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Data Article



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responses of multi-cell cultures treated with Porphyromonas gingivalis hemagglutinin B

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Dataset on the chemokine and cytokine

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ABSTRACT

Chemokines and cytokines produced in gingival tissues exposed to microorganisms and microbial products in dental plaque lead to local inflammation and tissue damage seen in periodontal disease. Bates et al. 2018 [1] reported that Porphyromonas gingivalis hemagglutinin B (HagB)-induced matrix metalloproteinase (MMP) responses of single cell cultures containing dendritic cells, gingival epithelial (GE) keratinocytes, or T cells were significantly different from the MMP responses of these same cells grown in multi-cell cultures. Here we report the concentrations (pg/ml) of HagB-induced IL1a, IL6, IL8, IL12(p40), GM-CSF, MIP1a, MIP1β, RANTES, TNFa, and VEGF produced by dendritic

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T cells Porphyromonas gingivalis Hemagglutinin B cells, GE keratinocytes, or T cells in single cell cultures, two-cell cultures, or three-cell cultures. © 2019 The Authors. Published by Elsevier Inc. This is an open access

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Specifications table

Subject area More specific subject area Type of data How data was acquired	Biology Oral biology and innate immunity Tables and graphs Luminex 100 IS instrument and xPonent v3.1 software (Luminex, Austin, TX USA); MILLIPLEX Analyst v5.1 software (Millipore, Bill- erica, MA USA)
Data format	Analyzed
Experimental factors	Treatment of dendritic cell, gingival epithelial (GE) keratinocyte, and T cell multi-cell cultures with <i>Porphyromonas gingivalis</i> hemagglu- tinin B (HagB)
Experimental features	Single cell and multi-cell cultures of dendritic cells, GE keratino- cytes, and T cells were treated with <i>P. gingivalis</i> HagB. At 0, 8, 16, 32, and 64 h tissue culture media was collected and the concentrations (pg/ml) of IL1α, IL6, IL8, IL12(p40), GM-CSF, MIP1α, MIP1β, RANTES, TNFα, and VEGF were determined
Data source location Data accessibility Related research article	College of Dentistry, The University of Iowa, Iowa City, IA 52241, USA The data are available with this article Data article is a companion paper to a research article [1]

Value of the data

- Chemokine and cytokine responses of single cell cultures are not reflective of the chemokine and cytokine responses seen in inflamed tissues.
- Multi-cell cultures are more representative of the host environment because they include the responses from multiple cell types found in inflamed tissues.
- Dendritic cells, GE keratinocytes, and T cells in single cell cultures produced different amounts of IL6, IL8, GM-CSF, MIP1α, MIP1β, RANTES, and TNFα after exposure to *P. gingivalis* HagB than the same cells grown in multi-cell cultures.

1. Data

Hemagglutinin B (HagB) is a non-fimbrial adhesion protein on the surface of *Porphyromonas gingivalis*. It is pro-inflammatory and induces chemokine and cytokine responses in a variety of cells. Bates et al. [1] reported that HagB-induced matrix metalloproteinase (MMP) responses of single cell cultures containing dendritic cells, gingival epithelial (GE) keratinocytes, or T cells were significantly different from the MMP responses of these same cells grown in multi-cell cultures.

Here we report the concentrations (pg/ml) of HagB-induced IL1 α , IL6, IL8, IL12(p40), GM-CSF, MIP1 α , MIP1 β , RANTES, TNF α , and VEGF produced by dendritic cells, GE keratinocytes, or T cells in single cell cultures, two-cell cultures, or three-cell cultures (Fig. 1, Fig. 2, Table 1). For example, HagB induced different MIP1 α responses in single cell and multi-cell cultures, which decreased after 32 h.



Fig. 1. *Porphyromonas gingivalis* hemagglutinin B (HagB)-induced IL1 α (a), IL8 (b), MIP1 α (c), and RANTES (d) responses in dendritic cells (DC), GE keratinocytes (Ker), and T cells (TC) cultivated 0–64 h in single cell cultures (DC, Ker, or TC), 2 cell cultures (DC+Ker, DC+TC, or Ker+TC), or 3 cell cultures (DC+Ker+TC). A no cell (NC) control was added that contained only media. Each point represents a replicate of 3.

