

Can disease management reduce greenhouse gas emissions?

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Summary

The majority of the greenhouse gas (GHG) costs of wheat production are associated with the cultivations and nitrogen (N) needed to establish and grow a green canopy. This investment is made prior to flowering in the expectation that photosynthesis will fill grain post-flowering. Epidemics of foliar diseases destroy green area and usually have their greatest effect during the yield forming period, after the emergence of new leaves has ceased. Hence, ineffective disease control decreases the GHG efficiency of wheat production. Maintaining the current level of UK production in the absence of fungicide treatment would use an additional 0.93 to 1.92 million tonnes of CO₂ equivalent per annum. In comparison, the GHG costs associated with disease control by disease resistant varieties or fungicides are small, provided any yield penalty associated with breeding for resistance is negligible. Effective disease management increases N use efficiency, but can also increase the economic optimum for N. If more N is applied as a result, some of the GHG benefits of good disease management are negated.

Introduction

The greenhouse gas (GHG) costs of wheat production are associated predominantly with the establishment and growth of a green canopy to intercept light during grain filling. The nitrogen (N) required to achieve this accounts for approximately 70% of the total GHG emissions from wheat production (Mortimer *et al.*, 2004), when calculated in terms of the equivalent global warming potential of CO₂. A further 10-15% of total emissions are accounted for by the cultivations and seed required to establish the canopy.

Hence, the majority of GHG and economic variable costs are incurred well before the start of yield formation, in the expectation that the investment in green plant tissues for photosynthesis will be repaid through dry matter assimilation during grain filling. Epidemics of foliar diseases destroy green area and usually have their greatest effect during the yield forming period, after the emergence of new leaves has ceased.

Figure 1 shows a typical example of the effect of disease (*Septoria tritici*) on green leaf area of a susceptible wheat crop during grain filling. Green area usually reaches a maximum just prior to flowering (time=0). In the absence of disease (dose=1), green area persists for a period and then declines through natural senescence. Without control (dose=0) disease-induced loss of green area (which follows a sigmoidal pattern) causes a premature decline in leaf area. Increasing dose causes a dose-response curve of green area on dose – most clearly seen at mid-grain filling around day 30. A more disease resistant variety would require a lower dose to avoid green area loss. The effects of N on

canopy growth are complete by the start of this diagram (time=0). In contrast virtually all of the effect of fungicide treatment is expressed after flowering (hence dose has little effect on green leaf area at time=0), despite treatments starting early in stem extension.

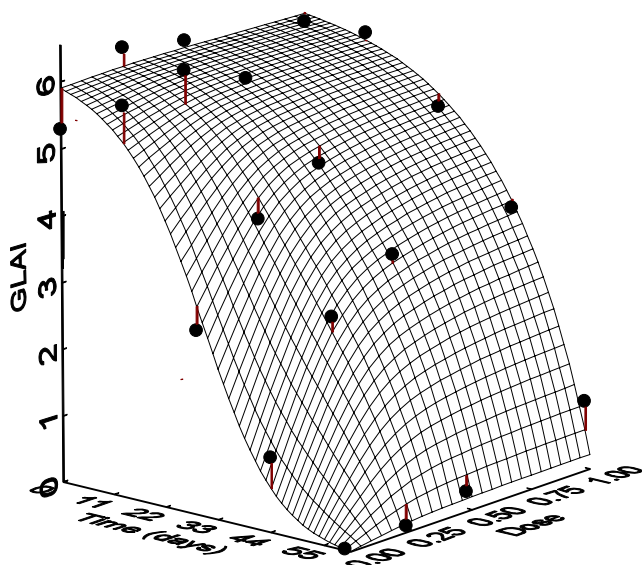


Figure 1. Change in green leaf area index (GLAI) of a wheat crop from flowering (time=0) to complete senescence, at a range of fungicide dose inputs (dose=1 represents a full fungicide spray programme designed to control foliar diseases). Closed circles are measured values, to which the surface (grid) has been fitted.

Given that the GHG costs of growing green canopy are large, and the GHG costs of fungicides (Table 1) or disease resistance to prevent it from being lost prematurely are small, wheat production should be more GHG efficient if diseases are well controlled.

Table 1. Crop inputs used in wheat production in the UK and greenhouse gas emissions factors

Input	Rate used	Emissions factor
[†] N fertiliser manufacture	185 ^a kg/ha	7.11 ^d kg CO ₂ eq/kg N
N fertiliser N ₂ O on application	185 ^b kg/ha	6.16 ^e kg CO ₂ eq/kg N
Fungicides	1.07 ^c kg ai/ha	3.9 ^f kg CO ₂ eq/kg ai

[†]Ammonium nitrate

Sources: ^aAnon. (2006b), ^bAnon. (2006b), ^cGarthwaite *et al.* (2005), ^dAnon. (2007a), ^eAnon. (2006a), ^fLal (2004).

