[Home-Based Wrinkle Reduction Using a Novel Handheld Multisource Phase-Controlled Radiofrequency Device (researchgate.net)](https://www.researchgate.net/publication/269872982_Home-Based_Wrinkle_Reduction_Using_a_Novel_Handheld_Multisource_Phase-Controlled_Radiofrequency_Device)

62 participants aged 37-72 took home a home care radiofrequency hand held tool for 1MHZ power to use on their face at least 4XMonth for 3 months, and the results were checked weekly and 1 month and 3 months post-op. The wrinkles significantly diminished as well as skin pigmentation disorders as measured by a dermatologist and using skin imaging technology. There were no side effects or disorders noticed or reported

[What are Radio Frequency bands and its uses? - RF Page](https://www.rfpage.com/what-are-radio-frequency-bands-and-its-uses/)

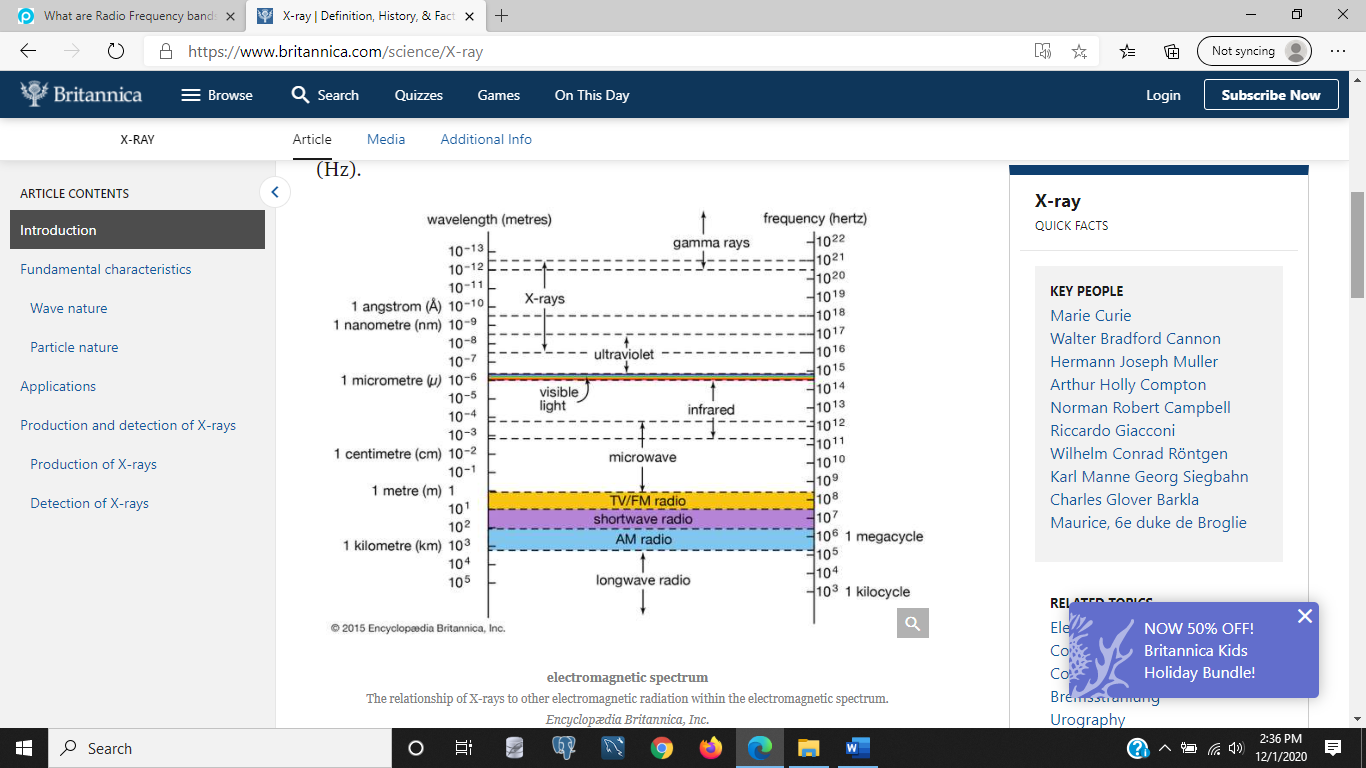
The 30kHZ to 3MHZ radio frequencies are on the light (30kHZ-300kHZ) and medium(300kHZ-3MHZ) frequencies of the electromagnetic spectrum respectively. The light frequency wavelength or height of the curve is 1-10 km and the medium wavelength is 100m-1km high. The apparatuses associated with these two frequencies are maritime for low frequencies around 40kHZ, aviation and am radio for medium frequencis around 1MHZ, and navigation for both frequency groups.

