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Data Article

Gene expression data of inflammatory mediators in apical periodontitis in 129 (wild type) and 5-lipoxygenase knockout mice

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ABSTRACT

Apical periodontitis is an immune inflammatory response around periapical tissues as a result of pathogens invasion into the root canal. The host immunoinflammatory response could determine the progression of this disease, which involves the recruitment of immune cells, and the release of several cytokines in the lesion site. The 5-lipoxygenase pathway has been activated in some osteolytic diseases due to its capacity to interfere in the proliferation and differentiation of bone cells, including the osteoclasts. As mean to understand the inflammatory genes regulation in the apical periodontitis progression, we evaluated the network of 66 genes related to cytokines, chemokines and other inflammatory mediators and receptors in the wild-type (WT) and 5-lipoxygenase enzyme genetically deficient mice (KO). This article presents data not published but related to the research article "Effects of 5-lipoxygenase gene

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disruption on inflammation, osteoclastogenesis and bone resorption in polymicrobial apical periodontitis" .

Specifications Table

Subject	Dentistry, Oral Surgery and Medicine
Specific subject area	Endodontics
Type of data	Gene expression (fold change) relative to control
How data were acquired	RNA extraction followed by reverse transcription Amplification in qRT-PCR machine (40 cycles) Instruments: Step one Plus (Applied Biosystems), GeNeCK web server and Software R
Data format	Raw Graphs Figures Table in Excel datasheet Report relative expression Analyzed
Parameters for data collection	Jaw samples with apical periodontitis and control (contralateral jaw with healthy teeth without lesion) from 5-lipoxygenase enzyme knockout mice and wild-type mice were collected as described [1,2]. Several inflammatory mediators were evaluated by qRT-PCR [1].
Description of data collection	Inflammatory mediators gene expression array was compared at different time points of apical periodontitis in the 5-lipoxygenase enzyme knockout mice and compared to wild-type mice.
Data source location	Institution: Universidade de São Paulo City/Town/Region: Ribeirão Preto Country: Brazil
Data accessibility	Full data is host in a public repository. Repository name: Universidade de São Paulo. Direct URL to data: http://repositorio.uspdigital.usp.br/handle/item/336
Related research article	Paula-Silva, F., Arnez, M., Petean, I., Almeida-Junior, L. A., da Silva, R., da Silva, L., & Faccioli, L. H. (2020). Effects of 5-lipoxygenase gene disruption on inflammation, osteoclastogenesis and bone resorption in polymicrobial apical periodontitis. *Archives of oral biology, 112*, 104,670. 10.1016/j.archoralbio.2020.104670

Value of the Data

- The data shows a panorama of inflammatory genes profile in the apical periodontitis progression in the wild-type and 5-lipoxygenase enzyme knockout mice.
- This data provides a valuable tool for studying the apical periodontitis development by comparing the inflammatory genes expression modulation in both animals using heatmap and gene regulatory networks in both models.
- The data could contribute to interpretation of 5-lipoxygenase enzyme to the inflammatory genes expression network during the apical periodontitis development.

1. Data Description

The 5-lipoxygenase enzyme deficiency in mice can result in changes in the immunoinflammatory markers gene expression during the apical periodontitis development. Furthermore, the absence of this enzyme affects gene interaction resulting in a broader network at 28 days of the disease. Table 1 integrates symbol and nomenclature of genes evaluated by qRT-PCR. The raw data of qRT-PCR analysis of each gene can be found in the Supplementary file. Fig. 1 shows a heatmap with 66 inflammatory mediator genes evaluated in the apical periodontitis of WT and KO at different stages of the disease, after 7, 14, 21 and 28 days of apical periodontitis. Gene regulatory network was evaluated in wild type (Fig. 2) and in knockout mice (Fig. 3), both at 28 days after apical periodontitis induction. In Figs. 2 and 3, the regulatory genes (Hub gene) and connected genes (nodes genes) of each group were shown.