However, there are interactions between disease and N which are likely to affect GHG efficiency. Larger canopies, resulting from higher N uptake, are better able to maintain light interception (and hence grain filling) despite the loss of a given amount of green area to disease. However, N increases the absolute rate at which disease epidemics grow, causing more green area to be lost (e.g. Neumann *et al.*, 2004). The latter effect generally predominates. Hence, the yield response from a given N input will usually be increased by

effective disease control. Figure 2 illustrates this effect with a hypothetical example. The lower curve represents the N response curve with poor or no disease control. The upper curve, representing the N response with good disease control, tends to diverge (because disease control has a larger effect on yield at high N inputs).

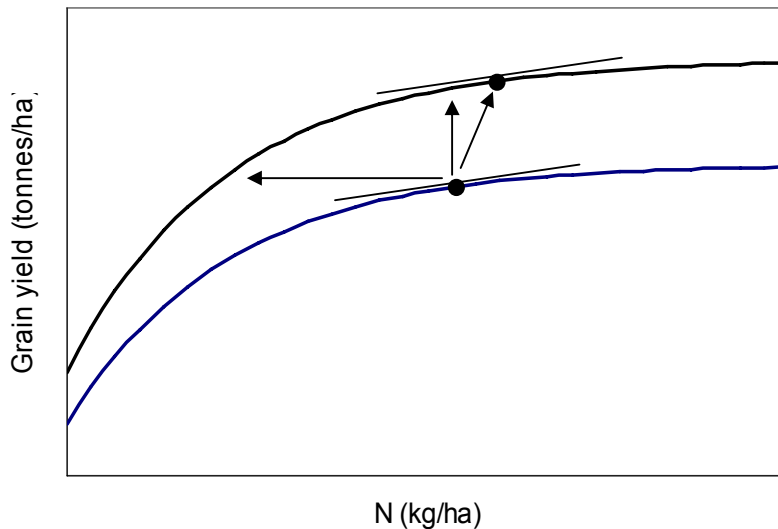


Figure 2. Hypothetical response curves of yield on N input with poor (lower line) and good (upper line) disease control. Tangents on the curves both show the ratio of N cost to grain value, which determines the position of the economic optimum (solid circle) for N inputs of each curve.

These curves can be interpreted in three ways. 1: The horizontal arrow suggests that a given level of yield can be achieved by two combinations: low N and good disease control or high N and poor disease control. The former will result in substantially lower GHG emissions per hectare and per tonne of grain produced. 2: The vertical arrow suggests that a given level of N input can result in low or high yield, depending on the degree of disease control. Good control will not affect GHG emissions per hectare, but should result in lower GHG emissions per tonne of grain. 3: The diagonal arrow shows that divergence of the response curves results in a higher economic optimum for N where disease control is good (because there is a better ‘pay back’ through light interception during grain filling, for a given investment in N). If the optimum level of N is applied for each curve, then the effect of disease control on GHG emissions per tonne of grain will depend on the relationship between the difference in N input and the difference in yield.

Unless recent reports of the beneficial effects of phytoliths are substantiated, carbon fixed by annual crops may have little net effect on atmospheric CO₂, because the carbon removed from the atmosphere during growth is re-emitted when the plant product is consumed, burned or decomposes. Hence, the largest effect of disease on climate is likely to be through increasing the area of cropped land required to satisfy demand for grain, thus increasing the GHG emissions per tonne of grain.

These interactions between N, disease, GHG emissions, land use and economics have not been adequately quantified. Here we address a preliminary question: are the effects big enough to matter?

HGCA Recommended List data for winter wheat were used to calculate the size of the effects represented by the vertical arrow. An analytical method was developed to quantify the potential net effect of disease and disease control on GHG emissions per hectare of crop and per tonne of crop produce (grain or bioethanol). The method was used to: (i) compare fungicide treated and untreated crops of elite cultivars, to calculate the contribution made by fungicides to minimising GHG emissions, (ii) calculate the relative GHG efficiency of cultivars which contrast in their levels of disease resistance, and (iii) calculate the maximum potential improvement which might be achieved if diseases could be controlled completely.