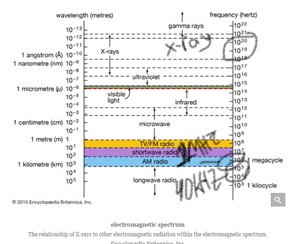
[Bipolar radiofrequency in the treatment of dermatologic imperfections: clinicopathological and immunohistochemical aspects - PubMed (nih.gov)](https://pubmed.ncbi.nlm.nih.gov/17941360/#:~:text=Bipolar%20radiofrequency%20in%20the%20treatment%20of%20dermatologic%20imperfections%3A,treated%20area%20caused%20by%20excessively%20high%20RF%20settings.)

30 patients were subjected to the bipolar radiofrequency device on their skin with wrinkles and other skin conditions to test the effects of the device over two weeks with 6-8 sessions using before and after images and 15 patients' biopsies before and after. Side effects from the various higher radio frequency settings left a rash or ecchymosis (similar to a bruise but sounds like a cupping mark where blood escapes cappilaries into soft tissue leaving a purplish color or hue, where bruises are purple hematomas from trauma to the blood capillaries) and few showed blisters at the treatment site from higher settings. The radiofrequency device when applied to the skin creates an electical change in the area that creates an electrical movement that the soft tissue resists and thus creates heat.

[X-ray | Definition, History, & Facts | Britannica](https://www.britannica.com/science/X-ray)

X-rays are ultra high frequency at 1/10,000,000 - 1/100,000,000,000 meters in length to 10,000,000,000 MHZ-10,000,000,000,000 MHZ in frequency.





[Vibration enhanced cell growth induced by surface acoustic waves as in vitro wound-healing model - PubMed (nih.gov)](https://pubmed.ncbi.nlm.nih.gov/33257581/)

The use of sound acoustic waves (SAW) using 100MHZ of radio frequenc on cells to stimulate wound healing showed noticeable wound-healing compared to a baseline of 135 give or take 85%.

[jleejd01218 1..5 (aestheticmarket.co.uk)](https://aestheticmarket.co.uk/media/9262/clinical-experience-with-tripollar_dr-levenberg.pdf)

the fat reduction and wrinkle reduction of radio frequency on skin contouring and aesthetics did not produce any changes in liver function or lipid profile indicators after testing 5 patient blood samples from the 37 females aged 23-82 years. Tripollar radiofrequency with 3 or more electrodes to deliver RF current deep into the dermis and subcutaneous layers to generate heat. The design and distance between nodes determines the depth of the current's reach. The Apollo radiofrequency system was used. The radiofrequency used was up to a max of 1MHZ on various areas: abs, buttocks, arms, and thighs. The treatments were 1Xweek up to 7 weeks. The blood samples were taken initially and post procedure of two treatments from volunteers aged 33-56 years. The lipid profile tested HDL and LDL triglycerides and the liver function tested total bilirubin and ALKP, ALT, AST, and GGT. All patients showed cm reductions in areas of the abs, arms, and thighs, with some having noticeable changes, the face produced edema but went away with visual reductions in appearance of wrinkles.

