work on the podium during the Poster Award Teaser Session. The presentation format will be forwarded to the finalists.

2026 Abstract Awards

The 2026 Annual Meeting Planning Committee will review all posters and podium presentations and select the recipients of the following awards. You may select all awards that are applicable; but no single abstract will receive more than one award.

1. Best Paper (Podium) Presentation (1st Place, $750; 2nd Place, $500; 3rd Place, $250
2. Best Poster (1st Place, $750; 2nd Place, $500; 3rd Place, $250)
3. Best ESI Paper (Podium) Presentation (1st Place, $750; 2nd Place, $500; 3rd Place, $250
4. Best ESI Poster (1st Place, $750; 2nd Place, $500; 3rd Place, $250)
5. Best ESI Research Proposal Award (1st Place, $750; 2nd Place, $500; 3rd Place, $250)

2026 Abstract Categories - Only one category can be selected for each abstract.

NEW for 2026 abstract submissions, Early-Stage Investigators (ESI) are defined as A Corresponding Author & Principal Investigator, First author-Corresponding author, or First author -Principal Investigator within six (6) years of their first faculty appointment at the time of the abstract submission.

ESIs can submit using either a one-page Specific Aims Research Plan (Category 6 below) or with a traditional abstract (Category 7 below). **All ESI submissions, regardless of the research topic, should be entered under one of the Early-Stage Investigator categories.**

1. **Bone Tumors** (Benign & Malignant)
2. **Soft Tissue Tumors** (Benign & Malignant)
3. **Metastases & Tumor-Like Diseases**
4. **Interactive Cases** ***New***: Disasterplasty, Technical/Conceptual Innovation, Complications & Management, Onco-plastics, Multi-Disciplinary Tumor Board (Pathology). Interactive cases may have a small sample size, but interactive educational value with respect to clinical judgement or care is very high. Experts in complex reconstructive surgeries and pathology will be moderators.
5. **Translational Science & Technology**: Digital Health, AI, Biology, Pathology, Immuno-Oncoogy
6. **Early-Stage Investigators (ESI)** ***New*** – **One-page Specific Aims Research Plan**. (0.5-inch margin, 11 arial-font, single-spaced). (Please see page 4 and 5 for Sample Specific Aims forms). Real-time feedback will be provided by MSTS members with prior federal funding. Mentor-mentee will be matched if desired. The goal of this program is to facilitate grant applications for future publications for MSTS ESIs. Only MSTS candidate- and active- members can submit Specific Aims for scholarly mentorship by MSTS Established Investigators.
7. **Early-Stage Investigators (ESI)** ***New*** **Abstract**– Traditional abstracts as described in page 2.

Thank you for your interest in submitting an abstract to the MSTS 2026 Annual Meeting. Please contact the MSTS office with any questions via email at info@msts.org or by phone at (847) 698-1625.

Thank you,

2026 Annual Meeting Program Committee
Musculoskeletal Tumor Society
1515 East Woodfield Road, Suite 345
Schaumburg, IL 60173

SPECIFIC AIMS (K08 Specific Aims Page; Kindly Provided by Dr. Christopher Collier, K08 Recipeint)
Cachexia, a multifactorial syndrome characterized by systemic muscle wasting and bone loss, is responsible for 20 to 40% of cancer-related deaths[51]. At autopsy up to 70% of cancer patients have metastatic bone disease[18]. Recent evidence suggests that bone metastases have systemic consequences, including cachexia[53,54]. Current treatment of bone metastases is mostly palliative, yet as cancer patients live longer and these systemic effects are better understood, this paradigm will likely evolve to include more aggressive interventions such as targeted medical or surgical therapies. There is a *critical need* to describe the contribution of metastatic bone disease to cachexia and define the key mediators involved to optimize care of persons living with metastatic bone disease.

