



Biochemical contents of apple peel and flesh affect level of partial resistance to blue mold



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ABSTRACT

Apple fruit contains a wide range of chemical compounds that may contribute to resistance against blue mold caused by *Penicillium expansum*. In the present study, contents of total titratable acidity, malic acid, total phenols and 10 individual phenolic compounds were quantified in peel and flesh fractions of both control and blue mold-inoculated fruits of 24 apple cultivars. In addition to the significant variation among cultivars in terms of all quantified compounds, correlation analysis revealed a significant impact of total phenols and individual phenols like flavonols and procyanidins B2 in the peel fraction, on blue mold resistance in the inoculated fruits. Multivariate analyses on data for chemical compounds in peel tissue of inoculated fruits, could also separate resistant and susceptible cultivars. These findings can be useful in breeding programs since higher levels of phenolic compounds may indicate better resistance in apple cultivars.

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1. Introduction

Apple fruits contain several constituents with health-related benefits such as organic acids, sugar alcohols and phenolic compounds (Boyer and Liu, 2004). In order to provide the market with healthy and attractive fruits, harvesting and storage conditions must be carefully optimized to avoid damage due to postharvest fungal diseases. Some of these are difficult to avoid, like blue mold caused by *Penicillium expansum*. Infection by this pathogen is mainly wound-mediated and can occur both in the orchard and during harvesting and storage. In general, the symptoms do however not appear until the fruit has been kept in cold storage for some weeks, thus causing serious economic loss for the growers. Blue mold is easily identified by the rounded and pale straw-colored lesions with white to bluish-green spores. Symptoms increase fast and the entire fruit is soon destroyed by internal rotting. In addition to lowering the commercial value of the apple harvest, this fungus also produces the powerful mycotoxin patulin (Konstantinou et al., 2011).

Traditionally, serious economic loss due to blue mold and other storage diseases has been avoided by postharvest application of fungicides. This procedure is, however, not allowed in organic apple production, and is prohibited also in conventional apple production in an increasing number of countries like Scandinavia and Great Britain (Tahir and Nybom, 2013). Moreover, the storage disease problems are likely to increase even further due to global warming (Weber, 2009). In this context, the identification of highly resistant apple cultivars becomes very desirable. Considerable inter-cultivar variability in the amount of damage caused by postharvest decay has been reported, both when evaluated after natural infection (Janisiewicz and Peterson, 2004) and after inoculation of harvested fruit with *P. expansum* (Jurick et al., 2011; Ahmadi-Afzadi et al., 2013; Tahir et al., 2015). No sources of highly efficient, genetically determined resistance have yet been identified but factors like level of fruit maturity (Prusky et al., 2004; Vilanova et al., 2012; Vilanova et al., 2014b; Vilanova et al., 2014c), initial firmness and rate of softening during storage (Nybom et al., 2008a; Johnston et al., 2009; Ahmadi-Afzadi et al., 2013) show strong associations with the amount of contracted damage.

In addition to the above-mentioned fruit texture-related traits, chemical constituents of the fruit flesh and peel can also be expected to play a role in resistance to storage diseases. Many plants can respond to pathogens either by accumulation of pre-

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formed compounds (phytoanticipins, i.e., chemicals that are already present in different concentrations and forms) or by production of new compounds (phytoalexins) due to induction of genes involved in the defense system. Phytoanticipins and phytoalexins frequently consist of phenolic compounds. These compounds belong to a large group of chemicals, many of which are involved in the natural defense reactions of plants, and can be toxic to invading organisms (Grayer and Kokubun, 2001; Lattanzio et al., 2006). Polyphenols are usually divided into different classes, e.g., flavanols (catechin, epicatechin and procyanidins), flavonols (quercetin glycosides), dihydrochalcones (phloridzin), and hydroxycinnamic acids (chlorogenic acid). Accumulation of phenolic compounds as a response to infection by apple scab caused by *Venturia inaequalis* has been reported (Mikulic-Petkovsek et al., 2009, 2011) while another study showed that total phenols and phenylalanine-ammonia lyase increased during the first week after inoculation of apple fruit by *P. expansum*, followed by a period of decreasing contents (Schovan-kova and Opatova, 2011).

Identification of cultivars with a high level of pre-formed protective chemical compounds would be especially valuable for plant breeding. Healthy fruit of different apple cultivars have thus been shown to vary significantly in content and composition of polyphenolic compounds (Bushway et al., 2002; Khanizadeh et al., 2008; Nybom et al., 2008a; Roen et al., 2009). Associations between these compounds and level of genetically determined resistance to diseases in apple have been investigated but with somewhat contradictory results. A positive correlation of chlorogenic acid, flavanols and coumaric acid with level of resistance to apple scab was thus reported by Picinelli et al. (1995) whereas amount of pre-formed flavan-3-ols showed no association with resistance to apple scab in other studies (Sierotzki and Gessler, 1993; Roen et al., 2009).

The objectives of this study were to (1) quantify the chemical compounds in peel and flesh of several apple cultivars in both uninfected (control) and infected fruits, and (2) investigate a possible association between these chemical compounds and the level of resistance to blue mold.

Table 1
Average of TTA, MA and TPH measurements in flesh and peel of control and inoculated fruits in different cultivars in 2012 and 2013.