KIM 8 Slimming System

[KIM-8-SLIMMING-SYSTEM-BALANCE-HOLISTIC-STUDIO.jpg (640×640) (bugibba-malta.com)](https://bugibba-malta.com/wp-content/uploads/2017/03/KIM-8-SLIMMING-SYSTEM-BALANCE-HOLISTIC-STUDIO.jpg)

cavitation+vacuum+laster multipole RF

Manual

summarized by me, but their summary is pretty close to the below

The fat abolation, bursting of fat from creating vacuum pockets that heat the fat, loosen the fat cell membranes and empty out the contents of fat cells or burst them ( to be picked up by the veinous system and lymphatic system) using the 1MHZ vacuum and RF tool, the 40kHZ RF tool, or the six electrode RF tool to do the same actions against fat cells, to shrink, explode, collapse, empty, etc. (into veinous or lymphatic system). The four and three pronged tools are also RF tools but only for the face and skin rejuvenation of discolorations, acne, and wrinkles.

https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE132936

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| Status | Public on Jun 19, 2019 |
| Title | Transcriptome Analysis and Functional Identification of Adipose-Derived Mesenchymal Stem Cells in Secondary Lymphedema |
| Organism | [Homo sapiens](https://www.ncbi.nlm.nih.gov/Taxonomy/Browser/wwwtax.cgi?mode=Info&id=9606) |
| Experiment type | Expression profiling by high throughput sequencing |
| Summary | We isolated adipose-derived mesenchymal stem cells (ASCs) from the lymphedema adipose tissue from liposuction specimens of 10 patients with malignancy-related extremity lymphedema, and we used adipose tissue from the normal upper abdomen of the same patients as control tissue. We compared the proliferation and adipogenic differentiation capacity between the two kinds of ASCs, and we explored the transcriptomic differences between them. We found that lymphedema-associated ASCs had more rapid proliferation and a higher adipogenic differentiation capacity. CDK1 inhibitors could return the abnormal biological characteristics of these cells to normal phenotype, suggesting that CDK1 is a key driver of proliferation and adipogenic differentiation in these cells, which might expound the accumulation of adipose tissue extensively observed in secondary lymphedema, indicating the CDK1 may be a potential target for lymphedema therapy. On the other hand, our finding showed that ASCs from lymphedema adipose tissues have higher immunosuppressive effect, and the inhibition of up-regulated cytokine CHI3L1 may be clinically beneficial. In summary, explore the underlying mechanisms of fat deposition in lymphedema may provide powerful strategies for the treatment of lymphedema. |
|  |  |
| Overall design | mRNA sequencing of ASCs from the affected thighs of 10 patients with lymphedema, and as control, ASCs from the normal upper abdomen of the same patients were also sequenced. |

[Mesenchymal Stem Cell - an overview | ScienceDirect Topics](https://www.sciencedirect.com/topics/neuroscience/mesenchymal-stem-cell)

https://www.sciencedirect.com/topics/neuroscience/mesenchymal-stem-cell

## [Mesenchymal Stem Cells](https://www.sciencedirect.com/science/article/pii/B9780123814227100173)

Zulma Gazit, ... Dan Gazit, in [Principles of Regenerative Medicine (Second Edition)](https://www.sciencedirect.com/book/9780123814227/principles-of-regenerative-medicine), 2011

This chapter provides an introduction to [mesenchymal stem cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/mesenchymal-stem-cell) (MSCs). MSCs are widely defined as a plastic-adherent cell population that can be directed to differentiate in vitro into cells of osteogenic, chondrogenic, adipogenic, myogenic, and other lineages. MSCs proliferate and give rise to daughter cells that have the same pattern of gene expression and phenotype and, therefore, maintain the “stemness” of the original cells. Self-renewal and differentiation potential are two criteria that define MSCs as real stem cells. MSCs exhibit the potential to differentiate into the osteogenic, chondrogenic, adipogenic, tenogenic, myogenic, or stromal lineages. Application of MSCs requires their isolation and directing the differentiation of these cells into the appropriate lineage. Immunoisolation is a method to isolate noncultured MSCs based on [cell surface markers](https://www.sciencedirect.com/topics/medicine-and-dentistry/cell-surface-marker). Immunodepletion is a “negative selection” approach in which the MSC population is enriched by washing out the cells labeled with antibodies, mostly directed against hematopoietic markers. Recently, more specific and pure populations are isolated utilizing a combination of immunoisolation and immunodepletion based on different surface markers. MSCs can either be systemically administered using intravenous (iv) injection or directly implanted in the [bone defect](https://www.sciencedirect.com/topics/medicine-and-dentistry/bone-defect) site. The systemic approach assumes that MSCs have the capability of migrating across the [endothelium](https://www.sciencedirect.com/topics/medicine-and-dentistry/endothelium) and homing to injured tissues in a manner similar to the migration of [leukocytes](https://www.sciencedirect.com/topics/medicine-and-dentistry/leukocyte) to sites of inflammation.

