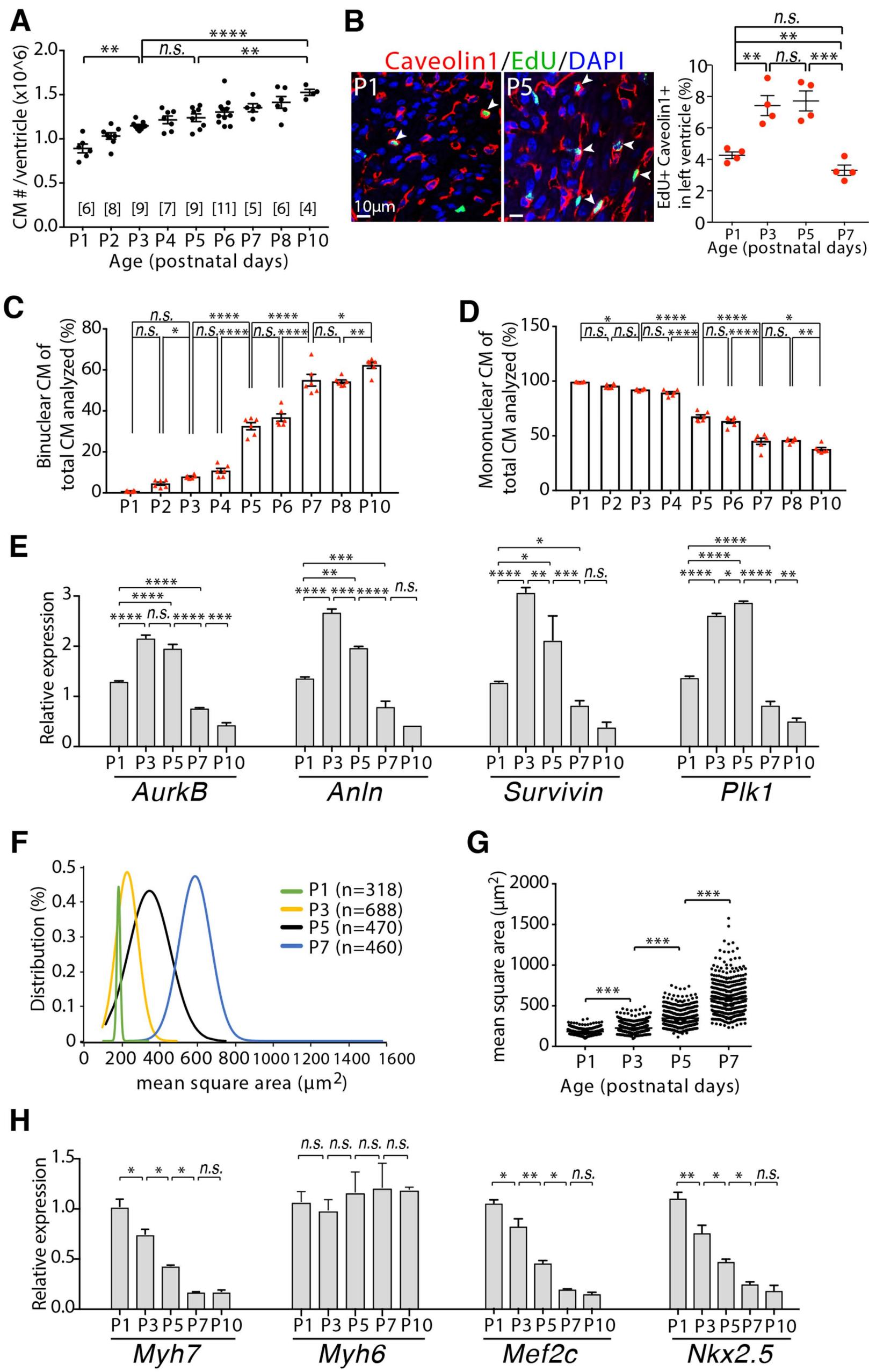


Supplemental Information:

**Fatty acid oxidation promotes cardiomyocyte proliferation rate but does not change
cardiomyocyte number in infant mice**

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Supplemental Figure 1. Infant mouse hearts show evidence for cardiomyocyte cycling, hypertrophic growth and maturation. (A) Total number of cardiomyocytes (CM) in both cardiac ventricles of mice. (B) Confocal images of EdU+ cardiomyocytes on tissue sections using Click-iT EdU Alexa Fluor (green) and co-immunostaining with antibody against caveolin1. Arrows point to EdU+Caveolin1+ cells. Graph on the right showing quantification of EdU+Caveolin1+ cells as percentage of total caveolin1+ cells analyzed per field. (C and D) Percentage of binuclear (C) and mononuclear (D) cardiomyocytes (CM) in the heart ventricles of infant mice. (E and H) Expression of indicated genes by qRT-PCR analysis of the mRNA of isolated heart ventricles at indicated time points (n= 5-6 per time point). (F and G) The frequency distribution (F) and mean square areas (G) of the surface area of cardiomyocytes isolated from infant mouse heart ventricles. *P* value was calculated using one-way ANOVA.

Supplemental Table 1. qRT-PCR primer sequences used in this study.

	Forward	Reverse
Myh6	CCACTTCTCCTGGTCCACTATG	ACAAACCCACCACCGTCTCA
Myh7	AAGGGCCTGAATGAGGAGTAGCTC	GCAAAGGCTCCAGGTCTGA
Mef2c	GCCAGCACTG ACATGGATAAG	CCATTGAGGCCCTTCTTCT
Gata4	CCGGGCTGTCATCTCACTATG	TTCAGAGCAGACAGCACTGGAT
Nkx2.5	TGACCCAGCCAAAGACCCCT	CCATCCGTCTCGGCTTTGT
Acta1	TGAAGATGGGTTAACGGAG	TTCGTCGCACATGGTGTCTA
Nppa	GGGTAGGATTGACAGGATTGG	CTCCTTGGCTGTTATCTTCGG
Nppb	CTGAAGGTGCTGTCCCAGAT	CCTTGGTCCTCAAGAGCTG
Acaca	CTGGGACAAAGAACCATCCA	ATAATCTGGATGCCCAAG
Acacb	CCGAGTTGTCACTCGGTT	GCATACACTGACCGCAGC
Acadm	AGCTCTAGACGAAGCCACGA	TGAGCCTAGCGAGTTCAACC
Acadl	AACGTCTGGACTCCGGTTCT	CGGGTACTCCCACATGTACC
Cpt1b	TCTCCATGGACTGGTCGAT	ACCATGCTGAGAAGTGCCTC
PPar α	CAGTCCATCGGTGAGGAGAG	CTGGAAGCTGGAGAGAGGGT
Pdk4	CGTTCCCTCACACCTTCACC	GGTCAAGGAAGGACGGTTTT
Ccnd1	TTCCTCTCCAAAATGCCAGA	AGGGTGGGTTGGAAATGAAC
Ccnd2	GAACCTGGCCCGCAGTCACCC	CGACGGCGGGTACATGGCAA
Bcl2	GTGGATGACTGAGTACCTGAAC	GAGACAGCCAGGAGAAATCAA
Bax	GTGGTTGCCCTCTTCTACTTT	CAGCCCATGATGGTCTGAT
18S	TCAAGAACGAAAGTCGGAGG	GGACATCTAAGGGCATCAC