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Evaluation of Mass-based Scaling Law for Brain Injury Criteria: A Preliminary Study

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Abstract:

Numerous in vivo impacts tests have been conducted on animal heads for the investigation of the traumatic brain injuries results from real-world impact accidents. The mass-based scaling law has long been used as a principal method for the scaling of input loadings as well injury thresholds between surrogate animal brains and human brain in those animal impact tests. The purpose of this study was to evaluate the performance of the mass-based scaling law for brain injury criteria.

Coronal plane rotational impacts were simulated using a rat head FE model and a human head FE model. Amplitudes and durations of rotational accelerations were defined based on prior in vivo experimental tests on rats. In total, 18 simulations, including 9 simulations using the rat brain model and 9 simulations using the human brain model, were conducted. Strain responses were extracted from four anatomical regions of interest (ROIs): parietal cortex, hippocampus, thalamus, and hypothalamus.

From the comparisons of peak strains of those ROIs, different strain patterns could be observed between those two head FE models. Meanwhile, the effects of the rotational acceleration durations on the peak strains of those ROIs were weaker in the human head FE model than the corresponding effects in the rat head FE model. Similar effects of the rotational acceleration amplitudes on the peak strains of those ROIs were observed between those two head FE models.

The simulations demonstrated that, the mass-based scaling law could be used for the scaling of the amplitude of inputted rotational accelerations, but it has limits for the scaling of the acceleration durations. Moreover, region-specific characteristics should be considered in scaling procedures of brain injuries between animals and human.

Keywords: traumatic brain injury; scaling law; finite element simulation; brain injury criteria

1 Introduction

Traumatic brain injury (TBI) is a serious public health problem in our modern society. A summary of 23 European reports showed that, for every 100,000 population in Europe, there were about 235 people suffered a TBI [1]. In the United States, 1.7 million people sustained TBI annually, and 52,000 civilians died from TBI-related injuries every year on average between 1997 and 2007 [2]. It has been revealed that approximately 60 percent of TBI cases are a result of road traffic injuries while the percentage of motor-vehicle-related TBI will grow continuously in the next decade along with the development of motorization in the developing countries [3]. Clearly understood injury mechanisms and reliable injury criteria for TBI could be helpful for the measurement of the brain injury risks in traffic accidents as well as the development of relative protection device.

Considering the similar anatomical structures of the brains between human and other mammals, in vivo animal impacts have been used as an effective tool to investigate the TBI. Not only the pathology of specific TBI, but also the tolerance of mechanical loadings as well the intracranial dynamical responses could be studied via those in vivo animal

impacts. How to scale the mechanical loadings that sustained by human head from traffic accidents to those animal head and how to apply those animal experiments to human brain protection are two essential issues that should be clearly understood. A mass-based scaling law (or Holbourn's law) for the brain injuries caused by rotational accelerations was proposed by Ommya et al. [4] and further improved by Margulies et al. [5] and Gutierrez et al. [6]. The injury scaling model was base on scaling angular head acceleration by a ratio of brain mass to the power of -2/3. By applying this mass-scaling law, diffuse brain injuries were produced in the rabbit brain while the rabbit head sustained to a sagittal plane rotational acceleration [6]. Similarly, Davisson et al. [7] investigated the diffuse axonal brain injuries in the rat brain in which study sagittal plane rotational accelerations were scaled from the corresponding accelerations for human brain injuries. Inversely, Fijalkowski et al. [8] proposed a tolerance for the concussion of the human brain based on the investigations of diffuse brain injuries in the rat brain. Moreover, a rotational injury criterion was proposed by Margulies and Thibault [9] using this scaling procedure based on the results of a primate injury model [10]. However, this scaling law was proposed based on assumptions that the brain tissue was elastic and there was no geometry difference between the human head and surrogate-animal heads [4]. In spite of the assumptions, this scaling law has not been exactly validated, especially when small mammals were used, like the rabbit and rat.

