

## SoundToys DevilLoc Deluxe 109 ((FREE))

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## SoundToys DevilLoc Deluxe 109

SoundToys DevilLoc Deluxe 109...1. Field of the Invention The invention relates to an apparatus for extinguishing fires, which includes a chamber formed at least partially by a housing, which chamber accommodates an extinguishing agent and a buffer volume, which is connected to the chamber by an openable and closable connection, and wherein the extinguishing agent is supplied to the buffer volume from a source of extinguishing agent and at least one valve is provided in the buffer volume to prevent overflow of the buffer volume. 2. Related Art DE-PS 26 48 281 discloses a device for extinguishing fires, in which extinguishing agent is discharged from a chamber through a manually actuated opening, which opening has a cross section in a shape corresponding to the cross section of a discharge conduit formed in a housing, and is connected to the chamber by a filling channel. The connecting channel provides a buffer volume, in which flow conditions for the extinguishing agent can be determined as desired for optimum operation. The disadvantage of the described design is that no control is provided over the flow of the extinguishing agent or over the pressure level in the buffer volume, so that the process conditions cannot be varied in an optimum manner.

CAF, a Signaling Hub for Smad1-Independent TGF- $\beta$ -Mediated Growth Arrest. Transforming growth factor- $\beta$  (TGF- $\beta$ ) is an important signal involved in cell fate determination, tissue homeostasis, and, in the case of pathological states, cell transformation. How the effects of TGF- $\beta$  in such contexts are mediated is largely unknown. We report here that the cancer-associated factor (CAF) is a novel signaling hub in TGF- $\beta$ -mediated growth arrest. CAF is a signal protein that was previously shown to influence Snail-mediated epithelial-to-mesenchymal transition (EMT). We show that CAF is a ligand that is required for TGF- $\beta$ -mediated growth arrest and that of its carboxyl-terminal region is the molecular determinant for this function. Silencing CAF in TGF- $\beta$ -treated cells results in disruption of Smad1-independent TGF- $\beta$ -mediated growth arrest, rendering cells resistant to TGF- $\beta$ -induced growth arrest. These findings establish CAF as a novel Smad1-independent signaling module that mediates TGF- $\beta$ -stimulated growth arrest by linking TGF- $\beta$  receptor signaling with Smad1-independent TGF- $\beta$  signaling.

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