## [Mesenchymal Stem Cells](https://www.sciencedirect.com/science/article/pii/B9780123814227100173)

Zulma Gazit, ... Dan Gazit, in [Principles of Regenerative Medicine (Second Edition)](https://www.sciencedirect.com/book/9780123814227/principles-of-regenerative-medicine), 2011

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[View chapter](https://www.sciencedirect.com/science/article/pii/B9780123814227100173)[Purchase book](https://www.elsevier.com/books/T/A/9780123814227)

## [Mesenchymal Stem Cells](https://www.sciencedirect.com/science/article/pii/B9780124079151000027)

Elena A. Jones PhD, ... Dennis McGonagle PhD, FRCPI, NIHR, in [Mesenchymal Stem Cells and Skeletal Regeneration](https://www.sciencedirect.com/book/9780124079151/mesenchymal-stem-cells-and-skeletal-regeneration), 2013

### **2.1 Discovery**

The history of the biology of [Mesenchymal Stem Cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/mesenchymal-stem-cell) (MSCs) owes it conception and birth to the earlier discovery of its more illustrious bone marrow (BM) resident sibling—the [hematopoietic stem cell](https://www.sciencedirect.com/topics/medicine-and-dentistry/hematopoietic-stem-cell) (HSC), whose existence was first proposed by Maximov in 1909. By 1960, there was a great interest in applying new emergent knowledge on HSCs toward BM transplantation strategies. In vitro cell culture and subsequently in vivo animal model assays showed the potential to transplant freshly isolated HSCs into primary and secondary recipients [43]. Based on these assays, new definitions of stem cells emerged, the main defining principle being an ability of a stem cell to self-renew, as illustrated by the HSCs to fully reconstitute [hematopoiesis](https://www.sciencedirect.com/topics/medicine-and-dentistry/hematopoiesis) in secondary recipients. This new knowledge paved the way for the widespread adoption of BM transplantation in [lymphoproliferative disease](https://www.sciencedirect.com/topics/medicine-and-dentistry/lymphoproliferative-disease) where aggressive antitumor ablation with irradiation and chemotherapy, followed by transplantation with healthy HSCs from tissue-compatible donors, could rescue the marrow [44].

In the Soviet Union, similar work was conducted at the Gamaleya’s Institute in Moscow where scientists in Dr Alexander Friedenstain’s group studied cells of BM [microenvironment](https://www.sciencedirect.com/topics/medicine-and-dentistry/microenvironment) and their resistance to severe regimes of irradiation, a procedure used to “ablate” patient’s marrow prior to transplantation [45]. In a course of their studies, Friedenstein et al. [46] noted that BM seeded in glass flasks and maintained in fairly basic culture media produced an intriguing adherent population of nonhematopoietic cells that formed colonies and were transplantable. Furthermore, this occurred at a single-colony level with transplanted colonies being capable of self-renewal and also forming mature nonhematopoietic tissues in recipient animals, such as bone, cartilage, and fibrous tissue thus indicating their potentially true stem cell nature [47].

Arnold Caplan and Darwin Prockop [35, 48][35][48] were among the first to recognize the importance of Friedentein’s discoveries in the West. Additionally, Caplan proposed a concept of “mesengenesis,” similar to a concept of hematopoiesis in the HSC field and was the first to coin the term “mesenchymal” stem cell (MSC) [48]. Below this ancestral stem cell, he placed a hierarchy of more mature “progenitor” cells, differentiation potentials of which were restricted to a narrower range of tissues, for example, [osteo-](https://www.sciencedirect.com/topics/medicine-and-dentistry/osteoprogenitor-cell) and chondro-progenitors that gave rise to bone or cartilage tissues, respectively [49]. The first marker of a clonogenic marrow [stromal cell](https://www.sciencedirect.com/topics/medicine-and-dentistry/stromal-cell), Stro-1, was described as early as 1991 [50]. Paulo Bianco and Pamela Robey [51] later proposed putative pericyte/reticular cell topography of MSCs in the BM.