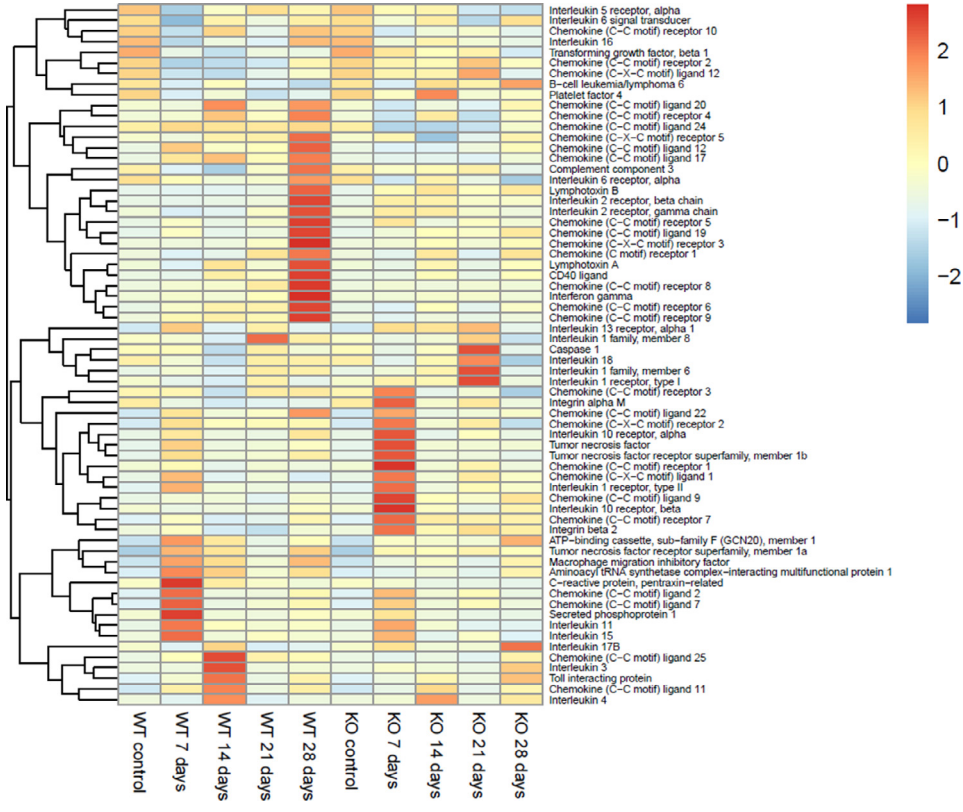

Fig. 1. Heatmap and cluster analysis of kinetic of 66 genes expression in the apical periodontitis of WT and KO mice at 7, 14, 21 and 28 days of lesion and their respective control groups. Color codes in each panel refer to *blue* for low expression and *red* for the highest expression levels.

Table 1
National Center for Biotechnology Information (NCBI) of the transcriptome, Bank access number, Symbol and Nomenclature of genes evaluated by qRT-PCR array that encode mouse inflammatory cytokines and receptors.

