This data article contains supporting information regarding the research article entitled “High butter-fat diet and bisphenol A additively impair male rat spermatogenesis” (P. Tarapore, M. Hennessy, D. Song, J. Ying, B. Ouyang, V. Govindarajah, et al.) [1]. Sprague-Dawley females were fed AIN, high fat butter, 17α-ethinyl estradiol, or high fat butter plus four bisphenol A doses (2500 μg/kg bw-d, 250 μg/kg bw-d, 25 μg/kg bw-d, and 2.5 μg/kg bw-d) before and during pregnancy. All diets were switched to AIN after the pups were born. Male offspring received testosterone (T)- and estradiol-17β (E2)-filled implants from postnatal day 70–210 for 20 weeks (T+E2 rat model). The testes were weighed, and examined for impairments in spermatogenesis.

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1. Data

We conducted a dose-response analyses to determine the minimal BPA dose that impedes spermatogenesis (Fig. 1) in male offspring exposed in utero to diets with bisphenol A (BPA) and high fat butter (HFB). Details on diets, animal groups and approach are outlined in Fig. 1A. The number of seminiferous tubules (STs) within the testis (per animal) with progression of spermatogenesis upto the round spermatids (Fig. 1B) or upto spermatozoa (Fig. 1C), was scored and plotted (T+E2 model). The body weights and the weights of testis and spleen were scored (Figs. 2A–C). In a separate work, data is presented for body weight and weight of the testis, epididymis, spleen, and kidney for offspring prenatally exposed to AIN, BPA, HFB, high fat olive oil (HFO), HFB+BPA, or HFO+BPA diets (Figs. 2D–G) and T+E2.

We examined the STs of the testis for presence of clusters of cells (using BRDT staining, Fig. 3) and for ERα (Fig. 4) and CYP19 (aromatase, Fig. 5) expression between the diet groups.

2. Experimental design, materials and methods

2.1. Diets and animals

Sprague–Dawley females were fed AIN, high fat butter (39 kcal% fat, HFB), 17α-ethinyl estradiol (EE2 (0.5 µg/kg bw-d), or HFB plus four BPA doses (2500 µg/kg bw-d, 250 µg/kg bw-d, 25 µg/kg bw-d,
and 2.5 μg/kg bw-d) before and during pregnancy (Fig. 1A). All diets were switched to AIN after the pups were born. At postnatal day (PND 70), prenatally exposed pups from each diet group were treated with T+E2 via SilasticTM implants [2,3] (T+E2 rat model) for 20 weeks. The animals were weighed, the testis, epididymis, spleen, and kidney were weighed, fixed, paraffin embedded, stained with hematoxylin and eosin and tubules examined for spermatogenesis (Figs. 1B and C). More details on the T+E2 model, tissue collection and data analyses are outlined in Tarapore et al., 2016 [1]. For Figs. 2D–G, the BPA administered to the dams in diet was 25 μg/kg bw-d. The sham-implanted, gestational exposed groups exhibited normal spermatogenesis on PND210 (100% offspring showed presence of spermatozoa in > 14% of STs).
2.2. Immunohistochemistry staining

The procedure and antibody sources are as outlined in Tarapore et al. [1].

2.3. Statistical analysis

For Figs. 1 and 2, significance was analyzed with one-way ANOVA and Dunnett’s multiple comparison test using the GraphPad Prism software.
Fig. 3. Offspring exposed to BPA, HFB and HFB+BPA diets contain ST with cell clusters in the T+E2 model. Sections were stained with anti-BRDT antibody. Red arrows point to clusters. RS round spermatids; PS pachytene Spermatocytes. Bar = 60 μm.

Fig. 4. Representative pictures illustrating ERα (ESR1) expression in the Leydig cells and STs of animals exposed in utero to indicated diets in the T+E2 model. Black arrows point to Leydig cells. Bar = 60 μm.
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Transparency document. Supporting information

Transparency data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j. dib.2016.10.025.

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[1] P. Tarapore, M. Hennessy, D. Song, J. Ying, B. Ouyang, V. Govindarajah, et al., High butter-fat diet and bisphenol A additively impair male rat spermatogenesis, Reprod. Toxicol. (2016), In press, PMID: 27658648.
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