I am an orthopedic surgeon who has dedicated my career to understanding the interaction between cancer and the musculoskeletal system. My *long-term scientific goal* is to develop cachexia treatments in the setting of metastatic bone disease. Such therapies can mitigate the impact cancer has on the musculoskeletal system and improve patient quality of life and survival. Renal cell carcinoma (**RCC**) is of great interest for this application since it frequently metastasizes to bone, 40% of RCC patients develop cachexia, and the prognosis is relatively favorable to allow time for therapeutic benefit. My *overall objective* is to determine the contribution of metastatic bone disease to cachexia, and the factors responsible, in RCC. Circulating factors such as IL-6 drive cachexia and are released directly by the tumor cells or indirectly by the microenvironment. My preliminary data suggests that muscle loss greater than 20% per year is the single greatest predictor of overall survival for persons living with RCC and is associated with osseous metastases at diagnosis. Additional preliminary data in mice and human patients with RCC metastatic to bone demonstrate increased muscle wasting, serum IL-6, and JAK/STAT3 signaling in muscle, compared to those with extraosseous disease. Therefore, my *central hypothesis* is that *metastatic bone disease in RCC increases systemic muscle wasting and bone loss by IL-6 mediated activation of JAK/STAT3 signaling in muscle.* The following specific aims will test this hypothesis:

AIM 1: Determine the impact of metastatic bone disease on systemic muscle wasting and bone loss in RCC *in vivo*. I *hypothesize* that metastatic bone disease increases systemic muscle wasting and bone loss, compared to localized or extraosseous metastatic disease. The human metastatic cell line Caki-1 will be implanted into mice by varying initial cell number and tumor location: kidney (localized), lung (extraosseous), or tibia (osseous). Tumor-bearing and sham mice will then be assessed for cachexia: body composition by weight and EchoMRI; muscle function and wasting by *in vivo* functional analysis, muscle weight, and fiber analysis; and bone loss by μCT and biomechanics. To account for variation in tumor burden across models, tissue-specific and overall tumor burden will be assessed by *in vivo* PET/MRI and real-time quantitative PCR.

AIM 2: Determine the effect of metastatic bone disease on the IL-6 pathway and the impact of IL-6 blockade on systemic muscle wasting and bone loss in RCC. For Subaim 2A, I *hypothesize* that metastatic bone disease, compared to localized or extraosseous disease, increases serum IL-6 levels and downstream JAK/STAT3 signaling in muscle. Mouse sera from Aim 1 will be analyzed by multiplex immunoassay. IL-6 and other pro-cachectic factor concentrations will be compared. Mouse muscle will be assessed for STAT3 phosphorylation by western blot. For Subaim 2B, I *hypothesize* that IL-6 blockade reduces systemic muscle wasting and bone loss in a model of metastatic bone disease. Caki-1 cells, with or without IL-6 deletion by CRISPR/Cas9, will be implanted into mice tibiae and treated with or without an antibody selective for mouse IL6. These experiments will compare the impact of tumor- vs microenvironment-derived IL-6 on cachexia and tumor burden, and provide important data for possible future studies using FDA-approved IL-6 pathway inhibitors.

AIM 3: Establish the contribution of metastatic bone disease to systemic muscle wasting, bone loss, and its effect on the IL-6 pathway in RCC *patients*. I *hypothesize* that RCC patients undergoing surgery for metastatic bone disease, compared to those undergoing nephrectomy for localized disease, will show increased plasma IL-6 and downstream JAK/STAT3 muscle signaling. Plasma concentrations of IL-6 and other factors will be determined by proteomic analysis after global protein quantification. Muscle will be evaluated by histomorphometry, western blotting and RNAseq for IL-6-related and catabolic signaling pathways. These results will be interpreted alongside clinical factors including demographic, tumor characteristics, treatment history, and body composition analysis by artificial intelligence-assisted evaluation of abdominal CT scans.

IMPACT: I *expect* these studies will elucidate the roles of metastatic bone disease and the IL-6 pathway in driving cachexia in RCC. This work should identify new pathways underpinning cachexia and explore how the bone microenvironment shapes the systemic responses to cancer. *My long-term career goal* is to become an independent, NIH-funded orthopaedic surgeon-scientist. The work and mentored experiences outlined in this application will allow me to develop needed skills in musculoskeletal and cancer biology, and position me to compete successfully for subsequent R01 funding focusing on cachexia and metastatic bone disease.