Unigene	GeneBank	Symbol	Nomenclature
Mm.329022	NM_013854	*Abcf1*	ATP-binding cassette, sub-family F (GCN20), member 1
Mm.347398	NM_009744	*Bcl6*	B-cell leukemia/lymphoma 6
Mm.491799	NM_007551	*Cxcr5*	Chemokine (C-X-C motif) receptor 5
Mm.19131	NM_009778	*C3*	Complement component 3
Mm.1051	NM_009807	*Casp1*	Caspase 1
Mm.4686	NM_011330	*Ccl11*	Chemokine (C–C motif) ligand 11
Mm.867	NM_011331	*Ccl12*	Chemokine (C–C motif) ligand 12
Mm.41988	NM_011332	*Ccl17*	Chemokine (C–C motif) ligand 17
Mm.490604	NM_011888	*Ccl19*	Chemokine (C–C motif) ligand 19
Mm.290320	NM_011333	*Ccl2*	Chemokine (C–C motif) ligand 2
Mm.116739	NM_016960	*Ccl20*	Chemokine (C–C motif) ligand 20
Mm.12895	NM_009137	*Ccl22*	Chemokine (C–C motif) ligand 22
Mm.31505	NM_019577	*Ccl24*	Chemokine (C–C motif) ligand 24
Mm.7275	NM_009138	*Ccl25*	Chemokine (C–C motif) ligand 25
Mm.341574	NM_013654	*Ccl7*	Chemokine (C–C motif) ligand 7
Mm.416125	NM_011338	*Ccl9*	Chemokine (C–C motif) ligand 9
Mm.274927	NM_009912	*Ccr1*	Chemokine (C–C motif) receptor 1
Mm.6272	NM_009915	*Ccr2*	Chemokine (C–C motif) receptor 2
Mm.57050	NM_009914	*Ccr3*	Chemokine (C–C motif) receptor 3
Mm.1337	NM_009916	*Ccr4*	Chemokine (C–C motif) receptor 4
Mm.14302	NM_009917	*Ccr5*	Chemokine (C–C motif) receptor 5
Mm.8007	NM_009835	*Ccr6*	Chemokine (C–C motif) receptor 6
Mm.2932	NM_007719	*Ccr7*	Chemokine (C–C motif) receptor 7
Mm.442098	NM_007720	*Ccr8*	Chemokine (C–C motif) receptor 8
Mm.442383	NM_009913	*Ccr9*	Chemokine (C–C motif) receptor 9
Mm.28767	NM_007768	*Crp*	C-reactive protein, pentraxin-related
Mm.21013	NM_008176	*Cxcl1*	Chemokine (C-X-C motif) ligand 1
Mm.303231	NM_021704	*Cxcl12*	Chemokine (C-X-C motif) ligand 12
Mm.332490	NM_019932	*Pf4*	Platelet factor 4
Mm.12876	NM_009910	*Cxcr3*	Chemokine (C-X-C motif) receptor 3
Mm.8021	NM_007721	*Ccr10*	Chemokine (C–C motif) receptor 10
Mm.240327	NM_008337	*Ifng*	Interferon gamma
Mm.379327	NM_008348	*Il10ra*	Interleukin 10 receptor, alpha
Mm.4154	NM_008349	*Il10rb*	Interleukin 10 receptor, beta
Mm.35814	NM_008350	*Il11*	Interleukin 11
Mm.24208	NM_133,990	*Il13ra1*	Interleukin 13 receptor, alpha 1
Mm.490053	NM_008357	*Il15*	Interleukin 15
Mm.10137	NM_010551	*Il16*	Interleukin 16
Mm.59313	NM_019508	*Il17b*	Interleukin 17B
Mm.1410	NM_008360	*Il18*	Interleukin 18
Mm.133095	NM_019450	*Il1f6*	Interleukin 1 family, member 6
Mm.45901	NM_027163	*Il1f8*	Interleukin 1 family, member 8
Mm.896	NM_008362	*Il1r1*	Interleukin 1 receptor, type I
Mm.1349	NM_010555	*Il1r2*	Interleukin 1 receptor, type II
Mm.384038	NM_008368	*Il2rb*	Interleukin 2 receptor, beta chain
Mm.2923	NM_013563	*Il2rg*	Interleukin 2 receptor, gamma chain
Mm.983	NM_010556	*Il3*	Interleukin 3
Mm.276360	NM_021283	*Il4*	Interleukin 4
Mm.3448	NM_008370	*Il5ra*	Interleukin 5 receptor, alpha
Mm.2856	NM_010559	*Il6ra*	Interleukin 6 receptor, alpha
Mm.4364	NM_010560	*Il6st*	Interleukin 6 signal transducer
Mm.234466	NM_009909	*Cxcr2*	Chemokine (C-X-C motif) receptor 2
Mm.262106	NM_008401	*Itgam*	Integrin alpha M
Mm.1137	NM_008404	*Itgb2*	Integrin beta 2
Mm.87787	NM_010735	*Lta*	Lymphotoxin A
Mm.1715	NM_008518	*Ltb*	Lymphotoxin B
Mm.2326	NM_010798	*Mif*	Macrophage migration inhibitory factor
Mm.235137	NM_007926	*Aimp1*	Aminoacyl tRNA synthetase complex-interacting
Mm.288474	NM_009263	*Spp1*	multifunctional protein 1

(*continued on next page*)

Table 1 (*continued*)

Unigene	GeneBank	Symbol	Nomenclature
Mm.248380	NM_011577	*Tgfb1*	Secreted phosphoprotein 1
Mm.1293	NM_013693	*Tnf*	Transforming growth factor, beta 1
Mm.474976	NM_011609	*Tnfrsf1a*	Tumor necrosis factor
Mm.235328	NM_011610	*Tnfrsf1b*	Tumor necrosis factor receptor superfamily, member 1a
Mm.4861	NM_011616	*Cd40lg*	Tumor necrosis factor receptor superfamily, member 1b
Mm.103551	NM_023764	*Tollip*	CD40 ligand
Mm.390241	NM_011798	*Xcr1*	Toll interacting protein
Mm.3317	NM_010368	*Gusb*	Chemokine (C motif) receptor 1
Mm.299381	NM_013556	*Hprt*	Glucuronidase, beta
Mm.2180	NM_008302	*Hsp90ab1*	Hypoxanthine guanine phosphoribosyl transferase
Mm.304088	NM_008084	*Gapdh*	Heat shock protein 90 alpha, class B member 1
Mm.391967	NM_007393	*Actb*	Glyceraldehyde-3-phosphate dehydrogenase Actin, beta