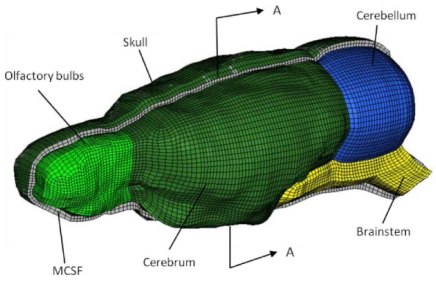
Thus, the purpose of the current study was to evaluate the performance of the mass-based scaling law for brain injury via the finite element (FE) simulations. Intracranial strain responses were calculated and compared between FE head models of the rat and human. The simulations could be helpful for the future studies on the application of the head impacts from animal experiments to human.

2 Materials and Methods

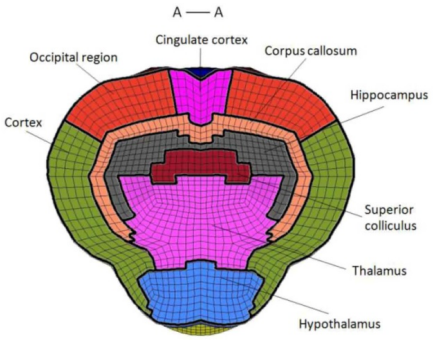
2.1 Finite element models

2.1.1 Rat head FE model

The rat head FE model (Figure 1) was developed by Baumgartner et al. [11] and previously improved and validated by Lamy et al. [12] and Ren et al. [13]. The model consisted of 150,390 nodes, 143,776 Hexahedral elements and 1 shell elements with an average edge size of 0.25 mm. the rat brain model was organized into 25 parts to present most of those essential anatomical feature of the rat brain, containing the skull, skull-brain interface (MCSF), cerebellum, olfactory bulbs, brainstem and 20 parts of the cerebrum.



(a) Global view



(b) Middle coronal cection view of the cerebrum

Figure 1. The rat head FE model

2.1.2 Human head FE model

A 3D human head FE model that developed by Yang et al. [14] was used in this study (Figure 2). The human head FE model consisted of 38 components, 57,043 nodes and 80,627 elements, with an effect mass of 4.4 kg. The model presented all essential anatomical features of a 50th percentile male head, including scalp, skull – with outer table, diploe, and inner table – dura, falx cerebri, tentorium, falx cerebelli, pia, cerebrospinal fluid (CSF), cerebrum, cerebellum, brain stem, and ventricles.

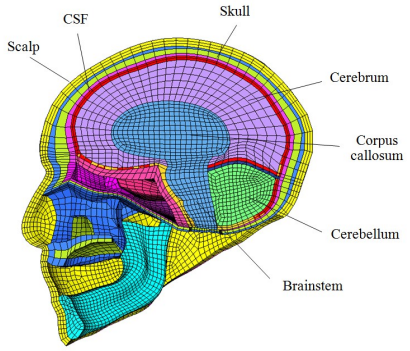


Figure 2 Middle sagittal plane view of the human FE head model

To eliminate the effect of the difference of material model/properties between these two head FE models, in the current study, same material model (LS-DYNA 971 Material Type 6) with the material properties of the rat brain were assigned to the brain tissue of the rat and human head models.

2.2 Scaling law for brain injury criteria and FE simulations

2.2.1 Scaling law for brain injury criteria

The mass based scaling law, which was suggested by Ommaya et al. [4] and further improved by Margulies et al. [5] and Gutierrez et al. [6] provides a relationship between rotational acceleration (α) and mass (m) of human and rat brains as:

$$\alpha_H = \alpha_R \left(\frac{m_R}{m_H}\right)^{2/3} \quad (1)$$

where the subscripts H and R are human brain and rat brain respectively. Provided that the human and rat brains have similar density and geometrics and are incompressible, elastic, homogeneous, and isotropic, the mass in Eq. (1) can be

substituted with the cube of the length ratio between the human and rat:

