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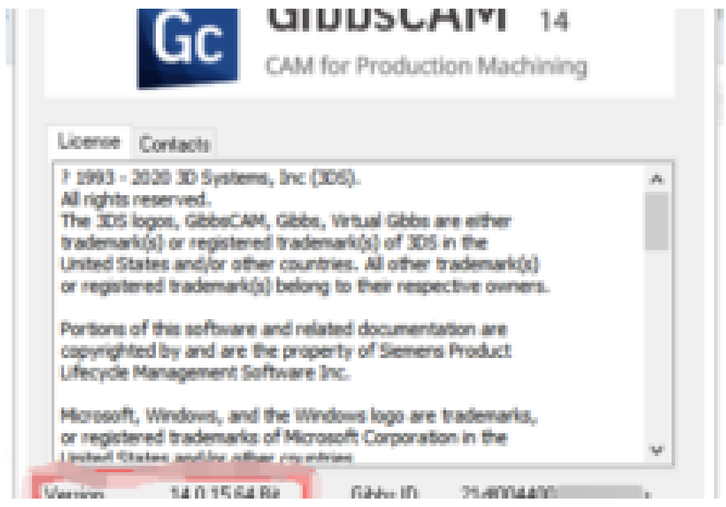

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Pulmonary arterial hypertension (PAH) is a life-threatening illness in which increased pulmonary vascular resistance results in right ventricular failure and death \[[@CR1]\]. Therapy for PAH is intended to prevent the development of right ventricular failure. However, the complex pathophysiology of PAH makes a positive clinical outcome difficult to predict. To tailor therapy and prevent the transition to right heart failure, physicians need to identify patients who are likely to have poor outcome and/or who are candidates for experimental therapies. Preclinical studies provide a critical means to evaluate the efficacy of potential therapies for PAH. Preclinical models of PAH have been developed, including the monocrotaline rat model, which mimics human disease \[[@CR2]--[@CR5]\]. Although this model is often considered the gold standard for preclinical studies of PAH, it has several limitations. First, the monocrotaline rat model of PAH is not considered a homogeneous model. The use of monocrotaline and the pulmonary vascular phenotype of the animal varies between institutions \[[@CR6]\]. Second, the development of right ventricular failure is variable in the monocrotaline model, and it is not sensitive to many classes of therapies \[[@CR6]\]. Therefore, alternative models, such as the Sugen's Sugen-hypoxia rat model, have been developed to overcome these limitations \[[@CR7], [@CR8]\]. Although the Sugen model is increasingly popular, it has not been adopted by the entire PAH research community and, therefore, few data are available on the effects of this model on different disease characteristics. After evaluating the effects of the Sugen model on several disease characteristics, we compared patient responses to the Sugen model with monocrotaline and Sugen's Sugen-hypoxia models in a retrospective study of data obtained from the American Heart Association Pulmonary Hypertension Registry. The primary endpoint was the degree of right ventricular failure and the secondary endpoints were the numbers of patients that were eligible for an experimental therapy and the survival rates at the end of the study period. In addition, we used a crossover design to evaluate the effect of treatment on the primary endpoint in the Sugen model. To the best of our knowledge, this is the first retrospective analysis using the PAH Registry database to compare the Sugen model to monocrotaline and Sugen's Sugen f3e1b3768c