clearly evident by the reduction in number of oviposition per female and proportion of well-developed F1 larvae. The maternally transferred BDE-47 in F1 offspring had embryotoxic effect as demonstrated by the higher number of broods with non-viable F1 embryos which failed to develop normally through early cleavage stages (see Figure 1). Lastly, the transcriptomic analysis provided potential mechanistic explanations on some of the phenotypic changes observed above. Namely, the malformed velar lobes might be cause be dysregulation in genes related to cilia; the delay in male emergence might be the result of dysregulation of steroid hormones; failure of embryos to perform cleavage at early stage could be due to the suppression of RHO protein, and potentially related to the suppression of Wnt signaling pathway.

Finding from the present study has highlighted the need for long-term exposure study of toxicants because the parental transfer effect of pollutants may cause serious and subtle impact to the offspring at their early stage.

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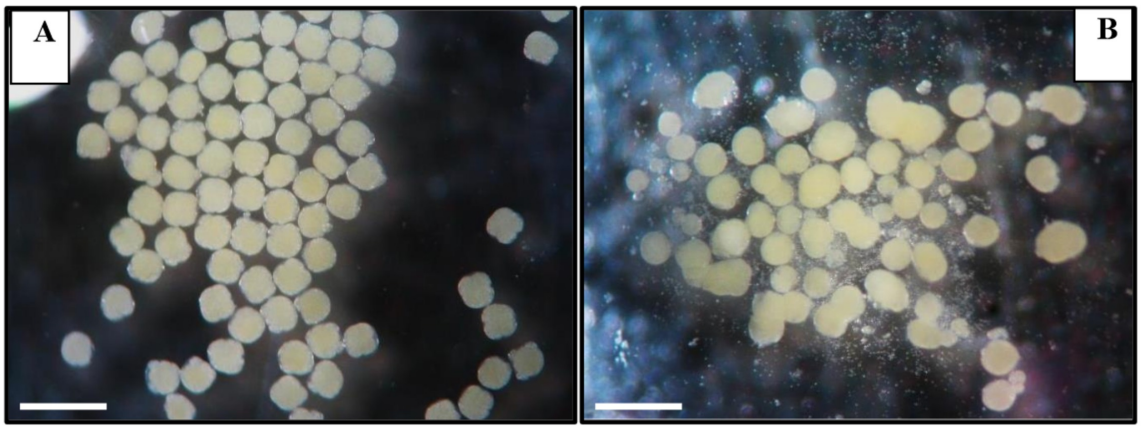

Figure 1. Some broods of F1 *Crepidula onyx* embryos developed abnormally during blastula to gastrula stage and became non-viable. (A) Normal embryos at blastula stage and (B) non-viable embryos with yolky materials of irregular shapes and sizes. Bar represents 0.5 mm.