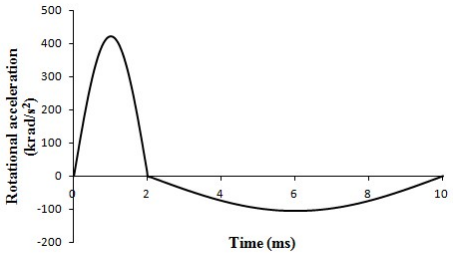
$$\alpha_H = \alpha_R (\frac{r_R}{r_H})^2 \tag{2}$$

where rH represents the radius of the human, and rR represents the radius of the rat. And for the duration (t) of the acceleration pulse:

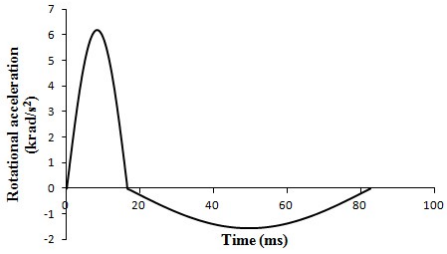
$$t_H = t_R (\frac{r_H}{r_R}) \tag{3}$$

2.2.2 Simulations of coronal plane rotational impacts

Coronal plane rotational impacts were applied to evaluate performance of the mass based scaling method on mild traumatic brain injury. Typical shapes of the applied rotational acceleration curve included a half sine-wave acceleration pulse and a half sine-wave deceleration pulse (Figure 3). Amplitudes of the acceleration pulses were defined as 295 krad/s2 for low case (M1), 425 krad/s2 for medium case (M2), and 535 krad/s2 for high case (M3). Durations of the acceleration pulses were defined as 1 ms for low case (D1), 2 ms for medium case (D2), and 3 ms for high case (D3). Additionally, hese amplitudes/durations of rotational accelerations have been applied for the production of mild brain injuries (concussions) in the rat brain [8]. To stop the rotational motion mildly, amplitudes of deceleration pulses were defined as quarter as amplitudes of corresponding acceleration pulse, while durations of deceleration pulses were defined four times as durations of corresponding accelerations. Amplitudes and durations of acceleration curves for human head were scaled from corresponding values for rat head according to mass based scaling law (where the measured rH and rR were approximately 70 mm and 8.5 mm at the middle coronal plane of those two head FE models, respectively): M1 was 4.35 krad/s2, M2 was 6.19 krad/s2, and M3 was 7.89 krad/s2;. D1 was 8.24 ms, D2 was 16.5 ms , and D3 was 24.7 ms.



(a) Rotational acceleration for the rat head model



(b) Rotatiaon acceleration for the human head model

Figure 3. Sample of Rotational acceleration pulses: half sine-wave acceleration pulses followed by half sine-wave deceleration pulses (M2D2).

Intracranial strain responses (maximum principal strain) were calculated for the rat head model and the human head model, respectively. Peak strains were extracted from four anatomical regions of interest (the parietal cortex, hippocampus, thalamus, and hypothalamus) and compared between those two head models.

3 Results

Totally, 18 simulations were conducted: 9 simulations using the rat brain FE model and 9 simulations using the human head FE model, respectively. Intracranial strain responses were calculated and the maximum strains were extracted from these four anatomical regions of interest (ROIs), respectively.

3.1 Intracranial strain responses of the rat head FE model

A typical strain contour in the middle coronal plane section of the rat head FE model (sustained the acceleration with medium amplitude and medium duration-M2D2) is illustrated in Figure 4. High strain responses can be observed at up, middle and low areas in the coronal section, where the parietal cortex, hippocampus/thalamus, and hypothalamus are located, respectively. As a sample, the elemental time historical strain responses in the parietal cortex with medium amplitude and medium duration acceleration are illustrated in Figure 5.

Peak strain responses of the parietal cortex, hippocampus, thalamus and hypothalamus are illustrated below. As shown in Figure 6, peak strain responses are increased with the increase of the amplitudes of impact accelerations, but reduced slightly with the increase of the acceleration durations. This trend was consistent in all these four ROIs.