The recognition that the high proliferative potential of culture-expanded MSCs offered an enormous potential for therapeutics in the fields of skeletal regeneration was first exploited by Osiris Therapeutics, a Baltimore-based biotechnology company, which brought MSCs to the forefront of scientific and public attention in a Science article entitled “Multilineage potential of adult human mesenchymal stem cells” [41]. This work synthesized the available knowledge from seemingly unrelated fields of bone, cartilage, and fat differentiation to develop novel in vitro functional assays for MSCs, which still form the basis for MSC characterization. Furthermore, the Science article proposed a set of markers to characterize MSCs retrospectively (i.e., following culture) and also provided first evidence of MSC heterogeneity at the single-cell level [41].

Finally, work emanating from the Osiris group and others also showed that culture-expanded MSCs appeared to be immunomodulatory and were capable of suppressing an array of inflammatory reactions [52]. Proof-of-concept studies of MSC immunomodulatory properties have been later performed using human cells [53]; shortly it proved to be instrumental in explaining some mechanisms of action by transplanted MSCs in many seemingly unrelated disease applications.

[View chapter](https://www.sciencedirect.com/science/article/pii/B9780124079151000027)[Purchase book](https://www.elsevier.com/books/T/A/9780124079151)

## [Mesenchymal Stem Cells](https://www.sciencedirect.com/science/article/pii/B978012809880600014X)

Zulma Gazit, ... Dan Gazit, in [Principles of Regenerative Medicine (Third Edition)](https://www.sciencedirect.com/book/9780128098806/principles-of-regenerative-medicine), 2019

### **Abstract**

The authors review the current status of research on [mesenchymal stem cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/mesenchymal-stem-cell) (MSCs), a plastic-adherent cell population that can be directed to differentiate in vitro into cells of osteogenic, chondrogenic, adipogenic, myogenic, and other lineages. Characteristics of MSCs that lend to their being used in stem cell applications are described. Various tissues containing MSCs are specified and the methods by which these cells can be isolated are explicated. The relationship between MSCs and the immune system are discussed. In addition, the authors offer a review of current knowledge of MSC [exomes](https://www.sciencedirect.com/topics/medicine-and-dentistry/exome) and their place in stem cell–driven therapy, as well as a discussion of induced pluripotent stem cell–derived MSCs.

[View chapter](https://www.sciencedirect.com/science/article/pii/B978012809880600014X)[Purchase book](https://www.elsevier.com/books/T/A/9780128098806)

## [Mesenchymal Stem Cells](https://www.sciencedirect.com/science/article/pii/B9780128018880000114)

Amit N. Patel, ... Thomas E. Ichim, in [Stem Cell and Gene Therapy for Cardiovascular Disease](https://www.sciencedirect.com/book/9780128018880/stem-cell-and-gene-therapy-for-cardiovascular-disease), 2016

Cardiovascular disease is a large global problem with limited options. [Mesenchymal stem cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/mesenchymal-stem-cell) have been used in [regenerative medicine](https://www.sciencedirect.com/topics/medicine-and-dentistry/regenerative-medicine) to treat a number of diseases including cardiovascular. Mesenchymal stem cells act as a repair cell that is stimulated by physiological need.Chronic inflammation plays an integral role in the cascade leading to heart failure and mesenchymal stem cells may be further developed to function as a biological anti-inflammatory.The mechanisms of action are diverse including immunomodulation, anti-apoptosis, and allogeneic utilization. Specifically these cells function to inhibit post-acute [myocardial infarction](https://www.sciencedirect.com/topics/medicine-and-dentistry/myocardial-infarction) remodeling, stimulate regeneration of injured cardiac tissue, and induce [coronary artery](https://www.sciencedirect.com/topics/medicine-and-dentistry/coronary-artery) [angiogenesis](https://www.sciencedirect.com/topics/medicine-and-dentistry/angiogenesis). There are various methods of [mesenchymal stem cell](https://www.sciencedirect.com/topics/medicine-and-dentistry/mesenchymal-stem-cell) administration that include intracoronary, epicardial, and [intravenous routes](https://www.sciencedirect.com/topics/medicine-and-dentistry/intravenous-route).Randomized trials have demonstrated significant improvement in [left ventricular ejection fraction](https://www.sciencedirect.com/topics/medicine-and-dentistry/heart-left-ventricle-ejection-fraction) with reduced infarct size and left ventricular [end-systolic volume](https://www.sciencedirect.com/topics/medicine-and-dentistry/end-systolic-volume) following administration of mesenchymal stem cells.Changes in cellular expression have advanced the overall homing abilities of these cells improving identification of the targeted tissue.Clinical use of mesenchymal stem cells is conceptually based on cellular revitalization and [rejuvenation](https://www.sciencedirect.com/topics/medicine-and-dentistry/rejuvenation).The concept behind cellular augmentation does not focus on cell replacement, but is mediated by trophic, angiogenic, anti-inflammatory, and [anti-apoptotic](https://www.sciencedirect.com/topics/medicine-and-dentistry/antiapoptotic) effects.