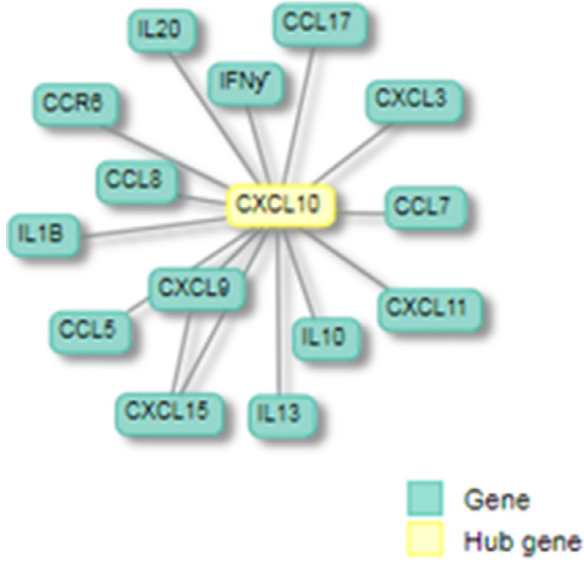

Fig. 2. Gene regulatory network (GRN) using the Graphical Lasso ($\lambda = 0.300$) method of WT mice at 28 days of lesion. Yellow circles indicate regulatory genes (hub gene) and light blue circles indicate poorly connected genes (nodes genes). There is 1 hub gene: CXCL10 participates in gene regulation and biological processes.

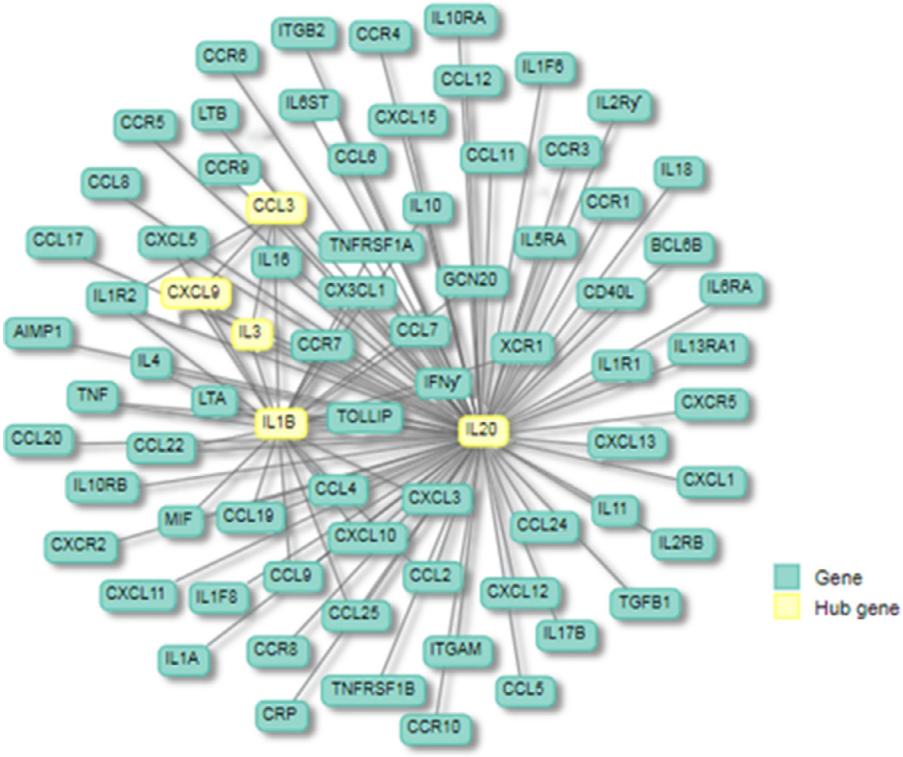

Fig. 3. Gene regulatory network (GRN) using the Graphical Lasso ($\lambda = 0.300$) method of KO mice at 28 days of lesion. Yellow circles indicate regulatory genes (Hub gene) and light blue circles indicate poorly connected genes (nodes genes). There are 5 hub genes: IL-1β, IL-3, IL-20, CXCL9 and CCL3 participate in gene regulation and biological processes.

2. Experimental Design, Material and Methods

2.1. Animals

Twenty four knockout (KO) mice for 5-lipoxygenase enzyme (129-Alox5^{tm1Fun}; 129-Alox5$^{-/-}$; The Jackson Laboratory, Bar Harbor, ME, USA) and 24 wild-type 129 mice for the control group were used in this study. Mice were male and adult (6–8 week-old). For the operative procedures the animals were anesthetized by intraperitoneal injections of ketamine hydrochloride (150 mg/kg, Ketamine 10%, Agener União Química Farmacêutica Nacional S/A, Embu-Guaçu, SP) and xylazine (7.5 mg/kg, Dopaser, Laboratorios Calier S/A, Barcelona, Spain).