In contrast, IL8 responses increased beginning at 32 h. At 64 h, concentrations of IL1 α and IL12(p40) were less than 10.0 pg/ml, while concentrations of IL6, IL8, GM-CSF, MIP1 α , MIP1 β , RANTES, TNF α , and VEGF were more than 25 pg/ml in some cultures.

At 64 h, HagB-induced IL1 α , IL6, IL8, IL12(p40), GM-CSF, MIP1 α , MIP1 β , RANTES, TNF α , and VEGF responses from single cell cultures of dendritic cells, GE keratinocytes, or T cells were compared (Table 1, Fig. 2). There were no significant differences (p > 0.05) among groups producing IL1 α , IL12 (p40), and VEGF (Table 1, Fig. 2). There were significant differences (p < 0.05) among groups producing groups producing IL1 α , IL12 (p40), and VEGF (Table 1, Fig. 2). There were significant differences (p < 0.05) among groups producing groups producing IL6, IL8, GM-CSF, MIP1 α , MIP1 β , GM-CSF, RANTES, and TNF α (Table 1, Fig. 2).

Some responses were driven by a single cell type in multi-cell cultures. For example, HagB induced IL6, IL8, GM-CSF, MIP1 α , MIP1 β , and TNF α responses in dendritic cells, and HagB induced RANTES responses in T cells (Fig. 2). There were similar HagB-induced responses in dendritic cell + GE keratinocyte and dendritic cell + GE keratinocyte + T cell multi-cell cultures (Fig. 2). Overall, there were more dynamic changes in HagB-treated multi-cell cultures than in HagB-treated single cell cultures.



Fig. 2. *Porphyromonas gingivalis* hemagglutinin B (HagB) induced IL1 α (a), IL6 (b), IL8 (c), GM-CSF (d), MIP1 α (e), MIP1 β (f), RANTES (g), TNF α (h), and VEGF (i) responses in dendritic cells (DC), GE keratinocytes (Ker), and T cells (TC) cultivated 64 h in single cell cultures (DC, Ker, or TC), 2 cell cultures (DC+Ker, DC+TC, or Ker+TC), or 3 cell cultures (DC+Ker+TC). Shared letters indicate no significant difference between groups (p > 0.05, n = 9) and differing letters indicate significantly different groups (p < 0.05, n = 9).

2. Experimental design, materials and methods

2.1. Purification of HagB

Recombinant HagB was prepared by cloning *hagB* of *P. gingivalis* (1.4 kb) into the vector pQE31 (QIAGEN Inc., Valencia, CA USA) as described [1]. *Escherichia coli* M15(pREP4)pQE-31-TX1 was grown at 37 °C in selective Luria-base (LB) broth supplemented with ampicillin and kanamycin A. After 4 h, 1 mM Isopropyl B-D-1-thiogalactopyranoside was added to induce HagB expression. After 4 h of

Table 1

HagB-induced chemokine and cytokine responses of dendritic cells (DC), GE keratinocytes (Ker), and T cells (TC) cultivated 64 h in single cell cultures (DC, Ker, or TC), 2 cell cultures (DC+Ker, DC+TC, or Ker+TC), or 3 cell cultures (DC+Ker+TC). Observed responses were log10-transformed and an analogous two-way fixed effect ANOVA was fitted to the log-transformed concentrations. Pairwise group comparisons were conducted using the method of Tukey's Honest Significant Differences (HSD). A 0.05 level was used to determine statistically significant differences. The mean (standard error of the mean) for each biomarker in each group is listed below. Shared letters within each row indicate no significant differences among groups (p > 0.05, n = 3) and differing letters within each row indicate significant differences among (p < 0.05, n = 3).