My personal summary of mesenchymal stem cells, they are cells that can be stemmed from original cells of fat, cartilage, blood, and muscle tissue as well as heart tissue and other organ tissues so that they don't have the immunosuppresant markers that compromise a person's immune system in healing from damage due to a wound or injury or disease. For more information on stem cells, there are a number of helpful topics to search online, and I base this summary of stem cells on the information made available at https://www.sciencedirect.com/topics/neuroscience/mesenchymal-stem-cell.

[RPKM, FPKM and TPM, clearly explained | RNA-Seq Blog (rna-seqblog.com)](https://rna-seqblog.com/rpkm-fpkm-and-tpm-clearly-explained/)

https://rna-seqblog.com/rpkm-fpkm-and-tpm-clearly-explained/

(TPM-is a feature of this data on lymphedema gene expression RNA profiling)

It used to be when you did RNA-seq, you reported your results in RPKM (Reads Per Kilobase Million) or FPKM (Fragments Per Kilobase Million). However, TPM (Transcripts Per Kilobase Million) is now becoming quite popular. Since there seems to be a lot of confusion about these terms, I thought I’d use a StatQuest to clear everything up.

These three metrics attempt to normalize for sequencing depth and gene length. Here’s how you do it for RPKM:

1. Count up the total reads in a sample and divide that number by 1,000,000 – this is our “per million” scaling factor.
2. Divide the read counts by the “per million” scaling factor. This normalizes for sequencing depth, giving you reads per million (RPM)
3. Divide the RPM values by the length of the gene, in kilobases. This gives you RPKM.

FPKM is very similar to RPKM. RPKM was made for single-end RNA-seq, where every read corresponded to a single fragment that was sequenced. FPKM was made for paired-end RNA-seq. With paired-end RNA-seq, two reads can correspond to a single fragment, or, if one read in the pair did not map, one read can correspond to a single fragment. The only difference between RPKM and FPKM is that FPKM takes into account that two reads can map to one fragment (and so it doesn’t count this fragment twice).

TPM is very similar to RPKM and FPKM. The only difference is the order of operations. Here’s how you calculate TPM:

1. Divide the read counts by the length of each gene in kilobases. This gives you reads per kilobase (RPK).
2. Count up all the RPK values in a sample and divide this number by 1,000,000. This is your “per million” scaling factor.
3. Divide the RPK values by the “per million” scaling factor. This gives you TPM.

So you see, when calculating TPM, the only difference is that you normalize for gene length first, and then normalize for sequencing depth second. However, the effects of this difference are quite profound.

When you use TPM, the sum of all TPMs in each sample are the same. This makes it easier to compare the proportion of reads that mapped to a gene in each sample. In contrast, with RPKM and FPKM, the sum of the normalized reads in each sample may be different, and this makes it harder to compare samples directly.

Here’s an example. If the TPM for gene A in Sample 1 is 3.33 and the TPM in sample B is 3.33, then I know that the exact same proportion of total reads mapped to gene A in both samples. This is because the sum of the TPMs in both samples always add up to the same number (so the denominator required to calculate the proportions is the same, regardless of what sample you are looking at.)

With RPKM or FPKM, the sum of normalized reads in each sample can be different. Thus, if the RPKM for gene A in Sample 1 is 3.33 and the RPKM in Sample 2 is 3.33, I would not know if the same proportion of reads in Sample 1 mapped to gene A as in Sample 2. This is because the denominator required to calculate the proportion could be different for the two samples.