Year	Cultivars	TTA (%)				MA (mg g ⁻¹)				TPH (mg g ⁻¹ GAE FW)				LD/W ^b (mm)
		Flesh		Peel		Flesh		Peel		Flesh		Peel		
		C ^a	I ^a	C	I	C	I	C	I	C	I	C	I	
2012	Apelsinoe	33	27	0.13	0.17	6.94	4.42	2.50	1.81	0.41	0.48	7.74	5.96	5.02
	Aroma	40	28	0.09	0.10	7.94	5.15	4.97	4.62	0.96	0.82	7.58	7.67	2.41
	Bersis	33	17	0.07	0.08	7.22	2.22	1.85	1.07	0.86	1.19	6.59	6.03	1.84
	Björka	29	24	0.07	0.11	5.68	4.01	4.20	3.44	0.92	0.71	9.03	6.11	2.39
	Discovery	33	30	0.13	0.11	6.73	5.43	7.58	4.30	1.12	0.86	7.19	4.25	5.70
	Elise	51	31	0.10	0.10	11.3	5.11	2.39	4.06	1.39	0.88	5.76	4.63	3.52
	Gloster	30	29	0.06	0.13	6.52	4.52	1.36	2.47	0.38	0.86	8.07	9.59	2.60
	Göteborgs Flickäpple	46	27	0.11	0.13	9.16	4.99	4.74	5.27	1.00	0.84	6.71	5.42	3.70
	Gravensteiner	35	29	0.10	0.12	7.32	4.57	4.49	4.72	0.90	0.78	8.34	6.08	2.54
	Ingrid Marie	34	32	0.10	0.10	7.21	4.88	4.00	3.55	0.55	0.77	10.54	6.8	3.15
	Katja	39	29	0.14	0.13	6.60	4.28	3.70	1.97	1.29	1.16	9.02	5.28	2.43
	Konsta	28	31	0.11	0.12	5.27	5.17	5.29	5.35	0.55	0.49	8.24	3.99	5.77
	Luke	53	38	0.13	0.14	10.80	5.70	6.78	6.85	1.31	1.25	11.12	6.49	4.48
	Olga	24	25	0.12	0.08	5.70	3.36	2.08	2.58	0.68	0.69	8.73	8.07	1.58
	Raja	45	33	0.06	0.15	9.95	6.30	2.61	7.20	0.29	0.36	5.72	3.10	4.74
	Santana	29	24	0.12	0.12	6.77	4.32	5.07	4.36	0.66	0.68	8.62	5.41	4.45
	Sariola	22	23	0.07	0.11	4.82	3.57	1.67	3.31	0.79	0.80	5.54	3.02	5.55
Sörmlandsäpple	35	35	0.11	0.10	6.38	3.86	2.30	2.01	0.82	0.75	8.58	4.52	2.54	
Tönnes	9	28	0.08	0.13	5.71	3.83	3.15	3.21	0.65	0.94	9.14	8.79	1.60	
Williams' Pride	32	25	0.09	0.11	6.16	4.07	1.44	4.71	0.45	0.41	8.93	3.67	4.42	
2013	Barchatnoje	35	37	0.06	0.06	7.64	7.04	4.81	3.47	0.73	0.88	7.99	6.25	5.18
	Bersis	36	35	0.06	0.08	9.09	7.84	1.94	2.88	0.91	0.91	8.84	6.98	4.74
	Elise	28	28	0.06	0.09	7.48	6.02	1.73	2.52	0.21	0.44	5.86	6.52	4.35
	Gloster	33	41	0.05	0.11	8.17	8.73	1.50	4.01	0.55	0.61	9.01	7.68	2.77
	Gravensteiner	41	43	0.06	0.05	9.79	9.96	5.15	2.92	1.17	0.72	6.53	9.02	4.86
	Ingrid Marie	32	32	0.09	0.11	8.46	7.07	4.59	3.34	0.62	0.68	9.26	7.42	3.36
	Juuso	49	43	0.07	0.07	12.3	9.94	6.43	5.37	0.72	0.74	8.54	6.29	6.05
	Luke	49	42	0.09	0.10	12.5	9.26	5.48	3.84	0.88	1.01	6.44	7.17	3.98
	Olga	25	23	0.06	0.08	6.84	4.95	1.77	1.72	0.84	0.89	11.25	9.88	3.01
	Pepin Schafranovij	39	41	0.08	0.11	10.6	9.10	4.43	4.03	1.16	1.74	5.95	6.04	2.41
	Raja	32	32	0.06	0.08	8.21	7.28	2.17	4.75	0.25	0.30	5.91	4.43	6.83
	Sandra	11	24	0.07	0.08	2.56	4.09	2.00	1.95	0.78	0.77	8.23	8.10	5.56
	Santana	25	31	0.08	0.10	5.95	6.64	3.18	3.16	0.49	0.55	6.62	4.69	5.96
Average of CV ^c (%) in 2012	3.7	2.9	6.4	6.5	4.3	4.5	6.0	5.1	5.3	5.9	6.5	4.6	9.8	
Average of CV (%) in 2013 (%)	1.9	2.2	6.1	6.7	2.8	3.3	7.5	5.5	5.3	4.9	5.6	5.8	5.0	

^a C, Control samples; I, Inoculated samples.

^b LD/W, Lesion Diameter/week.

^c CV, Coefficient of variation.

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