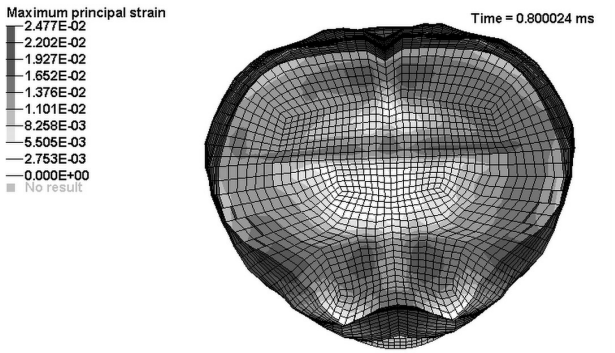


Figure 4. Strain responses in the middle coronal plane section of the rat head FE model (M2D2).

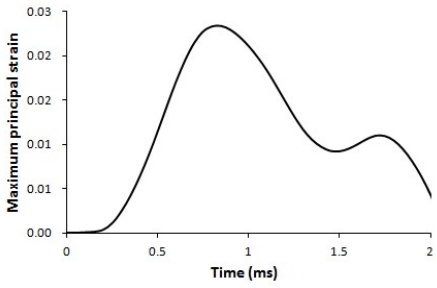


Figure 5. Sample of the elemental time history curve of the maximum principal strain in the parietal cortex of the rat head FE model (M2D2).

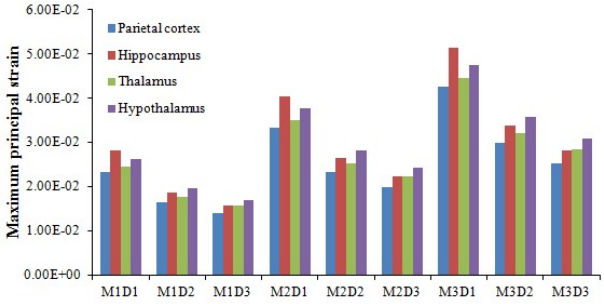


Figure 6. Peak strains in the parietal cortex, hippocampus, thalamus, and hypothalamus of the rat head model.

3.2 Intracranial strain responses of the human head FE model

A typical strain contour in the middle coronal plane section of the human head FE model is illustrated in Figure 7. High strain responses can be observed in cortex, middle area of corpus callosum, and especially basis areas. A typical time history curve of the elemental maximum principal strain is illustrated in Figure 5.

Peak strains of the parietal cortex, hippocampus, thalamus and hypothalamus calculated. As shown in Figure 9, peak strains are increased with the acceleration amplitudes, but decreased with the acceleration durations. And also, this trend was consistent in all these four regions.

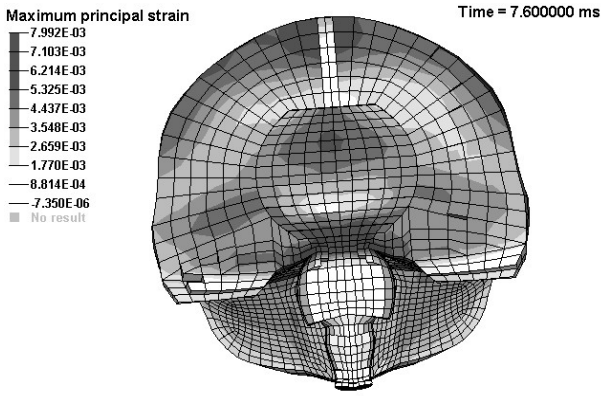


Figure 7. Strain responses in coronal plane section of the human head FE model (M2D2).

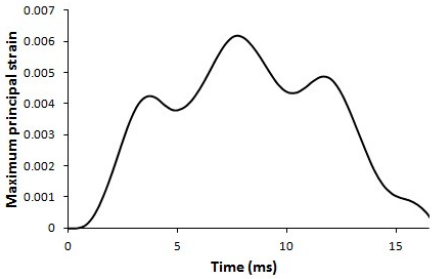
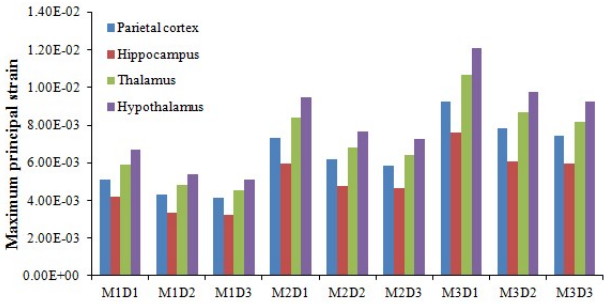


Figure 8. Sample of the elemental time history curve of the maximum principal strain in the parietal cortex of the human head FE model (M2D2).