Methods

Greenhouse gas emissions were calculated for the production of a hectare of wheat, a tonne of grain, and a tonne of bioethanol. Account was also taken of co-products, including straw and dried distillers grains (DDGS; a co-product of converting grain into bioethanol), as these have several end uses which affect GHG emissions.

The global warming potential (GWP) resulting from emissions of CO₂, N₂O and CH₄ was calculated in terms of the equivalent GWP of CO₂ (CO₂ eq). Over a 100 year timescale, one kg of N₂O was assumed to have a GWP of 296 times greater than one kg of CO₂. The GWP of CH₄ is 23 times greater than CO₂ (Anon, 2006a). The GHG emissions per hectare associated with producing the grain and straw were estimated by allocation based on the relative economic value of grain and straw. For baled straw the relative values of the grain and the straw produced per hectare (minus baling costs) were used. Default prices were assumed to be £80 per tonne of wheat grain (85% dry matter), £20 per tonne of straw (Nix, 2007) and baling costs were £50/ha (Nix, 2007). For incorporated straw the value of straw was estimated in terms of its fertiliser value, as £3.8 per tonne.

The gross GHG emissions associated with converting wheat grain into bioethanol using a combined heat and power (CHP) system have been estimated at 320 kg CO₂ eq per tonne of grain (85% dm) (Punter *et al.*, 2006). This system produces surplus electricity which was assumed to be exported to the national grid giving a GHG cost of -231 kg CO₂ eq per tonne of grain (85% dm) (Punter *et al.*, 2006). The net GHG emissions for converting wheat grain to bioethanol were therefore estimated at 89 kg CO₂ eq per tonne of grain. The GHG emissions associated with the production of each tonne of wheat grain and its conversion to bioethanol were allocated to bioethanol on the basis of the relative economic values of bioethanol and DDGS.

Data

Grain yield data for wheat crops grown with and without fungicides were obtained from the HGCA Recommended List trials (Anon. 2007b). These data were from experiments at eight or nine UK sites per year in 2004, 2005 and 2006. Each experiment tested 32 to 35 cultivars, grown without fungicides and with a full programme of fungicides (see www.hgca.com for protocol details). The predominant foliar and stem-base pathogens controlled were *Mycosphaerella graminicola* (*Septoria tritici*), *Puccinia triticina* (brown rust), *Puccinia striiformis* (yellow rust), *Blumeria graminis* (powdery mildew), *Oculimacula*

acuformis and *O. yallundae* (eyespot). These diseases are also reasonably well controlled in commercial crops in the UK by a combination of host resistance and fungicides (Hardwick *et al.*, 2001).

The fungicide treatments contained approximately 2 kg of active ingredient per hectare. All crop management was the same for both fungicide treatments, and was designed to ensure that yields were not limited by pests or lack of nutrients. To calculate the GHGs associated with fungicide treated crops the weight of active ingredient for the fungicide treatments was used together with the measured yields. For the untreated crops zero fungicide active ingredient was used with the measured yields. In addition, the GHGs associated with the field operations were reduced by 8.8 kg CO₂/ha for each fungicide application (Williams *et al.*, 2006) that was omitted (compared with the treated crops).

Results

Production of one hectare of wheat was estimated to be associated with emissions of 3187 kg CO₂ eq based on the default values described. Production of one tonne of grain was associated with 408 kg CO₂ eq assuming grain and straw yields of 8 t/ha and 5 t/ha respectively. Production of one tonne of bioethanol was associated with the emission of 1477 kg CO₂ eq. Changes in grain yield due to disease and its control were estimated to have a substantial effect on the GHG emissions per tonne of grain or bioethanol (Figure 3). The GHG emissions per tonne were inversely related to grain yield.

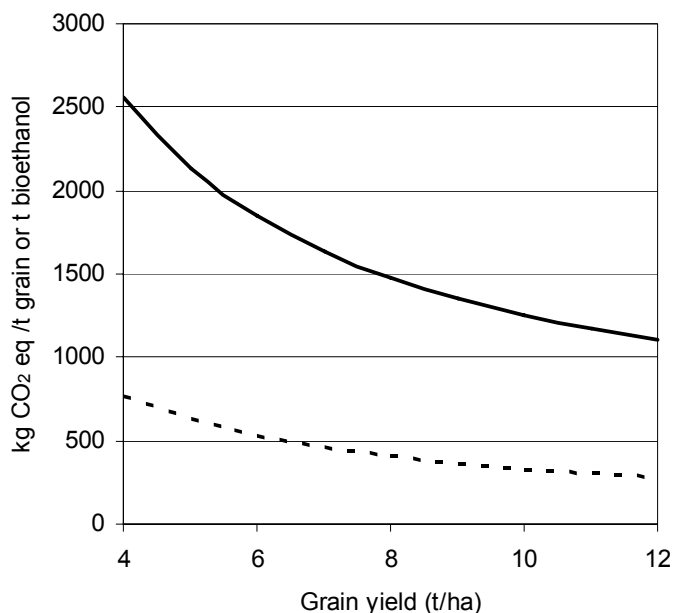


Figure 3. Calculated relationship between grain yield per hectare and the greenhouse gas emissions associated with the production of a tonne of grain (solid line) or bioethanol (dashed line).