Sample of NIH-Funded One-Page Specific Aims for MSTS Early-Stage Investigators (OREF/NCI R01)

Significance (***Should this study be done?; Why? Is the proposed study important in the field?)*** This proposal focuses on impaired pathological fracture healing in the presence of breast cancer cells, not on the entire complex sequence of cancer spread from the breast to bone. Metastatic cancer cells can settle anywhere in the bone including bone marrow, cortical bone, or outside of the bone. Cancer cells are spilled in and out of bone at the time of fracture. Pathological fracture calluses are deficient and very often fail to ossify despite adjuvant therapies such as radiation therapy and anti-resorptive agents including denosumab or bisphosphonates. Failed fracture healing is not solely a result of excessive bone resorption. There is no mechanistic understanding as to why fractures do not heal well in the presence of certain types of breast cancer cells. If there is a way of addressing impaired fracture repair while also inhibiting cancer growth in bone, the clinical care for pathological fractures will be immensely impacted. One of the most common types of pathological fracture is caused by metastatic breast cancers. We conducted whole transcriptome bulk RNAsequencing of several different types of breast cancer cells that are grown in the breast and bone separately. We observed that breast cancer cells that inhibit fracture repair. Our long-term goal is to develop a novel therapeutic strategy to improve healing of pathological fractures and local cancer control.

Innovation (***What's 'New'?, Technically, Conceptually, or Clinically New?: 'Repeat' Study?)***
• A murine pathological femoral fracture healing model: We established a clinically relevant animal model representing fracture repair in the presence or absence of breast cancer cells at the fracture sites. The model was rigorously characterized with radiographs, microCT, histology, immunoblotting, and gene expression.
• 3D capturing of early-stage fracture callus repairing cells and inflammatory cells in the fracture hematoma from the Day 1-4 fracture sites: Ex vivo experiments for molecular-interaction and drug screening.
• Mechanistic rescue of impaired fracture healing in a setting of personalized precision medicine: We plan to use genetic and pharmacological loss-of-function approaches to rescue impaired pathological fracture healing.

Hypothesis-driven Study or Technology-driven Study: '*Our goal is to develop/establish a xx technique'*:
We posit a **central hypothesis** that highly inflammatory pERK1/2-high breast cancer cells inhibit callus formation and maturation by causing prolonged hyper-inflammation and subsequent derangements of callus spatial transcriptomics. We propose 2 Specific Aims.

Approach (***Feasibility: Can this be done?; Rigor: Control/Intervention/Data Readouts/Statistics?***)
Aim 1. To define temporal and spatial transcriptomic changes of inflammatory, osteogenic, and chondrogenic cells in structurally intact fracture callus in the presence of breast cancer cells *in vivo*.
We will define how MEK1-pERK1/2-high breast cancers with pro-inflammatory features inhibit the formation and maturation of soft and hard callus with respect to expression of inflammatory cytokines and bone-acting proteins using a spatial transcriptomic approach alongside multiplex immunohistochemistry, radiographs, microCT, histology, and torsional biomechanical testing at various time points from early to late stages of fracture healing.

Aim 2. In Vivo Pre-clinical Therapeutic Translation: To establish a mechanism-based rescue of impaired pathological fracture healing by targeting specific pro-inflammatory kinases *in vivo*.
We will conduct blinded therapeutic trials in murine femoral fractures in the presence of pro-inflammatory breast cancer cells. Treatment groups will receive a specific allosteric reversible MEK1-pERK1/2 inhibitor. Bisphosphonates will be used to decouple the potential inhibition of osteoclasts from the kinase inhibitors. We will assess the formation and ossification of fracture calluses by radiographs, microCT, biomechanical torsional testing, and femur histology in cancer-bearing mice with and without therapeutic drug treatments at early, middle, and late stages of fracture healing after inoculation of $1x10^4$ breast cancer cells at the fracture sites. We will define specific transcriptomic changes that are reversed by MEK1-pERK1/2 inhibition in the structurally intact fracture callus. Statistical validation and power analysis will be conducted using SAS and R packages.

Overall Impact (Sustained influence on MSK tumors/clinical practice?): We are poised to unravel novel temporal and spatial transcriptomic changes during normal and impaired pathological fracture healing. We expect to develop a novel therapeutic paradigm for cancer patients suffering from pathological fractures.
Career Goals & Publication/Grant Application Plans*(This section is not usually included in the standard grant format. This section is to provide a moment to think about a long-term program, not many unrelated projects)*: I will seek an academic clinician track at the ABC Cancer Center. I will generate one or two sets of preliminary data and will apply for an MSTS New Investigator Grant, OREF, and MTF grant over the next 2 years. I will prepare one or two manuscripts in the next 2 years.