Materials/Methods: 1 participant ran on a treadmill that was accelerating between speeds of 0 and 18 Km/h in 2 minutes. CODA automated motion analysis system was used to collect 3D kinematic data from 14 markers placed on the head, shoulders, elbows, wrists, hips, knees, ankles of the participant (sampling frequency 100 Hz, trial length 120 s). Marker position and acceleration data were analysed in the x, y and z directions.
Individual and combined sensor measurements were resampled at 4000Hz to generate audible waveforms. Sonograms were then computed using moving Hanning windows for all the sound signals computed for each marker and combination of markers.
Results: Sonification of individual and combined markers are shown in Figure 1. The transition between the walking and running gaits is clearly visible in all of the sonograms (Figure 1). Sonification of individual markers (Figure 1, top left) shows the frequencies underpinning the marker movement. Combining the markers, sonification shows the cancellation and enhancement of frequencies involved in the gait as a result of coupling the marker waveforms (Figure 1, top right and bottom).

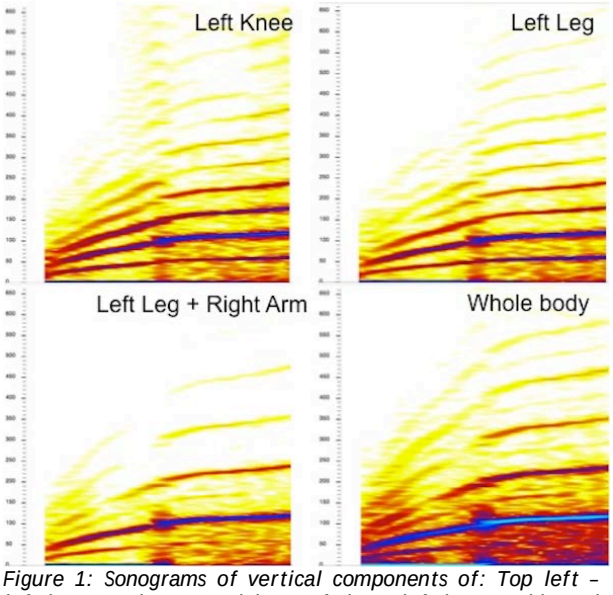

Figure 1: Sonograms of vertical components of: Top left – left knee marker; Top right – Left leg = left knee, ankle and hip markers; Bottom left – Left leg combined with Right arm = right wrist, elbow and shoulder markers; Bottom right – Whole body = combined left leg, right leg, left arm, right arm and head markers

Conclusion: Sonification provides a measure that clearly shows the phase transition between walking and running gaits. Furthermore, this measure is individual specific and situation specific. It is proposed that this method could be a used as a key tool for understanding and identifying and tracking changes in pathological or perturbed gaits; informing health practice.

Keywords: clinical gait analysis, sonification, movement analysis, coordination, biomechanical modeling

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Inhibiting the actions of cathepsin L effects both tumour initiation and metastasis formation.
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Purpose: Cathepsin L (CTSL) has been shown to play a role in tumour development and progression through its proteolytic activities. An increased expression and secretion of CTSL from cancerous cells enhances the tumour cell migration, aids in tumour invasion and promotes angiogenesis. In the present study we have investigated the role of the small molecule cathepsin L inhibitor, KGP94, on tumour initiation and metastatic spread in murine tumour models.
Methods: The C3H mammary carcinoma was implanted subcutaneously into the foot of male CDF1 mice. The SCCVII squamous cell carcinoma was either implanted into the foot or intravenously injected into the tail vein of C3H/HeNHsd mice. KGP94 was prepared by dissolving in a mixture of 10% Tween 80 and 90% HEPES-buffer. It was intraperitoneally (i.p.) injected at 0.01ml/g mouse bodyweight. Various doses (1-20mg/kg) were administered starting from the day of tumour implantation/injection. Tumour response was assessed by either determining the tumour growth time for foot tumours or the number of lung metastasis for the injections. Tumour growth time was assessed using a caliper and was the time in days to reach a volume of 500mm^3 (TGT_{500}). Lung metastasis were assessed after 2-3 weeks, where mice were euthanized, lungs were excised, weighed, and stained in Bouin's solution. Results are listed as Mean (± Standard Error). One-way ANOVA comparison of group means was performed, and a $P<0.05$ was considered significant.
Results: The TGT_{500} for control animals was 18.3 days (± 0.4) for the C3H mammary carcinoma and 13.6 days (± 0.7) for the SCCVII carcinoma. Treating the C3H mammary carcinoma with KGP94 significantly increased the TGT_{500} when doses were at 5.0 mg/kg or higher. Similar results were found with the SCCVII-carcinoma, except that a dose of 5.0 mg/kg did not have a significant effect on TGT. At lower doses of KGP94 neither of the tumour models showed significant growth delay. Studies on metastasis formation showed that 50% of animals in the control group developed metastasis within 2-3 weeks. The mean number of metastasis in these mice was 16 (±15). When mice were treated with KGP94 (10mg/kg) on a daily basis only 30% developed metastasis, and the mean number of metastasis in these mice was 5 (±4).
Conclusion: We have shown that inhibiting cathepsin L during the tumour initiation stage significantly delays tumour growth in the C3H and SCCVII murine tumour models. Furthermore, our metastasis study showed decreased metastasis formation in KGP94 treated animals. This suggests that KGP94 treatment can affect both tumour initiation and metastasis formation.

Keywords: Tumour growth inhibition; metastasis formation, cathepsin inhibitor KGP94.

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New hypoxia probe development based on mass spectrometry
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Many human tumors contain substantial regions of low oxygen (hypoxia), which promotes metastasis and resistance to most forms of therapy. Unfortunately, the methods available to assess cellular hypoxia are unable to detect the fluctuating oxygen concentrations that are proposed to be an important source of these cellular phenotypes, and similarly cannot detect changes in hypoxia as a consequence of treatment. We have established a novel method that enables measurement of dynamic changes in hypoxia at the cellular level. We developed a series of small molecule probes with identical chemical structures but containing different isotopes of tellurium that can be independently quantified by mass cytometry (MC) and imaging mass cytometry (IMC). This