The RL data on fungicide treated and untreated yields for 842 cultivar/site/season combinations show that, on average, disease reduced yield from 10.20 t/ha to 8.42 t/ha. This level of disease-induced yield loss, after allowing for the reduction in GHGs

associated with the manufacture and application of fungicides, resulted in a net increase in GHG emissions from 327 to 386 kg CO₂ eq per tonne of grain. GHG emissions per tonne of bioethanol were estimated to increase from 1235 to 1421 kg CO₂ eq/t for crops grown without fungicide.

There were large differences ($P < 0.001$) between cultivars in the amount of yield lost to disease (Figure 4). Seventeen cultivars were common across the three years of data, of which cv Consort experienced the greatest disease-induced yield loss of 2.47 t/ha and cv Hereward experienced the smallest loss of 1.27 t/ha (Figure 4). Despite the large differences in disease resistance, GHG emissions from the untreated cultivars were never lower than from the treated cultivars (Figure 4).

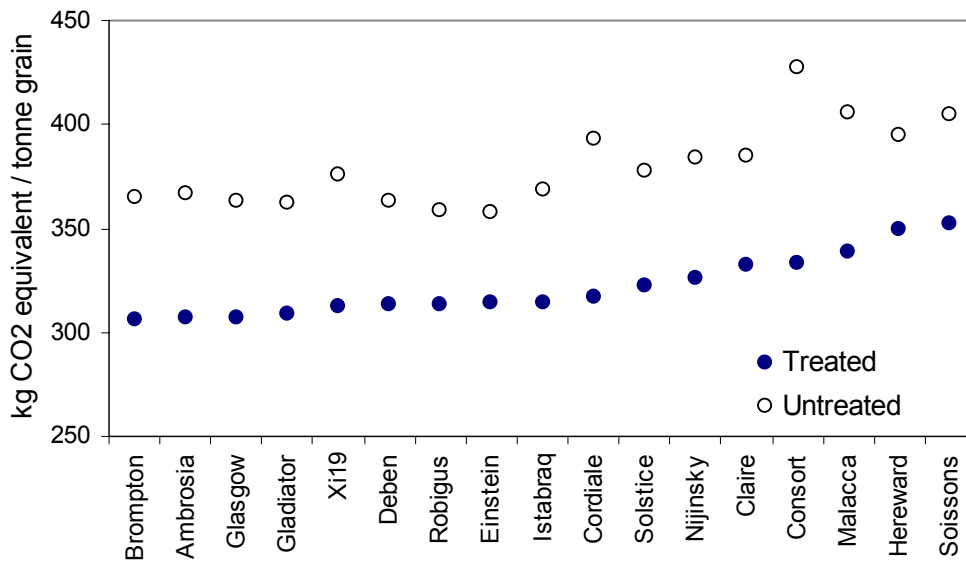


Figure 4. Calculated greenhouse gas emissions from fungicide treated and untreated grain yield data from wheat HGCA Recommended List cultivar evaluation trials (for cultivars present in all trials from 2004 to 2006 inclusive, ranked according to treated emissions).

Little information is available on the effect of disease control on straw yield, but data were obtained from six experiments across a range of wheat varieties at ADAS Rosemaund in 1999, 2000, 2005 and 2006, and at ADAS Terrington in 1999 and 2000, where growth analysis was undertaken. Disease significantly reduced straw yield in three out of the six experiments. The greatest reduction in straw yield was seen at ADAS Rosemaund in 2006, in which disease reduced straw yield from 8.10 t/ha to 6.86 t/ha ($P < 0.01$). Across the six experiments disease reduced straw yield from 6.73 t/ha to 6.14 t/ha. Approximately two thirds of the above-ground non-grain material measured in these experiments will equate to a farmer’s straw yield because not all of the straw and chaff is collected during baling. Accounting for the reduction in straw yield made little difference to the GHG emissions associated with grain production. However, disease can significantly increase the GHGs associated with the production of straw. In the Rosemaund 2006 experiment, disease was estimated to increase GHGs from 87 to 105 kg CO₂ eq per tonne of straw.