	none	DC	Ker	тс	DC+Ker	DC+TC	Kr+TC	DC+Ker+TC
IL1α	0.0221	0.0235	0.1766	0.0033	0.3548	0.0127	0.3862	0.2168
	(0.1040)	(0.1040)	(0.1040)	(0.1040)	(0.1040)	(0.1040)	(0.1040)	(0.1040)
	a	a	a	a	a	a	a	a
IL6	0.0085	1.0212	0.0014	0.0010	1.4084	0.9476	0.1220	1.1953
	(0.1811)	(0.1811)	(0.1811)	(0.1811)	(0.1811)	(0.1811)	(0.1811)	(0.1811)
	b	a	b	b	a	a	b	a
IL8	0.0081	3.5956	0.0942	0.2050	3.7917	3.5624	0.1802	3.7443
	(0.0761)	(0.0761)	(0.0761)	(0.0761)	(0.0761)	(0.0761)	(0.0761)	(0.0761)
	b	a	b	b	a	a	b	a
IL12(p40)	0.0970	0.0874	0.0408	0.0998	0.2050	0.1038	0.1080	0.1147
	(0.0602)	(0.0602)	(0.0602)	(0.0602)	(0.0602)	(0.0602)	(0.0602)	(0.0602)
	a	a	a	a	a	a	a	a
GM-CSF	0.0695	1.1949	0.0000	0.5432	1.4160	1.1081	0.5905	1.3737
	(0.1351)	(0.1351)	(0.1351)	(0.1351)	(0.1351)	(0.1351)	(0.1351)	(0.1351)
	c	a	c	b,c	a	a,b	b,c	a
MIP1a	0.0181	2.1030	0.0748	0.5450	2.6268	2.2430	0.5944	2.5552
	(0.1312)	(0.1312)	(0.1312)	(0.1312)	(0.1312)	(0.1312)	(0.1312)	(0.1312)
	b	a	b	b	a	a	b	a
MIP1β	0.0312	1.5717	0.0247	0.4912	2.0420	1.7403	0.7724	1.9423
	(0.1219)	(0.1219)	(0.1219)	(0.1219)	(0.1219)	(0.1219)	(0.1219)	(0.1219)
	c	a	c	b,c	a	a	b	a
RANTES	0.1363	0.4459	0.1552	1.1529	0.9688	1.1947	1.1130	1.3509
	(0.1547)	(0.1547)	(0.1547)	(0.1547)	(0.1547)	(0.1547)	(0.1547)	(0.1547)
	c	b,c	c	a	a,b	a	a,b	a
ΤΝFα	0.0181	1.6519	0.0808	0.2299	2.1189	1.6008	0.4067	2.0476
	(0.0980)	(0.0980)	(0.0980)	(0.0980)	(0.0980)	(0.0980)	(0.0980)	(0.0980)
	d	b,c	d	d	a	c	d	a,b
VEGF	0.7955	0.6462	1.2031	0.6477	0.7427	0.3281	0.7177	1.0756
	(0.2908)	(0.2908)	(0.2908)	(0.2908)	(0.2908)	(0.2908)	(0.2908)	(0.2908)
	a	a	a	a	a	a	a	a

incubation, 10.0 ml of 1.54 M sodium azide was added to arrest growth. *E. coli* was lysed in 6.0 M Guanidine-HCl, 0.1 M NaH₂PO₄, 0.01 M Tris Acid, pH 8.0. HagB was isolated from the *E. coli* lysate using a Profinity IMAC Ni-charged resin as described [1]. HagB was refolded using 6.0 M Urea, 0.5 M NaCl, 0.01 M Tris acid, 20.0% glycerol, pH 7.4 and then 3.0 M Urea, 0.5 M NaCl, 0.01 M Tris acid, 20.0% glycerol, pH 7.4 and then 0.25 M imidazole in 0.01 M Tris, 0.5 M NaCl, 20.0% glycerol, pH 7.4 and dialyzed against 0.01 M Tris containing 0.5 M NaCl and 20.0% glycerol, pH 7.4 at

4 °C. The final concentration of LPS was 2.44 ng LPS/1.0 μg HagB (QCL-1000, Chromogenic Limulus Amebocyte Lysate Assay, Lonza, Inc. Walkersville, MD USA).

