

**Table 4. Rescue of *ftk* phenotype by *nkx2.5* mRNA**

<i>nkx2.5</i> mRNA, pg	<i>ecx</i> MO, ng	No. surviving, <i>n</i>	<i>ftk</i> phenotype, %	Non- <i>ftk</i> phenotypes, %		
				Edema*	No tail	No head
Injection into wild-type embryos						
0	2	328	96.6	0.9	0	0
10	2	262	2.7	3.5	0	0
25	2	207	3.9	43.0	6.8	0
50	2	157	1.9	54.8	19.7	7.0
10	0	194	0	11.4	0	0
25	0	195	0	48.2	5.1	0
50	0	161	0	50.5	22.4	8.7
Buffer	0	99	0	0	0	0
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Injection into embryos obtained from hetero ( <i>ftk/+</i> ) × hetero ( <i>ftk/+</i> )						
0	-	316	22.8	0	0	0
10	-	261 <sup>†</sup>	3.1	3.4	0	0
25	-	217	2.8	48.4	5.5	0
50	-	149	2.0	65.1	10.1	15.4
Buffer	-	100	27.0	0	0	0

Either *nkx2.5* mRNA alone or *nkx2.5* mRNA plus *ecx* MO was injected into fertilized wild-type eggs. Phenotype was scored at 48 hpf.

\*Pericardiac edema caused by *nkx2.5* overexpression was distinguishable from that of *ftk*; even overexpression of *nkx2.5* itself produced pericardiac edema in zebrafish [Chen JN, Fishman MC (1996) Zebrafish tinman homolog demarcates the heart field and initiates myocardial differentiation. *Development* 122:3809–3816].

<sup>†</sup>Genotyping was performed on embryos with wild-type phenotype. Nineteen embryos out of 96 examined were confirmed to be genotypically *ftk* homozygous mutants.