Discussion

GHG emissions associated with untreated yields were estimated at 386 kg CO₂ eq per tonne of grain decreasing to 327 kg CO₂ eq for treated yields and to 313 kg CO₂ eq if disease-free crops could be achieved. The last value was calculated using survey data (Hardwick *et al.*, 2001), which indicated an average 3.6% yield loss due to disease remaining in crops despite fungicide treatment. If the disease-free yield could be achieved then about 4.70 million tonnes of CO₂ eq would be associated with the production of the typical UK wheat output of 15 million tonnes. This is estimated to increase to 5.84 Mt CO₂ eq if diseases were not controlled. Therefore controlling wheat diseases has the potential to save up to 1.14 Mt CO₂ eq per annum.

In the UK, 2123 tonnes of fungicide active ingredient are applied per annum using on average 2.9 spray applications per hectare (Garthwaite *et al.*, 2005). This amounts to 0.058 Mt CO₂ eq assuming 3.9 kg CO₂ eq is associated with the production of each kg ai (Lal, 2004) and 8.8 kg CO₂ eq is associated with the field operations for each application (Williams *et al.*, 2006). If fungicides were not used then, in the absence of varieties with improved disease resistance, an additional 0.93 Mt CO₂ eq would be emitted to produce 15 Mt of wheat. This estimate assumes that the additional wheat area required would displace other annual crops or temporary set-aside/fallow land in the UK which would result in little change in GHG emissions due to changing land use. However, if additional wheat area, or additional area of other crops to substitute for wheat grain, were to displace grassland or forest land, then additional GHG emissions would occur primarily as a result of changes in carbon storage in the soil and vegetation. It has been estimated that replacing grassland or forest with annual crop land releases 1.7 to 29.2 t CO₂ eq/ha over a 25 year period (carbon reporting within RTFO). Hence, the estimates of the impacts of disease are sensitive to assumptions about demand for grain and how it might be met.

A further reduction in emissions would occur if diseases could be controlled below the levels found in commercial crops currently. Improved control could be achieved through cultivars with more effective or durable disease resistance, or by improved fungicide treatment. The GHG implications of these two approaches differ. The net benefit from improved disease resistance would be sensitive to any associated yield penalty. 'Yield drag' may occur directly, through the physiological costs to the host of resistance responses, indirectly, through deleterious genes closely linked with resistance loci, or by diversion of finite plant breeding resources from selection for yield traits. In production systems where fungicide treatment is not available, the benefits to yield of improved resistance are likely to outweigh any yield drag and, therefore, provide a substantial net benefit. In production systems where fungicide treatment is widely used, the GHG benefits of improved disease resistance could potentially accrue by reducing the amount of fungicide required to achieve a given level of disease control, or by reducing the amount of disease remaining after a given level of fungicide input. However, the GHG benefits of either may be outweighed by even a very small yield penalty associated with disease resistance. With currently available fungicide active substances, the dose which would minimise GHG emissions per tonne of grain substantially exceeds the dose required to minimise the economic unit costs of production (because the GHG cost of fungicides is very small in comparison to the yield benefit, whereas their economic cost is significant). However, an increase in fungicide use, besides being economically deleterious, would be limited by regulatory constraints on the maximum total dose which can be applied. An

increase in the number of fungicide applications to each crop would increase the risk of fungicide resistance (Brent & Hollomon, 2007). New, more effective, active substances could provide a net benefit to emissions, provided the GHG costs associated with their manufacture were not substantially higher than those for existing products. In practice, the crucial aim must be to maintain effective disease control, despite evolution in pathogen populations towards virulence and fungicide insensitivity, by integrating disease resistance and fungicide treatment.

Yield losses resulting from uncontrolled disease in the UK were estimated in this study to increase GHG emissions per tonne of bioethanol from 1196 to 1421 kg CO₂ eq/t. Compared to fossil-derived petrol, bioethanol from wheat has the potential to reduce GHG emissions by 59% for each MJ of energy produced (Mortimer *et al.*, 2004). If disease is not controlled then we calculate that the GHG savings from bioethanol would decline to 51%.

The analysis reported here assumed that nitrogen fertiliser use was unaffected by disease (i.e. the calculation quantified the effect of the vertical arrow in Figure 2). However, disease control can affect the economic optimum for N, for the reasons described earlier. The potential impacts of disease on GHG emissions appear sufficiently large to justify analysis of the interactions between N, disease and GHGs.

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