2.2. Dendritic cells, GE keratinocytes, and T cells

Non-peripheral blood human myeloid dendritic cells (AllCells, LLC, Alameda, CA USA) were thawed and suspended in Lymphocyte Growth Medium-3 (LGM-3, Lonza, Walkersville, MD USA). The GE keratinocytes were previously isolated from healthy, non-smoking individuals in compliance with a protocol approved by the University of Iowa Institutional Review Board for the Use of Human Subjects in Research (number 199811030, November 6, 2005 to August 25, 2012) as previously described [2]. CD4+ T cells were obtained from StemCell Technologies, Inc. (Vancouver, BC, Canada). T cells were activated using a human T cell Activation/Expansion kit (Miltenyi Biotec, San Diego, CA USA) and grown in TexMACS medium (Miltenyi Biotec, San Diego, CA USA) for three days at 37 °C with 5% CO₂. All three cell types were suspended in LGM-3 to contain 1.0×10^5 viable cells/ml.

2.3. Multi-cell cultures

A diffusible 3 tier transwell platform was constructed that could contain all three cell types simultaneously [1]. The inserts from a 24-well transwell plate (No. 3414, Corning, Inc., Corning, NY USA) were put into the inserts from a 12 well transwell plate (No. 3401, Corning, Inc., Corning, NY USA), and both inserts were put into the 12 well transwell plate bottom. 800 μ l of LGM-3 was added to the bottom of each 12 well transwell plate, and 200 μ l of the dendritic cell suspension was added to the bottom of the 12 well transwell plate. 300 μ l of LGM-3 and 200 μ l of GE keratinocyte suspension were added to the middle insert. 200 μ l of T cell suspension was added to the top insert. The total volume was 1,700 μ l/well. LGM-3 was added in place of cell suspensions not present in a well. The plates were incubated for 2 h at 37 °C to allow the cells to settle and adjust.

Cells were exposed to either 10.0 μ g/ml HagB or the buffer used to elute HagB, placed on a shaker, and incubated at 37 °C with 5% CO₂ for 64 h. Samples were removed at 0, 2, 4, 8, 16, 32, and 64 h to assess the effect of HagB on chemokine and cytokine responses in cultures of single cell types (e.g., dendritic cells, GE keratinocytes, or T cells alone); in multi-cell cultures of two cell types (e.g., dendritic cells + GE keratinocytes, dendritic cells + T cells, and GE keratinocytes + T cells), and in multi-cell cultures of three cell types (e.g., dendritic cells + GE keratinocytes (e.g., dendritic cells + GE keratinocytes + T cells). 200 μ l of culture media was removed from each well, and 200 μ l of LGM-3 was added back to replace the media that was removed. Cultures of single cell types and multi-cell cultures of two cell types were made to compare with the transwell co-cultures containing three cell types. The co-culture experiment was repeated three times and three repeats of each condition were plated on the immunoassay to increase validity (N = 9).

2.4. Determination of chemokine and cytokine concentrations

Concentrations (pg/ml) of IL1 α , IL6, IL8, IL12(p40), GM-CSF, MIP1 α , MIP1 β , RANTES, TNF α , and VEGF were determined in cell culture media using a Milliplex immunoassay (Millipore, Billerica, MA USA) as described [1].

2.5. Transformation of data and statistical analysis

Chemokine and cytokine concentrations were interpolated from standard curves prepared by plotting concentrations of the known chemokine or cytokine standard versus its respective MFI value using xPonent v3.1 (Luminex, Austin, TX USA) and MILLIPLEX Analyst v5.1 (Millipore, Billerica, MA USA).

Concentrations of chemokines and cytokines detected in culture media from cells treated with buffer only were subtracted from the concentrations of chemokines and cytokines detected in culture media from cells treated with HagB. The concentrations of chemokines and cytokines determined in each replication (N = 3) were pooled from each of the 3 replications into one dataset at 64 h (N = 9).

The data was transformed by adding 1.0 pg/ml to each value and a log10-transformation was applied to all chemokine and cytokine values to attenuate the positive skew in the distributions and make the normality assumption more defensible.

A two-way fixed effect ANOVA was then fit to the log-transformed concentrations (JMP10, Version 10.0, SAS, Cary, NC USA). Pairwise group comparisons were conducted using the post-hoc method of Tukey's Honest Significant Differences (HSD). A 0.05 level was used to determine statistically significant differences.

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Transparency document. Supporting information

Transparency data associated with this article can be found in the online version at https://doi.org/ 10.1016/j.dib.2018.12.087.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at https://doi. org/10.1016/j.dib.2018.12.087.

References

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