2.2. Apical periodontitis model

The protocol of apical periodontitis was previously described in Da Silva et al. [2]. Mice were placed in a surgical table in order to promote the immobilization of the animals, maintenance of the mouth opened, and the visualization of the molar teeth. The upper first molar pulps were exposed using a 1011 spherical diamond tip (KG Sorensen Ind. Com. Ltda., Barueri, SP) and a type K file #06 (Les Fils d'Auguste Maillefer S/A, Switzerland). The exposed root canals were left open to the oral environment, as previously described [3]. The teeth without pulp exposure

were used as controls. Mice were euthanized on days 7, 14, 21 and 28 after experimental apical periodontitis induction ($n = 6$ teeth per period).

2.3. Evaluation of gene expression by global qRT-PCR arrays to demarcation of inflammatory event

A guanidine thiocyanate protocol (RNeasy® Mini, Qiagen Inc., Valencia, USA) was used for RNA extraction from two pools of three teeth each. The evaluation of the total RNA quality was performed by electrophoresis on 1% agarose gel (Sigma-Aldrich Corp.) containing ethidium bromide (Sigma-Aldrich Corp.) using 1x concentrated TBE buffer (Tris-Borate-EDTA). The estimate of the amount of nucleic acids and their purity were assessed by spectrophotometry in NanoDrop 1000 (Thermo Fisher Scientific Inc., Wilmington, USA). The synthesis cDNA via reverse transcription reaction was performed by using 2 µg of total RNA and the First Strand RT^2 kit (Qiagen Inc.).

RT-PCR arrays (Inflammatory Cytokines and Receptors PAMM-011Z, Qiagen Inc.) were used for the analysis of 66 target sequence genes (Table 1). As reference genes, *Gusb, Hprt, Hsp90ab1, Actb* and *Gapdh* were evaluated. Controls for detecting mouse genomic DNA contamination (MGDC), controls for the efficiency of the reverse transcription reaction (RTC) and the positive controls (PPC) consisting of a passive artificial DNA sequence to be detected during the reaction. The qRT-PCR reactions were performed using SybrGreen, consisting of a duplicate in an Eppendorf Mastercycler® ep Realplex (Eppendorf AG). Amplification was done under the following conditions: denaturation at 95 °C for 10 min; followed by 40 cycles of 95 °C for 15 s and 60 °C for 1 min. The specificity of the primers was analyzed using the dissociation curve, considering the melting temperature of the amplicon under the following conditions: temperature increase to 95 °C for 15 s, followed by decrease the temperature to 60 °C for 15 s, gradually increasing the temperature to 95 °C for 20 min and maintained at 95 °C for 15 min. The $\Delta\Delta$Ct method was used for relative quantification.

2.4. Data presentation and analysis

The qRT-PCR data of 66 gene expressions were plotted in MS Excel for the data normalization. The relative quantification of all experimental groups were analyzed by R statistical package version 4.0.3. For data analysis, a heatmap was used in order to show the magnitude of the fold change of each gene in a color scale. The columns correspond to the experimental groups and the rows the genes evaluated.

2.5. Gene regulatory networks

The gene-gene association network of the same 66 genes was evaluated in the WT and KO group, both with 28 days of apical periodontitis, using the Graphical Lasso method ($\lambda = 0.300$) by GeneCK, a web server for building gene networks and visualization [4]. These graphs shows the nodes representing the genes and the edges representing the gene-gene interaction.

Ethics Statement

All experiments using animals were performed following the guidelines for animal research at University of São Paulo (USP). The experimental protocols were approved by the Ethics Committee on Animal Use from the School of Dentistry of Ribeirão Preto (process# 12.1.60.53.8).

Declaration of Competing Interest

The authors declare no conflict of interest for this article.

CRediT Author Statement

Thaise Mayumi Taira: Software, Data curation, Writing – review & editing; **Vítor Luís Ribeiro:** Software, Data curation, Writing – review & editing; **Yuri Jivago Silva Ribeiro:** Writing – review & editing; **Raquel Assed Bezerra da Silva:** Supervision, Writing – review & editing; **Léa Assed Bezerra da Silva:** Supervision, Writing – review & editing; **Marília Pacífico Lucisano Politi:** Supervision, Writing – review & editing; **Lúcia Helena Faccioli:** Conceptualization, Supervision; **Francisco Wanderley Garcia Paula-Silva:** Conceptualization, Methodology, Supervision, Writing – review & editing.

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Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.dib.2021.107787.

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