Fiugre 9. Peak strains in the parietal cortex, hippocampus, thalamus, and hypothalamus of the human head model.

4 Discussions

The objective of this study was to evaluate the mass-based scaling law for brain injuries caused by rotational accelerations. In spite of the amplitude of the rotational acceleration, the acceleration duration also has significant effect on the severity of the brain injuries while larger durations could induce longer unconsciousness [15]. Thus, angular acceleration pulses with three levels of the amplitude and three levels of the duration were imposed on the head FE models.

Generally, calculated peak strains of those four selected regions in the human head FE model were lower than those corresponding values in the rat brain model. In these four ROIs of the human head FE model, the highest peak strains were observed at the hypothalamus, followed by the peak strains of the thalamus, parietal cortex and hippocampus. In the rat head FE model, higher peak strains were located at the hippocampus and hypothalamus, followed by the peak strains of the thalamus and parietal cortex. The observed difference of the intracranial strain patterns (of those ROIs) between the rat and human head FE models could result different brain injury patterns in the rat brain and human brain, respectively. This difference also reflected one of the limitations of the mass-based scaling law that region-specific responses as well region-specific injury mechanisms/tolerances were not considered.

Effect of the durations of the rotational accelerations: The peak strains of those ROIs decreased with the durations of the imposed rotational acceleration pulses in both the rat and human head FE models. With the durations increased from D1 to D2, the percentage changes of the peak strains were -29.8%, -34.3%, -27.7 and -24.9% of the parietal cortex, hippocampus, thalamus and hypothalamus in the rat head FE model, respectively. The corresponding percentage changes were lower in the human head FE model with the corresponding values ranged from -15% to -20% of those ROIs. With the durations increased from D2 to D3, the percentage changes of the peak strains ranged from -11.6% to -16.0% of those ROIs in the rat head FE model, while the corresponding values of the human head FE model ranged from -2.3% to -5.9%. The difference of the effect of the durations of the rotational accelerations on the peak strains between the rat and human head FE models demonstrated that, the mass-based scaling law (Eq. 3) has limits for the scaling of the durations of rotational acceleration pulses.

Effect of the amplitudes of the rotational accelerations: The peak strain values of those ROIs increased with the amplitudes of the rotational acceleration in both the rat brain model and the human brain model. Meanwhile, the percentage increases of the peak strains in all ROIs were close to each other as the amplitude of the rotational acceleration increased (in both the rat brain model and the human head model). Moreover, the percentage changes of the peak strains associated with the acceleration amplitudes in the rat head model were similar as the corresponding percentage changes in the human head FE model. With the amplitudes increased from M1 to M2, the peak strain in these ROIs increased approximately 42%. With the amplitudes increased from M2 to M3, the percentage increase of those peak strain values were approximately 27%. In spite of the difference of the calculated peak strain values between

those two head models, the similar trends of the peak strains associated the amplitudes of the rotational accelerations demonstrated that mass-based scaling law (Eq. 2) were partly effective for the scaling of the amplitudes of rotational acceleration pulses.

Considering the different modeling strategies between the rat and human head FE models, additional errors could be induced into the simulations. For example, the falx cerebri was not represented in the current rat head FE model which could reduce the strains of the brain tissue adjacent to the falx cerebri [10]. Additionally, the generally lower strain responses of the human head FE model (compared with the corresponding strains of the rat head FE model) may also caused by different modeling strategies between the rat and human head FE models (figures 6 and 9).

In summary, the mass-based scaling method could be used for the scaling of the amplitudes of inputted rotational accelerations, but it has limits for the scaling of the duration of such accelerations. Meanwhile, region-specific characteristics of different anatomical regions should also be considered future studies.

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