

Critique a paper literature review

[Science](#), [Biology](#)



The title and abstract suggest that the paper is an attempt to show that in the cartilage growth plate, abnormal chondrocyte apoptosis is influenced by genetic background as well as deletion of CHOP. The research is interesting because it enables the discovery of whether whether the abnormal chondrocyte apoptosis that is seen in the mutant mice growth plate is CHOP-mediated. The research builds on the research done by Delot, King, Briggs, Wilcox & Cohn (1999) and Hecht et al. (2001), who demonstrate that type 3 recurring mutations are responsible for most of the Pseudoachondroplasia (PSACH) cases, which are believed to lead to misfolding of the protein bearing mutant characteristics and its retention inside the endoplasmic reticulum (ER). This paper is an extension of previous work The authors of this paper sought to find out why Pseudoachondroplasia (PSACH) cases lead to misfolding of the protein bearing mutant characteristics and its retention inside the endoplasmic reticulum (ER). To achieve this, they hypothesized that the abnormal chondrocyte apoptosis present in the COMP mouse growth plate (T585M) was responsible for CHOP-mediated ER stress prompted by the trafficking and folding of mutant COMP protein (T585M).

The experiment involved the crossing of the mild PSACH CTD-COMP mouse model and CHOP null mice to obtain a phenotype, which was then analyzed. Mass spectrometry analysis was done on xiphoid cartilage protein from T585M COMP mutant mice. The TUNEL assay was a major part of the experiment. The assay was applied to sections of 3-week old mice limbs. The dependent variables for this experiment are lengths of the pelvis, tibia and femur for the CHOP null and wild-type mice. The wild-type mice were the controls for this experiment they were used as reference points to indicate

the changes in the variable of the CHOP null mice.

Results provided include mass spectrometry analysis figures; head measurements of the different mice categories used; TUNEL assay staining quantification measures and Immunohistochemistry data for 3-week COMP. Overall, the results showed a decline in the level of the major bone structural components. Through this experiment, the authors confirm that CHOP is crucial in bone biology. The results demonstrated that the dysregulation of apoptosis in the growth plate proliferative zones in mutant mice (T585M COMP) is not mediated by CHOP in a direct manner. The author cites journals such as the American Journal of Medical Genetics and the Journal of Biochemistry, which are reputable. In addition, the data collection, sample choice and size as well as the statistical methods employed are acceptable. The sample comprises of mice used CHOP null mice and homozygous T585M COMP mice. The measurements include bone lengths, which was done using Growbase software. The data was analyzed statistically using t-tests and one way ANOVA.

The significance of these findings is that they enable the authors to confirm the importance of CHOP in bone biology. This is because the results demonstrate that the dysregulation of apoptosis in the growth plate proliferative zones is not mediated by CHOP in a direct manner in mutant mice (T585M COMP). An interesting finding in this research is that there was a decrease in the levels of stress-inducible ER genes. This suggest that CHOP has a potential role in the pathogenesis of PSACH. A major undoing for this paper is that the authors do not address the implication of the research in terms of what it means for the field of biology in general sense. The research

is convincing because it concludes that Chondrocyte proliferation was unaffected in [CHOP-/-/COMPm/m] mice. This is in-keeping with previous studies on different types of CHOP mice.